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**RELATIONSHIP BETWEEN DYSFUNCTIONAL EATING  
PATTERNS AND BINGE DRINKING IN YOUNG PEOPLE  
AND ASSOCIATED RISK FACTORS**

TESIS DOCTORAL

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*Als meus pares i iaies, gràcies per tant*



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## RESUM

La joventut es considera un període d'especial vulnerabilitat en el qual s'observen dos grans problemes: problemes alimentaris i problemes de consum d'alcohol i drogues (Bava i Tapert, 2010). En particular, s'observa amb freqüència un elevat consum d'alcohol i aliments rics en greixos no saludables (greixos trans i saturats) i sucres (Delegació del Govern per al Pla Nacional sobre Drogues, 2018; Fleming-Milici i Harris, 2020; Inchley i cols., 2018; Navarro-Cruz i cols., 2017).

Sovint el problema no radica només en el tipus de consum, sinó també en com es consumeix. L'afartament és una de les maneres més habituals en què els joves poden consumir aliments o alcohol. Es caracteritza per la ingestió de grans quantitats d'aliment (afartament de menjar- A) o alcohol (afartament d'alcohol - AA) de manera incontrolada i en un període de temps curt (Associació Americana de Psiquiatria, 2013; Paul i cols., 2011). Ambdós problemes solen ser altament comòrbids i estar associats a conseqüències molt greus per a la salut, com ara trastorns posteriors de l'alimentació i consum d'alcohol, conductes de risc, agressió, diabetis, sobrepès i obesitat, baix rendiment acadèmic, trastorns psicològics i altes taxes de mortalitat (Fazzino i cols., 2017; Ferriter i Ray, 2011; Galicia-Paredes i cols., 2017; Guitart i cols., 2011; Hudson i cols., 2007; Isorna i cols., 2015; Keski-Rahkonen i Mustelin, 2016; Kessler i cols., 2013; Kuntsche i cols., 2017; Moral i Ovejero, 2011; Raevuori i cols., 2015; Striegel-Moore i Franko, 2008).

A més d'aquestes complicacions, hi ha un concepte estretament relacionat amb l'A que ha guanyat protagonisme en la recerca en els darrers anys per la seua elevada prevalença en els joves: l'addicció al menjar (AM). L'AM es refereix a la ingesta excessiva i desregulada d'aliments rics en greixos i/o sucre, i els símptomes dels quals s'assemblen als problemes d'abús de substàncies, encara que en aquest cas en relació als aliments (Schulte i cols., 2015). Per exemple, hi ha un desig intens de menjar aquests aliments, intents infructuosos de reduir el consum d'aquests aliments, ús continuat malgrat les conseqüències, etc. Aquest problema és molt freqüent en els joves i també té un impacte molt negatiu en la seua salut i benestar (Rostanzo i Aloisi, 2021; Steele i cols., 2016).

Atesa l'elevada prevalença d'aquestes conductes en els joves i les conseqüències per a la salut observades, així com la seua elevada comorbiditat reportada, calen estudis que analitzen la relació entre aquestes conductes i els factors de risc que hi poden estar associats, per tal de dissenyar programes de prevenció i tractament adequats. Per tant, l'objectiu principal d'aquesta Tesi Doctoral és estudiar la relació entre aquests patrons alimentaris disfuncionals i el consum excessiu d'alcohol en els joves, i examinar els factors de risc associats a aquestes conductes.

Diversos estudis han intentat investigar la comorbiditat entre l'A i l'AA, les característiques compartides i les conseqüències comunes (Bahji i cols., 2019; Davis i cols., 2017; Ferriter i Ray, 2011). Algunes teories apunten a causes i factors de risc similars en ambdues conductes, així com a una relació evident entre elles (Ferriter i Ray, 2011; Laghi i cols., 2014). Tanmateix, segons el que sabem, no hi ha estudis fins ara que hagen revisat els punts en comú, les diferències i les influències entre aquests dos problemes. Per tant, el primer objectiu d'aquesta Tesi Doctoral va ser realitzar una revisió narrativa per identificar els punts en comú i les diferències entre l'A i l'AA, i la influència que exerceix un comportament sobre l'altre i viceversa.

Aquesta revisió (estudi #1) va assenyalar que l'A i l'AA comparteixen molts aspectes comuns (inici i naturalesa del problema, conseqüències associades, emocions negatives associades i factors de personalitat i socioculturals). A més, s'ha demostrat la direccionalitat entre l'A i l'AA. En particular, s'ha demostrat que l'AA pot estimular la ingesta d'aliments (Caton i cols., 2004, 2005, 2007; De Castro i Orozco, 1990; Westerterp-Plantenga i Verwegen, 1999; Yeomans, 2010). Tanmateix, encara no hi ha molts estudis a la literatura que hagen examinat si aquesta relació també es produeix a la inversa, és a dir, si els patrons alimentaris disfuncionals poden influir en el consum d'alcohol. Alguns estudis han indicat que l'A pot predir el consum futur de drogues (Sonneville i cols., 2013), encara que la influència de l'A sobre l'AA és menys clara. Només hem trobat un estudi que apunta a una clara influència de l'A i la ingesta de greixos en l'AA (Blanco-Gandía, Ledesma, i cols., 2017), però es va realitzar en models animals i els resultats no es poden generalitzar als humans. Per tant, aquesta revisió apunta a la necessitat d'estudis translacionals que analitzen la influència dels patrons alimentaris disfuncionals en l'AA en humans per entendre millor l'etiologia i el desenvolupament de l'AA en els joves.

Per investigar la interacció entre aquests dos comportaments, calen mesures que avaluen de manera fiable i vàlida els constructes. En el cas dels patrons alimentaris disfuncionals, els investigadors han desenvolupat alguns instruments per operar i avaluar l'A i l'AM. La *Binge Eating Scale (BES)* és l'escala més utilitzada a tot el món per identificar els A, amb bones propietats psicomètriques (Gormally i cols., 1982). La *modified Yale Food Addiction Scale 2.0 (mYFAS 2.0)* també és coneguda a tot el món per avaluar AM segons els criteris DSM-5 i ha obtingut excel·lents propietats psicomètriques (Schulte i Gearhardt, 2017). Tanmateix, fins ara no hi ha estudis que n'hagen analitzat les propietats psicomètriques en població jove espanyola. Per tant, el nostre següent objectiu va ser validar la *BES* per mesurar A (estudi #2) i la *mYFAS 2.0* (estudi #4) per mesurar AM, en població jove espanyola. Es van realitzar dos estudis empírics, i a partir de les troballes, es va concloure que tant la *BES* com la *mYFAS 2.0* tenen bones propietats psicomètriques i són mesures d'autoinforme fiables i vàlides per avaluar A i AM respectivament en la població juvenil espanyola.

Com s'ha esmentat anteriorment, la nostra revisió va assenyalar la necessitat de més investigacions sobre la influència entre l'A i l'AA, per tal d'entendre més clarament la seua etiologia i desenvolupament. Com s'ha comentat, la influència de l'AA en la ingesta d'aliments és ben coneguda, però encara no hi ha molts estudis que examinen si aquesta relació també es produeix a la inversa, és a dir, si la ingesta d'aliments influeix en el consum d'alcohol. Alguns estudis assenyalen una relació entre el consum de sucre i el consum d'alcohol (Bouhlal i cols., 2018; Kampov-Polevoy i cols., 2014). Un estudi amb models animals ha assenyalat el paper predictiu de la ingesta de greixos i l'A en el consum excessiu d'alcohol (Blanco-Gandía, Ledesma, i cols., 2017). Tanmateix, es desconeix la relació entre la ingesta de greixos/sucres i el consum d'alcohol en els joves. Calen estudis translacionals que avaluen l'impacte d'aquests hàbits dietètics en la vulnerabilitat al consum d'alcohol dels joves. Per tant, el nostre següent objectiu va ser analitzar el paper predictiu dels patrons alimentaris disfuncionals (A i ingesta de greixos) en l'AA en joves (Estudi #3). A més, la literatura ha assenyalat alguns factors que poden estar associats amb la ingesta de greixos i l'A (per exemple, gènere, edat, Índex de Massa Corporal (IMC), estils alimentaris, etc.) (Camilleri i cols., 2014; Kakoschke i cols., 2015; Linardon i Messer, 2019; Mason i Lewis, 2014; Sultson i cols., 2017), i per tant pot estar relacionat indirectament amb l'AA. Per tant, també es va examinar si aquests factors individuals (gènere, IMC,

impuls per la primesa, insatisfacció corporal, estils d'alimentació, impulsivitat i AM) podien predir l'AA a través de la seua relació amb la ingesta de greixos i l'A.

Aquest estudi va mostrar per primera vegada evidències de la influència de l'A i la ingesta de greixos en l'AA en joves. També és la primera vegada que es proporciona evidència de la relació indirecta dels estils d'alimentació (alimentació emocional, externa i restrictiva) i l'AM amb l'AA, mediada per l'A i la ingesta de greixos. Aquests resultats ens poden ajudar a prevenir l'AA en joves mitjançant intervencions dirigides a joves amb patrons alimentaris disfuncionals.

Com s'ha comentat anteriorment, l'AM ha esdevingut molt rellevant en els darrers anys. L'A comparteix molta variància amb l'AM (Gearhardt, White, i cols., 2011), i com que és un constructe tan prevalent i nociu en els joves, cal conèixer-ne els factors de risc per tal de dissenyar les campanyes de prevenció i intervenció més adequades possibles.

S'ha trobat que molts factors estan fortament associats amb l'AM i, per tant, poden ser factors de risc per a aquest problema alimentari. Aquests inclouen l'edat, sent les persones més joves les que tenen més probabilitats de ser addictes als aliments (Schiestl i Gearhardt, 2018; Wiss i Brewerton, 2020). Un altre factor és el gènere. Les dones tenen més probabilitats que els homes de tenir aquest problema (Gearhardt i cols., 2016; Pursey i cols., 2014). A més a més, també s'ha observat una associació positiva entre els estils d'alimentació (alimentació emocional, externa i restrictiva) i l'AM (Pepino i cols., 2014; Schiestl i Gearhardt, 2018; Schulte i Gearhardt, 2017; Wardle i cols., 1992; Wiss i Brewerton, 2020). Finalment, però no menys important, s'ha observat una relació molt forta entre l'A i l'AM (Escrivá-Martínez i cols., 2019; Gearhardt i cols., 2016; Gearhardt i cols., 2012), i entre la bulímia i l'AM (Meule i cols., 2014). Fins ara, s'ha demostrat que totes aquestes variables estan associades a l'AM, però es desconeix si poden actuar com a factors de risc d'aquesta. Es necessita més literatura per indicar si aquests factors poden ser factors de risc per a l'AM i veure fins a quin punt tots poden explicar aquest problema. Per tant, un altre dels nostres objectius també era analitzar el poder predictiu del gènere, l'edat, l'IMC, els estils alimentaris, l'A i la bulímia sobre l'AM.

Aquest estudi va demostrar per primera vegada la predicció de l'IMC, estils

alimentaris, A i bulímia sobre l'AM en joves, amb totes les variables que expliquen més de la meitat de la variància en AM. Aquests resultats assenyalen la importància de considerar totes aquestes variables en la prevenció de l'AM en els joves.

Finalment, i tenint en compte el moment especial en què es va desenvolupar aquesta tesi, no podem obviar una situació que podria haver tingut un gran impacte en la conducta alimentaria: el confinament per la COVID-19. L'aparició de la COVID-19 amb les seues mesures per frenar el contagi podria haver afectat especialment els estils de vida de les persones i, concretament, el comportament alimentari de la població jove. Diversos estudis han mostrat troballes molt variades. Mentre que alguns estudis han reportat un augment en el consum d'aliments no saludables, així com un IMC més elevat (Ammar i cols., 2021; Pellegrini i cols., 2020; Phillipou i cols., 2020; Sidor i Rzymiski, 2020), altres estudis reportats han trobat una millora en els hàbits alimentaris i cap canvi en l'IMC (Di Renzo i cols., 2020; Haddad i cols., 2021; Rodríguez-Pérez i cols., 2020). Aquests estudis s'han limitat a preguntar sobre hàbits dietètics anteriors de manera retrospectiva, cosa que podria donar lloc a un biaix d'informació. Fins ara, no hi ha treballs que avaluen les conductes alimentaries abans i durant el confinament, és a dir, amb dades reals tant abans com durant el període de confinament domiciliari. Així, el següent objectiu d'aquesta Tesi Doctoral era avaluar les diferències en els estils d'alimentació (alimentació emocional, externa i restrictiva), A, ingesta de greixos i IMC abans del confinament per COVID-19 (novembre de 2019) i durant el confinament per COVID-19 (abril de 2020) (Estudi #5). A més a més, conèixer els factors de risc d'aquests patrons alimentaris en moments d'estrès elevat ens pot ajudar a prevenir-los en moments futurs. Per tant, també ens vam proposar analitzar el paper predictiu dels estils d'alimentació (alimentació emocional, externa i restrictiva) sobre l'IMC, la ingesta de greixos i l'A durant el període de confinament estricte de la COVID-19.

Els resultats del nostre estudi van indicar que el confinament per COVID-19 va tenir un impacte positiu en els comportaments alimentaris, millorant l'A i la ingesta de greixos dels joves. Els estils de menjar es van mantenir estables, com era d'esperar (Meiselman i cols., 1998), i també es va demostrar l'estabilitat de l'IMC. Això pot ser perquè els joves es van adherir més als patrons d'alimentació saludable durant el confinament (Di Renzo i cols., 2020). També s'ha observat que només els individus

que tenien problemes de pes anteriors van augmentar de pes durant el confinament (Di Renzo i cols., 2020), i la nostra mostra tenia un IMC normal abans del confinament. Pel que fa al poder predictiu dels estils alimentaris sobre aquests comportaments, es va demostrar que l'alimentació emocional va predir l'IMC i l'A durant el confinament. L'alimentació externa va predir la ingesta de greixos durant el confinament, i l'alimentació restrictiva va predir l'A, i va predir negativament l'IMC i la ingesta de greixos durant el confinament. Aquest estudi va ser el primer a comparar els comportaments alimentaris abans i durant el confinament per COVID-19, sense incloure dades retrospectives. També va ser el primer a analitzar el paper predictiu dels estils alimentaris sobre aquestes conductes. Aquests resultats poden ajudar les futures polítiques de salut a centrar la prevenció en la millora dels estils alimentaris per frenar els comportaments alimentaris disfuncionals i l'IMC en moments d'estrès elevat.

Finalment, el nostre últim objectiu va ser avaluar si els estils alimentaris moderaven la relació entre l'estrès percebut i l'A durant el confinament per COVID-19 (Estudi #6). Hem dissenyat una aplicació mòbil per avaluar els comportaments alimentaris durant el confinament en temps real, evitant el biaix de record i millorant la validesa ecològica. Els nostres resultats van mostrar que els tres estils d'alimentació van moderar la relació entre l'estrès percebut i l'A durant el confinament per COVID-19. Es demostra per primera vegada la influència dels estils alimentaris en la relació entre l'estrès percebut i l'A, amb la força d'haver-se realitzat en el context d'una pandèmia i utilitzant una avaluació ecològica momentània. És important saber quines persones tenen més risc d'A en situacions d'estrès, de manera que es beneficiaran més de les intervencions destinades a reduir l'A en moments d'estrès.

En conclusió, aquesta Tesi Doctoral presenta 6 estudis, amb l'objectiu d'augmentar el coneixement sobre la relació entre els patrons alimentaris disfuncionals i l'AA en els joves. A més a més, s'han proposat diferents factors de risc que poden influir en aquests patrons alimentaris disfuncionals i en l'AA en els joves. Esperem que aquesta investigació ajude a dissenyar la prevenció, la intervenció i el tractament de tots aquests comportaments tan perjudicials per als joves de hui.



## ABSTRACT

Youth is considered a period of special vulnerability in which two major problems are observed: food problems and problems with alcohol and drug use (Bava & Tapert, 2010). In particular, high consumption of alcohol and food rich in unhealthy fats (trans and saturated fats) and sugars are frequently observed (Government Delegation for the National Plan on Drugs, 2018; Fleming-Milici & Harris, 2020; Inchley et al., 2018; Navarro-Cruz et al., 2017).

Often the problem lies not only in the type of consumption, but also in how it is consumed. Binge is one of the most common ways in which young people can consume food or alcohol. It is characterized by the ingestion of large amounts of food (binge eating - BE) or alcohol (binge drinking - BD) in an uncontrolled manner and in a short period of time (American Psychiatric Association, 2013; Paul et al., 2011). Both problems are often highly comorbid and associated with very serious health consequences, such as subsequent eating and alcohol use disorders, risk behaviors, aggression, diabetes, overweight and obesity, poor academic performance, psychological disorders and high mortality rates (Fazzino et al., 2017; Ferriter & Ray, 2011; Galicia-Paredes et al., 2017; Guitart et al., 2011; Hudson et al., 2007; Isorna et al., 2015; Keski-Rahkonen & Mustelin, 2016; Kessler et al., 2013; Kuntsche et al., 2017; Moral & Ovejero, 2011; Raevuori et al., 2015; Striegel-Moore & Franko, 2008).

In addition to these complications, there is a concept closely related to BE that has gained prominence in research in recent years due to its high prevalence in young people: the food addiction (FA). FA refers to the excessive and dysregulated intake of foods high in fat and/or sugar, and whose symptoms resemble those of substance abuse problems, albeit in this case in relation to food (Schulte et al., 2015). For instance, there is an intense desire to eat these foods, unsuccessful attempts to reduce the consumption of these foods, continued use despite the consequences, etc. This problem is very prevalent in young people and also has a very negative impact on their health and well-being (Rostanzo & Aloisi, 2021; Steele et al., 2016).

Since the high prevalence of these behaviors in young people and the health consequences observed, as well as their high reported comorbidity, studies are

needed to analyze the relationship between these behaviors and the risk factors that may be associated with them, in order to design appropriate prevention and treatment programs. Therefore, the main objective of this Doctoral Thesis is to study the relationship between these dysfunctional eating patterns and excessive alcohol consumption in young people, and to examine the risk factors associated with these behaviors.

Several studies have attempted to investigate the comorbidity between BE and BD, the shared characteristics and common consequences (Bahji et al., 2019; Davis et al., 2017; Ferriter & Ray, 2011). Some theories point to similar causes and risk factors in both behaviors, as well as to an evident relationship between them (Ferriter & Ray, 2011; Laghi et al., 2014). However, to our best knowledge, there are no studies to date that have reviewed the commonalities, differences, and influences between these two problems. Therefore, the first aim of this Doctoral Thesis was to conduct a narrative review to identify the commonalities and differences between BE and BD, and the influence exerted by one behavior on the other and vice versa.

This review (study #1) pointed out that BE and BD share many commonalities (onset and nature of the problem, associated consequences, associated negative emotions, and personality and socio-cultural factors). In addition, evidence for directionality between BD and BE has been demonstrated. In particular, it has been shown that BD can stimulate food intake (Caton et al., 2004, 2005, 2007; De Castro & Orozco, 1990; Westerterp-Plantenga & Verwegen, 1999; Yeomans, 2010). However, there are not yet many studies in the literature that have examined whether this relationship also occurs in reverse, i.e., whether dysfunctional eating patterns may influence alcohol consumption. Some studies have indicated that BE may predict future drug use (Sonneville et al., 2013), although the influence of BE on BD is less clear. We found only one study that points to a clear influence of BE and fat intake on BD (Blanco-Gandía, Ledesma, et al., 2017), but it was conducted in animal models and the results cannot be generalized to humans. Hence, this review points to the need for translational studies analyzing the influence of dysfunctional eating patterns on BD in humans to better understand the etiology and development of BD in young people.

To investigate the interaction between these two behaviors, measures that reliably and validly assess the constructs are needed. In the case of dysfunctional

eating patterns, researchers have developed some instruments to operationalize and assess BE and FA. The Binge Eating Scale (BES) is the most widely used scale worldwide to identify binge eaters, with good psychometric properties (Gormally et al., 1982). The modified Yale Food Addiction Scale 2.0 (mYFAS 2.0) is also well known worldwide for assessing FA according to DSM-5 criteria, and has obtained excellent psychometric properties (Schulte & Gearhardt, 2017). However, so far there are no studies that have analyzed its psychometric properties in young Spanish population. Therefore, our next objective was to validate the BES to measure BE (study #2) and the mYFAS 2.0 (study #4) to measure FA, in young Spanish population. Two empirical studies were carried out, and based on the findings, it was concluded that both the BES and the mYFAS 2.0 have good psychometric properties and are reliable and valid self-report measures for assessing BE and FA respectively in the Spanish youth population.

As previously mentioned, our review pointed out a need for further research on the influence between BE and BD, in order to understand more clearly their etiology and development. As commented, the influence of BD on food intake is well known, but there are not yet many studies that examine whether this relationship also occurs in reverse, i.e., whether food intake influences alcohol consumption. Some studies point to a relationship between sugar consumption and alcohol consumption (Bouhlal et al., 2018; Kampov-Polevoy et al., 2014). A study using animal models has pointed to the predictive role of fat intake and BE on excessive alcohol consumption (Blanco-Gandía, Ledesma, et al., 2017). However, the relationship between fat/sugar intake and alcohol consumption in young people is unknown. Translational studies assessing the impact of these dietary habits on vulnerability to alcohol consumption in young people are needed. Therefore, our next aim was to analyze the predictive role of dysfunctional eating patterns (BE and fat intake) on BD in young people (Study #3). Furthermore, the literature has pointed to some factors that may be associated with fat intake and BE (e.g., gender, age, Body Mass Index (BMI), eating styles, etc.) (Camilleri et al., 2014; Kakoschke et al., 2015; Linardon & Messer, 2019; Mason & Lewis, 2014; Sultson et al., 2017), and thus may be indirectly related to BD. Therefore, it was also examined whether these individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) could predict BD through their relationship with fat intake and BE.

This study showed for the first-time evidence of the influence of BE and fat intake on BD in young people. It is also the first time that evidence is provided for the indirect relationship of eating styles (emotional, external, and restrictive eating) and FA on BD, mediated by BE and fat intake. These results may help us to prevent BD in young people through interventions targeting young people with dysfunctional eating patterns.

As discussed above, FA has become very relevant in recent years. BE shares much variance with FA (Gearhardt, White, et al., 2011), and because it is such a prevalent and harmful construct in young people, it is necessary to know its risk factors in order to design the most appropriate prevention and intervention campaigns possible.

Many factors have been found to be strongly associated with FA, and therefore may be risk factors for this eating problem. These include age, with younger people being more likely to be addicted to food (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020). Another factor is gender. Females are more likely than males to have this problem (Gearhardt et al., 2016; Pursey et al., 2014). Moreover, a positive association between eating styles (emotional, external and restrictive eating) and FA has also been observed (Pepino et al., 2014; Schiestl & Gearhardt, 2018; Schulte & Gearhardt, 2017; Wardle et al., 1992; Wiss & Brewerton, 2020). Last but not least, a very strong relationship has been observed between BE and FA (Escrivá-Martínez et al., 2019; Gearhardt et al., 2016; Gearhardt et al., 2012), and between bulimia and FA (Meule et al., 2014). So far, all these variables have been shown to be associated with FA, however, it is unknown whether they may act as risk factors for FA. More literature is needed to indicate whether these factors may be risk factors for FA, and to see how much all of them can explain this problem. Therefore, another of our aims was also to analyze the predictive power of gender, age, BMI, eating styles, BE and bulimia on FA (Study #4).

This study demonstrated for the first time the prediction of BMI, eating styles, BE, and bulimia on FA in young people, with all variables explaining more than half of the variance in FA. These results point to the importance of considering all these variables in the prevention of FA in young people.

Finally, and considering the special time in which this thesis was developed, we

could not ignore one situation that could have had a major impact on eating behavior: the COVID-19 confinement. The appearance of COVID-19 with its measures to curb contagion could have particularly affected people's lifestyles and, specifically, the eating behavior of the young population. Several studies have shown very mixed findings. Whereas some studies have reported an increase in the consumption of unhealthy foods, as well as a higher BMI (Ammar et al., 2021; Pellegrini et al., 2020; Phillipou et al., 2020; Sidor & Rzymiski, 2020), other studies reported have found an improvement in eating habits and no change in BMI (Di Renzo et al., 2020; Haddad et al., 2021; Rodríguez-Pérez et al., 2020). These studies have been limited to asking about past dietary habits retrospectively, which could result in a reporting bias. To date, there are no works assessing eating behaviors before and during confinement, i.e., with actual data both before and during the period of home confinement. Thus, the next aim of this Doctoral Thesis was to assess differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before COVID-19 confinement (November 2019) and during COVID-19 confinement (April 2020) (Study #5). Furthermore, knowing the risk factors for these eating patterns at times of elevated stress may help us in their prevention at future moments. Therefore, we also aimed to analyze the predictive role of eating styles (emotional, external, and restrictive eating) on BMI, fat intake and BE during the period of strict COVID-19 confinement.

The results of our study indicated that COVID-19 confinement had a positive impact on eating behaviors, improving BE and fat intake of the youngsters. Eating styles remained stable, as would be expected (Meiselman et al., 1998), and stability in BMI was also demonstrated. This may be because young people adhered more to healthy eating patterns during confinement (Di Renzo et al., 2020). It has been also observed that only individuals who had previous weight problems gained weight during confinement (Di Renzo et al., 2020), and our sample had a normal BMI before confinement. Regarding the predictive power of eating styles on these behaviors, emotional eating predicted BMI and BE during confinement. External eating predicted fat intake during confinement, and restrictive eating predicted BE, and negatively predicted BMI and fat intake during confinement. This study was the first to compare eating behaviors before and during COVID-19 confinement, without including retrospective data. It was also the first to analyze the predictive role of eating styles on these behaviors. These results may help future health policies to focus prevention on

improving eating styles to curb dysfunctional eating behaviors and BMI at times of high stress.

Finally, our last aim was to assess whether eating styles moderated the relationship between perceived stress and BE during COVID-19 confinement (Study #6). We designed a mobile application to assess eating behaviors during confinement in real time, avoiding recall bias and improving ecological validity. Our results showed that the three eating styles moderated the relationship between perceived stress and BE during COVID-19 confinement. The influence of eating styles on the relationship between perceived stress and BE is demonstrated for the first time, with the strength of having been conducted in the context of a pandemic and using an Ecological Momentary Assessment (EMA). It is important to know which individuals are most at risk of BE in stressful situations, so that they will benefit most from interventions aimed at reducing BE in stressful times.

In conclusion, this Doctoral Thesis presents 6 studies, with the aim to increase knowledge about the relationship between dysfunctional eating patterns and BD in young people. In addition, different risk factors have been proposed that may influence these dysfunctional eating patterns and BD in young people. We hope that this research will help to design prevention, intervention, and treatment for all these behaviors that are so harmful to young people today.

## PREFACE

This Doctoral Thesis is based on the following six studies:

- Escrivá-Martínez, T., Herrero, R., Molinari, G., Rodríguez-Arias, M., Verdejo-García, A., & Baños, R. M. (2020). Binge eating and binge drinking: A two-way road? An integrative review. *Current pharmaceutical design*, 26(20), 2402-2415. <https://doi.org/10.2174/1381612826666200316153317> (**study 1**)
- Escrivá-Martínez, T., Galiana, L., Rodríguez-Arias, M., & Baños, R. M. (2019). The Binge Eating Scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Frontiers in Psychology*, 10, 530. <https://doi.org/10.3389/fpsyg.2019.00530> (**study 2**)
- Escrivá-Martínez, T., Galiana, L., Herrero, R., Rodríguez-Arias, M., & Baños, R. M. (2020). Understanding the Influence of Eating Patterns on Binge Drinking: A Mediation Model. *International Journal of Environmental Research and Public Health*, 17(24), 9451. <https://doi.org/10.3390/ijerph17249451> (**study 3**)
- Food addiction and their relationships with other eating behaviors in a college sample. In preparation (**study 4**)
- Escrivá-Martínez, T., Herrero, R., Rodríguez-Arias, M., & Baños, R. M. (2021). Eating behaviors, eating styles and body mass index during COVID-19 confinement in a college sample: a predictive model. *Journal of Behavioral Medicine*. Submitted (**study 5**)
- Escrivá-Martínez, T., Ciudad-Fernández, V., Herrero, R., Rodríguez-Arias, M., & Baños, R. M. (2021). Eating styles moderate the relationship between stress and binge eating: A study conducted during COVID-19 lockdown. *Foods*. Submitted (**study 6**)





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## ABREVIATURES

<b>Abreviatura</b>	<b>Significat</b>
A	Afartament de menjar
AA	Afartament d'Alcohol
AM	Addicció al Menjar
BES	Binge Eating Scale
IMC	Índex de Massa Corporal
mYFAS 2.0	Modified Yale Food Addiction Scale 2.0
YFAS	Yale Food Addiction Scale



## ABBREVIATIONS

<b>Abbreviation</b>	<b>Meaning</b>
<b>AUDIT</b>	Alcohol Use Disorders Identification Test
<b>BD</b>	Binge Drinking
<b>BE</b>	Binge Eating
<b>BES</b>	Binge Eating Scale
<b>BIS-15S</b>	Barrat Impulsiveness Scale-15 Spanish
<b>BMI</b>	Body Mass Index
<b>CFA</b>	Confirmatory Factor Analyses
<b>CFI</b>	Comparative Fit Index
<b>CI</b>	Confidence Interval
<b>DEBQ</b>	Dutch Eating Behavior Questionnaire
<b>EDI-3 RF</b>	Eating Disorder Inventory-3 Referral Form
<b>EMA</b>	Ecological Momentary Assessment
<b>FA</b>	Food Addiction
<b>MIMIC</b>	Multiple Indicators Multiple Causes
<b>mYFAS</b>	Modified Yale Food Addiction Scale
<b>mYFAS 2.0</b>	Modified Yale Food Addiction Scale 2.0
<b>PSS-4</b>	Perceived Stress Scale-4
<b>RMSEA</b>	Root Mean Squared Error of Approximation
<b>YFAS</b>	Yale Food Addiction Scale





# **1. INTRODUCCIÓ**



# Introducció general



## Introducció general

L'afartament de menjar (A) i l'afartament d'alcohol (AA) són problemes molt freqüents en els joves i s'associen amb conseqüències molt negatives (Bava i Tapert, 2010; Ferriter i Ray, 2011; Steele i cols., 2016). Atesa l'alta prevalença i les conseqüències relacionades amb la salut i la qualitat de vida, hi ha hagut un creixent conjunt d'investigacions que s'han interessat a estudiar les característiques compartides i la relació entre l'A i l'AA (Bahji i cols., 2019; Ferriter i Ray, 2011). Tanmateix, segons el nostre millor coneixement, cap estudi ha revisat els punts comuns, les diferències i la direccionalitat entre els dos comportaments. El primer objectiu d'aquesta Tesi va ser realitzar una revisió narrativa per identificar els punts en comú entre l'A i l'AA, les diferències i la influència d'un comportament sobre l'altre i viceversa (estudi 1).

Per avaluar l'A en població espanyola jove, es necessita una escala espanyola fiable i vàlida que mesurara el constructe d'A. La *Binge Eating Scale (BES)* s'utilitza habitualment per identificar els A (Gormally i cols., 1982), però, cap estudi ha analitzat les propietats psicomètriques de l'escala en població general espanyola. Per tant, el segon objectiu va ser validar la *BES* per mesurar l'A en població jove espanyola (estudi 2).

Els estudis epidemiològics han demostrat repetidament que l'alcohol pot estimular el consum d'aliments (Caton i cols., 2004, 2005, 2007; De Castro i Orozco, 1990; Westerterp-Plantenga i Verwegen, 1999; Yeomans, 2010). Tanmateix, la influència de la ingesta dietètica en el consum d'alcohol és menys coneguda. Un estudi recent en models animals ha demostrat el paper predictiu de la ingesta de greixos i l'A en l'AA (Blanco-Gandía, Ledesma, i cols., 2017), tot i que no hi ha estudis humans que hagen avaluat el paper predictiu d'aquests patrons de ingesta i dieta sobre l'AA. Es necessiten estudis translacionals que avaluen l'impacte de la ingesta de greixos i l'A sobre la vulnerabilitat a l'AA en els joves. Per tant, el tercer objectiu era examinar el paper predictiu dels patrons alimentaris disfuncionals (A i ingesta de greixos) sobre l'AA en els joves (estudi 3).

La literatura ha assenyalat per separat alguns factors que poden estar associats amb la ingesta de greixos, A, addicció al menjar (AM) i AA en joves (per exemple,

gènere, edat, IMC, estils alimentaris, etc.) (Camilleri i cols., 2014; Kakoschke i cols., 2015; Linardon i Messer, 2019; Mason i Lewis, 2014; Sultson i cols., 2017). No obstant això, encara no s'ha investigat si aquestes variables juntes poden actuar com a factors de risc de comportaments alimentaris disfuncionals (ingesta de greixos i A) i de consum excessiu d'alcohol en joves. Per tant, el quart objectiu d'aquesta Tesi Doctoral era analitzar si diferents variables individuals (gènere, IMC, impuls per la primesa, insatisfacció corporal, estils alimentaris, impulsivitat i AM) podien predir l'AA a través de la seua relació amb la ingesta de greixos i l'A (estudi 3).

Per avaluar l'AM, també calia utilitzar una escala fiable i vàlida que mesurara AM en joves espanyols. La *modified Yale Food Addiction Scale 2.0 (mYFAS 2.0)* és coneguda arreu del món per avaluar AM i ha obtingut propietats psicomètriques adequades (Schulte i Gearhardt, 2017). Tot i que ha estat validada en molts idiomes, encara no s'ha validat en espanyol. Per tant, el cinquè objectiu de la present Tesi Doctoral va ser validar la *mYFAS 2.0* per mesurar l'AM en població jove espanyola (estudi 4). El sisè objectiu va ser analitzar el poder predictiu del gènere, edat, IMC, estils alimentaris, A i bulímia sobre l'AM (estudi 4).

Els patrons alimentaris es poden modificar en moments d'estrès. La pandèmia de la COVID-19 i les mesures adoptades per frenar les infeccions van tenir un gran impacte en l'estil de vida dels joves, especialment en els hàbits alimentaris (Ammar i cols., 2021; Pellegrini i cols., 2020; Phillipou i cols., 2020; Sidor i Rzymiski, 2020). Segons el nostre millor coneixement, no hi ha estudis que avaluen els comportaments alimentaris abans i durant el confinament, és a dir, amb dades reals tant abans com durant el període de confinament domiciliari. Per tant, el setè objectiu va ser avaluar les diferències en els estils alimentaris (alimentació emocional, externa i restrictiva), A, ingesta de greixos i IMC abans i durant el confinament per COVID-19 (estudi 5). El vuitè objectiu va ser analitzar per primera vegada el paper predictiu dels estils alimentaris sobre l'IMC, la ingesta de greixos i l'A durant el període de confinament estricte de la COVID-19 (estudi 5).

És important saber quins factors poden predir aquests patrons alimentaris disfuncionals en moments d'estrès elevat, ja que això ens pot ajudar en la seua prevenció i tractament en futures pandèmies o en moments d'estrès futurs. Per tant, el novè i últim objectiu va ser avaluar si els estils alimentaris moderaven la relació

entre l'estrès percebut i l'A durant el confinament per COVID-19 (estudi 6). Es va dissenyar una aplicació mòbil per mesurar l'A i l'estrès percebut durant el confinament en temps real, evitant el biaix de record i millorant la validesa ecològica. Tot i que aquesta relació s'havia estudiat en temps normals, encara no s'havia provat en èpoques de gran estrès. Amb aquest estudi, pretenem respondre a la pregunta: "Poden els estils alimentaris millorar la relació entre l'estrès percebut i l'A durant el confinament per COVID-19?".

La introducció es divideix en cinc apartats. En el primer apartat es defineixen els principals problemes d'alimentació i beguda dels joves, i es presenten la prevalença i les conseqüències associades a cada problema. També es presenten els instruments més utilitzats per avaluar A i AM.

En el segon apartat, es discuteixen els punts en comú entre A i AA, i les variables personals i els mecanismes neurobiològics que poden predir ambdues conductes.

En el tercer apartat, s'explora la relació entre A i AA, discutint la influència d'un sobre l'altre i viceversa.

En el quart apartat, es presenten les principals variables que s'han demostrat que estan associades a patrons alimentaris disfuncionals (ingesta de greixos i A) i que, per tant, també poden influir indirectament en l'aparició i desenvolupament de l'AA en els joves. També es presenten les variables que estan més relacionades amb l'AM i que també poden actuar com a factors de risc en aquesta problemàtica.

A la cinquena i última secció, s'explora la influència del confinament estricte de la COVID-19 en els hàbits alimentaris dels joves. També es comenten els principals factors de risc que poden haver influït en els patrons alimentaris disfuncionals dels joves durant el confinament per la COVID-19.





# 1.1. Joventut i hàbits inadequats en relació als patrons alimentaris i al consum d'alcohol



## **1.1. Joventut i hàbits inadequats en relació als patrons alimentaris i al consum d'alcohol**

La joventut (18-30 anys) es considera un període de vulnerabilitat, en la qual és freqüent observar en els joves problemes en els hàbits alimentaris i un augment del consum d'alcohol. En particular, s'observen sovint trastorns alimentaris, consum d'alcohol i drogues i una tendència més gran a buscar noves sensacions i comportaments de risc (Bava i Tapert, 2010).

El consum d'alcohol és un factor de risc molt important per a la salut dels joves. Actualment, l'alcohol és la droga més consumida entre els joves (Delegació del Govern per al Pla Nacional sobre Drogues, 2018; Inchley i cols., 2018). Tanmateix, el cervell encara està acabant de madurar a aquesta edat i l'alcohol produeix un efecte neurotòxic que pot danyar greument la salut dels joves. Així, els estudis indiquen que l'alcohol produeix múltiples conseqüències que poden arribar a ser irreversibles en els joves, com ara la conducció insegura, l'agressivitat, la conducta sexual i de risc, els problemes emocionals, el baix rendiment acadèmic i els trastorns per consum d'alcohol en l'edat adulta (Cservenka i Brumback, 2017; Jones i cols., 2018; Kuntsche i cols., 2017).

Un altre problema que ha crescut exponencialment els darrers anys i s'ha convertit en un greu problema de salut pública és l'alimentació. Els patrons alimentaris disfuncionals són cada vegada més freqüents entre els joves, caracteritzats per beure moltes begudes ensucrades, saltar-se menjars o menjar grans quantitats d'aliments processats rics en greixos no saludables, és a dir, greixos saturats i trans, així com sucres (Fleming-Milici i Harris, 2020; Navarro-Cruz i cols., 2017). Viure amb companys de la universitat, les dificultats econòmiques o la capacitat reduïda per cuinar bé poden ser els responsables d'aquests patrons alimentaris disfuncionals. Aquests hàbits alimentaris estan associats amb el sobrepès i l'obesitat (Drewnowski, 2007), que al seu torn estan fortament associats a complicacions greus de salut com la diabetis, el càncer o les malalties cardiovasculars (Swinburn, 1998). Aquests problemes també comporten costos significatius per a la societat, que es reflecteixen en l'augment de la càrrega sanitària, la disminució de la productivitat laboral i l'augment de les baixes per malaltia (Kleinman i cols., 2014).

Generalment, el problema no radica només en el consum d'alcohol o d'un tipus concret de dieta, sinó també en el patró de consum, és a dir, el fenomen conegut com a "afartament". El terme "afartament" s'utilitza habitualment per descriure un patró caracteritzat per un consum gran i incontrolat, que es pot aplicar tant a l'alcohol (afartament d'alcohol - AA) com als aliments (afartament de menjar - A) (Associació Americana de Psiquiatria, 2013; Paul i cols., 2011).

L'AA es defineix com el patró de consum que augmenta la concentració d'alcohol en sang a  $\geq 0,08$  g per cent, i correspon específicament al consum de més de quatre begudes en dones (cinc o més en homes) en un període de dues hores (Tapia-Rojas i cols., 2017). Aquest patró de consum té una major incidència en la població juvenil, es manifesta per igual en ambdós sexes i la seua forma habitual de consum s'associa a una baixa percepció de risc i una sensació de pèrdua de control sobre aquesta ingesta (Balodis i cols., 2009; Carlson i cols., 2010; Galán i cols., 2014; Soler-Vila i cols., 2014). Aquest tipus de patró s'ha convertit en la forma preferida dels joves de consumir alcohol (Dawson i cols., 2015). Una quarta part dels joves informa consumir alcohol (Kanny i cols., 2018) i la meitat informa haver consumit cinc o més copes en una ocasió (Wechsler i Nelson, 2008). Aquest problema té múltiples implicacions a nivell individual, familiar, social i comunitari (Ferriter i Ray, 2011; Laghi i cols., 2014). Una de les conseqüències més preocupants d'aquest consum és el risc d'accident de trànsit en els joves, ja que quasi la meitat dels joves que han mort en accidents de trànsit han consumit alcohol (Galicia-Paredes i cols., 2017). A Espanya, l'AA també s'associa a altes taxes de morbiditat i mortalitat, emergències, dependència de l'alcohol, intoxicació aguda, consum d'altres drogues emergents, comportaments sexuals de risc, conflictes interpersonals i agressivitat (Galicia-Paredes i cols., 2017; Guitart i cols., 2011; Isorna i cols., 2015; Moral i Ovejero, 2011).

L'A es defineix com la ingesta objectiva d'una quantitat excessiva d'aliments en un període curt de temps i una sensació de pèrdua de control sobre aquesta ingesta, sovint acompanyada d'angoixa emocional, com la vergonya o la culpa, i l'absència de conductes compensatòries (Associació Americana de Psiquiatria, 2013). Aquest concepte està íntimament lligat al consum d'aliments rics en greixos i en sucres, ja que l'A sol presentar-se amb aquests aliments rics en calories (Allison i Timmerman,

2007; Avena i cols., 2009). La prevalença d'A en els joves és alta (0,3%-3,1%) (Sonneville i cols., 2013), i ha mostrat un augment en les últimes dècades (Hudson i cols., 2007; Smink i cols., 2014), especialment en l'edat adulta jove (Goldschmidt i cols., 2014). S'han reportat dos pics d'inici d'A en la joventut, el primer després de la pubertat (al voltant dels 14 anys) i el segon al final de l'adolescència (19-24 anys) (Smink i cols., 2014). Tot i que l'A pot estar present en els trastorns de la conducta alimentària (anorèxia, bulímia i trastorn d'A) així com en persones amb sobrepès i obesitat, és molt freqüent que les persones tinguen fartades de menjar sense ser obesas; a més a més, la majoria de les persones obesas no tenen A (Hudson i cols., 2007; Kessler i cols., 2013). Aquests episodis també tenen conseqüències molt negatives, com ara afeccions mèdiques com la diabetis, la hipertensió, l'obesitat, la malaltia celíaca, la síndrome metabòlica, així com problemes psicològics, com els trastorns depressius i d'ansietat (Hudson i cols., 2007; Keski-Rahkonen i Mustelin, 2016; Kessler i cols., 2013).

L'elevada prevalença de l'A associada als joves i les conseqüències observades han provocat la necessitat de desenvolupar mesures d'autoinforme de l'A. La *Binge Eating Scale (BES)* (Gormally i cols., 1982) és l'escala més coneguda per mesurar A. Ha demostrat una gran sensibilitat i especificitat per detectar els A (Grupski i cols., 2013) i la seua validesa s'ha confirmat en mostres clíniques i no clíniques (Duarte i cols., 2015; Hood i cols., 2013). Malgrat la seua rellevància, l'estructura del factor és controvertida, amb alguns estudis que informen que només hi ha un factor d'A, i altres estudis que informen que la *BES* consta de dos factors: A cognitiu i A conductual (Duarte i cols., 2015; Zúñiga i Robles, 2006). L'escala s'ha validat en molts idiomes però encara no s'ha validat en espanyol a la població general. Es necessiten més investigacions sobre l'estructura factorial de l'escala. A més a més, atesa l'elevada prevalença d'aquest problema en els joves i les conseqüències associades, calen escales fiables que mesuren l'A en la població general espanyola per tenir una mesura fiable i vàlida en aquesta població.

Un concepte molt relacionat amb l'A i que ha esdevingut molt popular en les últimes dècades és el concepte d'addicció al menjar (AM) (Gearhardt, White, i cols., 2011). Concretament, la literatura és extensa sobre el debat de paral·lelismes entre els trastorns per ús de substàncies i el trastorn d'A (Gearhardt i cols., 2009a). Aquest

debat especula sobre la qüestió de si els aliments altament processats (per exemple, xocolata o pizza) tenen un potencial addictiu comparable a les drogues (Gearhardt, Davis, i cols., 2011), que poden reflectir correlacions neuronals comuns (per exemple, l'activació de les mateixes vies de recompensa cerebral). En aquest sentit, l'AM es defineix com la ingesta excessiva i desregulada d'aliments rics en greixos i/o sucres (Schulte i cols., 2015), entre d'altres: disminució del control sobre el consum d'aquests aliments, desig intens d'aquests aliments, intents fallits d'eliminar aquest consum, ús continuat malgrat les conseqüències negatives, etc.

El concepte d'AM continua sent un tema debatut (Avena i cols., 2012). Un punt important de debat és que no hi ha una definició clara d'AM. Els opositors al concepte assenyalen ràpidament que alguns símptomes d'AM (abstinència o tolerància) es limiten als models animals i no es produeixen en el comportament alimentari humà (Rippe i Marcos, 2016). Altres investigadors argumenten que el nucli de la psicopatologia de l'alimentació és diferent del de la dependència de substàncies, per exemple, en els problemes alimentaris hi ha preocupacions sobre la forma i el pes que no estan presents en el consum de substàncies (Ziauddeen i cols., 2012). Tanmateix, en suport del concepte d'AM, altres assenyalen que els paradigmes utilitzats en models animals poden ser comparables al comportament alimentari d'individus amb problemes alimentaris i que els criteris d'abús i dependència de drogues es poden transposar perfectament als aliments (Meule, 2014).

Per superar aquest debat, es va desenvolupar la *Yale Food Addiction Scale* (YFAS; Gearhardt i cols., 2009b). Aquest instrument mesura la presència de símptomes d'AM a partir dels criteris del trastorn per consum de substàncies del DSM-IV, amb l'addició de criteris de significació clínica, és a dir, malestar o deteriorament significatiu en diferents àmbits (personal, social, laboral, etc.). Es pot fer un diagnòstic d'AM quan es compleixen tres criteris i hi ha angoixa o deteriorament significatiu. Aquesta escala i les seues respectives versions han mostrat bones propietats psicomètriques i han estat ben recolzades en la investigació. La nova versió de l'escala, la *YFAS 2.0*, reflecteix els canvis del DSM-5 en els criteris de diagnòstic del trastorn per consum de substàncies (Associació Americana de Psiquiatria, 2013; Gearhardt i cols., 2016) i sembla tenir una millor consistència interna que la *YFAS* (Gearhardt i cols., 2016). La versió curta d'aquesta escala, la *mYFAS 2.0*, ja ha estat

desenvolupada i validada, i ha obtingut propietats psicomètriques adequades (Schulte i Gearhardt, 2017). La *mYFAS 2.0* s'ha validat en molts altres idiomes (anglès, àrab, portuguès, txec, italià, francès). Tanmateix, encara no s'ha validat en espanyol. Es necessiten escales curtes validades en espanyol que avaluen l'AM d'acord amb els criteris del DSM-5, per tal d'observar si la AM és clínicament prevalent en els joves en el context espanyol, i desenvolupar estratègies per prevenir-la o tractar-la.

En relació a la prevalença d'AM, els resultats són alarmants. Quasi una quarta part de les persones amb obesitat compleixen els criteris d'AM (Pursey i cols., 2014), i també és molt freqüent en joves de pes normal, amb alguns estudis que indiquen que l'11-25% dels estudiants universitaris tenen AM (Gearhardt i cols., 2009b; Murphy i cols., 2014; Pursey i cols., 2014; Rostanzo i Aloisi, 2021). Aquest problema representa greus conseqüències per a la salut de les persones, amb un augment del consum d'aliments processats i les seues conseqüències associades (Steele i cols., 2016), que també impacten indirectament en els costos de la salut pública.

Com es pot veure en aquest apartat, els problemes de consum d'alcohol i les conductes alimentaries disfuncionals poden augmentar la vulnerabilitat dels joves a desenvolupar diferents patologies mèdiques i de salut mental (càncer, diabetis, sobrepès, obesitat, problemes de salut mental greus, etc.). Aquestes conductes han augmentat de manera exponencial en els darrers anys, sobretot en els joves, i constitueixen un greu problema de salut pública. És important establir polítiques de salut que ajuden a frenar aquestes conductes i reduir els danys associats a curt i llarg termini. Per a això, cal més investigació sobre la seua prevalença, conseqüències, comorbiditat, així com els factors de risc que poden estar precipitant aquestes conductes.

L'elevada prevalença d'A i d'AA en els joves i les conseqüències sobre la salut i la qualitat de vida han fet que s'haja augmentat l'interès per investigar les característiques compartides i la relació entre ambdós (Bahji i cols., 2019; Ferriter i Ray, 2011). A la secció següent, proporcionem un marc per entendre l'A i l'AA, discutint les similituds entre ambdós, les característiques personals i els mecanismes neurobiològics que poden subjure a ambdós comportaments.





## 1.2. Aspectes comuns entre l'afartament i l'afartament d'alcohol i els factors que hi contribueixen



## **1.2. Aspectes comuns entre l'afartament i l'afartament d'alcohol i els factors que hi contribueixen**

La primera part d'aquesta secció se centra en les similituds trobades entre l'A i l'AA. La segona part se centra en els contribuents rellevants comuns per explicar l'ocurrència de l'A i l'AA.

L'A i l'AA es defineixen com el consum d'una substància, que pot ser aliment o alcohol, en quantitats molt grans i durant un període de temps curt (Associació Americana de Psiquiatria, 2013; Paul i cols., 2011). La literatura indica que ambdues conductes tenen potencial addictiu (Benjamin i Wulfert, 2005), ja que es caracteritzen per una resposta incontrolable i repetitiva (Ferriter i Ray, 2011; Laghi i cols., 2014) que s'associa amb efectes negatius personals, acadèmics i socials (Ferriter i Ray, 2011).

Les investigacions indiquen que l'A i l'AA poden ser un factor de risc per als trastorns de l'alimentació i els trastorns per l'ús de substàncies (Grant i cols., 2001). A més de la seua alta comorbiditat, l'A pot ser predictiu de problemes d'alcohol (Field i cols., 2012; Sonnevile i cols., 2013), i l'AA pot predir un augment dels patrons alimentaris disfuncionals (Nelson i cols., 2009).

En relació a la seua edat d'inici, tant l'A com l'AA tendeixen a començar a l'adolescència, i s'observen amb més freqüència en l'edat adulta jove (Kanny i cols., 2018; Kessler i cols., 2013). Aquestes conductes de risc són molt freqüents entre els estudiants universitaris (Ferriter i Ray, 2011), amb quasi la meitat dels estudiants que consumeixen una quantitat més gran d'aliments que altre en circumstàncies similars (Kelly-Weeder, 2011) i una pèrdua de control sobre aquesta ingesta (Lipson i Sonnevile, 2017). El mateix nombre s'observa en la prevalença de joves que consumeixen alcohol (Croteau i Morrell, 2019; Ferriter i Ray, 2011). Aquesta etapa de desenvolupament és fonamental en la maduració del cervell i els canvis cognitius i fisiològics que es produeixen, fet que pot explicar per què els joves són més vulnerables a totes aquestes amenaces ambientals (Harris i Fleming-Millici, 2019).

La literatura també ha assenyalat un altre aspecte comú entre l'A i l'AA: les conseqüències negatives associades. Ambdues conductes produeixen conseqüències molt similars en les persones, incloent problemes mentals, com la depressió i l'ansietat, o problemes mèdics, com la diabetis, el sobrepès o l'obesitat,

així com una major probabilitat d'involucrar-se en conductes d'alt risc i un baix rendiment acadèmic (Fazzino i cols., 2017; Ferriter i Ray, 2011; Kuntsche i cols., 2017; Raevuori i cols., 2015; Striegel-Moore i Franko, 2008).

A més d'examinar les similituds en ambdós, també cal centrar-se en les característiques personals comunes i els mecanismes psicofisiològics que poden contribuir a una millor comprensió de la causa d'ambdós problemes (Ferriter i Ray, 2011; Ventura-Cots i cols., 2017).

Les perspectives que han examinat els problemes d'alimentació i els problemes de consum d'alcohol o substàncies apunten a la desregulació de l'estat d'ànim com un dels factors causals d'ambdues conductes. S'ha trobat que els problemes d'estat d'ànim són rellevants per predir l'AA (Lamis i cols., 2010) i l'A (Phillips i cols., 2016; Wheeler i cols., 2005). L'estrès, entès com un esdeveniment incontrolable que afecta els processos adaptatius bàsics per recuperar l'homeòstasi (Sinha, 2008), juga un paper central com a factor de risc tant en els problemes d'alcoholisme juvenil (Kenney i cols., 2013) com en l'A (Phillips i cols., 2016). Una possible explicació de la relació entre l'estrès amb A i AA és el cortisol, una hormona alliberada en moments d'estrès que pot tenir un paper important com a reforçador d'aliments saborosos i drogues (Naish i cols., 2018; Sinha, 2001). Encara que el mecanisme subjacent a la relació entre l'A i l'AA amb la desregulació de l'estat d'ànim segueix sent desconegut, s'ha suggerit que ambdues conductes s'utilitzen com a mecanismes d'afrontament per fer front a l'estrès i regular les emocions negatives (Laghi i cols., 2009, 2014; Pompili i Laghi, 2017).

Els factors socioculturals també tenen un paper important en l'aparició i el desenvolupament de l'A i l'AA. Aquests inclouen la pressió dels companys, ja que els individus tenen més probabilitats de menjar i beure en grans quantitats per complir amb les normes del grup, millorar el seu estatus social i, finalment, ser més acceptats pel seu grup d'iguals (Laghi i cols., 2012, 2015; Lai i cols., 2013). En canvi, també hi ha factors que poden reduir el risc d'ambdues conductes, la més important de les quals és la família. És ben sabut que les relacions familiars saludables i els alts nivells de calidesa dels pares redueixen la probabilitat d'A i d'AA (Cleveland i cols., 2008; Langdon-Daly i Serpell, 2017).

També hi ha diversos mecanismes neurobiològics principals que poden ser la base d'ambdós comportaments. Els problemes alimentaris i l'addicció a l'alcohol i les substàncies comparteixen alteracions en els mateixos sistemes cerebrals, és a dir, els sistemes dopaminèrgic, glutamatèrgic, serotoninèrgic i opioide endògen (Fletcher i Kenny, 2018; Hadad i Knackstedt, 2014; Harrop i Marlatt, 2010; Pearlstein, 2002; Rothman i cols., 2008). Així, el circuit de recompensa mesolímbic és responsable del desig i la regulació de la motivació per consumir substàncies gratificants, i pot ser activat tant per drogues com per aliments (Davis i cols., 2009; Volkow i cols., 2008). Així, tant l'alimentació per plaer com l'abús de substàncies activen vies de recompensa comunes. Al seu torn, el sistema opioide, relacionat específicament amb l'experiència sensorial del gust, sembla que també hi intervé (Berridge, 2009). A més a més, diversos estudis suggereixen que els factors genètics poden tenir un paper important en la comorbiditat d'ambdues conductes (Pearlstein, 2002; Peveler i Fairburn, 1990; von Ranson i cols., 2003).

Un altre component que pot influir en ambdós comportaments és el *craving* (desig) per la substància o el menjar. El *craving* és ben conegut per ser un component essencial dels problemes d'addicció (Potenza i Grilo, 2014), sent un dels principals criteris de diagnòstic dels trastorns per consum de substàncies (Associació Americana de Psiquiatria, 2013). Els desencadenants que generen el *craving*, que poden ser senyals ambientals que recorden la substància (droga o aliment), produeixen una alliberació de dopamina que impulsa a buscar i consumir la substància (Volkow i cols., 2008). Per exemple, el *craving* de la substància induïda per indicis ambientals s'ha relacionat amb regions cerebrals implicades en el paper de la recompensa, com l'hipocamp o l'amígdala (Bonson i cols., 2002; Schneider i cols., 2001).

Finalment, els trets de personalitat també poden ser mecanismes subjacents per a l'aparició d'ambdues conductes. Els factors precipitants inclouen el neuroticisme, la impulsivitat i la sensibilitat a la recompensa. La desregulació de l'estat d'ànim pot augmentar la probabilitat d'A i d'AA, com s'ha comentat anteriorment (Lamis i cols., 2010; Phillips i cols., 2016; Wheeler i cols., 2005). Aquest concepte està íntimament lligat al tret de personalitat del neuroticisme. El neuroticisme es defineix com la tendència a experimentar emocions negatives (Costa i McCrae, 1980). S'ha trobat que els individus neuròtics que solen experimentar afectes depressius o ansiosos

participen en A i AA en major mesura (Davis i Jamieson, 2005; Kuntsche i cols., 2008).

La impulsivitat és una de les característiques de la personalitat més implicades en l'A i l'AA. La impulsivitat es defineix com la resposta als estímuls sense planificació i sense tenir en compte els efectes adversos que poden derivar d'un comportament impulsiu (Moeller i cols., 2001). Es conceptualitza com un tret multidimensional que consta de diversos trets, com ara la urgència, la falta de planificació i la recerca de sensacions (Whiteside i Lynam, 2001). La investigació indica que el tret d'urgència, entès com la tendència a actuar precipitadament en resposta a estats emocionals negatius, és una característica comuna entre l'A i l'AA (Fischer i cols., 2004). Per tant, es podria especular que l'A i l'AA poden ser una resposta precipitant a alts nivells d'emocionalitat negativa.

Finalment, la teoria de la sensibilitat a la recompensa planteja la hipòtesi que les persones que mengen en excés i prenen drogues fan servir la substància (en aquest cas, aliments o substància d'abús) per experimentar una recompensa, mentre que altres no necessiten la substància perquè senten la recompensa d'altres estímuls (Volkow i cols., 1999). Per tant, les persones més sensibles a les recompenses buscaran la substància per augmentar el plaer, que no augmenta per altres estímuls (Loxton i Dawe, 2007).

Aquesta secció proporciona un marc per a una comprensió més clara dels A i dels AA. D'una banda, es destaquen els mecanismes comuns entre els A i els AA, com ara l'aparició i naturalesa del problema i les conseqüències negatives associades. D'altra banda, es comenten els mecanismes causals d'ambdues conductes, com ara el paper de la desregulació de l'estat d'ànim, els factors socioculturals, els mecanismes neurobiològics, el *craving* i els trets de personalitat.

Tenint en compte les característiques compartides de l'A i l'AA i la seua alta comorbiditat, els estudis han intentat dilucidar si un podria influir en l'altre i viceversa. A la secció següent, revisem els estudis que suggereixen la influència de l'AA en l'A i viceversa, alhora que discutim les limitacions dels estudis i les implicacions per a la pràctica clínica.

### 1.3. Relació entre l'afartament i l'afartament d'alcohol





### 1.3. Relació entre l'afartament i l'afartament d'alcohol

Com s'ha indicat anteriorment, hi ha una clara comorbiditat entre l'A i l'AA. A més de conèixer la seua comorbiditat, és important saber com poden influir-se mútuament, per entendre amb més precisió l'etiologia d'ambdues conductes. En aquesta següent secció, intentem oferir una visió general de l'evidència que apunta la influència d'una sobre l'altra.

En relació a la influència de l'AA en l'A, l'evidència és més forta. La investigació que ha intentat abordar com l'alcohol exerceix un efecte sobre el consum d'aliments ha considerat diverses característiques de consum d'aliments i alcohol, com ara el tipus d'aliment consumit i la quantitat, el tipus d'alcohol consumit i la quantitat i el temps de consum.

Molts estudis que han intentat analitzar aquesta relació assenyalen que l'alcohol té el potencial d'estimular el consum d'aliments quan es consumeix abans o durant els menjars (Caton i cols., 2004, 2005, 2007; Westerterp-Plantenga i Verwegen, 1999; Yeomans, 2010). En línia amb això, diversos estudis epidemiològics indiquen que es consumeix més aliments els dies en què es consumeix més alcohol (De Castro i Orozco, 1990; De Castro, 2009). Això es pot explicar per diversos mecanismes, per exemple, l'activació reduïda del sistema que controla la sacietat. S'ha trobat que la ingesta d'alcohol redueix els senyals de sacietat després del consum d'alcohol (Raben i cols., 2003; Röjdmarm i cols., 2001). Un altre mecanisme explicatiu d'aquesta relació radica en els sistemes de recompensa. L'alcohol estimula tant el gust (entès com el plaer del gust dels aliments) com el *craving* (entès com la motivació per menjar; aquest component depèn més del sistema dopaminèrgic) (Melis i cols., 2009). Altres estudis indiquen que es mengen més aliments després de beure alcohol a causa de la pèrdua d'autocontrol (Caton i cols., 2015; Yeomans, 2010).

La investigació també ha intentat dilucidar si el tipus d'aliment influeix en la relació entre el consum d'alcohol i la posterior ingesta d'aliments. Diversos estudis semblen indicar que la ingesta d'alcohol augmenta la probabilitat de consumir aliments (Caton i cols., 2007; Yeomans, 2010). Relacionat amb això, un estudi va explorar l'efecte de l'alcohol en la ingesta d'aliments ad libitum (específicament, en la condició de dieta alta en greixos i la condició de dieta baixa en greixos) (Tremblay i cols., 1995).

L'estudi va demostrar que l'alcohol no inhibeix la ingesta d'aliments, és a dir, quan es consumeix alcohol durant els menjars, el contingut energètic consumit no es compensa amb una disminució de la ingesta energètica. Això també corrobora la hipòtesi que l'alcohol té un impacte en la ingesta d'aliments grassos. Un estudi molt recent també va indicar que els bevedors excessius tenen una ingesta més gran de calories i un consum de greixos totals i saturats que els bevedors moderats o baixos (Rosen i cols., 2021).

També és important tenir en compte la quantitat d'alcohol consumida que es necessita perquè l'alcohol tinga un impacte en el consum d'aliments. Tot i que la literatura en aquesta àrea és escassa, un estudi va trobar que la quantitat d'alcohol influeix en la quantitat d'aliments consumits (Caton i cols., 2004). Així, com més gran siga la dosi d'alcohol, més gran serà la ingesta d'energia. Si la quantitat d'alcohol és mínima, no té cap efecte sobre la ingesta d'aliments. Això podria corroborar la teoria que el principal mecanisme pel qual es menja més aliments després de beure alcohol és la pèrdua d'autocontrol (Caton i cols., 2015; Yeomans, 2010). S'entén que amb dosis baixes d'alcohol no hi ha pèrdua de control, i per tant, no es tendeix a consumir més aliments.

En resum, els estudis que intenten dilucidar com l'alcohol pot afectar la ingesta d'aliments han explorat tant el tipus i la quantitat d'aliments com el tipus i la quantitat de consum d'alcohol. Aquests estudis indiquen que l'alcohol pot estimular el consum d'aliments quan es menja o quan es beu abans de menjar. A més a més, sembla que hi ha una relació dosi-resposta, ja que, si la quantitat d'alcohol és mínima, aquest mecanisme no es produeix, però si la quantitat d'alcohol és més alta, aquesta relació sí que es produeix. Diferents mecanismes poden explicar aquesta relació, inclosa la pèrdua d'autocontrol després del consum d'alcohol (Caton i cols., 2015; Yeomans, 2010), els efectes de l'alcohol en la reducció dels senyals de sacietat (Raben i cols., 2003; Röjdmak i cols., 2001) i la propietat de l'alcohol per augmentar el valor de recompensa dels aliments (Berridge, 2009; Cooper, 2005). Per tant, tenint en compte aquests estudis, es podria concloure que hi ha una relació unidireccional de l'AA a l'A. Tanmateix, i al revés, l'A també pot influir en l'AA? En els paràgrafs següents, es dedica una atenció especial a la influència dels hàbits alimentaris en l'AA.

La literatura també ha intentat analitzar la influència dels patrons d'alimentació

en el consum d'alcohol. Un estudi prospectiu de quasi 17.000 adolescents va trobar que l'A no va predir més AA, però sí que va predir el consum futur de drogues (Sonneville i cols., 2013). En aquest estudi, l'A es va definir com el consum de grans quantitats d'aliments en un curt període de temps i la pèrdua de control sobre aquesta ingesta (Associació Americana de Psiquiatria, 2013). L'AA es va definir com la freqüència de consum de quatre o més begudes en poques hores l'any anterior. Un altre estudi de quasi 500 dones adolescents va assenyalar, d'acord amb l'anterior, que els símptomes primerencs del trastorn alimentari prediuen més abús de substàncies (Measelle i cols., 2006).

Aquests resultats van ser corroborats per un altre estudi similar realitzat amb més d'11.000 joves al Regne Unit (Micali i cols., 2015). En aquest cas, l'A es va definir segons el DSM-5 (A setmanal, malestar intens sobre l'A, i almenys tres dels següents símptomes: menjar més ràpid de l'habitual, menjar grans quantitats, menjar fins a omplir-se, menjar i no tenir gana, menjar sol a causa de la vergonya per la quantitat menjada i sentir fàstic amb un mateix). El subllindar d'A fa referència a l'A mensual. L'AA es va definir amb la pregunta: "Has pres sis o més begudes alcohòliques almenys una vegada l'any passat?" Els resultats van indicar que tampoc hi havia cap associació entre l'A i el subllindar d'A amb l'AA.

En canvi, un estudi de quasi 9.000 dones va avaluar si l'A, el trastorn de purga, la bulímia nerviosa i el trastorn alimentari no especificat d'una altra manera (A mensual o episodi de purga o A sense perdre el control) són predictors del consum freqüent d'alcohol (Field i cols., 2012). Els resultats van revelar que els participants amb trastorn de purga tenien un risc significativament més elevat de consum de drogues i AA, de manera que el comportament alimentari pot influir en el consum d'alcohol.

Estudis anteriors apunten a una relació positiva i unidireccional entre els patrons alimentaris disfuncionals i el posterior abús de substàncies. Això podria ser perquè l'A pot produir sentiments negatius, com ara vergonya o culpa, i el consum de substàncies pot ser un comportament d'afrontament per a aquestes emocions negatives (Lai i cols., 2013). No obstant això, la relació predictiva dels patrons d'alimentació inadaptats sobre l'AA és contradictòria. Tot i que alguns estudis apunten a una relació unidireccional (Field i cols., 2012), altres no troben cap relació (Micali i cols., 2015; Sonneville i cols., 2015). Això pot ser degut a problemes en les definicions del

constructe, ja que, mentre que alguns estudis han avaluat els trastorns de l'alimentació, d'altres han avaluat la simptomatologia de l'alimentació. A més a més, alguns estudis utilitzen variables contínues i altres variables categòriques, que també poden tenir un efecte diferent en els resultats (Puccio i cols., 2016). Així mateix, alguns estudis inclouen el consum d'alcohol dins del consum de substàncies, mentre que d'altres no, i alguns es van realitzar abans de l'aparició del DSM-5, mentre que altres es van realitzar després del DSM-5.

Donada l'elevada comorbiditat entre ambdues conductes, i ateses les limitacions anteriors, les línies d'investigació recents han continuat avaluant aquesta relació. Concretament, han volgut observar si el consum de greixos i sucres i l'A prediuen el consum posterior d'alcohol.

Un estudi recent va trobar que tenir una major propensió a consumir aliments agradables (alt en greixos i sucre) en els nens va predir un major risc de consum d'alcohol durant la joventut (Mehlig i cols., 2018). Un altre estudi recent també va demostrar que les persones que preferien els dolços tenien més desitjos d'alcohol que les que no preferien els dolços (Bouhlal i cols., 2018). En línia amb això, també s'ha trobat que tenir un fenotip de gust dolç s'associa a problemes futurs d'alcohol per a aquells que ja han iniciat el consum d'alcohol (Kampov-Polevoy i cols., 2014).

Una nova línia d'investigació utilitzant models animals també ha suggerit aquesta relació. Concretament, han observat que l'A i la ingesta de greix prediuen un augment del consum excessiu d'etanol i cocaïna en ratolins adolescents (Blanco-Gandía, Cantacorps, i cols., 2017; Blanco-Gandía, Ledesma, i cols., 2017). Per entendre-ho millor, els investigadors van avaluar les conseqüències de l'exposició a una dieta alta en greixos durant l'adolescència sobre els efectes de reforç de l'etanol i la cocaïna (Blanco-Gandía, Cantacorps, i cols., 2017; Blanco-Gandía, Ledesma, i cols., 2017). L'administració de la dieta alta en greixos es va avaluar de dues maneres diferents: el model de consum continu (ad libitum) i el model de consum intermitent i limitat, en forma d'A. En el model d'A, els ratolins alimentats amb una dieta estàndard durant la seua adolescència i als quals se'ls va permetre l'accés a la dieta alta en greixos durant només dues hores al dia durant tres dies a la setmana van desenvolupar un patró d'A. Aquest equip de recerca va observar que el consum continuat d'una dieta alta en greixos produeix canvis metabòlics que condueixen a l'augment de pes i la

hiperleptinèmia, però no augmenta la sensibilitat als efectes reforçants de la cocaïna i l'alcohol. En canvi, la ingesta excessiva de greixos en un curt període de temps no indueix canvis metabòlics, però al contrari del que es va observar en el consum continuat, sí que prediu el consum posterior de cocaïna i etanol, augmentant així la vulnerabilitat a aquestes substàncies (Blanco-Gandía, Cantacorps, i cols., 2017; Blanco-Gandía, Ledesma, i cols., 2017).

Aquests resultats confirmarien la comorbiditat observada entre els problemes alimentaris i l'abús d'alcohol i substàncies. A més, aquests estudis demostren que els patrons alimentaris disfuncionals modifiquen no només el metabolisme del nostre cos, sinó també com el nostre cervell respon a les drogues d'abús.

Tot i que els estudis epidemiològics demostren l'impacte d'ambdós problemes en els joves, no hi ha estudis que s'hagen centrat a avaluar la relació entre la ingesta i els patrons dietètics i el consum d'alcohol en els joves. Es necessiten estudis de revisió per oferir una visió general actualitzada de l'A i l'AA. A més, calen estudis translacionals per avaluar l'impacte dels hàbits alimentaris, especialment el consum de dietes altes en greixos i sucres i l'A, en la susceptibilitat al consum d'alcohol dels joves. Aquests futurs estudis tindran implicacions per a la prevenció i el tractament de l'AA en els joves, un problema que, com s'ha assenyalat en apartats anteriors, és molt prevalent i genera moltes conseqüències negatives en la joventut actual.

Com ha demostrat aquesta secció, els patrons alimentaris disfuncionals poden influir en el consum excessiu d'alcohol. Cal treballar més per ajudar a ampliar el coneixement sobre els factors de risc que poden influir en aquests comportaments alimentaris disfuncionals en els joves, ja que, a més de totes les conseqüències no desitjades dels hàbits alimentaris disfuncionals, també poden influir indirectament en el consum d'alcohol dels joves. En la següent secció, es comenten les principals variables que s'han demostrat que estan associades a conductes alimentaries disfuncionals.



## 1.4. Variables que poden influir en els patrons alimentaris disfuncionals i la beguda excessiva





#### **1.4. Variables que poden influir en els patrons alimentaris disfuncionals i la beguda excessiva**

Com passa amb altres problemes o trastorns, no tots els éssers humans corren el mateix risc de desenvolupar patrons disfuncionals de menjar i beure. Com s'ha assenyalat anteriorment, hi ha molts factors i característiques implicades en aquests processos que poden determinar la vulnerabilitat de les persones a desenvolupar aquestes conductes. A la primera part d'aquest apartat, presentem les principals variables que s'han demostrat que estan associades a patrons alimentaris disfuncionals (ingesta de greixos i A) i que, per tant, també poden influir indirectament en l'aparició i desenvolupament de l'AA en els joves. En la segona part d'aquest apartat, presentem les variables que estan més relacionades amb l'AM i que també poden actuar com a variables de risc en aquest problema.

Com hem vist a l'apartat anterior, la literatura indica que tant la ingesta de greixos com l'A poden influir directament en l'AA en la joventut. És concebible que les variables que poden influir en la ingesta de greixos i en l'A també puguin exercir una influència indirecta en l'AA en els joves, a través de la relació amb aquestes variables dietètiques.

Hi ha moltes variables que poden influir en la ingesta de greixos i l'A. Per exemple, es pot observar la influència del gènere en els hàbits alimentaris, tot i que depèn de si parlem del tipus d'aliment (aliments grassos) o del tipus d'ingesta (A). D'una banda, la literatura ha assenyalat que l'A és més elevada en les dones que en els homes (Associació Americana de Psiquiatria, 2013), potser perquè a les dones els costa més controlar la quantitat d'aliments (Striegel-Moore i Franko, 2008). Aquesta desigualtat generalment s'ha atribuït a una varietat de factors genètics i biològics, per exemple, el paper de les hormones reproductives femenines (Halmi, 2001). També pot ser degut a factors ambientals i culturals, per exemple, les pressions que han patit les dones per obtenir "el cos ideal" (Stice, 1994), la qual cosa pot portar-los a restringir la seua alimentació i, en conseqüència, tenir un efecte rebot que condueix a l'A. D'altra banda, els homes tenen més probabilitats de consumir aliments grassos que les dones en l'edat adulta jove (Li i cols., 2012). Això pot ser perquè les dones consideren que el contingut nutricional és important en major mesura que els homes (Li i cols., 2012), tot i que com s'ha comentat anteriorment, les dones poden menjar més en forma d'A

perquè perden el control dels aliments a causa de l'efecte rebot de la dieta. Relacionat amb això, l'impuls per la primesa i la insatisfacció corporal també són dues variables que s'associen negativament amb la ingesta de greixos (Liebman i cols., 2001; Ribeiro-Silva i cols., 2018), però positivament associat amb l'A (Gordon i cols., 2012), pels mateixos motius que s'han comentat anteriorment.

Un IMC més alt també s'ha associat amb un A més alt (Mason i Lewis, 2014) i una major ingesta de greixos (Wang i cols., 2020), com s'esperaria de les calories implicades en els aliments grassos i en el consum de grans quantitats d'aliments.

Un altre factor que s'ha associat habitualment amb l'A i la ingesta de greixos és la impulsivitat. La impulsivitat s'entén com una predisposició a actuar sense planificar i a reaccionar davant dels estímuls sense tenir en compte les conseqüències adverses (Barrat, 1985; Moeller i cols., 2001). La investigació ha indicat que ser impulsiu pot precipitar l'A i el consum d'aliments processats (Meule, 2013; Vogeltanz-Holm i cols., 2000). Tot i que té sentit que la tendència a actuar sense tenir en compte les conseqüències et faça participar més en comportaments poc saludables, encara es desconeix el mecanisme a través del qual la impulsivitat pot conduir a uns hàbits alimentaris més pobres.

Els estils alimentaris es caracteritzen per la tendència a menjar com a resposta a emocions negatives (alimentació emocional), a senyals externs com la vista o l'olfacte (alimentació externa), o a no menjar per reduir o mantenir el pes (alimentació restrictiva). Aquests tres estils d'alimentació també poden ser factors de risc per a l'A i la ingesta de greixos (Mason i Lewis, 2014; Racine i cols., 2011; Rolls i cols., 2007; Stice i cols., 2002). Així, els que mengen emocionalment poden utilitzar els aliments per fer front a les seues emocions negatives (van Strien i cols., 1986). Els consumidors externs poden consumir quantitats més grans d'aliments poc saludables a causa de les propietats atractives dels aliments (Anschutz i cols., 2009; van Strien i cols., 1986). El menjar restrictiu, encara que s'associa amb una ingesta menor de greixos (Anschutz i cols., 2009), s'associa amb un augment d'A (Fairburn i cols., 2003; Stice i cols., 2000), en part per l'efecte rebot de la dieta. En la mateixa línia, l'AM també s'ha vinculat als A (Linardon i Messer, 2019) i la ingesta de greixos (Ruddock i cols., 2017), per la qual cosa també pot ser un factor de risc per als dos comportaments alimentaris.

Fins ara, la investigació bàsica indica que la ingesta de greixos i l'A poden influir en l'AA. Calen estudis que avaluen l'impacte dels hàbits dietètics en la vulnerabilitat a l'AA en joves. Com s'ha comentat anteriorment, hi ha variables que s'han relacionat amb la ingesta de greixos i l'AA i, per tant, també poden ser factors de risc indirectes per a l'AA. Seria important analitzar si aquestes variables (gènere, IMC, impuls per la primesa, insatisfacció corporal, alimentació emocional, alimentació externa, alimentació restrictiva, impulsivitat i AM) poden influir indirectament en l'AA a través de la seua relació amb la ingesta de greixos i l'A en joves.

L'AM és una variable que ha adquirit especial rellevància en els darrers anys. Es tracta d'un constructe que comparteix moltes característiques amb l'A i donada la seua elevada prevalença en els joves, cal seguir investigant i identificant quines persones són més vulnerables a patir-la, per tal de dissenyar la seua prevenció i intervenció de la manera més adequada.

Diversos factors s'han relacionat habitualment amb l'AM i, per tant, poden ser factors que augmenten la vulnerabilitat a l'AM. Com a constructe que comparteix tanta variància amb l'A, les variables seran molt semblants a les comentades anteriorment. Entre elles està la joventut. Les investigacions indiquen que les persones més joves tenen més simptomatologia d'AM que les persones grans (Schiestl i Gearhardt, 2018; Wiss i Brewerton, 2020). La joventut és un període de vulnerabilitat que et fa més susceptible als efectes del medi ambient. A més, és més difícil avaluar els riscos, cosa que fa més difícil resistir-se a consumir aliments poc saludables. Les dones també poden tenir més probabilitats de tenir AM (Gearhardt i cols., 2016; Pursey i cols., 2014), tot i que sembla que és el sobrepès o l'obesitat observat en aquestes mostres de dones el que realment està relacionat amb l'AM (Pursey i cols., 2014), ja que alguns estudis assenyalen que no hi ha diferències de gènere en AM en poblacions no clíniques (Ahmed i Sayed, 2017; Hauck i cols., 2017; Pursey i cols., 2014; Schulte i Gearhardt, 2017). Això pot ser perquè les persones que tenen un risc genètic de ser obeses tenen més probabilitats de consumir aliments poc saludables (Gearhardt, White, i cols., 2011; Rapuano et al., 2017).

La literatura també ha assenyalat una relació positiva entre els estils alimentaris i l'AM. És a dir, s'ha observat que les persones que tendeixen a menjar com a resposta a emocions negatives (p. ex., estrès i ansietat) i com a resposta a estímuls ambientals

(p. ex., aliments apetitosos) també tenen més probabilitats de ser addictes als aliments (Pepino i cols., 2014; Schulte i Gearhardt, 2017). Això pot ser perquè les persones que mengen en resposta a emocions negatives necessiten aliments per calmar-se, amb la qual cosa poden necessitar cada vegada més menjar per sentir-se millor (Dingemans i cols., 2009), que al seu torn pot contribuir a l'AM. De la mateixa manera, els menjadors externs, ja que tendeixen a menjar quantitats més grans d'energia (Wardle i cols., 1992), això pot fer que el seu sistema de recompensa cerebral siga més sensible i, per tant, necessiten menjar més aliments per sentir-se bé, cosa que també pot contribuir a l'AM. Els consumidors restrictius també tenen més probabilitats de tenir AM, potser a causa de la dieta que els porta a menjar més i a tornar-se més addictes als aliments (Schiestl i Gearhardt, 2018; Wiss i Brewerton, 2020).

Finalment, s'observa una de les relacions més fortes entre l'A i l'AM (Escrivá-Martínez i cols., 2019; Gearhardt i cols., 2012; Gearhardt i cols., 2016) i entre la bulímia i l'AM (Meule i cols., 2014). Aquestes tres variables comparteixen moltes coses, inclosa la ingesta molt alta d'aliments o el malestar després d'una ingesta elevada d'aliments. Aquesta variància compartida pot fer-los tan estretament relacionats. Una altra hipòtesi podria ser que el comportament d'A i la bulímia poden sensibilitzar el sistema de recompensa i necessitar seguir menjant per sentir-se satisfet, contribuint així a l'AM (Robinson i cols., 2015).

Fins ara, diversos estudis han informat d'associacions o prediccions entre aquestes variables i l'AM, però no hi ha cap estudi que haja analitzat la predicció de totes aquestes variables conjuntament sobre l'AM. És important que els estudis futurs analitzen com es relacionen totes aquestes variables i fins a quin punt poden explicar conjuntament l'AM, amb l'objectiu de considerar-les totes en la prevenció i tractament d'aquesta problemàtica tan prevalent en els joves.

Finalment, no podem oblidar una de les variables que ha tingut i continua tenint un major impacte en els estils de vida de les persones: l'estrès. L'estrès es defineix com el conjunt de reaccions psicològiques i fisiològiques que experimenten les persones davant de fortes exigències ambientals (Selye, 1956). L'estrès pot originar-se de fonts internes, per exemple, la manera com les persones gestionen i resolen els problemes, el temperament o la salut mental i física. També pot tenir el seu origen en agents externs, com ara l'excés de treball o l'estudi excessiu, problemes familiars o

alguna cosa totalment externa i imprevisible, per exemple, viure una pandèmia global.

El març del 2020, el món sencer es va veure atrapat en el que seria un dels períodes més estressants del segle passat: la pandèmia de la COVID-19. Una pandèmia global que seria causada pel virus SARS-CoV-2 i que provocaria milions d'hospitalitzacions i morts a tot el món. Aquesta pandèmia i les mesures que s'havien d'adoptar per fer-hi front anaven a ser molt estressants i provocarien un canvi en l'estil de vida de les persones, sobretot en els comportaments dietètics. En el següent apartat, veurem com la COVID-19 i el període de confinament van afectar els hàbits alimentaris. A més, també veurem fins a quin punt l'estrès i els estils alimentaris poden afectar aquests comportaments alimentaris.



## 1.5. COVID-19 i hàbits alimentaris





## 1.5. COVID-19 i hàbits alimentaris

L'estrès és una variable que pot influir en els hàbits alimentaris de la població. En la primera part d'aquest apartat es parla de la influència del confinament de la COVID-19 en els hàbits alimentaris dels joves. En la segona part d'aquest apartat es comenten els principals factors de risc que poden haver influït en els patrons alimentaris disfuncionals dels joves durant el confinament per la COVID-19.

La pandèmia de salut global provocada per la propagació del SARS-COV-2, que causa la malaltia anomenada COVID-19, ha provocat un nombre molt elevat d'infeccions i, fins avui, un total de 5.248.747 morts a tot el món (Centre de Ciència i Enginyeria de Sistemes, 2021). Es va detectar per primera vegada el desembre de 2019 a Wuhan (Xina) i es va estendre a la majoria de països, inclosa Espanya. L'evolució de la malaltia des que es va conèixer va ser molt alarmant, i les autoritats sanitàries es van veure obligades a declarar l'estat d'alerta sanitària i el confinament dels ciutadans a la majoria de països del món (Anderson i cols., 2020).

A Espanya, 88.159 persones han mort fins ara a causa de la COVID-19 (Centre de Ciència i Enginyeria de Sistemes, 2021). Entre les mesures preventives que va posar en marxa el govern espanyol hi havia el confinament domiciliari des del 14 de març de 2020 fins al 2 de maig de 2020. Durant aquest període, només es van permetre activitats a l'exterior, i va ser a partir del 2 de maig de 2020 quan tots els ciutadans van poder eixir a fer esport, regulant la seua presència fora de casa en diferents edats i franges horàries.

El període de contenció de la COVID-19 va ajudar molt a reduir les taxes d'infecció i morts, però també va suposar una alteració dels estils de vida de la població, especialment els hàbits alimentaris.

Una població d'interès a mirar és l'edat adulta jove (18-30 anys) (Society for Adolescent Health and Medicine, 2017). Són especialment vulnerables al canvi, ja que es troben en un període crític de desenvolupament entre l'adolescència i l'edat adulta i tenen un alt nivell d'activitat professional i educativa. A més, aquesta població es caracteritza per nivells més alts d'estrès, consum d'aliments poc saludables i estils de vida sedentaris, la qual cosa la fa més vulnerable a problemes mèdics (per exemple, obesitat, càncer) o mentals (per exemple, depressió, ansietat). Això, sumat als ràpids

canvis econòmics, socials i tecnològics i a la dificultat per assumir rols familiars i laborals, els converteix en un col·lectiu vulnerable que val la pena analitzar. Cal tenir en compte que aquesta població mai s'ha enfrontat a una pandèmia, la qual cosa pot estar associada a una capacitat reduïda per fer front a l'estrès d'una pandèmia de COVID-19.

La investigació que ha descobert com el confinament afectava els hàbits alimentaris de la població jove en general ha apuntat a resultats molt contradictoris. D'una banda, hi ha estudis que apunten a un augment del consum d'aliments processats (rics en sucres i greixos), que pot provocar un augment de l'IMC, sobretot en poblacions amb sobrepès i obesitat (Ammar i cols., 2021; Pellegrini i cols., 2020; Sidor i Rzymiski, 2020). A més, l'A també va augmentar durant el període de confinament (Phillipou i cols., 2020). Es podria pensar que romandre tant de temps a l'interior sense poder eixir, tenir grans quantitats d'aliment a la vista i l'avorriment i l'estrès podria provocar un augment d'aquests patrons alimentaris disfuncionals. Els humans som éssers socials, així que romandre sols sense poder eixir tant de temps, juntament amb la por a la malaltia COVID-19, podria haver augmentat les emocions negatives, com l'estrès, la tristesa o la por, i per tant, podrien haver utilitzat el menjar per fer front a aquestes emocions negatives.

No obstant això, d'altra banda, hi ha investigacions que apuntaven a un augment del consum d'aliments saludables i una disminució del menjar ràpid, especialment en els joves (Di Renzo i cols., 2020; Rodríguez-Pérez i cols., 2020). A més, no era habitual observar canvis en l'IMC durant el confinament a la població general (Di Renzo i cols., 2020; Haddad i cols., 2021). En aquest cas, es podria pensar que l'augment de la disponibilitat de temps durant el confinament va portar els més joves a cuinar aliments més saludables i a fer exercici en major mesura, la qual cosa va tenir un impacte positiu en el seu estil de vida i hàbits de salut. Encara que es desconeix, també és possible que els joves es cuidaren més per por a emmalaltir de la COVID-19 i que les conseqüències foren més negatives, ja que, per exemple, era ben sabut que el sobrepès estava associat amb una major probabilitat d'hospitalització i mort per COVID-19 (Simonnet i cols., 2020).

En relació als estils d'alimentació (alimentació emocional, externa i restrictiva) s'esperaria que no hi haguera cap canvi durant el confinament, ja que s'ha demostrat

que els estils alimentaris són estables en circumstàncies adverses en els joves (Meiselman i cols., 1998). No obstant això, fins ara no hi ha estudis que hagen avaluat si els estils d'alimentació es van modificar durant el confinament a casa per la COVID-19.

A partir de la literatura, és difícil treure una conclusió precisa sobre com va afectar el confinament a casa en els hàbits alimentaris dels joves. A més, els estudis que comparen els hàbits alimentaris abans i durant la pandèmia tenen limitacions importants. La principal limitació és la mesura dels hàbits alimentaris i l'IMC durant el confinament sense disposar de dades reals d'abans de la pandèmia. Els estudis es limiten a preguntar sobre els hàbits alimentaris de manera retrospectiva, cosa que podria conduir a un biaix en els informes, ja que responen setmanes o mesos després de l'aparició d'aquests comportaments. Fins ara, no disposem d'estudis que avaluen les conductes alimentaries abans i durant el confinament, és a dir, amb dades reals tant abans com durant el període de confinament a domicili. És important analitzar els canvis en els hàbits alimentaris durant el període de pandèmia, ja que podem estar involucrats en situacions d'estrès similars en el futur i saber què passa durant aquests períodes ens pot ajudar a prevenir futurs comportaments de salut no desitjats en els joves.

És ben sabut que no totes les persones corren el mateix risc de desenvolupar patrons alimentaris disfuncionals en moments de gran estrès. Moltes característiques poden influir en la vulnerabilitat a desenvolupar diferents hàbits alimentaris disfuncionals. És important saber quins factors o característiques poden predir aquests patrons alimentaris disfuncionals en moments d'estrès elevat, ja que això ens pot ajudar en la seua prevenció i tractament en futures pandèmies o en moments d'estrès futurs.

Com s'ha comentat en seccions anteriors, els estils d'alimentació (alimentació emocional, externa i restrictiva) s'associen habitualment amb patrons alimentaris disfuncionals (per exemple, A i consum d'aliments rics en greixos i sucre) (Mason i Lewis, 2014; Racine i cols., 2011; Rolls i cols., 2007; Stice i cols., 2002). La literatura indica que el paper dels estils alimentaris en els patrons alimentaris disfuncionals pot ser crucial en moments d'estrès elevat. Per exemple, els individus que mengen com a resposta a emocions negatives (menjadors emocionals) mostren una major

desregulació de les seues emocions durant els moments d'estrès i poden menjar més per fer front a aquests estats negatius (Spoor i cols., 2007). Dit d'una altra manera, els que mengen emocionalment en moments d'estrès busquen recompensa per les seues emocions negatives, i el menjar és una recompensa emocional important per a ells (Cecchetto i cols., 2021). Això pot tenir conseqüències negatives, ja que s'ha trobat que el menjar emocional afecta l'IMC (Czepczor-Bernat i Brytek-Matera, 2021).

L'alimentació externa és un altre estil d'alimentació que també pot afectar els hàbits alimentaris en moments d'estrès (Okumus i Ozturk, 2021). Aquest estil d'alimentació s'ha associat amb el consum d'aliments poc saludables i un augment del consum d'aliments (Anschutz i cols., 2009). Els consumidors externs tendeixen a menjar més quan s'enfronten a aliments que són agradables a la vista o a l'olfacte (van Strien i cols., 1986). Durant el confinament, es van acumular grans quantitats d'aliments a casa, per la qual cosa es pot esperar que aquells amb una alimentació externa més alta menjaren més i pitjor durant el confinament a casa.

Finalment, l'alimentació restrictiva també pot haver afectat els hàbits alimentaris durant el confinament. S'ha observat que l'estrès pot augmentar l'A en els consumidors restrictius (Woods i cols., 2010). Els consumidors restrictius tenen com a objectiu mantenir o reduir el pes, per tant, menjaran menys i més aliments saludables per assolir el seu objectiu (Anschutz i cols., 2009). Tanmateix, aquesta privació dietètica s'associa habitualment amb l'A, a causa de la incapacitat de mantenir aquesta dieta al llarg del temps (Fairburn i cols., 2003; Stice i cols., 2000). Per tant, pot ser que durant el confinament hi haja una major probabilitat que els consumidors restrictius augmenten l'A i que aquesta relació s'accentue per l'estrès.

Tot i que la lògica portaria a creure que els estils alimentaris poden haver influït directament en els patrons alimentaris disfuncionals i l'IMC durant el confinament, fins ara no hi ha estudis que ho hagen examinat. Calen estudis que examinen la capacitat predictiva dels estils alimentaris (alimentació emocional, externa i restrictiva) sobre els patrons alimentaris disfuncionals (A, ingesta de greixos i IMC) durant el confinament. Aquests resultats poden ajudar a millorar les intervencions enfocades a millorar els comportaments alimentaris en moments d'estrès elevat i, al seu torn, a prevenir l'obesitat i els problemes de salut tan freqüents en la joventut actual.

Finalment, cal destacar que el confinament va ser un període estressant per a moltes persones, però no podem oblidar les diferències individuals. No totes les persones han sentit estrès durant el confinament. Tenint en compte tot l'anterior, sorgeix la següent pregunta: És possible que només les persones que van percebre l'estrès augmentaren els seus patrons d'alimentació inadaptats? Si és així, en quins tipus de persones va ser més forta aquesta relació?

Els estils de menjar ens poden ajudar a entendre la relació entre l'estrès percebut i l'A. Per exemple, s'ha observat una relació positiva entre l'alimentació emocional, l'estrès i el consum d'aliments no saludables (Hou i cols., 2013). També s'ha conclòs que l'alimentació emocional s'associa amb un augment de l'A durant el confinament (dos Santos Quaresma i cols., 2021), que es podria explicar pel fet que els devoradors emocionals recorren a l'A per reduir l'estrès del confinament. Els que mengen externament també poden tenir dificultats per allunyar-se dels estímuls apetitius en moments d'estrès elevat, augmentant la probabilitat d'A (Touyz i cols., 2020). Pel que fa als consumidors restrictius, és possible que en situacions d'estrès disminuisquen la seua pròpia autoregulació, el que els porta a augmentar la seua ingesta alimentària (Baumeister i cols., 1993).

Per tant, tenint en compte que l'estrès pot influir en l'A (Crowther i cols., 2001), sorgeix la següent pregunta: Per a quins tipus de persones la relació entre estrès i A es fa més forta? Tenint en compte la literatura, és possible que l'alimentació emocional, externa i restrictiva modere la relació entre l'estrès percebut i l'A? Es necessiten estudis per examinar per a quins tipus de persones la relació entre l'estrès percebut i l'A durant el confinament és més forta. Com s'ha comentat anteriorment, aquests estudis ens poden ajudar en la prevenció i tractament de l'A en moments d'estrès elevat, i així prevenir en la mesura del possible les conseqüències associades a aquesta conducta alimentària.

Com s'ha assenyalat a la secció introductòria, els patrons alimentaris disfuncionals (ingesta de greixos, A i AM) i AA són molt freqüents en els joves d'avui i estan associats amb conseqüències molt negatives a curt i llarg termini. És ben sabut que hi ha una alta comorbiditat entre l'A i l'AA, però, cap estudi ha revisat els punts comuns, les diferències i la direccionalitat entre els dos comportaments. Cal conèixer l'estat de l'art de la relació entre l'A i l'AA, i veure si es poden influir mútuament.

Estudis bàsics i epidemiològics han demostrat la influència de la ingesta de greixos i l'A en l'AA. Tanmateix, no hi ha estudis que hagen avaluat la relació entre la dieta i els patrons d'ingesta i l'AA en joves. Es necessiten estudis translacionals que avaluen l'impacte dels hàbits alimentaris, especialment el consum de dietes altes en greixos i sucres i l'A, sobre la vulnerabilitat a l'AA en els joves.

A més, s'ha demostrat que moltes variables dietètiques i de personalitat estan relacionades amb els patrons alimentaris disfuncionals i el consum d'alcohol en els joves i, per tant, poden influir directament o indirectament en aquestes conductes. Encara no hi ha estudis que hagen analitzat en profunditat quins factors poden predir l'aparició i desenvolupament de la ingesta de greixos, l'A, l'AM i l'AA en els joves. Cal més coneixement sobre els factors de risc que poden influir en aquestes conductes prevalents en els joves per tal de contribuir a una millor comprensió, prevenció i tractament.

Finalment, també cal conèixer com es poden modificar aquests patrons alimentaris en condicions d'estrès i quins factors de risc poden influir en aquestes conductes en condicions d'estrès. La pandèmia de la COVID-19 i les mesures adoptades per frenar la malaltia de la COVID-19 poden haver afectat especialment els comportaments alimentaris dels joves. Tanmateix, cap estudi fins ara no ha avaluat diferències en els estils d'alimentació (alimentació emocional, externa i restrictiva), A, ingesta de greixos i IMC abans i durant el confinament per COVID-19. A més, tampoc hi ha estudis que hagen examinat el paper predictiu dels estils alimentaris en aquests patrons alimentaris disfuncionals durant el període de confinament estricte per COVID-19. Tampoc cap estudi ha respost la pregunta següent: és possible que només les persones que percebien l'estrès augmentaren els seus patrons alimentaris disfuncionals? Si és així, en quines persones va ser més forta aquesta relació?

Per cobrir aquests buits de la literatura, aquesta Tesi Doctoral proposa alguns objectius que es tractaran en l'apartat següent.

# **1. INTRODUCTION**





# General introduction



## **General introduction**

Binge eating (BE) and binge drinking (BD) are highly prevalent problems in young people and associated with very negative consequences (Bava & Tapert, 2010; Ferriter & Ray, 2011; Steele et al., 2016). Given the high prevalence and linked health and quality of life consequences, there has been a growing body of research that has been interested in studying the shared characteristics and relationship between BE and BD (Bahji et al., 2019; Ferriter & Ray, 2011). However, to our best knowledge, no study has reviewed the commonalities, differences, and directionality between the two behaviors. The first aim of this Thesis was to conduct a narrative review to identify the commonalities between BE and BD, the differences, and the influence of one behavior on the other and vice versa (study 1).

To assess BE in young Spanish population, a reliable and valid Spanish scale is needed to measure the construct of A. The Binge Eating Scale (BES) is commonly used to identify binge eaters (Gormally et al., 1982), however, no study has analyzed the psychometric properties of the scale in the general Spanish population. Therefore, the second objective was to validate the BES to measure BE in young Spanish population (study 2).

Epidemiological studies have repeatedly shown that alcohol can stimulate food consumption (Caton et al., 2004, 2005, 2007; De Castro & Orozco, 1990; Westerterp-Plantenga & Verwegen, 1999; Yeomans, 2010). However, the influence of dietary intake on alcohol consumption is less known. A recent study in animal models have demonstrated the predictive role of fat intake and BE on BD (Blanco-Gandía, Ledesma, et al., 2017), although there are no human studies that have assessed the predictive role of these intake and diet patterns on BD. Translational studies assessing the impact of fat intake and BE on vulnerability to BD in young people are needed. Therefore, the third aim was to examine the predictive role of dysfunctional eating patterns (BE and fat intake) on BD in young people (study 3).

The literature has separately pointed to some factors that may be associated with fat intake, BE, FA and BD in young people (e.g., gender, age, BMI, eating styles, etc.) (Camilleri et al., 2014; Kakoschke et al., 2015; Linardon & Messer, 2019; Mason & Lewis, 2014; Sultson et al., 2017). However, whether these variables together may act

as risk factors for dysfunctional eating behaviors (fat intake and BE) and excessive alcohol consumption in young people has not yet been investigated. Therefore, the fourth aim of this Doctoral Thesis was to analyze whether different individual variables (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) could predict BD through their relationship with fat intake and BE (study 3).

To assess FA, it was also necessary to use a reliable and valid scale to measure FA in young Spaniards. The modified Yale Food Addiction Scale 2.0 (YFAS 2.0) is well known worldwide for assessing FA and has obtained adequate psychometric properties (Schulte & Gearhardt, 2017). Although it has been validated in many languages, it has not yet been validated in Spanish. Therefore, the fifth objective of the present Doctoral Thesis was to validate the mYFAS 2.0 to measure FA in young Spanish population (study 4). The sixth objective was to analyze the predictive power of gender, age, BMI, eating styles, BE and bulimia on FA (study 4).

Eating patterns may be modified in times of stress. The COVID-19 pandemic and the measures taken to curb infections had a major impact on young people's lifestyles, especially eating habits (Ammar et al., 2021; Pellegrini et al., 2020; Phillipou et al., 2020; Sidor & Rzymiski, 2020). To our best knowledge, there are no studies assessing eating behaviors before and during confinement, i.e., with real data both before and during the period of home confinement. Therefore, the seventh aim was to assess differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before and during COVID-19 confinement (study 5). The eighth objective was to analyze for the first time the predictive role of eating styles on BMI, fat intake and BE during the period of strict COVID-19 confinement (study 5).

It is important to know which factors can predict these dysfunctional eating patterns at times of elevated stress, as this may help us in their prevention and treatment in future pandemics or at future stressful times. Therefore, the ninth and final aim was to assess whether eating styles moderated the relationship between perceived stress and BE during COVID-19 confinement (study 6). A mobile application was designed to measure BE and perceived stress during confinement in real time, avoiding recall bias and improving ecological validity. Although this relationship had been studied in normal times, it had not yet been tested in times of high stress. With this study, we aim to answer the question: "Can eating styles enhance the relationship

between perceived stress and BE during COVID-19 confinement?”.

The introduction is divided into five sections. In the first section, the main eating and drinking problems of young people are defined, and the prevalence and consequences associated with each problem are presented. The most used instruments for assessing BE and FA are also presented.

In the second section, the commonalities between BE and BD, and the personal variables and neurobiological mechanisms that may predict both behaviors are discussed.

In the third section, the relationship between BE and BD is explored, discussing the influence of one on the other and vice versa.

In the fourth section, the main variables that have been shown to be associated with dysfunctional eating patterns (fat intake and BE) and that may therefore also indirectly influence the onset and development of BD in young people are presented. The variables that are most related to FA and that may also act as risk factors in this problem are also presented.

In the fifth and final section, the influence of strict COVID-19 confinement on young people's eating habits is explored. The main risk factors that may have influenced the dysfunctional eating patterns of young people during COVID-19 confinement are also discussed.



## 1.1. Youth and inadequate habits in relation to dietary patterns and alcohol consumption





## **1.1. Youth and inadequate habits in relation to dietary patterns and alcohol consumption**

Youth (from 18 to 30 years old) is considered a period of vulnerability, in which problems in eating habits and an increase in alcohol consumption are frequently observed in young people. In particular, eating disorders, alcohol and drug use and an increased tendency to seek new sensations and risky behaviors are frequently observed (Bava & Tapert, 2010).

Alcohol consumption is a very important risk factor for young people's health. Alcohol is currently the most widely consumed drug among young men and women (Government Delegation for the National Plan on Drugs, 2018; Inchley et al., 2018). However, the brain is still finishing maturing at this age and alcohol produces a neurotoxic effect that can seriously damage young people's health. Thus, studies indicate that alcohol produces multiple consequences that may become irreversible in young people, such as unsafe driving, aggression, sexual and risky behavior, emotional problems, poorer academic performance and alcohol use disorders in adulthood (Cservenka & Brumback, 2017; Jones et al., 2018; Kuntsche et al., 2017).

Another problem that has grown exponentially in recent years and has become a serious public health issue is diet. Dysfunctional eating patterns are increasingly common among youth, characterized by drinking a lot of sugary drinks, skipping meals or eating large amounts of processed food rich in unhealthy fats, i.e. saturated and trans fats, as well as sugars (Fleming-Milici & Harris, 2020; Navarro-Cruz et al., 2017). Living with university peers, financial hardship, or reduced ability to cook well may be responsible for these dysfunctional eating patterns. These eating habits are associated with overweight and obesity (Drewnowski, 2007), which in turn are strongly associated with serious health complications such as diabetes, cancer or cardiovascular disease (Swinburn, 1998). These problems also entail significant costs to society, reflected in increased health care burden, decreased work productivity and increased sick leave (Kleinman et al., 2014).

Generally, the problem lies not only in the consumption of alcohol or a specific type of diet, but also in the pattern of consumption, i.e., the phenomenon known as "binge". The term "binge" is commonly used to describe a pattern characterized by

large and uncontrolled consumption, which can be applied to both alcohol (BD) and food (BE) (American Psychiatric Association, 2013; Paul et al., 2011).

BD is defined as drinking pattern that raise the blood alcohol concentration to  $\geq 0.08$  g per cent, and specifically corresponds to the consumption of more than four drinks in women (five or more in men) in a two-hour period (Tapia-Rojas et al., 2017). This pattern of consumption has a higher incidence in the youth population, it manifests itself equally in both sexes and its habitual form of consumption is associated with a low perception of risk and a feeling of loss of control over that intake (Balodis et al., 2009; Carlson et al., 2010; Galán et al., 2014; Soler-Vila et al., 2014). This type of pattern has become a favorite way for young people to consume alcohol (Dawson et al., 2015). A quarter of young people report abusing alcohol (Kanny et al., 2018) and half report consuming five or more drinks on one occasion (Wechsler & Nelson, 2008). This problem has multiple implications at individual, family, societal and community levels (Ferriter & Ray, 2011; Laghi et al., 2014). One of the most worrying consequences of this consumption is the risk of traffic accidents in young people, with almost half of the young people who have died in car accidents having consumed alcohol (Galicia-Paredes et al., 2017). In Spain, BD is also associated with high rates of morbidity and mortality, emergencies, alcohol dependence, acute intoxication, use of other emerging drugs, risky sexual behavior, interpersonal conflicts and aggression (Galicia-Paredes et al., 2017; Guitart et al., 2011; Isorna et al., 2015; Moral & Ovejero, 2011).

BE is defined as the objective intake of an excessive amount of food in a short period of time and a sense of loss of control over that intake, often accompanied by emotional distress, such as shame or guilt, and an absence of compensatory behaviors (American Psychiatric Association, 2013). This concept is closely linked to the consumption of high-fat and high-sugar foods, as BE usually occurs with these high-calorie foods (Allison & Timmerman, 2007; Avena et al., 2009). The prevalence of BE in young people is high (0.3%-3.1%) (Sonneville et al., 2013), and has shown an increase in recent decades (Hudson et al., 2007; Smink et al., 2014), especially in young adulthood (Goldschmidt et al., 2014). Two peaks of onset of BE in youth have been reported, the first after puberty (around 14 years of age) and the second at the end of adolescence (19-24 years) (Smink et al., 2014). Although BE can be present in

eating disorders (anorexia, bulimia and BE disorder) as well as in overweight and obese people, it is very common for people to get fed up with eating without being obese; in addition, most obese people do not have BE (Hudson et al., 2007; Kessler et al., 2013). These episodes also have very negative consequences, including medical conditions such as diabetes, hypertension, obesity, coeliac disease, metabolic syndrome, as well as psychological problems, such as depressive and anxiety disorders (Hudson et al., 2007; Keski-Rahkonen & Mustelin, 2016; Kessler et al., 2013).

The high prevalence of BE associated with young people and the observed consequences have led to the need for the development of self-report measures of BE. The BES (Gormally et al., 1982) is the most widely known scale for measuring BE. It has demonstrated high sensitivity and specificity for detecting BE (Grupski et al., 2013) and its validity has been confirmed in clinical and non-clinical samples (Duarte et al., 2015; Hood et al., 2013). Despite its relevance, the factor structure is controversial, with some studies reporting that there is only one BE factor, and other studies reporting that BE consists of two factors: cognitive BE and behavioral BE (Duarte et al., 2015; Zúñiga & Robles, 2006). The scale has been validated in many languages but has not yet been validated in Spanish in the general population. Further research on the factor structure of the scale is needed. Furthermore, given the high prevalence of this problem in young people and the associated consequences, reliable scales that measure BE in the general Spanish population are needed in order to have a reliable and valid measure in this population.

A concept that is closely related to BE and has become very popular in recent decades is the concept of FA (Gearhardt, White, et al., 2011). Specifically, the literature is extensive on the debate of parallels between substance use disorders and BE disorder (Gearhardt et al., 2009a). This debate speculates on the question of whether highly processed foods (e.g. chocolate or pizza) have an addictive potential comparable to drugs (Gearhardt, Davis, et al., 2011), which may reflect common neural correlates (e.g. activation of the same brain reward pathways). In this sense, FA is defined as the excessive and dysregulated intake of high-fat and/or high-sugar foods (Schulte et al., 2015), including among others: decreased control over the consumption of such foods, intense craving for such foods, failed attempts to eliminate such

consumption, continued use despite negative consequences, etc.

The concept of FA remains a hotly debated topic (Avena et al., 2012). An important point of debate is that there is no clear definition of FA. Opponents of the concept are quick to point out that some symptoms of FA (withdrawal or tolerance) are limited to animal models and do not occur in human eating behavior (Rippe & Marcos, 2016). Other researchers argue that the core of eating psychopathology is different from that of substance dependence, for instance, in eating problems there are concerns about shape and weight that are not present in substance use (Ziauddeen et al., 2012). However, in support of the concept of FA, others point out that the paradigms used in animal models may be comparable to the eating behavior of individuals with eating problems and that the criteria for drug abuse and dependence can be perfectly transposed to food (Meule, 2014).

To overcome this debate, the Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009b) was developed. This instrument measures the presence of FA symptoms based on the DSM-IV substance use disorder criteria, with the addition of criteria for clinical significance, i.e., significant distress or impairment in different areas (personal, social, occupational, etc.). A diagnosis of FA can be made when three criteria are met, and significant distress or impairment is present. This scale and its respective versions have shown good psychometric properties and have been well supported in research. The new version of the scale, the YFAS 2.0, reflects the DSM-5 changes in diagnostic criteria for substance use disorder (American Psychiatric Association, 2013; Gearhardt et al., 2016) and appears to have better internal consistency than the YFAS (Gearhardt et al., 2016). The short version of this scale, the mYFAS 2.0, has already been developed and validated, and has obtained adequate psychometric properties (Schulte & Gearhardt, 2017). The mYFAS 2.0 has been validated in many other languages (English, Arabic, Portuguese, Czech, Italian, French). However, it has not yet been validated in Spanish. There is a need for short scales validated in Spanish that assess FA according to DSM-5 criteria, in order to observe whether FA is clinically prevalent in young people in the Spanish context, and to develop strategies to prevent or treat it.

In relation to the prevalence of FA, the results are alarming. Nearly a quarter of people with obesity meet criteria for FA (Pursey et al., 2014), and it is also highly prevalent in young people of normal weight, with some studies indicating that 11-25%

of university students have FA (Gearhardt et al., 2009b; Murphy et al., 2014; Pursey et al., 2014; Rostanzo & Aloisi, 2021). This problem represents serious consequences for people's health, with an increase in processed food consumption and its associated consequences (Steele et al., 2016), which also indirectly impact on public health costs.

As can be seen in this section, alcohol consumption problems and dysfunctional eating behaviors can increase the vulnerability of young people to develop different medical and mental health pathologies (cancer, diabetes, overweight, obesity, severe mental health problems, etc.). These behaviors have increased exponentially in recent years, especially in young people, and constitute a serious public health problem. It is important to establish health policies that help to curb these behaviors and reduce the associated harms in the short and long term. To this end, further research is needed on their prevalence, consequences, co-morbidity, as well as the risk factors that may be precipitating these behaviors.

The high prevalence of BE and BD in young people and the consequences on health and quality of life have led to a growing interest in investigating the shared characteristics and relationship between the two (Bahji et al., 2019; Ferriter & Ray, 2011). In the following section, we provide a framework for understanding BE and BD, discussing the similarities between the two, the personal characteristics and the neurobiological mechanisms that may underlie both behaviors.



## 1.2. Commonalities between binge eating and binge drinking and contributing factors





## **1.2. Commonalities between binge eating and binge drinking and contributing factors**

The first part of this section focuses on the similarities found between BE and BD. The second part focuses on the common relevant contributors to explain the occurrence of BE and BD.

BE and BD are both defined as the consumption of a substance, which can be food or alcohol, in very large quantities and over a short period of time (American Psychiatric Association, 2013; Paul et al., 2011). The literature indicates that both behaviors have addictive potential (Benjamin & Wulfert, 2005), as they are characterized by an uncontrollable and repetitive response (Ferriter & Ray, 2011; Laghi et al., 2014) that is associated with negative personal, academic and social effects (Ferriter & Ray, 2011).

Research indicates that BE and BD may be a risk factor for eating disorders and substance use disorders (Grant et al., 2001). In addition to their high comorbidity, BE may be predictive of alcohol problems (Field et al., 2012; Sonnevile et al., 2013), and BD may predict an increase in dysfunctional eating patterns (Nelson et al., 2009).

In relation to their age of onset, both BE and BD tend to begin in adolescence, and are most commonly observed in young adulthood (Kanny et al., 2018; Kessler et al., 2013). These risk behaviors are highly prevalent in college students (Ferriter & Ray, 2011), with nearly half of students consuming a greater amount of food than would someone else in similar circumstances (Kelly-Weeder, 2011) and a loss of control over that intake (Lipson & Sonnevile, 2017). The same number is observed in the prevalence of young people who binge drink (Croteau & Morrell, 2019; Ferriter & Ray, 2011). This stage of development is critical in brain maturation and the cognitive and physiological changes that occur, which may explain why young people are more vulnerable to all these environmental threats (Harris & Fleming-Milici, 2019).

The literature has also pointed to another commonality between BE and BD: the associated negative consequences. Both behaviors produce very similar consequences in individuals, including mental problems, such as depression and anxiety, or medical problems, such as diabetes, overweight or obesity, as well as an increased likelihood of engaging in high-risk behaviors and poor academic

performance (Fazzino et al., 2017; Ferriter & Ray, 2011; Kuntsche et al., 2017; Raevuori et al., 2015; Striegel-Moore & Franko, 2008).

In addition to examining similarities in both, it is also necessary to focus on common personal characteristics and psychophysiological mechanisms that may contribute to a better understanding of the cause of both problems (Ferriter & Ray, 2011; Ventura-Cots et al., 2017).

Perspectives that have examined eating problems and alcohol or substance use problems point to mood dysregulation as one of the causal factors for both behaviors. Mood problems have been found to be relevant in predicting BD (Lamis et al., 2010) and BE (Phillips et al., 2016; Wheeler et al., 2005). Stress, understood as an uncontrollable event that affects basic adaptive processes to regain homeostasis (Sinha, 2008), plays a central role as a risk factor in both youth alcohol problems (Kenney et al., 2013) and BE (Phillips et al., 2016). One possible explanation for the relationship between stress with BE and BD is cortisol, a hormone released in times of stress that may play an important role as a reinforcer of palatable foods and drugs (Naish et al., 2018; Sinha, 2001). Although the mechanism underlying the relationship between both BE and BD with mood dysregulation remains unknown, it has been suggested that both behaviors are used as coping mechanisms to deal with stress and regulate negative emotions (Laghi et al., 2009, 2014; Pompili & Laghi, 2017).

Socio-cultural factors also play an important role in the onset and development of BE and BD. These include peer pressure, as individuals are more likely to eat and drink in large quantities in order to comply with group norms, improve their social status and ultimately be more accepted by their peer group (Laghi et al., 2012, 2015; Lai et al., 2013). In contrast, there are also factors that can reduce the risk of both behaviors, the most important of which is the family. It is well known that healthy family relationships and high levels of parental warmth reduce the likelihood of BE and BD (Cleveland et al., 2008; Langdon-Daly & Serpell, 2017).

There are also several main neurobiological mechanisms that may underlie both behaviors. Eating problems and alcohol and substance addiction share alterations in the same brain systems, namely the dopaminergic, glutamatergic, serotonergic and endogenous opioid systems (Fletcher & Kenny, 2018; Hadad & Knackstedt, 2014;

Harrop & Marlatt, 2010; Pearlstein, 2002; Rothman et al., 2008). Thus, the mesolimbic reward circuitry is responsible for the craving and regulation of motivation to consume rewarding substances, and can be activated by both drugs and food (Davis et al., 2009; Volkow et al., 2008). Thus, both pleasure eating and substance abuse activate common reward pathways. In turn, the opioid system, specifically related to the sensory experience of liking, also seems to be involved (Berridge, 2009). In addition, several studies suggest that genetic factors may play an important role in the comorbidity of both behaviors (Pearlstein, 2002; Peveler & Fairburn, 1990; von Ranson et al., 2003).

Another component that can influence both behaviors is the craving for the substance or the food. Craving is well known to be an essential component of addictive problems (Potenza & Grilo, 2014), being one of the main diagnostic criteria for substance use disorders (American Psychiatric Association, 2013). The triggers that generate craving, which can be environmental cues reminiscent of the substance (drug or food), produce a dopamine release that drives to seek and consume the substance (Volkow et al., 2008). For instance, craving for the substance induced by environmental cues has been linked to brain regions involved in the role of reward, such as the hippocampus or the amygdala (Bonson et al., 2002; Schneider et al., 2001).

Finally, personality traits may also be underlying mechanisms for the occurrence of both behaviors. Precipitating factors include neuroticism, impulsivity, and reward sensitivity. Mood dysregulation may increase the likelihood of BE and BD, as discussed previously (Lamis et al., 2010; Phillips et al., 2016; Wheeler et al., 2005). This concept is closely linked to the personality trait of neuroticism. Neuroticism is defined as the tendency to experience negative emotions (Costa & McCrae, 1980). Neurotic individuals who usually experience depressive or anxious affect have been found to engage in BE and BD to a greater extent (Davis & Jamieson, 2005; Kuntsche et al., 2008).

Impulsivity is one of the personality characteristics most involved in BE and BD. Impulsivity is defined as responding to stimuli without planning and without consideration of the adverse effects that may result from impulsive behavior (Moeller et al., 2001). It is conceptualized as a multidimensional trait consisting of several traits, such as urgency, lack of planning and sensation seeking (Whiteside & Lynam, 2001).

Research indicates that urgency trait, understood as the tendency to act rashly in response to negative emotional states, is a common feature between BE and BD (Fischer et al., 2004). Therefore, it could be speculated that BE and BD may be a precipitating response to high levels of negative emotionality.

Finally, reward sensitivity theory hypothesizes that people who overeat and take drugs use the substance (in this case, food or substance of abuse) to experience reward, while others do not need the substance because they feel reward from other stimuli (Volkow et al., 1999). Therefore, people who are more reward-sensitive will seek the substance to increase pleasure, which is not increased by other stimuli (Loxton & Dawe, 2007).

This section provides a framework for a clearer understanding of BE and BD. On the one hand, the common mechanisms between BE and BD are highlighted, such as the onset and nature of the problem and the associated negative consequences. On the other hand, the causal mechanisms of both behaviors are discussed, such as the role of mood dysregulation, sociocultural factors, neurobiological mechanisms, craving and personality traits.

Given the shared characteristics of BE and BD and their high comorbidity, studies have sought to elucidate whether one might influence the other and vice versa. In the following section, we review studies that suggest the influence of BD on BE and vice versa, while discussing the limitations of the studies and implications for clinical practice.

### 1.3. Relationship between binge eating and binge drinking



### **1.3. Relationship between binge eating and binge drinking**

As noted above, there is a clear comorbidity between BE and BD. In addition to knowing their comorbidity, it is important to know how they may influence each other, in order to understand more precisely the etiology of both behaviors. In this next section, we attempt to provide an overview of the evidence pointing to the influence of one on the other.

In relation to the influence of BD on BE, the evidence is stronger. Research that has attempted to address how alcohol exerts an effect on food consumption has considered various food and alcohol consumption characteristics, such as type of food consumed and quantity, type of alcohol consumed and quantity, and time of consumption.

Many studies that have attempted to analyze this relationship point out that alcohol has the potential to stimulate food consumption when consumed before or during meals (Caton et al., 2004, 2005, 2007; Westerterp-Plantenga & Verwegen, 1999; Yeomans, 2010). In line with this, several epidemiological studies indicate that more food is consumed on days when more alcohol is consumed (De Castro & Orozco, 1990; De Castro, 2009). This may be explained by several mechanisms, for instance, reduced activation of the system that controls satiety. Alcohol intake has been found to reduce satiety signals after alcohol consumption (Raben et al., 2003; Röjdmarm et al., 2001). Another explanatory mechanism for this relationship lies in the reward systems. Alcohol stimulates both taste (understood as the pleasure of the taste of food) and craving (understood as the motivation to eat; this component is more dependent on the dopaminergic system) (Melis et al., 2009). Other studies indicate that more food is eaten after drinking alcohol due to loss of self-control (Caton et al., 2015; Yeomans, 2010).

Research has also sought to elucidate whether the type of food influences the relationship between alcohol consumption and subsequent food intake. A number of studies appear to indicate that alcohol intake increases the likelihood of food intake (Caton et al., 2007; Yeomans, 2010). Related to this, one study explored the effect of alcohol on ad libitum food intake (specifically, on the high-fat diet condition and the low-fat diet condition) (Tremblay et al., 1995). The study showed that alcohol does not

inhibit food intake, i.e., when alcohol is consumed during meals, the energy content consumed is not compensated by a decrease in energy intake. This also corroborates the hypothesis that alcohol has an impact on the intake of fatty foods. A very recent study also indicated that binge drinkers have a higher intake of calories and consumption of total and saturated fat than moderate or low drinkers (Rosen et al., 2021).

It is also important to consider the amount of alcohol consumed that is needed for alcohol to have an impact on food consumption. Although the literature in this area is sparse, one study found that the amount of alcohol does influence the amount of food consumed (Caton et al., 2004). Thus, the higher the dose of alcohol, the higher the energy intake. If the amount of alcohol is minimal, it has no effect on food intake. This could corroborate the theory that the main mechanism by which more food is eaten after drinking alcohol is loss of self-control (Caton et al., 2015; Yeomans, 2010). It is understood that with low doses of alcohol there is no loss of control, and therefore, no tendency to consume more food.

In summary, studies trying to elucidate how alcohol may affect food intake have explored both the type and quantity of food and the type and quantity of alcohol consumption. These studies indicate that alcohol may stimulate food consumption when eating or when drinking before eating. In addition, there appears to be a dose-response relationship, since, if the amount of alcohol is minimal, this mechanism does not occur, but if the amount of alcohol is higher, this relationship does occur. Different mechanisms may explain this relationship, including the loss of self-control following alcohol consumption (Caton et al., 2015; Yeomans, 2010), the effects of alcohol in reducing satiety signals (Raben et al., 2003; Röjdmarm et al., 2001) and the property of alcohol to increase the reward value of food (Berridge, 2009; Cooper, 2005). Therefore, taking these studies into account, one could conclude that there is a unidirectional relationship from BD to BE. However, what about the other way round, can BE also influence BD? In the following paragraphs, special attention is devoted to the influence of eating habits on BD.

The literature has also tried to analyze the influence of eating patterns on alcohol consumption. A prospective study of almost 17,000 adolescents found that BE did not predict further BD, but did predict future drug use (Sonneville et al., 2013). In this study,



BE was defined as consuming large amounts of food in a short period of time and losing control over that intake (American Psychiatric Association, 2013). BD was defined as the frequency of consuming four or more drinks within a few hours in the previous year. Another study of almost 500 adolescent females noted, in line with the above, that early eating disorder symptoms do predict further substance abuse (Measelle et al., 2006).

These results were corroborated by another similar study conducted with more than 11,000 young people in the UK (Micali et al., 2015). In this case, BE was defined according to the DSM-5 (weekly BE, intense discomfort about BE, and at least three of the following symptoms: eating faster than usual, eating large amounts, eating until full, eating despite not being hungry, eating alone due to embarrassment about the amount eaten, and feeling disgusted with oneself). Subthreshold BE referred to monthly BE. BD was defined by the question: "Have you had six or more alcoholic drinks at least once in the past year?" The results indicated that there was also no association between BE and subthreshold BE with BD.

In contrast, a study of almost 9,000 women assessed whether BE, purging disorder, bulimia nervosa and eating disorder not otherwise specified (monthly BE or purging episode or BE without losing control) are predictors of frequent alcohol consumption (Field et al., 2012). The results revealed that participants with purging disorder had a significantly higher risk of drug use and BD, so eating behavior may influence alcohol consumption.

Previous studies point to a positive, unidirectional relationship between dysfunctional eating patterns and subsequent substance abuse. This could be because BE may produce negative feelings, such as shame or guilt, and substance use may be a coping behavior for these negative emotions (Lai et al., 2013). However, the predictive relationship of maladaptive eating patterns on BD is contradictory. While some studies do point to a unidirectional relationship (Field et al., 2012), others find no relationship (Micali et al., 2015; Sonnevile et al., 2015). This may be due to problems in construct definitions, as while some studies have assessed eating disorders, others have assessed eating symptomatology. In addition, some studies use continuous variables and others categorical variables, which may also have a different effect on the results (Puccio et al., 2016). Moreover, some studies include

alcohol use within substance use, while others do not, and some were conducted before the appearance of the DSM-5, while others were conducted after the DSM-5.

Given the high comorbidity between the two behaviors, and given the above limitations, recent lines of research have continued to assess this relationship. Specifically, they have sought to observe whether fat and sugar consumption and BE predict subsequent alcohol consumption.

A recent study found that having a higher propensity to consume palatable food (high in fat and sugar) in children predicted an increased risk of alcohol consumption during youth (Mehlig et al., 2018). Another recent study also showed that people who preferred sweets had higher cravings for alcohol than those who did not prefer sweets (Bouhlal et al., 2018). In line with this, having a sweet taste phenotype has also been found to be associated with future alcohol problems for those who have already initiated alcohol consumption (Kampov-Polevoy et al., 2014).

A new line of research using animal models has also suggested this relationship. Specifically, they have observed that BE and fat intake predict an increase in excessive ethanol and cocaine consumption in adolescent mice (Blanco-Gandía, Cantacorps, et al., 2017; Blanco-Gandía, Ledesma, et al., 2017). To better understand this, the researchers assessed the consequences of exposure to a high-fat diet during adolescence on the reinforcing effects of ethanol and cocaine (Blanco-Gandía, Cantacorps, et al., 2017; Blanco-Gandía, Ledesma, et al., 2017). The administration of the high-fat diet was evaluated in two different ways: the continuous (ad libitum) consumption model and the intermittent and limited consumption model, in the form of BE. In the binge model, mice fed a standard diet during their adolescence and allowed access to the high-fat diet for only two hours per day for three days per week developed a BE pattern. This research team observed that continued consumption of a high-fat diet produces metabolic changes that lead to weight gain and hyperleptinemia, but does not increase sensitivity to the reinforcing effects of cocaine and alcohol. On the contrary, excessive fat intake in a short period of time does not induce metabolic changes, but contrary to what was observed in continuous consumption, it does predict subsequent consumption of cocaine and ethanol, thus increasing vulnerability to these

substances (Blanco-Gandía, Cantacorps, et al., 2017; Blanco-Gandía, Ledesma, et al., 2017).

These results would confirm the comorbidity observed between eating problems and alcohol and substance abuse. Furthermore, these studies demonstrate that dysfunctional eating patterns modify not only our body's metabolism, but also how our brain responds to drugs of abuse.

Although epidemiological studies demonstrate the impact of both problems in young people, there are no studies that have focused on assessing the relationship between intake and dietary patterns and alcohol consumption in young people. Review studies are needed to provide an up-to-date overview of BE and BD. Moreover, translational studies are needed to assess the impact of eating habits, especially the consumption of high-fat and high-sugar diets and BE, on susceptibility to alcohol consumption in young people. These future studies will have implications for the prevention and treatment of BD in young people, a problem that, as noted in previous sections, is highly prevalent and generates many negative consequences in today's youth.

As this section has shown, dysfunctional eating patterns can influence excessive alcohol consumption. More work is needed to help expand knowledge about the risk factors that may influence these dysfunctional eating behaviors in young people, as, in addition to all the unintended consequences of dysfunctional eating habits, they may also indirectly influence young people's alcohol consumption. In the next section, the main variables that have been shown to be associated with dysfunctional eating behaviors are discussed.



## 1.4. Variables that may influence dysfunctional eating patterns and binge drinking



#### **1.4. Variables that may influence dysfunctional eating patterns and binge drinking**

As with other problems or disorders, not all human beings are at the same risk of developing dysfunctional eating and drinking patterns. As noted above, there are many factors and characteristics involved in these processes that may determine people's vulnerability to developing these behaviors. In the first part of this section, we present the main variables that have been shown to be associated with dysfunctional eating patterns (fat intake and BE) and may therefore also indirectly influence the onset and development of BD in young people. In the second part of this section, we present the variables that are most related to FA and that may also act as risk variables in this problem.

As we have seen in the previous section, the literature indicates that both fat intake and BE can directly influence BD in youth. It is conceivable that the variables that may influence fat intake and BE may also exert an indirect influence on BD in young people, through the relationship with these dietary variables.

There are many variables that can influence fat intake and BE. For instance, the influence of gender on eating habits can be observed, although it depends on whether we are talking about the type of food (fatty food) or the type of intake (BE). On the one hand, the literature has pointed out that BE is higher in women than in men (American Psychiatric Association, 2013), perhaps because women find it more difficult to control the amount of food (Striegel-Moore & Franko, 2008). This inequality has generally been attributed to a variety of genetic and biological factors, for instance, the role of female reproductive hormones (Halmi, 2001). It may also be due to environmental and cultural factors, for instance, the pressures women have been under to obtain "the ideal body" (Stice, 1994), which may lead them to restrict their eating and consequently have a rebound effect leading to BE. On the other hand, men are more likely to consume fatty foods than women in young adulthood (Li et al., 2012). This may be because women consider nutritional content to be important to a greater extent than men (Li et al., 2012), although as discussed above, women may binge eat more because they lose control over food due to the rebound effect of dieting. Related to this, drive for thinness and body dissatisfaction are also two variables that are negatively associated with fat intake (Liebman et al., 2001; Ribeiro-Silva et al., 2018), but positively associated with

BE (Gordon et al., 2012), for the same reasons as discussed above.

Higher BMI has also been associated with higher BE (Mason & Lewis, 2014) and higher fat intake (Wang et al., 2020), as would be expected from the calories involved in fatty foods and the consumption of large amounts of food.

Another factor that has been commonly associated with BE and fat intake is impulsivity. Impulsivity is understood as a predisposition to act without planning and to react to stimuli without regard for adverse consequences (Barrat, 1985; Moeller et al., 2001). Research has indicated that being impulsive can precipitate BE and consumption of processed foods (Meule, 2013; Vogeltanz-Holm et al., 2000). While it makes sense that the tendency to act without considering the consequences makes you engage more in unhealthy behaviors, the mechanism through which impulsivity may lead to poorer eating habits is still unknown.

Eating styles are characterized by the tendency to eat in response to negative emotions (emotional eating), to external cues such as sight or smell (external eating), or to not eat in order to reduce or maintain weight (restrictive eating). These three eating styles may also be risk factors for BE and fat intake (Mason & Lewis, 2014; Racine et al., 2011; Rolls et al., 2007; Stice et al., 2002). Thus, emotional eaters may use food to cope with their negative emotions (van Strien et al., 1986). External eaters may consume larger amounts of unhealthy food due to the attractive properties of the food (Anschutz et al., 2009; van Strien et al., 1986). Restrictive eating, although associated with lower fat intake (Anschutz et al., 2009), is associated with increased BE (Fairburn et al., 2003; Stice et al., 2000), partly because of the rebound effect of dieting. In the same vein, FA has also been linked to BE (Linardon & Messer, 2019) and fat intake (Ruddock et al., 2017), so it may also be a risk factor for both eating behaviors.

To date, basic research indicates that fat intake and BE may influence BD. There is a need for studies assessing the impact of dietary habits on vulnerability to BD in young people. As discussed above, there are variables that have been related to fat intake and BD and may therefore also be indirect risk factors for BD. It would be important to analyze whether these variables (gender, BMI, drive for thinness, body dissatisfaction, emotional eating, external eating, restrictive eating, impulsivity, and FA)



may indirectly influence BD through their relationship with fat intake and BE in young people.

FA is a variable that has become particularly relevant in recent years. It is a construct that shares many characteristics with BE and given its high prevalence in young people, it is necessary to continue researching and identifying which individuals are more vulnerable to suffer from it, in order to design its prevention and intervention in the most appropriate way.

Several factors have been commonly related to FA and may therefore be factors that increase vulnerability to FA. As a construct that shares so much variance with BE, the variables will be very similar to those discussed above. Among them is youth. Research indicates that younger people have more FA symptomatology than older people (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020). Youth is a period of vulnerability that makes you more susceptible to the effects of the environment. In addition, it is more difficult to assess risks, making it harder to resist consuming unhealthy food. Women may also be more likely to have FA (Gearhardt et al., 2016; Pursey et al., 2014), although it appears that it is the overweight or obesity observed in these samples of women that is actually related to FA (Pursey et al., 2014), as some studies note that there are no gender differences in FA in non-clinical populations (Ahmed & Sayed, 2017; Hauck et al., 2017; Pursey et al., 2014; Schulte & Gearhardt, 2017). This may be because people who have a genetic risk of being obese are more likely to consume unhealthy foods (Gearhardt, White, et al., 2011; Rapuano et al., 2017).

The literature has also pointed to a positive relationship between eating styles and FA. That is, it has been observed that people who tend to eat in response to negative emotions (e.g., stress and anxiety) and in response to environmental stimuli (e.g., appetitive foods) are also more likely to be addicted to food (Pepino et al., 2014; Schulte & Gearhardt, 2017). This may be because people who eat in response to negative emotions need food to calm down, thus requiring more and more food to feel better (Dingemans et al., 2009), which in turn can contribute to FA. Similarly, external eaters, as they tend to eat larger amounts of energy (Wardle et al., 1992), this can make their brain reward system more sensitive and therefore need to eat more food to feel good, which can also contribute to FA. Restrictive eaters are also more likely to

have FA, perhaps because of dieting leading them to binge eat more and become more addicted to food (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020).

Finally, one of the strongest relationships is observed between BE and FA (Escrivá-Martínez et al., 2019; Gearhardt et al., 2012; Gearhardt et al., 2016) and between bulimia and FA (Meule et al., 2014). These three variables share many things, including very high food intake or discomfort after high food intake. This shared variance may make them so strongly related. Another hypothesis could be that BE behavior and bulimia may sensitize the reward system and need to keep eating to feel satisfied, thus contributing to FA (Robinson et al., 2015).

To date, several studies have reported associations or predictions between these variables and FA, but there is no study that has analyzed the prediction of all these variables together on FA. It is important that future studies analyze how all these variables are related and how much they can explain FA together, with the aim of considering all of them in the prevention and treatment of this problem, which is so prevalent in young people.

Finally, we cannot forget one of the variables that has had and continues to have the greatest impact on people's lifestyles: the stress. Stress is defined as the set of psychological and physiological reactions that people experience in the face of strong environmental demands (Selye, 1956). Stress can originate from internal sources, for instance, the way people manage and solve problems, temperament, or mental and physical health. It can also originate from external agents, e.g., overwork or over-study, family problems, or something totally external and unpredictable, e.g., living through a global pandemic.

In March 2020, the whole world was caught up in what would be one of the most stressful periods of the last century: the COVID-19 pandemic. A global pandemic that would be caused by the SARS-CoV-2 virus and would result in millions of hospitalizations and deaths worldwide. This pandemic and the measures that were to be taken to deal with it were going to be very stressful and would cause a change in people's lifestyles, especially in dietary behaviors. In the next section, we will see how the COVID-19 and the period of confinement affected eating habits. In addition, we will also see to what extent stress and eating styles can affect these eating behaviors.

## 1.5. COVID-19 and eating habits



## **1.5. COVID-19 and eating habits**

Stress is a variable that can influence the dietary habits of the population. In the first part of this section, the influence of COVID-19 confinement on the eating habits of young people is discussed. In the second part of this section, the main risk factors that may have influenced the dysfunctional eating patterns of young people during the COVID-19 confinement are discussed.

The global health pandemic caused by the spread of SARS-COV-2, which causes the disease called COVID-19, has resulted in a very high number of infections and, to date, a total of 5,248,747 deaths worldwide (Center for Systems Science and Engineering, 2021). It was first detected in December 2019 in Wuhan (China), and spread to most other countries, including Spain. The progress of the disease since it became known was very alarming, and health authorities were forced to declare a state of health alert and the confinement of citizens in most countries in the world (Anderson et al., 2020).

In Spain, 88,159 people have died to date from COVID-19 (Center for Systems Science and Engineering, 2021). Among the preventive measures put in place by the Spanish government was home confinement from 14 March 2020 until 2 May 2020. During this period, only essential activities were allowed outside, and it was from 2 May 2020 when all citizens were allowed to go out for sport, regulating their presence outside the home at different ages and time slots.

The period of COVID-19 containment greatly helped to reduce infection rates and deaths, but it also meant an alteration of lifestyles across the population, especially dietary habits.

One population of interest to look at is young adulthood (18-30 years) (Society for Adolescent Health and Medicine, 2017). They are particularly vulnerable to change, as they are in a critical period of development between adolescence and adulthood and have a high level of professional and educational activity. In addition, this population is characterized by higher levels of stress, unhealthy food consumption and sedentary lifestyles, making them more vulnerable to medical (e.g., obesity, cancer) or mental (e.g., depression, anxiety) problems. This, coupled with rapid economic, social, and technological changes and the difficulty in assuming family and work roles, makes

them a vulnerable group worth analyzing. It should be borne in mind that this population has never faced a pandemic, which may be associated with a reduced ability to cope with the stress of a COVID-19 pandemic.

Research that has unraveled how confinement affected the eating habits of the general young population has pointed to very mixed results. On the one hand, there are studies that point to an increase in the consumption of processed foods (rich in sugars and fats), which can lead to an increase in BMI, especially in overweight and obese populations (Ammar et al., 2021; Pellegrini et al., 2020; Sidor & Rzymiski, 2020). In addition, BE also increased during the period of confinement (Phillipou et al., 2020). One might think that staying indoors for so long without being able to go out, having large amounts of food in sight, and boredom and stress could lead to an increase in these dysfunctional eating patterns. Humans are social beings, so staying alone without being able to go outside for so long, coupled with fear of COVID-19 disease, could have increased negative emotions, such as stress, sadness, or fear, and therefore, they could have used food to cope with these negative emotions.

However, on the other hand, there is research that pointed to an increase in the consumption of healthy food and a decrease in fast food, especially in youth (Di Renzo et al., 2020; Rodríguez-Pérez et al., 2020). Furthermore, it was not common to observe changes in BMI during confinement in the general population (Di Renzo et al., 2020; Haddad et al., 2021). In this case, it could be thought that the increased availability of time during confinement led younger people to cook healthier food and exercise to a greater extent, which had a positive impact on their lifestyle and health habits. Although it is unknown to date, it is also possible that young people took more care of themselves for fear of becoming ill from COVID-19 and that the consequences were more negative, as, for instance, it was well known that being overweight was associated with a higher likelihood of hospitalization and death from COVID-19 (Simonnet et al., 2020).

In relation to eating styles (emotional, external and restrictive eating) it would be expected that there would be no change during confinement, as eating styles have been shown to be stable under adverse circumstances in young people (Meiselman et al., 1998). However, there are no studies to date that have assessed whether eating styles were modified during home confinement by COVID-19.

From the literature, it is difficult to draw a precise conclusion about how home confinement affected young people's eating habits. Moreover, studies comparing eating habits before and during the pandemic have important limitations. The main limitation is the measurement of eating habits and BMI during confinement without having actual data from before the pandemic. The studies are limited to asking about eating habits retrospectively, which could lead to a reporting bias, as they answer weeks or months after the occurrence of such behaviors. To date, we do not have studies that assess eating behaviors before and during confinement, i.e., with actual data both before and during the period of home confinement. It is important to look at changes in eating habits during the pandemic period, as we may be involved in similar stressful situations in the future and knowing what happens during these periods may help us to prevent future undesirable health behaviors in young people.

It is well known that not all people are at the same risk of developing dysfunctional eating patterns in times of high stress. Many characteristics can influence vulnerability to developing different dysfunctional eating habits. It is important to know which factors or characteristics may predict these dysfunctional eating patterns at times of high stress, as this may help us in their prevention and treatment in future pandemics or at future stressful times.

As discussed in previous sections, eating styles (emotional, external and restrictive eating) are commonly associated with dysfunctional eating patterns (e.g., BE and consumption of foods high in fat and sugar) (Mason & Lewis, 2014; Racine et al., 2011; Rolls et al., 2007; Stice et al., 2002). The literature indicates that the role of eating styles on dysfunctional eating patterns can be crucial in times of high stress. For instance, individuals who eat in response to negative emotions (emotional eaters) show greater dysregulation of their emotions during stressful times and may eat more to cope with these negative states (Spoon et al., 2007). In other words, emotional eaters in times of stress seek reward for their negative emotions, with food being a major emotional reward for them (Cecchetto et al., 2021). This can lead to negative consequences, as emotional eating has been found to affect BMI (Czepczor-Bernat & Brytek-Matera, 2021).

External eating is another eating style that can also affect eating habits in times of stress (Okumus & Ozturk, 2021). This eating style has been associated with the

consumption of unhealthy foods and increased food consumption (Anschutz et al., 2009). External eaters tend to eat more when faced with foods that are pleasing to sight or smell (van Strien et al., 1986). During confinement, large amounts of food were accumulated at home, so it can be expected that those with higher external eating ate more and worse during home confinement.

Finally, restrictive eating may also have affected eating habits during confinement. It has been observed that stress can increase BE in restrictive eaters (Woods et al., 2010). Restrictive eaters aim to maintain or reduce weight, therefore, they will eat less and healthier food in order to achieve their goal (Anschutz et al., 2009). However, this dietary deprivation is commonly associated with BE, due to the inability to maintain that diet over time (Fairburn et al., 2003; Stice et al., 2000). It may therefore be that during confinement there is a greater likelihood that restrictive eaters will increase BE and that this relationship is accentuated by stress.

Although logic would lead one to believe that eating styles may have directly influenced dysfunctional eating patterns and BMI during confinement, there are no studies to date that have examined this. Studies examining the predictive ability of eating styles (emotional, external, and restrictive eating) on dysfunctional eating patterns (BE, fat intake and BMI) during confinement are needed. These results may help to improve interventions focused on improving eating behaviors in times of high stress, and in turn, to prevent obesity and health problems so prevalent in today's youth.

Finally, it is necessary to point out that confinement was a stressful period for many people, but we cannot forget individual differences. Not all people have felt stress during confinement. Taking all the above into account, the following question arises: Is it possible that only people who perceived stress increased their maladaptive eating patterns? If so, in which types of people was this relationship strongest?

Eating styles can help us understand the relationship between perceived stress and BE. For instance, a positive relationship has been observed between emotional eating, stress and consumption of unhealthy foods (Hou et al., 2013). It has also been concluded that emotional eating is associated with increased BE during confinement (dos Santos Quaresma et al., 2021), which could be explained by the fact that



emotional eaters resort to BE to reduce the stress of confinement. External eaters may also have difficulty distancing themselves from appetitive stimuli in times of high stress, increasing the likelihood of BE (Touyz et al., 2020). As for restrictive eaters, it is possible that in stressful situations they decrease their own self-regulation, leading them to increase their food intake (Baumeister et al., 1993).

Therefore, considering that stress can influence BE (Crowther et al., 2001), the following question arises: For which types of people does the relationship between stress and BE become stronger? Considering the literature, is it possible that emotional, external, and restrictive eating moderate the relationship between perceived stress and BE? Studies are needed to examine for which types of people the relationship between perceived stress and BE during confinement is stronger. As discussed above, such studies can help us in the prevention and treatment of BE at times of high stress, and thus prevent the consequences associated with this eating behavior as far as possible.

As noted in the introductory section, dysfunctional eating patterns (fat intake, BE and FA) and BD are very prevalent in young people today and are associated with very negative short- and long-term consequences. It is well known that there is a high comorbidity between BE and BD, however, no study has reviewed the commonalities, differences, and directionality between the two behaviors. There is a need to know the state of the art on the relationship between BE and BD, and to see whether they may influence each other.

Basic and epidemiological studies have demonstrated the influence of fat intake and BE on BD. However, there are no studies that have assessed the relationship between diet and intake patterns and BD in young people. There is a need for translational studies that assess the impact of eating habits, especially the consumption of high-fat and high-sugar diets and BE, on vulnerability to BD in young people.

In addition, many dietary and personality variables have been shown to be related to dysfunctional eating patterns and alcohol consumption in young people, and thus may directly or indirectly influence these behaviors. No studies have yet analyzed in depth which factors may predict the onset and development of fat intake, BE, FA and

BD in young people. More knowledge is needed on the risk factors that may influence these prevalent behaviors in young people in order to contribute to better understanding, prevention and treatment.

Finally, it is also necessary to know how these eating patterns may be modified under stressful conditions and what risk factors may influence these behaviors under stressful conditions. The COVID-19 pandemic and the measures taken to curb COVID-19 disease may have particularly affected the eating behaviors of young people. However, no studies to date have assessed differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before and during COVID-19 confinement. Furthermore, there are also no studies that have examined the predictive role of eating styles on these dysfunctional eating patterns during the period of strict COVID-19 confinement. Nor have any studies answered the following question: Is it possible that only people who perceived stress increased their dysfunctional eating patterns? If so, in which people was this relationship strongest?

To fill these gaps in the literature, this Doctoral Thesis proposes some objectives that will be discussed in the following section.

## **2. AIMS AND** **HYPOTHESES**



## 2. Aims and hypotheses

As can be seen in the previous section, dysfunctional eating patterns and excessive alcohol consumption are highly prevalent in young people and are associated with devastating negative consequences. Given the comorbidity found between eating problems and alcohol consumption and the similarities discussed above, there is a need to understand this relationship in more depth, as well as to see what risk factors may influence both dysfunctional behaviors in young people in order to contribute to their better understanding, prevention and treatment. Therefore, the overall aim of this Doctoral Thesis is to study the relationship between dysfunctional eating patterns and BD in young people and to explore the risk factors associated with both behaviors.

To achieve the above, the following specific aims are proposed:

- 1) To review the literature to identify the commonalities between BE and BD, the differences, and the relationship between the two.
- 2) To validate the BES to measure BE in a young Spanish population.
- 3) To analyze the predictive role of dysfunctional eating patterns (BE and fat intake) in BD in young people.
- 4) To analyze whether dysfunctional eating patterns (BE and fat intake) mediate the relationship between individual risk factors (gender; BMI; drive for thinness; body dissatisfaction; eating styles: emotional, external, and restrictive eating; impulsivity and FA) and BD in young people.
- 5) To validate the mYFAS 2.0 to measure FA in a young Spanish population.
- 6) To analyze the predictive power of gender, age, BMI, eating styles (emotional, external, and restrictive eating), BE and bulimia on FA in young people.
- 7) To analyze differences in eating styles (emotional, external, and restrictive eating), dysfunctional eating patterns (BE and fat intake) and BMI before and during COVID-19 confinement in young people.

8) To analyze the predictive power of eating styles (emotional, external, and restrictive eating) on dysfunctional eating patterns (BE and fat intake) and BMI during COVID-19 confinement in young people.

9) To assess whether eating styles (emotional, external, and restrictive eating) moderate the relationship between perceived stress and BE during COVID-19 confinement in young people.

Six studies were conducted to carry out the above objectives.

Study 1 (see article publication in annex 1) address Objective 1. Specifically, it aimed to analyze the commonalities between BE and BD, to analyze their differences, to explore the effects of BD on BE and to explore the effects of BE on BD. It is true that there is a relationship between the two (Ferriter & Ray, 2011; Laghi et al., 2014), however, no study so far had reviewed studies examining the directionality between the two behaviors. Therefore, this review was considered necessary to ascertain the state of the art in the literature on the relationship between BE and BD. **It was hypothesized that both BE and BD would share commonalities, differences, and a positive influence between them.**

Study 2 (see article publication in annex 2) address Objective 2. It aimed to validate the BES in a young Spanish population. This questionnaire has been widely used to identify binge eaters (Gormally et al., 1982), however, research on the BES is limited. There were no studies that had analyzed the psychometric properties in both sexes, and there were contradictions in its dimensions, in addition to the fact that it had not been validated in a Spanish sample. Considering that it was a variable of great interest for this Doctoral Thesis and that we needed to measure it adequately, it was decided to carry out study 2. **It was hypothesized that the BES would have a one-factor structure, measurement invariance between sexes and good psychometric properties (i.e., good convergent and discriminant validity with different variables).**

Study 3 (see article publication in annex 3) address Objectives 3 and 4. It was conducted to study how dysfunctional eating patterns (BE and fat intake) influenced BD. **It was hypothesized that dysfunctional eating patterns would positively influence BD.** Furthermore, we examined whether dysfunctional eating patterns

mediated the relationship between individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) and BD. **It was hypothesized that these individual factors could predict BD through their relationship with dysfunctional eating patterns.**

To improve ecological validity, we also set out to develop a mobile application to assess dysfunctional eating and drinking patterns (fat and alcohol intake, BE and BD) in real time and in the natural context (using EMA). The application is currently developed, and could be used to conduct study 6, but we have not yet been able to analyze eating behaviors in a period of normality due to the COVID-19 pandemic situation. We hope to carry out this objective when we return to a normal situation. This Thesis also aimed to conduct a functional neuroimaging study to experimentally demonstrate whether the administration of a high-fat diet can modify the brain and behavioral response to ethanol consumption. However, this study was to be carried out during the second stay of the Doctoral Thesis (Universitat de Monash, Melbourne, Austràlia), but the PhD student could not perform it due to mobility restrictions at COVID-19. We also hope to conduct it out when the COVID-19 pandemic stabilizes.

Study 4 (see article in annex 4) address Objectives 5 and 6. FA is another prevalent problem in young people and shares a close relationship with BE (Gearhardt et al., 2016). Moreover, it is possible that the behavioral act of overeating can become addictive for some people, with young people being more vulnerable to it (Schulte & Gearhardt, 2017). Therefore, we wanted to study it in depth and analyze which dietary factors could contribute to the emergence and development of FA in young people. To do so, we needed to use a reliable scale to measure FA in young people. Study 4 was conducted to meet all these objectives. **The mYFAS 2.0 was hypothesized to have a one-factor structure considering the original validation** (Schulte & Gearhardt, 2017). **Furthermore, it was hypothesized that BMI, eating styles, BE, and bulimia would directly affect FA.**

Study 5 (see article in annex 5) address Objectives 7 and 8. The pandemic context did not allow several of the planned studies to be carried out, but it was a good opportunity to see what changes in eating behavior occurred at times of high stress. Given that COVID-19 confinement meant changing the lifestyle habits of the population, we imagined that this would particularly affect young people, and so study

5 was conducted (see annex 5 for the full article). Since we already had dietary data assessed prior to COVID-19, differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before and during COVID-19 confinement could be analyzed. Furthermore, it was considered important to explore which risk factors could affect these behaviors during COVID-19 confinement, therefore, the predictive role of eating styles on these eating behaviors during COVID-19 confinement was also analyzed. **Changes in BE, fat intake, and BMI during confinement were hypothesized, and these variables were expected to be affected by maladaptive eating styles. However, no hypotheses were specified in either direction due to the exploratory nature of this study.**

Finally, study 6 (see article in annex 6) address Objective 9. It was conducted to test whether the relationship between perceived stress and BE in young people during COVID-19 confinement was moderated by eating styles. Perceived stress and BE were assessed using a mobile app designed to assess these behaviors in real time and in the natural context, which provided ecological validity to the study. **It was hypothesized that high scores in the three dysfunctional eating styles (emotional eating, external eating, and restrictive eating) would moderate the relationship between perceived stress and episodes of BE during confinement.**

The knowledge gained from this Doctoral Thesis will contribute to a better understanding of the mechanisms underlying the problems of dysfunctional eating patterns and BD in young people. Furthermore, the results obtained will help us to improve the prevention and intervention of these behaviors in young people.



# **3. METHODOLOGY** **AND RESULTS**



### **3. Methodology and results**

The six studies that make up this Doctoral Thesis were approved by the Ethics Committee of the Universitat de València (registration numbers H1513854038939; 1821046) (annex 7 and 8). All studies were conducted in accordance with the ethical standards of the 1964 Declaració de Helsinki. All participants were informed of the confidentiality of the data and voluntarily cooperated in the study. All participants gave informed consent prior to their inclusion in the studies.

The methodology and main results of the six studies of this Thesis are presented below. For ease of reading, each section pertains to one study.



### 3.1. Study 1. A narrative review of binge eating and binge drinking.



### **3.1. Study 1. A narrative review of binge eating and binge drinking.**

The overall aim of this narrative review was to provide an updated overview of BE and BD. The specific aims were: 1) To analyze the commonalities between BE and BD; 2) To analyze the differences between BE and BD; 3) To explore the effects of BD on BE; and 4) To explore the effects of BE on BD.

#### **Commonalities between binge eating and binge drinking.**

Firstly, both share the nature and occurrence of the problem. They are defined by the consumption of a substance (food and alcohol) in large quantities and over a short period of time (American Psychiatric Association, 2013). They usually begin in adolescence and occur most frequently in young adulthood (Kanny et al., 2018; Kessler et al., 2013). Moreover, they are highly prevalent in young people and tend to be persistent, despite their associated negative consequences (Ferriter & Ray, 2011; Kessler et al., 2013).

Second, they share several physical, psychological, social, and academic consequences. For instance, both behaviors are associated with symptoms of depression and anxiety, increased risk of being overweight or obese, diabetes problems, increased likelihood of engaging in high-risk behaviors and poorer academic performance (Bahji et al., 2019; Davis et al., 2017; Ferriter & Ray, 2011).

Third, both are associated with negative emotions. Recent studies indicate that depressive symptoms may influence BE and BD (Jin-Yi et al., 2017; Lamis et al., 2010).

Fourth, both are associated with several personality dimensions: impulsivity, sensation seeking, neuroticism and reward sensitivity. Negative urgency, defined as the tendency to act impulsively when upset, has been shown to predict BE and BD (Bø et al., 2016; Fischer et al., 2004; Smith & Cyders, 2016).

Fifth, social and cultural factors have also been shown to play an important role in the onset and development of both problems. For instance, peer pressure, comparison with their peer group and the media may influence young people to adopt these behaviors. This may be because young people want to adopt behaviors similar to their peer group in order to improve their social position and be socially accepted

(Laghi et al., 2012, 2015; Lai et al., 2013).

### **Differences between binge eating and binge drinking.**

In addition to the commonalities, BE and BD also have distinct and unique characteristics. The first is found with dietary restriction and body dissatisfaction. It appears that dieting or body dissatisfaction may be risk factors for BE (Fairburn et al., 2003; Stice et al., 2000). However, the relationship between dietary restraint and BD appears to be different. In particular, a trend has been observed in young people called "drunkorexia", i.e. the tendency to restrict caloric foods to compensate for the caloric intake of alcohol consumption and become intoxicated more quickly (Eisenberg & Fitz, 2014; Hunt & Forbush, 2016). The second is the importance of the substance. While the concept of alcohol addiction is well established, FA is not considered an addictive disorder in diagnostic classifications and remains the subject of controversy and debate. Related to the above, another difference is that there are still no human studies that have systematically examined the withdrawal phenomenon in processed food, whereas withdrawal is particularly relevant in alcohol consumption (Schulte et al., 2016, 2018; Ziauddeen & Fletcher, 2013).

It seems clear that BE and BD share a number of characteristics, but it is not known whether these behaviors are related unidirectionally or bidirectionally, i.e. whether BD influences how people eat or whether BE influences how people drink.

### **Effects of binge drinking on binge eating.**

Regarding the effects of BD on BE, evidence is scarce. Some studies show that alcohol consumption stimulates short-term food intake (Caton et al., 2004, 2005, 2007; Westerterp-Plantenga & Verwegen, 1999; Yeomans, 2010), which may be attributed to a loss of self-control (Caton et al., 2015; Yeomans, 2010) or that alcohol may increase the reward value of food (Melis et al., 2009). In addition, alcohol intake also appears to increase the intake of high-fat foods (Caton et al., 2007; Yeomans, 2010), and may be determined by the amount of alcohol consumed, as there is one study showing that low-dose alcohol does not produce this effect (Caton et al., 2004). In this sense, therefore, there appears to be a unidirectional relationship between BD and high food intake, although more research is needed to see whether BD predicts subsequent BE.



### **Effects of binge eating on binge drinking.**

In relation to the effects of BE on BD, mixed results are also observed. While some studies do observe a predictive relationship of BE on BD (Blanco-Gandía, Ledesma, et al., 2017; Field et al., 2012), other results claim no relationship (Micali et al., 2015; Sonnevile et al., 2013). This may be due to the number of definitions of BE and BD, as well as the fact that some studies operationalize eating pathology as an eating disorder, while others only focus on symptoms. There is a need for unified criteria to determine the direction of the effects of BE on BD.

It is concluded that BE and BD share many commonalities, as well as certain particularities. Furthermore, this study shows for the first-time evidence of directionality between the two behaviors, although the results are not conclusive. Further studies are needed to be able to affirm that BE can act as a risk factor for BD, and vice versa. To read the published article, please see Annex 1.



3.2. Study 2. Validation of the Spanish Binge Eating Scale and its relationship with food addiction, impulsivity, binge drinking, and body mass index.



### **3.2. Study 2. Validation of the Spanish version of the Binge Eating Scale and its relationship with food addiction, impulsivity, binge drinking, and body mass index.**

The aim of this study was to analyze the psychometric properties of the BES in a young Spanish population, following several steps: 1) To analyze its factor structure through a competing structural equation model, specifically, a confirmatory factor analysis (CFA); 2) To test measurement invariance between sexes; 3) To provide evidence of its internal consistency; 4) To provide evidence of its convergent and discriminant validity.

A total of 428 Spanish college students were recruited, 324 females (75.7%;  $M_{age} = 21.04$ ;  $SD = 4.22$ ) and 104 males (24.3%;  $M_{age} = 22.27$ ;  $SD = 5.39$ ). Females had a mean BMI of 21.91 ( $SD = 2.98$ ), and males had a mean BMI of 23.46 ( $SD = 2.84$ ). Most of the participants (77.1%) had a normal weight ( $18.5 \geq BMI \leq 24.99$ ).

Most of the participants were recruited in the classrooms of the Universitat de València. Participants were informed of the study design, the voluntary nature of their participation and the confidentiality of the data obtained. Subsequently, once informed consent had been collected, participants were asked to respond to an online survey. This survey was carried out through the Lime Survey web platform of the Universitat de València.

The Mexican version of the BES was used (Zúñiga & Robles, 2006). A bilingual translator and a native Spanish speaker reviewed the translated BES separately. Subsequently, the scale was administered to 40 Spanish students to see if the items were correctly understood. Finally, it was decided to use the same scale as the previous authors (Zúñiga & Robles, 2006), as it was an exact translation of the original English version and the items were correctly understood. Item 26 of the Eating Disorder Inventory-3 Referral Form (EDI-3 RF; Clausen et al., 2011) was also used to assess BE: "Have you ever engaged in BE (did you eat a lot of food and feel that you could not stop eating?". To assess FA, the modified Yale Food Addiction Scale was used (mYFAS; Flint et al., 2014). To measure impulsivity, the Barrat Impulsivity Scale-15S was used (BIS-15S; Spinella, 2007). To assess BD, the item most supported in the literature was used: "Considering all types of alcoholic beverages, did you consume

five or more drinks in a row (four if female) on at least one occasion in the last month? How many times in the last month?" (Paul et al., 2011). Finally, to assess BMI, current weight in kilograms was divided by the square of height in meters (World Health Organization, 2000).

**Factor structure.** The factorial validity of the BES was analyzed by CFAs. The two structures used in the literature were tested: the one-factor model of the BES and the two-factor model (behavioral BE and cognitive BE). The results showed that the two models fitted adequately; however, in the two-factor model, the correlation between the two factors was very high, so discriminant validity was not demonstrated. For this reason, it was finally decided to keep the one-factor model, since it fit the data better:  $\chi^2(104) = 374.35$  ( $p < 0.001$ ); CFI = 0.94; RMSEA = 0.08 [0.067, 0.084].

**Measurement of BES invariance between sexes and comparison between latent means.** An invariance measurement routine was tested to test whether BES was invariant between males and females. The scalar invariance model was the most parsimonious, demonstrating evidence of no measurement bias when comparing the two groups, as the significance of BE (item loadings and intersections) was the same in both groups. Because scalar invariance was maintained, latent means were compared between samples to test whether levels of BE were equal in the two groups. The fit of this model decreased significantly, so the results pointed to a difference between the means of the two groups. Therefore, this model showed statistically significant differences between sexes, with a higher prevalence of BE in females.

**Internal consistency.** Reliability evidence for the Spanish BES was provided through Cronbach's  $\alpha$  (Cronbach, 1951) and McDonald's  $\omega$  (McDonald, 1999). Cronbach's  $\alpha$  was 0.869 and McDonald's  $\omega$  was 0.915, indicating appropriate reliability estimates for the scale.

**Convergent and discriminant validity.** Convergent and discriminant validity was assessed through correlations between the BES and the EDI-3 BE indicator, the BIS-15S, the mYFAS, the BD indicator, and the BMI. Evidence of convergent validity was shown through the positive, high, and statistically significant correlation between the BES and the EDI-3 BE indicator ( $r = 0.621$ ,  $p < 0.001$ ), and between the BES and FA ( $r = 0.761$ ,  $p = 0.001$ ). Evidence of discriminant validity was provided by statistically

significant, but lower correlations between BES and impulsivity dimensions ( $r$  between 0.164 and 0.284,  $p = 0.001$ ), between BES and BD ( $r = 0.139$ ,  $p = 0.023$ ), and between BES and BMI ( $r = 0.243$ ,  $p < 0.001$ ).

After performing this validation, it can be confirmed that the Spanish BES is a reliable and valid self-report measure to assess BE in a young non-clinical population. The main contribution of this study is to validate for the first time the BES in non-clinical population and to test the measurement invariance between both sexes. To read the published article, please see Annex 2.





### 3.3. Study 3. Influence of eating behavior on binge drinking.



### **3.3. Study 3. Influence of eating behavior on binge drinking.**

The first objective of this study was to analyze the relationship between BE, fat intake and BD. It was hypothesized that BE and fat intake would positively influence BD.

Considering that there are factors related to BE and fat intake that may indirectly relate to BD, the second objective was to analyze whether BE and fat intake mediated the relationship between these individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) and BD. It was hypothesized that these individual factors could act on BD through their relationship with BE and fat intake. To carry out these objectives, a structural equation model was tested.

The sample consisted of 428 college students from Valencia (Spain), 324 females ( $M_{\text{age}} = 21.04$ ;  $SD = 4.22$ ) and 104 males ( $M_{\text{age}} = 22.27$ ;  $SD = 5.39$ ).

The design of this study was cross-sectional. Most of the participants were recruited in the classrooms of the Universitat de València. All participants were informed of the study design, the voluntary nature of their participation, and the confidentiality of the data obtained. They all received a link to complete the survey, in which they provided sociodemographic data and questionnaires. The survey was conducted through the Lime Survey web platform of the Universitat de València.

This structural model included the following variables:

Exogenous variables: sex (male and female); BMI (World Health Organization, 2000); drive for thinness and body dissatisfaction (EDI-3; Clausen et al., 2011; Elosua & López-Jáuregui, 2012); emotional, external and restrictive eating (Dutch Eating Behavior Questionnaire - DEBQ; Cebolla et al., 2014; van Strien et al., 1986); motor, unplanned and attentional impulsivity (BIS-15S; Orozco-Cabal et al., 2010; Spinella, 2007); and FA (mYFAS; Flint et al., 2014).

Mediating variables: BE (BES; Escrivá-Martínez et al., 2019; Gormally et al., 1982) and fat intake (SFQ; Dobson et al., 1993).

Dependent variable: BD. This variable was measured in two ways: with Alcohol Use Disorders Identification Test (AUDIT) item 3 "How often do you have six or more

drinks on a single occasion?" (Maurage et al., 2020) and with the BD indicator "Considering all types of alcoholic beverages, did you ever have five or more drinks (four if female) in a two-hour period (once) in the past month?" (Paul et al., 2011).

**Descriptive statistics.** All questionnaire scores were at the average. No clinical symptomatology was shown for BE, FA, and impulsivity. After analyzing AUDIT-3 scores, it was observed that 0.7% of participants reported BD daily or almost daily; 4% binge drank alcohol weekly; 22.8% monthly; and 37.8% less than once a month.

**Structural equation model.** The multiple indicators multiple causes (MIMIC) structural model presented a good fit:  $\chi^2(23) = 28.025$  ( $p = 0.214$ ), CFI = 0.992, RMSEA = 0.023 [90% CI = 0.000, 0.048]. BE and fat intake showed a positive and statistically significant effect with BD ( $p < 0.001$ ). However, the correlation between BE and fat intake was not significant ( $p = 0.684$ ).

The exogenous variables that were related to BE were: drive for thinness ( $p < 0.001$ ), body dissatisfaction ( $p = 0.019$ ), emotional eating ( $p < 0.001$ ), external eating ( $p = 0.004$ ), restrictive eating ( $p = 0.026$ ), and FA ( $p < 0.001$ ). The exogenous variables that were related to fat intake were: drive for thinness ( $p = 0.018$ ), external eating ( $p < 0.001$ ), restrictive eating ( $p < 0.001$ ), motor impulsivity ( $p = 0.013$ ) and FA ( $p = 0.008$ ).

As for the indirect effects of the exogenous variables on BD, emotional eating, external eating, and FA, they showed positive and statistically significant indirect relationships with BD, while the indirect relationship of restrictive intake on BD was negative.

Overall, the model explained 68.3% of food BE ( $R^2 = 0.683$ ,  $p < 0.001$ ), 27.0% of fat intake ( $R^2 = 0.270$ ,  $p < 0.001$ ) and 10.4% of BD ( $R^2 = 0.104$ ,  $p = 0.015$ ).

This study is the first to provide evidence of the direct relationship between BE and fat intake on BD in humans, as well as to provide evidence of the indirect relationship between different eating patterns (eating styles and FA) and BD, mediated by BE and fat intake. This study may help to improve the prevention and intervention of BD in young people, through strategies that promote an improvement in these eating patterns. To read the published article, please see Annex 3.

### 3.4. Study 4. Validation of the modified Yale Food Addiction Scale 2.0 and associated risk factors.



### **3.4. Study 4. Validation of the modified Yale Food Addiction Scale 2.0 and associated risk factors.**

The first objective of the study was to analyze the psychometric properties of the mYFAS 2.0 in a young Spanish population, following these two steps: 1) To analyze its factor structure through a competing structural equation model, specifically, a CFAs; 2) To provide evidence of its reliability. The second objective was to analyze the relationship between mYFAS 2.0 with other dietary variables (emotional eating, external eating, restrictive eating, BE and bulimia), sociodemographic characteristics (age and sex) and BMI. The third objective was to analyze the predictive power of some dietary variables (emotional eating, external eating, restrictive eating, BE, bulimia, and BMI) on mYFAS 2.0, through a structural equation model.

A sample of 400 Spanish college students was recruited, of whom 51% were female ( $M_{age} = 23.35$ ;  $SD = 4.65$ ) and 49% were male ( $M_{age} = 24.99$ ;  $SD = 7.26$ ). The women had a mean BMI of 21.90 ( $SD = 3.22$ ), and the men had a mean BMI of 23.87 ( $SD = 4.23$ ). Seventy-four percent of the participants had a normal weight ( $18.5 \geq BMI \leq 24.99$ ).

Participants were recruited from classrooms at the Universitat de València. They were invited to participate in an online survey, always informing them that participation was voluntary, and data were confidential. The survey was administered through the Lime Survey web platform of the Universitat de València. Participants were not financially compensated.

Permission was obtained from the original authors of the scale to translate and validate the mYFAS 2.0 into Spanish. The scale was translated into English by a bilingual translator. Subsequently, three Spanish reviewers reviewed the translated items. Finally, the mYFAS 2.0 was administered to 20 students to corroborate their understanding of the scale. The Spanish version was an exact translation of the original English version (Schulte & Gearhardt, 2017). The mYFAS 2.0 offers three ways of scoring: 1) original score: mean of the 11 items without the two items of clinical significance; 2) symptom count: each item is scored 0 or 1 depending on whether it meets the stated criterion or not; finally, the items are summed and a score between 0 and 11 is obtained; and 3) diagnostic score: the symptoms are counted and the

statistical significance criterion (impairment or distress) is added, obtaining a score that divides the sample as follows: does not meet FA criteria (one or fewer symptoms plus does not meet impairment or distress), mild FA (two to three symptoms plus impairment or distress), moderate (four or five symptoms plus impairment or distress), or severe (six or more symptoms plus impairment or distress).

The modified Yale Food Addiction Scale (mYFAS; Flint et al., 2014) was also used to assess FA; the DEBQ (Cebolla et al., 2014; van Strien et al., 1986) to assess emotional, external and restrictive eating; the BES (BES; Escrivá-Martínez et al., 2019; Gormally et al., 1982) to assess BE; and the EDI-3 RF (Clausen et al., 2011; Elosua & López-Jáuregui, 2012) to assess bulimia. Finally, to assess BMI, the following division was made: weight (kg)/height (meters<sup>2</sup>) (World Health Organization, 2000).

**Factor structure.** The factorial validity of the mYFAS 2.0 was analyzed by CFAs. Two single-factor models were tested, considering the original score and the symptom count score. The model for the original score was an excellent fit:  $\chi^2(44) = 116.93$  ( $p < 0.001$ ); CFI = 0.98; SRMR = 0.05; RMSEA = 0.06 [0.050, 0.079]. All factor loadings were statistically significant ( $p < 0.001$ ). The model for symptom count also fitted excellently:  $\chi^2(44) = 59.34$  ( $p = 0.061$ ); CFI = 0.99; SRMR = 0.07; RMSEA = 0.03 [0.000, 0.047]. All factor loadings were also statistically significant ( $p < 0.001$ ).

**Reliability.** Evidence of reliability of the Spanish mYFAS 2.0 was provided through Cronbach's  $\alpha$  (Cronbach, 1951) and McDonald's  $\omega$  (McDonald, 1999). With the original score, Cronbach's  $\alpha$  was 0.828 and the  $\omega$  was 0.916. With the symptom count scores, Cronbach's  $\alpha$  was 0.783 and the  $\omega$  was 0.921. Therefore, the scale showed adequate reliability with both types of scores.

**Prevalence of FA.** Using the original mYFAS 2.0 score, the mean was 8.23 ( $SD = 8.96$ ) (score between 0 and 44). Using the mYFAS 2.0 symptom count score, the mean was 3.58 ( $SD = 2.62$ ) (score between 0 and 11). Using the mYFAS 2.0 diagnostic score, 12.5% had mild, 11.3% moderate, and 20.6% severe FA. The diagnosis of FA was made if they presented more than three symptoms plus impairment or distress. Thus, 31.9% met the criteria for FA.

**Convergent validity.** Two CFAs were estimated to account for the fact that both mYFAS 2.0 items and mYFAS items measured the same construct. Both the original



score model and the symptom count model showed adequate fit:  $\chi^2(153) = 320.448$  ( $p < 0.001$ ); CFI = 0.96; SRMR = 0.06; RMSEA = 0.06 [0.050, 0.067], for the model using the original score, and  $\chi^2(153) = 187.683$  ( $p = 0.001$ ); CFI = 0.96; SRMR = 0.13; RMSEA = 0.031 [0.020, 0.041], for the model using the symptom count score. The mYFAS 2.0, measured using the original score and symptom count, was also positively and statistically significantly correlated with emotional eating, external eating, restrictive eating, BE, and bulimia ( $p < 0.001$ ). Using the mYFAS 2.0 diagnostic scores (no FA, mild, moderate, and severe FA), it was found that there were significant differences between the eating styles (emotional eating, external and restrictive eating) and the FA groups ( $p < 0.001$ ), with the most severe levels of FA having the highest scores across all three eating styles. BE showed the strongest association with FA ( $p < 0.001$ ); the more severe the FA, the higher the BE scores. Finally, significant differences were also found between bulimia and FA ( $p < 0.001$ ), with the most severe levels of FA having the most bulimia.

**Relationship between mYFAS 2.0 and sociodemographic characteristics (age, sex), and BMI.** The relationship between mYFAS 2.0 and age was negative and statistically significant ( $r = 0.127$ ,  $p = 0.011$ , for the original score) and ( $r = 0.163$ ,  $p = 0.001$ , for the symptom count score). Younger people present greater FA than older people. In relation to gender and BMI, there were no significant differences with the original score, symptom count or mYFAS 2.0 diagnostic score. That is, there was no difference between males and females on the mYFAS 2.0 score, and there was also no difference according to BMI ( $p > 0.05$ ).

**Structural equation model to predict FA.** Two structural equation models were tested, in which the aforementioned variables and their prediction of FA were tested simultaneously, using the original and symptom count scores from the mYFAS 2.0. The two models fitted the data well:  $\chi^2(104) = 284.089$  ( $p < 0.001$ ); CFI = 0.95; SRMR = 0.05; RMSEA = 0.07 [0.057, 0.075], for the model in which items with the original score were used; and  $\chi^2(104) = 207.344$  ( $p < 0.001$ ); CFI = 0.96; SRMR = 0.07; RMSEA = 0.05 [0.040, 0.060], for the model in which mYFAS 2.0 symptom count scores were used. When measured with the original score, all variables were statistically significant, with BE showing the strongest relationship with FA. In total, 44.5% of the variance in FA was explained ( $p < 0.001$ ). When measured with the

symptom count score, external and restrictive eating did not show statistically significant predictive power on FA, and BE again showed the strongest relationship. In this case, 56.6% of the variance in FA was explained ( $p < 0.001$ ).

After conducting this study, it can be confirmed that the Spanish mYFAS 2.0 is a reliable and valid self-report measure to assess FA in the general Spanish population. Furthermore, the role of dietary variables (BMI, emotional eating, external eating, restrictive eating, BE and bulimia) in the prediction of FA is demonstrated for the first time, all together explaining approximately 50% of their variance. All these variables should be considered to prevent FA, especially in young people. To read the full article, please see annex 4. This article is currently in preparation.

3.5. Study 5. Eating behaviors, eating styles and body mass index during COVID-19 confinement in a college sample: a predictive model.



### **3.5. Study 5. Eating behaviors, eating styles and body mass index during COVID-19 confinement in a college sample: a predictive model.**

The first objective of the study was to analyze differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before and during COVID-19 confinement. The second objective was to analyze whether eating styles predict BE, fat intake and BMI during COVID-19 confinement.

A sample of 146 Spanish college students residing in Valencia was recruited, of whom 71.2% were female ( $M_{\text{age}} = 22.20$ ;  $SD = 2.97$ ) and 28.8% were male ( $M_{\text{age}} = 24.74$ ;  $SD = 3.53$ ). Of all participants, 75.17% were of normal weight ( $18.5 \geq \text{BMI} \leq 24.99$ ) before confinement, and 71.72% were of normal weight during confinement. Inclusion criteria were being between 18 and 30 years old, living in Spain at the time of confinement by COVID-19, and having completed the surveys by November 2019. Exclusion criteria were having an eating disorder, presenting a medical condition that may affect eating behavior or mood, and having a diagnosis of severe mental disorder.

Students were invited to participate in a study by answering several questionnaires before and during confinement by COVID-19. The first survey was completed in November 2019 (Time 1, period of normality in Spain; T1) and the second survey, in April 2020 (Time 2, period of strict confinement in Spain; T2). All surveys were conducted using the Lime Survey web platform of the Universitat de València.

The following measures were analyzed: sociodemographic characteristics (sex, age, and marital status); BMI ( $\text{weight}/\text{height}^2$ ); eating styles: emotional, external, and restrictive eating (DEBQ; Cebolla et al., 2014; van Strien et al., 1986); BE (BES; Escrivá-Martínez et al., 2019; Gormally et al., 1982); and fat intake (SFQ; Dobson et al., 1993).

**To analyze differences in eating styles (emotional, external, and restrictive eating), body mass index, binge eating, and fat intake before and during COVID-19 confinement.**

Results showed no statistically significant differences for emotional eating between T1 ( $M = 25.27$ ,  $SD = 9.90$ ) and T2 ( $M = 25.42$ ,  $SD = 10.44$ ):  $t(143) = -0.42$ ,  $p = .672$ ,  $d = 0.035$ . Nor did statistically significant differences emerge for external eating

between T1 ( $M = 29.40$ ,  $SD = 7.20$ ) and T2 ( $M = 28.77$ ,  $SD = 7.17$ ):  $t(143) = 1.20$ ,  $p = .231$ ,  $d = 0.100$ . Finally, no differences were found for restrictive eating ( $t(143) = -0.83$ ,  $p = .408$ ,  $d = 0.069$ ), with a mean of 21.31 and standard deviation of 7.88 at T1 and a mean of 21.71 and standard deviation of 7.93 at T2. Individuals did show a decrease in their BE at T2 ( $M = 7.67$ ,  $SD = 6.33$ ) compared to their BE at T1 ( $M = 6.53$ ,  $SD = 5.82$ ):  $t(87) = 2.07$ ,  $p = .041$ ,  $d_R = 0.23$ . In addition, individuals also decreased their fat intake at T2 ( $M = 20.39$ ,  $SD = 8.17$ ) compared to their fat intake at T1 ( $M = 22.21$ ,  $SD = 7.70$ ):  $t(87) = 2.75$ ,  $p = .007$ ,  $d_R = 0.36$ . However, with regard to BMI, no difference was found between T1 ( $M = 22.66$ ,  $SD = 2.92$ ) and T2 ( $M = 22.92$ ,  $SD = 3.35$ ):  $t(87) = -0.68$ ,  $p = 0.499$ ,  $d_R = 0.21$ .

**To analyze whether eating styles (emotional, external, and restrictive eating) assessed prior to confinement predict binge eating score, fat intake, and body mass index during confinement by COVID-19.** Model results indicated excellent model fit to the data:  $\chi^2(21) = 22.04$ ,  $p = .397$ , CFI = .99, RMSEA = .018 (90%CI .000 - .073), SRMR = .036. Emotional eating positively predicted BMI ( $\beta = .105$ ,  $p = .036$ ) and BE at T2 ( $\beta = .159$ ,  $p = .041$ ). External eating positively and marginally predicted fat intake at T2 ( $\beta = .138$ ,  $p = .090$ ). Restrictive eating positively predicted BE at T2 ( $\beta = .165$ ,  $p = .004$ ), and negatively and marginally predicted BMI ( $\beta = -.080$ ,  $p = .071$ ) and fat intake at T2 ( $\beta = -.102$ ,  $p = .055$ ). The model explained 80.5% of the variance in BMI, 41.5% of the variance in BE, and 25.8% of the variance in fat intake during confinement by COVID-19.

This study demonstrates for the first time that COVID-19 confinement may have improved eating behaviors in youth, considering previously obtained scores. In addition, structural equation modeling has shown that eating styles have a direct impact on BE, fat intake and BMI during COVID-19 confinement.

This is the first study to compare eating habits before and during COVID-19 confinement, without including retrospective data that may affect the validity of the dietary data. In addition, it is the first study to analyze using structural equation modeling whether eating styles before confinement play an important predictive role in BE, fat intake and BMI during confinement. To read the full article, please see annex 5. This article is under review by the journal *Journal of Behavioral Medicine*.

3.6. Study 6. Eating styles moderate the relationship between perceived stress and binge eating during COVID-19 confinement.





### **3.6. Study 6. Eating styles moderate the relationship between perceived stress and binge eating during COVID-19 confinement.**

The aim of this study was to evaluate whether eating styles (emotional, external, and restrictive eating) moderated the relationship between perceived stress and BE during COVID-19 confinement.

A sample of 114 Spanish college students was recruited, of whom 73.5% were female and 26.5% were male ( $M_{age} = 20.50$ ;  $SD = 5.24$ ). The mean BMI was 22.63 ( $SD = 3.32$ ).

Participants were recruited in the classrooms of the Universitat de València and through social networks and email. All students answered sociodemographic data and a survey to assess eating styles, the DEBQ (Cebolla et al., 2014; van Strien et al., 1986). In addition, they participated in a 7-day EMA using a mobile application, which measured perceived stress each day through the Perceived Stress Scale-4 (PSS-4; Herrero & Meneses, 2006) and the number of BE episodes per week. BE was assessed through two questions after each meal: "Have you overeaten (eaten more than would be reasonable during a short period)?", "During the BE episode, did you feel that you were losing control or could not stop eating?" (1 = not at all, 5 = extremely). The mobile app sent them the notifications every weekday between 7 pm and 10 pm. The data were collected between April 22 and April 30, 2020 (period of strict confinement by COVID-19 in Spain).

**Descriptive analyzes of eating styles, stress, and binge eating.** Students showed mean scores for all three eating styles: external eating ( $M = 2.94$ ;  $SD = .71$ ), emotional eating ( $M = 1.96$ ;  $SD = .78$ ), and restrictive eating ( $M = 2.17$ ;  $SD = .76$ ). Mean levels of stress ( $M = 1.96$ ;  $SD = .63$ ) were also shown. However, the BE score was low ( $M = .16$ ;  $SD = .58$ ).

**Moderation analysis (moderator variable: emotional eating).** The emotional eating model adequately predicted BE [ $F(3,67) = 13.64$ ,  $R^2 = .38$ ;  $p < .0001$ ]. The interaction between stress and emotional eating contributed 11.6% in explaining the variance of the model [ $F(1,67) = 12.52$ ;  $R^2 = .12$ ;  $p = .0007$ ]. The results indicate that individuals with high emotional eating tend to have more BE episodes when reporting stress ( $t = 3.34$ ;  $p = .0014$ ), and this relationship does not occur in individuals with

moderate or low levels of emotional eating. The proposed model explained 38% of the variance in BE.

**Moderation analysis (moderator variable: external eating).** The external eating model adequately predicted BE [ $F(3,67) = 6.62$ ,  $R^2 = .23$ ;  $p = .0006$ ]. The interaction between stress and external eating contributed 7% in explaining model variance [ $F(1,67) = 6.05$ ;  $R^2 = .70$ ;  $p = .0165$ ]. The results indicate that individuals with moderate ( $t = 2.59$ ;  $p = 0.011$ ) or high ( $t = 3.31$ ;  $p = 0.0015$ ) external eating tend to have more BE episodes when reporting stress, and this relationship does not occur in individuals with low levels of external eating. The proposed model explained 23% of the variance in BE.

**Moderation analysis (moderator variable: restrictive eating).** The restrictive eating model adequately predicted BE [ $F(3,67) = 4.47$ ,  $R^2 = .17$ ;  $p = .0064$ ]. The interaction between stress and restrictive eating contributed 5% in explaining the variance of the model [ $F(1,67) = 4.32$ ;  $R^2 = .05$ ;  $p = .0416$ ]. The results indicate that individuals with moderate ( $t = 2.57$ ;  $p = 0.0123$ ) or high ( $t = 3.16$ ;  $p = 0.0023$ ) restrictive eating tend to have more episodes of BE when reporting stress, and this relationship does not occur in individuals with low levels of restrictive eating. The proposed model explained 17% of the variance in BE.

This is the first study to demonstrate the influence of eating styles on the relationship between perceived stress and BE, in the context of a pandemic and using EMA. Eating styles may act as risk factors for abnormal and maladaptive eating behaviors in stressful situations. To read the full article, please see annex 6. This article is under review by the journal *Foods*.

## **4. DISCUSSION**



# General discussion



## **General discussion**

As shown throughout the Doctoral Thesis, young people are currently facing two major issues: dysfunctional eating patterns (e.g., high intake of saturated and trans fats and/or sugars, BE and FA) and BD. A high comorbidity between BE and BD has been reported in the literature on several occasions. However, the commonalities between BE and BD, their differences, and the influence between the two are unknown. In addition, there are many sociodemographic, personality and dietary variables that are associated with these dysfunctional patterns and thus may influence BD. It is necessary to test whether these variables may act as risk factors for these problematic eating and drinking behaviors in young people, in order to design prevention and treatment in the most appropriate way possible

To fill this gap in the literature, this Doctoral Thesis was conceived to shed light on the relationship between dysfunctional eating patterns and BD in young people and on the risk factors associated with these behaviors. Therefore, the main objective of this Doctoral Thesis was to study the relationship between dysfunctional eating patterns and BD in young people and to explore the risk factors associated with these behaviors.

With these objectives in mind, six studies were conducted. First, a narrative review was carried out to identify the commonalities between BE and BD, the differences, and the influence of BE on BD and vice versa. Secondly, validation of the BES was carried out in order to reliably and validly assess BE in a young Spanish population. Third, a structural equation model was used to analyze whether dysfunctional eating patterns (fat intake and BE) were predictive of BD in young people. We also analyzed whether these dysfunctional eating patterns mediated the relationship between various individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) and BD. Fourth, the mYFAS 2.0 was validated to assess FA reliably and validly in young Spaniards. In addition, the predictive role of several sociodemographic and dietary factors (sex, age, BMI, eating styles, BE and bulimia) on FA in young people was analyzed. Fifth, differences in eating styles (emotional, external, and restrictive eating), dysfunctional eating patterns (BE and fat intake) and BMI before and during COVID-19 confinement were analyzed. The influence of eating styles on BE, fat intake and BMI during strict COVID-19

confinement was also analyzed. Finally, sixth, the moderating role of eating styles on the relationship between perceived stress and BE during COVID-19 confinement was evaluated.

In this chapter, the main results obtained from each study are summarized. Subsequently, the strengths and limitations of the present dissertation are discussed. Finally, the clinical implications, possible future directions and general conclusions of the whole work are presented.



## 4.1. Summary of main findings



#### 4.1.1. Narrative review of binge eating and binge drinking.

The aim of study 1 was to conduct a narrative review exploring the commonalities and differences of BE and BD, as well as the influence of one on the other and vice versa. In addition, we explore what are the reasons why young people tend to engage in both behaviors.

In summary, BE and BD have several commonalities, such as the onset and nature of the problem, prevalence, and associated consequences. In addition, several factors may act as risk variables in both behaviors, e.g., negative emotions and personality (depressive symptoms, impulsivity, neuroticism, sensation seeking or reward sensitivity) and sociocultural factors (e.g., being like one's peer group and being socially accepted). These commonalities point to the need to consider both behaviors when planning interventions targeting youth. In addition, transdiagnostic interventions may be more beneficial in reducing both behaviors by targeting their commonalities.

Regarding the differences between BE and BD, dietary restraint and body dissatisfaction appear to be risk factors for BE (Fairburn et al., 2003; Stice et al., 2000). The relationship between these two variables and BD appears to be different. In this context, there is a pattern that has been increasing in recent years, called "drunkorexia". This is the tendency to restrict food consumption in order to become intoxicated more quickly from alcohol (Eisenberg & Fitz, 2014; Hunt & Forbush, 2016). Another difference is found in the importance of the substance. While the concept of alcohol addiction is well established, the concept of FA is not yet established, and is not considered within addictive disorders. The last difference is found in the withdrawal phenomenon; while withdrawal is well known in alcohol consumption, there is no evidence of withdrawal symptoms in FA (Schulte et al., 2016, 2018; Ziauddeen & Fletcher, 2013).

To date, despite their comorbidity and commonalities, it is unknown whether these behaviors may influence each other. Regarding the effects of BD on BE, evidence is limited. Some studies have shown that alcohol stimulates short-term food intake when consumed before or with a meal. This effect may be attributed to a loss of self-control (Caton et al., 2015; Yeomans, 2010) or that alcohol may increase the rewarding value of food (Melis et al., 2009). In addition, alcohol intake also seems

to influence the intake of high-fat foods (Caton et al., 2007; Yeomans, 2010). There is a study that points out that this relationship is influenced by the amount of alcohol consumed, with only high doses producing such an effect (Caton et al., 2004). Therefore, in this sense, it seems that there is a direct influence of BD on BE.

In relation to the effects of BE on BD, mixed results are observed. Some research has observed a predictive relationship of BE on BD (Blanco-Gandía, Ledesma, et al., 2017; Field et al., 2012), however, other results do not confirm this relationship (Micali et al., 2015; Sonnevile et al., 2013). These inconclusive results may be due to the wide variety of definitions of BE and BD. There is a need to unify criteria to define both variables in order to determine more precisely the direction between both behaviors.

Therefore, the hypotheses stated that both BE and BD share many commonalities, differences and positive influence between them are fulfilled. This review demonstrates for the first-time evidence for directionality between the two behaviors, although further research is needed to affirm the mutual influence between these behaviors. This review may help to better understand the etiology, development, and maintenance of both behaviors.

#### 4.1.2. Psychometric properties of the Binge Eating Scale.

The aim of study 2 was to analyze the psychometric properties of the BES in a young Spanish population, specifically, to analyze its factor structure through a CFAs, to test the measurement invariance between sexes, and to analyze its reliability and its convergent and discriminant validity.

Considering the evidence of the factor structure of the BES, two structures were tested, the one-factor model of the BES and the two-factor model. The results supported the one-dimensionality of the scale, with the one-factor model being the best fit to the data. Although the original authors support the two-dimensionality of the scale, our results showed a high correlation between the two factors, being very difficult to demonstrate discriminant validity between the two factors. Moreover, this factor structure is also supported by other studies (Brunault et al., 2016; Duarte et al., 2015). For all these reasons, it was decided to opt for the one-factor model.

Once the factor structure was clear, an invariance measurement routine was tested to check whether the BES was invariant between sexes (men and women). The scalar invariance model was the most parsimonious and best fitting, showing evidence of no measurement bias when comparing the two groups, as the meaning of BE was the same for both men and women. It should be noted that men and women behave differently in the context of eating disorders, so sex invariance is necessary to make comparisons between groups (Kline, 2015). However, this is assumed in much of the research, as the authors assume that there is mean difference, without offering evidence of invariance (Gormally et al., 1982; Ricca et al., 2000).

Because scalar invariance was maintained, latent means were compared between samples to test whether BE levels were equal in the two groups. The fit of this model decreased significantly, so the results pointed to a difference between the means of the two groups, with females having the higher mean. Therefore, it can be concluded that there were statistically significant differences in BE between sexes, with a higher prevalence in women. This result is in line with other studies that point to a higher prevalence of BE in women (Kessler et al., 2013; Preti et al., 2009; Ricca et al., 2000). However, this is the first study to test this model in a measurement error-free context.

We also provided evidence of reliability and convergent and discriminant validity of the Spanish BES. Regarding internal consistency, reliability estimates were excellent. Regarding convergent and discriminant validity, evidence of convergent validity was shown through the positive, high, and statistically significant correlation between the BES and the EDI-3 BE indicator, and between the BES and FA. Evidence of discriminant validity was provided through the statistically significant, but lower correlations between BES and impulsivity dimensions, between BES and BD, and between BES and BMI. This is consistent with other previous findings (Duncan et al., 2017; Fouladi et al., 2015; Freitas et al., 2006; Kessler et al., 2013; Laghi et al., 2014; Mason et al., 2018; Steward et al., 2017; Villarejo et al., 2012). These results point to the importance of understanding the relationship between BE and addictive problems and opens doors for us to study the mechanisms underlying both behaviors.

This is the first study in the literature to test two competing models to analyze factor structure, suggesting a unidimensional structure, in accordance with DSM-5

criteria (American Psychiatric Association, 2013). In addition, it is the first time this scale is assessed in young population of both sexes, and the first to test measurement invariance between sexes. Based on the results of this study, all our hypotheses are confirmed. That is, the one-dimensionality of the scale is supported, there is evidence of measurement invariance between sexes and good convergent and discriminant validity.

The BES has good psychometric properties in young Spanish population, being therefore a reliable and valid self-report measure to assess BE in young Spanish population. The BES is one of the most widely used scales to assess BE, both in clinical and non-clinical samples. This study can help us to detect BE in young Spaniards, and thus to better understand and delve deeper into this problem, which is so prevalent in today's youth. It should be noted that the Spanish BES is freely available and can be downloaded in article 2 (see annex 2) and in this Doctoral Thesis (see annex 9).

#### 4.1.3. Influence of dysfunctional eating habits on binge drinking.

The first objective of study 3 was to analyze the relationship between BE and fat intake with BD. In addition, the literature points to several factors that are related to BE and fat intake and thus may be indirectly related to BD. Thus, the second aim of study 3 was to analyze whether BE and fat intake mediated the relationship between individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) and BD. For all of these, a MIMIC structural equation model was tested.

The MIMIC that was proposed showed a good fit. This model indicated that BE and fat intake were positively related to BD. These results are in line with other studies in animal models that point to a causal relationship between fat intake and BE with BD (Blanco-Gandía, Ledesma, et al., 2017). Although this is the first study pointing to this relationship in young people, there are previous studies that have already associated BE as risk factors in alcohol and drug abuse (Field et al., 2012; Measelle et al., 2006; Micali et al., 2015; Sonnevile et al., 2013). A plausible hypothesis would be that BE produces negative emotions such as shame or guilt, therefore, alcohol consumption could be used to regulate these emotions (Caton et al., 2015).

In relation to the second objective, the results pointed out that emotional eating was positively related to BD through its relationship with BE. There is evidence that emotional eating may be a predictor of BE (Mason & Lewis, 2014; Sultson et al., 2017), however, the mediating role of BE in the relationship between emotional eating and BD was unknown. External eating, in turn, was related to BD through its relationship with BE and fat intake. There is evidence of the relationship between external eating with fat intake (Anschutz et al., 2009; Camilleri et al., 2014) and with BE (Burton et al., 2007; Mason & Lewis, 2014). However, our study is the first to examine the mediating role of BE and fat intake in this relationship.

Restrictive eating was also related to BE, but in this case negatively, and this relationship was mediated by decreased fat intake. That is, people who restrict food consume less fat, and this lower fat intake leads them to consume less alcohol. One possible explanation is that both food and alcohol are high in calories, and restrictive eaters try to reduce their calories in order to maintain or reduce weight. A recent study points to the weak relationship between alcohol consumption and restrictive behaviors (Baker et al., 2017). Our study points out that fat intake may act as a mediator between both behaviors.

Finally, FA was also related to BD through its relationship with fat intake and BE. The relationship between food and alcohol addiction is well known (Gearhardt et al., 2009a, 2009b), however, this is the first study to suggest that fat intake and BE may play an important role in mediating this relationship.

The model fully explained 68.3% of BE, 27.0% of fat intake, and 10.4% of BD.

Therefore, as hypothesized, there is a positive influence of dysfunctional eating patterns (fat intake and BE) on BD. Furthermore, the hypothesis that individual factors (gender, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity, and FA) predict BD through their relationship with dysfunctional eating patterns (fat intake and BE) is also confirmed.

This study provides for the first-time evidence of the direct relationship between BE and fat intake on BD in young people through structural equation modeling. It is also the first time that evidence is provided for the indirect relationship between different eating patterns (eating styles and FA) and BD, mediated by BE and fat intake.

This study may help prevent BD in young people through interventions targeting young people with dysfunctional eating patterns. Furthermore, if strategies to reduce all these dysfunctional eating patterns are promoted, this could have a positive impact on reducing youth BD.

#### 4.1.4. Psychometric properties of the modified Yale Food Addiction Scale 2.0 and risk factors for food addiction.

The first objective of study 4 was to analyze the psychometric properties of the mYFAS 2.0 in a young Spanish population. Specifically, the factor structure was analyzed through a competing structural equation model and evidence of its internal consistency was provided. The second objective of study 4 was to analyze the relationship between mYFAS 2.0 with other dietary variables (emotional eating, external eating, restrictive eating, BE and bulimia), sociodemographic characteristics (age and sex) and BMI. The third objective was to analyze the predictive power of these dietary variables (emotional eating, external eating, restrictive eating, BE, bulimia and BMI) on mYFAS 2.0, also through structural equation modeling.

To carry out the first objective, the factor structure of the mYFAS 2.0 was assessed by CFAs. Two single-factor models were tested, considering the original score and the symptom count score. Both models fitted excellently. Therefore, evidence of a single factor of the mYFAS 2.0 was provided. This result is consistent with the original validation, which suggested a good fit for the single-factor model (Schulte & Gearhardt, 2017). Furthermore, the factor loadings of the two models were statistically significant, therefore, the one-factor solution was maintained. Regarding reliability, evidence was provided through Cronbach's  $\alpha$  and McDonald's  $\omega$ . Internal consistency was excellent with both the original score and the symptom count scores, indicating that the mYFAS 2.0 is a reliable measure to assess FA in young Spanish population.

The prevalence of FA in our sample was 31.9%. These results are higher than those described in other studies, which indicate a prevalence of between 11.4% and 21% in young university students (Gearhardt et al., 2009a; Murphy et al., 2014; Rostanzo & Aloisi, 2021). This may be due to the use of different scales to measure



FA. These results point to the need to further analyze whether there are sociodemographic and personal factors that may be causing these differences in the prevalence of FA.

Two CFAs were also estimated to explain that both mYFAS 2.0 items and mYFAS items measured the same construct. The results indicated that both scales measured FA. Due to the good psychometric properties exhibited by both scales and the similarity in their scoring form (Lemeshow et al., 2016; Schulte & Gearhardt, 2017), the results were as expected. This is the first time that the relationship between the mYFAS and the mYFAS 2.0 has been explored.

To carry out the second objective of this study, the relationships between FA and other relevant dietary variables, such as eating styles (emotional, external, and restrictive eating), BE and bulimia, were analyzed. FA was shown to be related to all these variables. This suggests that people who tend to eat more in response to negative emotions or in response to appetitive environmental stimuli are more likely to have FA. This may mean that food may play an important role in calming negative emotions, whereby people may increasingly need to consume more and more food to feel better (Dingemans et al., 2009), which in turn may contribute to FA. For their part, external eaters may also eat more because these, by consuming more energy (Wardle et al., 1992), the response of their reward system is less and less, and, therefore, need to eat more to feel better (Stice et al., 2013). We also found a relationship between restrictive eating and FA. BE may play an important role in this relationship, as dieting may precipitate BE (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020), and these in turn increase the likelihood of presenting FA.

Related to the above, a relationship between BE and FA was also observed, being the strongest relationship of all. FA was also related to bulimia, as would be expected. It could be thought that the excessive food intake that occurs in BE and bulimia may sensitize the brain reward system, which may result in a need to continue eating, as occurs with drugs of abuse (Robinson et al., 2015).

Regarding the relationship between FA and sociodemographic characteristics (age, gender) and BMI, it was found that young people presented higher scores in FA. This may be due to the greater vulnerability to addiction in young people, as they are

more suggestible to environmental effects than older people and have more difficulty controlling or resisting unhealthy food.

In relation to gender, there was no difference between men and women in FA, and there was also no difference according to BMI. There is evidence confirming no relationship between FA and gender (Ahmed & Sayed, 2017; Hauck et al., 2017; Schulte & Gearhardt, 2017). One might think that the gender differences obtained in some studies in relation to FA are due to the fact that the samples are usually composed of overweight or obese women (Pursey et al., 2014). Our sample is composed of young people, which may explain the absence of differences.

To carry out the third and final objective of this study, two structural equation models were tested to see if all the previously mentioned variables (emotional eating, external eating, restrictive eating, BE, bulimia, and BMI) predicted FA, using the original and symptom count scores of the mYFAS 2.0. Both models were adequately adjusted. When measured with the original score, all variables were statistically significant, with BE showing the strongest relationship with FA. In this model, 44.5% of the variance in FA was explained. When measured with the symptom count score, external and restrictive eating did not significantly predict FA, and BE again showed the strongest relationship. In this model, almost 60% of the variance in FA was explained. With all this, it was possible to demonstrate the predictive power of all the variables mentioned above (emotional eating, external eating, restrictive eating, BE, bulimia, and BMI) on FA.

Therefore, as hypothesized, the mYFAS 2.0 has a unifactorial structure, and there are several dietary factors that directly predict FA, such as eating styles, BE, bulimia, and BMI.

This study confirms for the first time that the Spanish mYFAS 2.0 is a reliable and valid self-report measure to assess FA in a young Spanish population. Furthermore, it demonstrates for the first time the role of BMI, eating styles, BE and bulimia in the prediction of FA in young people, all of them explaining approximately 50% of their variance. This is the first study to point out the importance of considering all these variables together in the prevention of FA in youth. It should be noted that the Spanish mYFAS 2.0 is freely accessible and can be downloaded in article 4 (see annex 4) and

in this Doctoral Thesis (see annex 10).

4.1.5. Dysfunctional eating patterns and body mass index during confinement and associated risk factors.

Study 5 aimed to analyze differences in eating styles (emotional, external, and restrictive eating), BE, fat intake and BMI before (Time 1, T1) and during COVID-19 confinement (Time 2, T2). The second objective was to analyze whether eating styles predicted BE, fat intake and BMI during COVID-19 confinement.

Regarding the first objective, the results showed no statistically significant differences for emotional eating, external eating, and restrictive eating between T1 and T2. These findings are supported by previous literature, which points out that eating styles are temporally stable despite negative circumstances (Meiselman et al., 1998). There are also studies suggesting that eating styles are trait factors, not state factors (Malesza & Kaczmarek, 2021). This is consistent, since if we ask young people "do you feel like eating when you are angry?" they are likely to respond similarly every time, regardless of whether they are facing adverse situations or not. Thus, this study reinforces the idea that eating styles are stable despite experiencing stressful circumstances, such as COVID-19 confinement.

Youth did reduce BE and fat intake during confinement. This may be due to the fact that youth adopted healthier habits during confinement (Di Renzo et al., 2020). For instance, there was an increase in physical activity during the confinement period (Romero-Blanco et al., 2020). In addition, the food was more homemade because it was not possible to go to restaurants, therefore, the probability of eating healthier food was higher (Di Renzo et al., 2020; Sinisterra-Loaiza et al., 2021).

Regarding BMI, no changes were observed before and during confinement. This may be related to the above, as young people adopted healthier habits, which may have helped to avoid weight gain during the quarantine period. There is a study that points out that people with normal weight have higher adherence to healthy dietary guidelines (Di Renzo et al., 2020). Considering that most of our sample had a normal BMI before confinement, it would be expected that they would improve their eating habits and no changes in their BMI would be reported.

Regarding the second objective, the results indicated that emotional eating positively predicted BMI and BE at T2 but did not predict fat intake at T2. Previous studies have pointed out that people who are emotional eaters find it difficult to differentiate real hunger from physiological signals that are determined by negative emotions (Bruch, 1964). Therefore, they respond by eating more when negative emotions are present. This can lead to an increase in their BMI (Hemmingsson, 2014). The fact that emotional eating influences only BE and not fat intake may be due to the fact that the consumption of unhealthy foods is not associated with negative emotions, as pointed out by some studies (Adriaanse et al., 2011). In addition, it could be speculated that emotional eating may be associated with impulsivity, the difficulty in controlling eating behavior. Therefore, it is possible that it has more influence on the amount eaten (BE) and not so much on the type of food (fat intake).

External eating was positively and marginally predictive of fat intake at T2. This is in line with other studies, which indicate an association between external eating and energy intake, especially fat (Anschutz et al., 2009). This is the first study to relate external eating to fat intake during confinement. These results are consistent, as external eaters tend to eat according to the external properties of food (van Strien et al., 1986), making them more likely to eat appetizing foods rich in fat. However, it was observed that external eating did not predict BE during confinement. There is a study that external eating may be a risk factor for BE only in people with depressive problems and high BMI (Mason & Lewis, 2014). Considering that our sample consists of people with normal BMI and no mental problems, one would expect that external eating would not predict BE. Finally, external eating also did not predict BMI. It is plausible that BMI is determined more by the response to negative emotions rather than by the response to environmental cues (van Strien et al., 2009). The mediating role of fat intake in the relationship between external eating and BMI has also been observed (Burton et al., 2007). In this study, external eating and BMI are directly related, so future studies should evaluate the mediating role of fat intake at times of elevated stress, such as COVID-19 confinement.

Finally, restrictive eating positively predicted BE at T2 and negatively and marginally predicted BMI and fat intake at T2. The goal of restrictive eaters is to lose or maintain weight, so it is plausible that they will eat less fat in order to achieve their

goal (Anschutz et al., 2009). Furthermore, as has been discussed on several occasions, dietary restriction can cause a rebound effect and precipitate BE (Fairburn et al., 2003; Stice et al., 2000). This is the first study to demonstrate the influence of dietary restriction on BE, fat intake and BMI in a stressful time, as was COVID-19 confinement. The model in total explained 80.5% of the variance in BMI, 41.5% of the variance in BE and 25.8% of the variance in fat intake during COVID-19 confinement.

Therefore, the hypotheses of changes in BE and fat intake during confinement were met, although BMI remained stable for the reasons discussed above. Furthermore, as expected, these variables (BE, fat intake, and BMI) were affected by maladaptive eating styles.

This study is the first to compare eating habits before and during COVID-19 confinement, without including retrospective data that may affect the validity of the data presented. It is also the first study to analyze the predictive role of eating styles on BE, fat intake, and BMI during COVID-19 confinement. The present study may help to reduce dysfunctional eating behaviors during times of high stress by focusing interventions on mindful eating techniques and techniques that help to reduce external and restrictive behaviors. It is necessary to understand the relationship between all these variables, in order to best prevent obesity and health problems in young people.

4.1.6. The role of eating styles in the relationship between perceived stress and binge eating during COVID-19 confinement.

Study 6 aimed to assess the moderating role of eating styles (emotional, external, and restrictive eating) in the relationship between perceived stress and BE during COVID-19 confinement. In this study, we chose to assess perceived stress and BE using EMA, with a mobile application measuring perceived stress and BE during seven days of confinement. This assessment avoids recall bias and improves ecological validity and is the first study to use this assessment to analyze these behaviors.

As hypothesized, results indicated that all three eating styles moderated the relationship between perceived stress and BE during COVID-19 confinement. First, it was shown that individuals with high emotional eating tend to have more BE episodes

when reporting stress, and this relationship does not occur in individuals with moderate or low levels of emotional eating. The proposed model explained 38% of the variance in BE. In this case, it could be concluded that emotional eaters resort to BE to relieve negative emotions, such as stress (Ricca et al., 2009), as these are the ones who have the most difficulty coping with these emotions and food can be a positive emotional reward for them (dos Santos Quaresma et al., 2021).

Second, the results indicated that individuals with moderate or high external eating tend to binge eat more when reporting stress, and this relationship does not occur in individuals with low levels of external eating. The proposed model explained 23% of the variance in BE. It could be thought that external eaters, having more difficulty distancing themselves from tasty stimuli in high stress situations (Hou et al., 2013; Kalkan-Uğurlu et al., 2021), increase the likelihood of BE. Furthermore, it should be considered that the confinement situation entailed a change in people's lifestyle, as daily activities were restricted, and they chose to buy large amounts of food and store them at home due to the fear of food shortages. Staying at home for so long and the high availability of so much food, coupled with the stress we were experiencing, may have also increased the likelihood of BE (Touyz et al., 2020).

Third and finally, it was shown that individuals with moderate or high restrictive eating also tend to have more episodes of BE when they report stress, and this relationship does not occur in individuals with low levels of restrictive eating. It is possible that individuals who tend to be restrictive eaters, in stressful situations, find it more difficult to self-regulate, which may lead them to consume more food (Baumeister et al., 1993). This is also consistent with disinhibition theory, in which restrictive eaters exhibit dichotomous or extreme thoughts about dieting. Because stress may lead them to skip the diet, they may think that they have blown the process and that it makes no difference what they eat, increasing the likelihood of overeating (Ruderman, 1986).

The pandemic was a stressful time when people may have engaged in episodes of BE to cope with stress. It is important to know "for whom" this relationship is strongest, as these individuals may benefit most from interventions aimed at reducing BE at times of high stress. Eating styles (emotional, external and restrictive eating) may play an important role as moderators of this relationship, as it has been shown on several occasions that these eating styles may increase the likelihood of BE (Černelič-

Bizjak & Guiné, 2021; Kim et al., 2018; Zunker et al., 2011). However, it was not known what happened in negative and prolonged stressful situations.

This is the first study to demonstrate the influence of eating styles on the relationship between perceived stress and BE, with the strength of having been conducted in the context of a pandemic and using an EMA. This study may lead to the conclusion that eating styles may act as risk factors for BE in stressful situations. It is important to know which individuals are most at risk of developing BE in high-stress situations, so that these individuals may benefit most from interventions aimed at reducing dysfunctional eating patterns during stressful times.





## 4.2. Strengths



## 4.2. Strengths

This Doctoral Thesis has several strengths which are necessary to highlight in order to better understand the main findings and implications.

Firstly, the dissertation consists of six studies that meet high methodological standards. As can be seen in the "Methodology and results" section, most of these studies use structural equation modelling to analyze both the factor structure of two scales (BES and mYFAS 2.0) and the predictive relationship between variables. Furthermore, all the studies presented comply with ethical standards and were approved by the ethics committee of the Universitat de València (registration numbers: H1513854038939; 1821046).

Secondly, all the research in this dissertation contributed novel aspects to scientific knowledge, as has been proven by the literature published to date. The first study reviews for the first time the directionality between BE and BD, demonstrating for the first-time evidence of directionality between the two behaviors. The second study is the first in the literature to validate the BES in the general Spanish population of both sexes. In addition, two competing models are tested for the first time to analyze the attempted structure of the BES, and it is also the first time that measurement invariance is tested in both sexes. The third study is the first translational study to demonstrate how fat intake and BE can directly influence BD in young people, as well as to provide evidence for an indirect relationship between different eating patterns (eating styles and FA) and BD, mediated by BE and fat intake. The fourth study is the first to validate the mYFAS 2.0 in Spanish and is also the first to demonstrate the role of all these dietary variables (BMI, emotional eating, external eating, restrictive eating, BE and bulimia) in predicting FA. The fifth study is the first in the literature to compare eating habits before and during COVID-19 confinement, without including retrospective data that may affect the validity of the dietary data presented. Furthermore, it is the first study to analyze using structural equation modelling whether eating styles (emotional, external, and restrictive eating) assessed before confinement play an important predictive role in BE, fat intake and BMI during confinement. Finally, the sixth study is also the first to demonstrate the influence of eating styles on the relationship between perceived stress and BE, in the context of a pandemic and using EMA.

Importantly, this Doctoral Thesis developed for the first time a mobile application to assess dysfunctional eating patterns (fat intake and BE), alcohol consumption and BD in real time and in the natural context. The app was implemented by participants during the confinement by COVID-19 to conduct study 6. However, we hope to return soon to a situation of normality in order to be able to assess longitudinally whether dysfunctional eating patterns can predict BD in young people.

## 4.3. Limitations



### **4.3. Limitations**

The present Doctoral Thesis is not without limitations. Firstly, the representativeness of the sample was limited, as all studies were carried out with college students. Therefore, the findings cannot be generalized to other demographic groups, such as young people of other educational levels or adults, who have also been found to exhibit dysfunctional eating and drinking patterns. Secondly, all studies used self-reported height and weight to calculate BMI. Research suggests that self-reported measures of height and weight should be viewed with caution, as individuals tend to overestimate height and underestimate body weight (Niedhammer et al., 2000), which may lead to underestimation of the BMI value. Along these lines, measures of eating behaviors in some studies (studies 3, 4 and 5) were also self-reported, which may be subject to self-report biases and yield different results than would be obtained with more structured assessments (Berg et al., 2012). Another limitation was the cross-sectional nature of the data in some studies (studies 3 and 4), which requires cautious interpretation of the direction of the associations obtained. Finally, study 6 has two specific limitations. On the one hand, the mobile application is only available for Android devices, which may bias the results. On the other hand, the full potential of the EMA could not be exploited, as the weekly measures of BE and stress were averaged. In the "future directions" section, several proposals for future studies are presented to address these limitations.





## 4.4. Clinical implications



#### **4.4. Clinical implications**

The results obtained from this Doctoral Thesis may have implications for researchers and health professionals interested in promoting health and preventing risky behaviors in young people. The work presented points to several conclusions. First, fat intake and BE may be a gateway to the onset and development of BD in young people. Eating styles (emotional, external, and restrictive eating) and FA may also influence BD through their relationship with fat intake and BE. Prevention and intervention strategies could target young people with high scores on these eating patterns to weaken the association between these eating patterns and BD. Secondly, it is shown that BMI, eating styles, BE, and bulimia may be risk factors for FA in young people. Therefore, these variables can be considered in the prevention of FA in this population. Thirdly, it is shown that eating styles can also directly influence BE, fat intake and BMI during COVID-19 confinement. Furthermore, eating styles may influence the relationship between perceived stress and BE during COVID-19 confinement. Therefore, prevention policies should focus their efforts on reducing eating styles, for instance, with mindful eating techniques, to curb dysfunctional eating behaviors and BMI problems during stressful times. This Doctoral Thesis may help us to understand the causal relationships between different eating behaviors. This could be a first step to prevent and treat obesity and behaviors that impact on the physical and mental health of young people.



## 4.5. Future directions



#### **4.5. Future directions**

This Doctoral Thesis helped to answer some relevant questions. However, the field is still poorly known, and more research and future research is needed to overcome the limitations mentioned above. Open questions and new challenges that could be addressed in future studies are outlined below.

Firstly, future studies could explore the psychometric properties of the BES and the mYFAS 2.0 in other Spanish samples to assess BE and FA in other populations (adolescents, older adults, eating disorders, etc.). Furthermore, it would be convenient to test for measurement invariance between sexes, in order to check whether the meaning of both constructs is the same among men and women. Related to the above, future studies should also explore causal relationships between eating behaviors and alcohol consumption in other populations and be able to generalize the results to other populations.

Secondly, it is proposed that future studies conduct longitudinal evaluations or time series analyzes with larger samples to explore more accurately the predictive relationships observed in this Doctoral Thesis. For instance, to analyze how fat intake and daily BE influence subsequent BD, or to analyze how eating styles (emotional, external, and restrictive eating) influence FA or BE at normal and stressful times. Longitudinal studies and studies with larger and more heterogeneous samples are needed to explore all these relationships in more depth.

Thirdly, although there are indicators that work well for measuring BD, such as AUDIT item 3 "how often do you have six or more drinks on a single occasion?" (Maurage et al., 2020) or the BD indicator "have you ever had four or more drinks (five if you are a woman) in a two-hour period in the past month?" (Kuntsche et al., 2006; Paul et al., 2011), we do not yet have a standardized measure to assess BD in young people. Therefore, it is recommended that future studies develop a standardized measure to measure this prevalent yet harmful behavior in young people.

Fourthly, future research should explore the role of other nutrients, such as carbohydrates, as they may also act as risk factors for alcohol consumption and eating behaviors, such as BE, FA, or BMI.





## 4.6. Conclusions



## 4.6. Conclusions

The main findings with respect to the aims of this Doctoral Thesis are presented below:

- After a comprehensive review of the literature, it is concluded that BE and BD share many commonalities. Furthermore, there is evidence of influence between both behaviors.
- The Spanish version of the BES is a reliable and valid self-report scale to assess BE in a young Spanish population. The significance of BE assessed with the BES does not vary between males and females.
- Fat intake and BE may act as a gateway to the onset and development of BD in young people.
- Eating styles and FA may indirectly influence BD through their relationship with fat intake and BE.
- The Spanish version of the mYFAS 2.0 is a reliable and valid self-report measure to assess FA in the general Spanish population.
- BMI, eating styles, BE, and bulimia may act as risk factors for FA in young people.
- COVID-19 confinement may have improved young people's eating behavior.
- Eating styles (emotional, external, and restrictive eating) may play an important predictive role in BE, fat intake and BMI during COVID-19 confinement.
- Eating styles (emotional, external, and restrictive eating) may influence the relationship between perceived stress and BE during COVID-19 confinement.



## **5. REFERENCES**



## 5. References

- Adriaanse, M. A., de Ridder, D. T. D., & Evers, C. (2011). Emotional eating: Eating when emotional or emotional about eating? *Psychology & Health, 26*(1), 23–39. <https://doi.org/10.1080/08870440903207627>
- Ahmed, A. Y., & Sayed, A. M. (2017). Prevalence of food addiction and its relationship to body mass index. *Egyptian Journal of Medical Human Genetics, 18*(3), 257–260. <https://doi.org/10.1016/j.ejmhg.2016.10.002>
- Allison, S., & Timmerman, G. M. (2007). Anatomy of a binge: Food environment and characteristics of nonpurge binge episodes. *Eating Behaviors, 8*(1), 31–38. <https://doi.org/10.1016/j.eatbeh.2005.01.004>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* (5th ed.). American Psychiatric Association.
- Ammar, A., Trabelsi, K., Brach, M., Chtourou, H., Boukhris, O., Masmoudi, L., Bouaziz, B., Bentlage, E., How, D., Ahmed, M., Mueller, P., Mueller, N., Hammouda, O., Paineiras-Domingos, L. L., Braakman-Jansen, A., Wrede, C., Bastoni, S., Pernambuco, C. S., Mataruna, L., ... Hoekelmann, A. (2021). Effects of home confinement on mental health and lifestyle behaviours during the COVID-19 outbreak: insights from the ECLB-COVID19 multicentre study. *Biology of Sport, 38*(1), 9–21. <https://doi.org/10.5114/BIOLSPORT.2020.96857>
- Anderson, R. M., Heesterbeek, H., Klinkenberg, D., & Hollingsworth, T. D. (2020). How will country-based mitigation measures influence the course of the COVID-19 epidemic? *The Lancet, 395*(10228), 931–934. [https://doi.org/10.1016/S0140-6736\(20\)30567-5](https://doi.org/10.1016/S0140-6736(20)30567-5)
- Anschutz, D. J., Van Strien, T., Van De Ven, M. O. M., & Engels, R. C. M. E. (2009). Eating styles and energy intake in young women. *Appetite, 53*(1), 119–122. <https://doi.org/10.1016/j.appet.2009.03.016>
- Avena, N. M., Gearhardt, A. N., Gold, M. S., Wang, G.-J., & Potenza, M. N. (2012). Tossing the baby out with the bathwater after a brief rinse? The potential downside of dismissing food addiction based on limited data. *Nature Reviews*

*Neuroscience*, 13(7), 514–514. <https://doi.org/10.1038/nrn3212-c1>

Avena, N. M., Rada, P., & Hoebel, B. G. (2009). Sugar and Fat Bingeing Have Notable Differences in Addictive-like Behavior. *The Journal of Nutrition*, 139(3), 623–628. <https://doi.org/10.3945/jn.108.097584>

Bahji, A., Mazhar, M. N., Hudson, C. C., Nadkarni, P., MacNeil, B. A., & Hawken, E. (2019). Prevalence of substance use disorder comorbidity among individuals with eating disorders: A systematic review and meta-analysis. *Psychiatry Research*, 273, 58–66. <https://doi.org/10.1016/j.psychres.2019.01.007>

Baker, J. H., Munn-Chernoff, M. A., Lichtenstein, P., Larsson, H., Maes, H., & Kendler, K. S. (2017). Shared familial risk between bulimic symptoms and alcohol involvement during adolescence. *Journal of Abnormal Psychology*, 126(5), 506–518. <https://doi.org/10.1037/abn0000268>

Balodis, I. M., Potenza, M. N., & Olmstead, M. C. (2009). Binge drinking in undergraduates: relationships with sex, drinking behaviors, impulsivity, and the perceived effects of alcohol. *Behavioural Pharmacology*, 20(5–6), 518–526. <https://doi.org/10.1097/FBP.0b013e328330c779>

Barrat, E. S. (1985). Impulsiveness subtraits: Arousal and information processing. In J. T. Spence & C. E. Izard (Eds.), *Motivation, Emotion and Personality* (pp. 137–146). Elsevier Science Publishers.

Baumeister, R. F., Heatherton, T. F., & Tice, D. M. (1993). When ego threats lead to self-regulation failure: Negative consequences of high self-esteem. *Journal of Personality and Social Psychology*, 64(1), 141–156. <https://doi.org/10.1037/0022-3514.64.1.141>

Bava, S., & Tapert, S. F. (2010). Adolescent brain development and the risk for alcohol and other drug problems. *Neuropsychol Rev*, 20(4), 398–413. <https://doi.org/10.1007/s11065-010-9146-6>

Benjamin, L., & Wulfert, E. (2005). Dispositional correlates of addictive behaviors in college women: Binge eating and heavy drinking. *Eating Behaviors*, 6(3), 197–209. <https://doi.org/10.1016/j.eatbeh.2003.08.001>



- Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2012). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *International Journal of Eating Disorders, 45*(3), 428–438. <https://doi.org/10.1002/eat.20931>
- Berridge, K. C. (2009). “Liking” and “wanting” food rewards: Brain substrates and roles in eating disorders. *Physiology and Behavior, 97*(5), 537–550. <https://doi.org/10.1016/j.physbeh.2009.02.044>
- Blanco-Gandía, M. C., Cantacorps, L., Aracil-Fernández, A., Montagud-Romero, S., Aguilar, M. A., Manzanares, J., Valverde, O., Miñarro, J., & Rodríguez-Arias, M. (2017). Effects of bingeing on fat during adolescence on the reinforcing effects of cocaine in adult male mice. *Neuropharmacology, 113*, 31–44. <https://doi.org/10.1016/j.neuropharm.2016.09.020>
- Blanco-Gandía, M. C., Ledesma, J. C., Aracil-Fernández, A., Navarrete, F., Montagud-Romero, S., Aguilar, M. A., Manzanares, J., Miñarro, J., & Rodríguez-Arias, M. (2017). The rewarding effects of ethanol are modulated by binge eating of a high-fat diet during adolescence. *Neuropharmacology, 121*, 219–230. <https://doi.org/10.1016/j.neuropharm.2017.04.040>
- Bø, R., Billieux, J., & Landrø, N. I. (2016). Which facets of impulsivity predict binge drinking? *Addictive Behaviors Reports, 3*, 43–47. <https://doi.org/10.1016/j.abrep.2016.03.001>
- Bonson, K. R., Grant, S. J., Contoreggi, C. S., Links, J. M., Metcalfe, J., Weyl, H. L., Kurian, V., Ernst, M., & London, E. D. (2002). Neural Systems and Cue-Induced Cocaine Craving. *Neuropsychopharmacology, 26*(3), 376–386. [https://doi.org/10.1016/S0893-133X\(01\)00371-2](https://doi.org/10.1016/S0893-133X(01)00371-2)
- Bouhlal, S., Farokhnia, M., Lee, M. R., Akhlaghi, F., & Leggio, L. (2018). Identifying and Characterizing Subpopulations of Heavy Alcohol Drinkers Via a Sucrose Preference Test: A Sweet Road to a Better Phenotypic Characterization? *Alcohol and Alcoholism, 53*(5), 560–569. <https://doi.org/10.1093/alcalc/agy048>
- Bruch, H. (1964). Psychological Aspects of Overeating And Obesity.

*Psychosomatics*, 5(5), 269–274. [https://doi.org/10.1016/S0033-3182\(64\)72385-7](https://doi.org/10.1016/S0033-3182(64)72385-7)

Brunault, P., Gaillard, P., Ballon, N., Couet, C., Isnard, P., Cook, S., Delbachian, I., Réveillère, C., & Courtois, R. (2016). Validation de la version française de la Binge Eating Scale : étude de sa structure factorielle, de sa consistance interne et de sa validité de construit en population clinique et non clinique. *Encephale*, 42(5), 426–433. <https://doi.org/10.1016/j.encep.2016.02.009>

Burton, P., J. Smit, H., & J. Lightowler, H. (2007). The influence of restrained and external eating patterns on overeating. *Appetite*, 49(1), 191–197. <https://doi.org/10.1016/j.appet.2007.01.007>

Camilleri, G. M., Méjean, C., Kesse-Guyot, E., Andreeva, V. A., Bellisle, F., Hercberg, S., & Péneau, S. (2014). The associations between emotional eating and consumption of energy-dense snack foods are modified by sex and depressive symptomatology. *Journal of Nutrition*, 144(8), 1264–1273. <https://doi.org/10.3945/jn.114.193177>

Carlson, S. R., Johnson, S. C., & Jacobs, P. C. (2010). Disinhibited characteristics and binge drinking among university student drinkers. *Addictive Behaviors*, 35(3), 242–251. <https://doi.org/10.1016/j.addbeh.2009.10.020>

Caton, S. J., Ball, M., Ahern, A., & Hetherington, M. M. (2004). Dose-dependent effects of alcohol on appetite and food intake. *Physiology and Behavior*, 81(1), 51–58. <https://doi.org/10.1016/j.physbeh.2003.12.017>

Caton, S. J., Bate, L., & Hetherington, M. M. (2007). Acute effects of an alcoholic drink on food intake: Aperitif versus co-ingestion. *Physiology and Behavior*, 90(2–3), 368–375. <https://doi.org/10.1016/j.physbeh.2006.09.028>

Caton, S. J., Marks, J. E., & Hetherington, M. M. (2005). Pleasure and alcohol: Manipulating pleasantness and the acute effects of alcohol on food intake. *Physiology and Behavior*, 84(3), 371–377. <https://doi.org/10.1016/j.physbeh.2004.12.013>

Caton, S. J., Nolan, L. J., & Hetherington, M. M. (2015). Alcohol, Appetite and Loss of Restraint. *Current Obesity Reports*, 4(1), 99–105.

<https://doi.org/10.1007/s13679-014-0130-y>

Cebolla, A., Barrada, J. R., van Strien, T., Oliver, E., & Baños, R. (2014). Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite*, *73*, 58–64. <https://doi.org/10.1016/j.appet.2013.10.014>

Cecchetto, C., Aiello, M., Gentili, C., Ionta, S., & Osimo, S. A. (2021). Increased emotional eating during COVID-19 associated with lockdown, psychological and social distress. *Appetite*, *160*, 105122.

<https://doi.org/10.1016/j.appet.2021.105122>

Center for Systems Science and Engineering. (2021). *COVID 19 Dashboard by the Center for Systems Science and Engineering (CSSE) at Johns Hopkins University of Medicine Coronavirus Resource Center.*

<https://coronavirus.jhu.edu/map.html>

Černelič-Bizjak, M., & Guiné, R. P. F. (2021). Predictors of binge eating: relevance of BMI, emotional eating and sensitivity to environmental food cues. *Nutrition & Food Science, ahead-of-p*(ahead-of-print). <https://doi.org/10.1108/NFS-02-2021-0062>

Clausen, L., Rosenvinge, J. H., Friborg, O., & Rokkedal, K. (2011). Validating the eating disorder inventory-3 (EDI-3): a comparison between 561 female eating disorders patients and 878 females from the general population. *Journal of Psychopathology and Behavioral Assessment*, *33*(1), 101–110.

<https://doi.org/10.1007/s10862-010-9207-4>

Cleveland, M. J., Feinberg, M. E., Bontempo, D. E., & Greenberg, M. T. (2008). The Role of Risk and Protective Factors in Substance Use Across Adolescence. *Journal of Adolescent Health*, *43*(2), 157–164.

<https://doi.org/10.1016/j.jadohealth.2008.01.015>

Cooper, S. J. (2005). Palatability-dependent appetite and benzodiazepines: New directions from the pharmacology of GABAA receptor subtypes. *Appetite*, *44*(2), 133–150. <https://doi.org/10.1016/j.appet.2005.01.003>

Costa, P. T., & McCrae, R. R. (1980). Influence of extraversion and neuroticism on subjective well-being: Happy and unhappy people. *Journal of Personality and*

*Social Psychology*, 38(4), 668–678. <https://doi.org/10.1037/0022-3514.38.4.668>

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests.

*Psychometrika*, 16(3), 297–334. <https://doi.org/10.1007/BF02310555>

Croteau, V., & Morrell, J. S. (2019). Prevalence of Binge Drinking Episodes Among Male and Female College Students (P18-008-19). *Current Developments in Nutrition*, 3(Supplement\_1), nzz039.P18-008-19.

<https://doi.org/10.1093/cdn/nzz039.P18-008-19>

Crowther, J. H., Sanftner, J., Bonifazi, D. Z., & Shepherd, K. L. (2001). The role of daily hassles in binge eating. *International Journal of Eating Disorders*, 29(4), 449–454. <https://doi.org/10.1002/eat.1041>

Cservenka, A., & Brumback, T. (2017). The burden of binge and heavy drinking on the brain: Effects on adolescent and young adult neural structure and function. *Frontiers in Psychology*, 8, 1111. <https://doi.org/10.3389/fpsyg.2017.01111>

Czepczor-Bernat, K., & Brytek-Matera, A. (2021). The impact of food-related behaviours and emotional functioning on body mass index in an adult sample. *Eating and Weight Disorders*, 26(1), 323–329. <https://doi.org/10.1007/S40519-020-00853-3>

Davis, C. A., Levitan, R. D., Reid, C., Carter, J. C., Kaplan, A. S., Patte, K. A., King, N., Curtis, C., & Kennedy, J. L. (2009). Dopamine for “Wanting” and Opioids for “Liking”: A Comparison of Obese Adults With and Without Binge Eating. *Obesity*, 17(6), 1220–1225. <https://doi.org/10.1038/oby.2009.52>

Davis, C., Mackew, L., Levitan, R. D., Kaplan, A. S., Carter, J. C., & Kennedy, J. L. (2017). Binge Eating Disorder (BED) in Relation to addictive behaviors and personality risk factors. *Frontiers in Psychology*, 8, 579.

<https://doi.org/10.3389/fpsyg.2017.00579>

Davis, R., & Jamieson, J. (2005). Assessing the functional nature of binge eating in the eating disorders. *Eating Behaviors*, 6(4), 345–354.

<https://doi.org/10.1016/j.eatbeh.2005.02.001>

- Dawson, D. A., Goldstein, R. B., Saha, T. D., & Grant, B. F. (2015). Changes in alcohol consumption: United States, 2001-2002 to 2012-2013. *Drug and Alcohol Dependence*, 148, 56–61. <https://doi.org/10.1016/j.drugalcdep.2014.12.016>
- De Castro, J. M. (2009). When, how much and what foods are eaten are related to total daily food intake. *British Journal of Nutrition*, 102(8), 1228–1237. <https://doi.org/10.1017/S0007114509371640>
- De Castro, J. M., & Orozco, S. (1990). Moderate alcohol intake and spontaneous eating patterns of humans: Evidence of unregulated supplementation. *American Journal of Clinical Nutrition*, 52(2), 246–253. <https://doi.org/10.1093/ajcn/52.2.246>
- Delegación del Gobierno para el Plan Nacional sobre Drogas. (2018). *Plan Nacional sobre Drogas. Encuesta sobre Uso de Drogas en Enseñanzas Secundarias en España (ESTUDES) 2018/2019*. [https://pnsd.sanidad.gob.es/profesionales/sistemasInformacion/sistemaInformacion/pdf/ESTUDES\\_2020\\_Informe.pdf](https://pnsd.sanidad.gob.es/profesionales/sistemasInformacion/sistemaInformacion/pdf/ESTUDES_2020_Informe.pdf)
- Di Renzo, L., Gualtieri, P., Pivari, F., Soldati, L., Attinà, A., Cinelli, G., Cinelli, G., Leggeri, C., Caparello, G., Barrea, L., Scerbo, F., Esposito, E., & De Lorenzo, A. (2020). Eating habits and lifestyle changes during COVID-19 lockdown: An Italian survey. *Journal of Translational Medicine*, 18(1), 229. <https://doi.org/10.1186/s12967-020-02399-5>
- Dingemans, A. E., Martijn, C., Jansen, A. T. M., & van Furth, E. F. (2009). The effect of suppressing negative emotions on eating behavior in binge eating disorder. *Appetite*, 52(1), 51–57. <https://doi.org/10.1016/j.appet.2008.08.004>
- Dobson, A. J., Blijlevens, R., Alexander, H. M., Croce, N., Heller, R. F., Higginbotham, N., Pike, G., Plotnikoff, R., Russell, A., & Walker, R. (1993). Short fat questionnaire: a self-administered measure of fat-intake behaviour. *Australian Journal of Public Health*, 17(2), 144–149. <https://doi.org/10.1111/j.1753-6405.1993.tb00123.x>
- dos Santos Quaresma, M. V., Marques, C. G., Magalhães, A. C. O., & dos Santos, R.

- V. T. (2021). Emotional eating, binge eating, physical inactivity, and vespertine chronotype are negative predictors of dietary practices during COVID-19 social isolation: A cross-sectional study. *Nutrition*, *90*, 111223.  
<https://doi.org/10.1016/j.nut.2021.111223>
- Drewnowski, A. (2007). The Real Contribution of Added Sugars and Fats to Obesity. *Epidemiologic Reviews*, *29*(1), 160–171. <https://doi.org/10.1093/epirev/mxm011>
- Duarte, C., Pinto-Gouveia, J., & Ferreira, C. (2015). Expanding binge eating assessment: Validity and screening value of the Binge Eating Scale in women from the general population. *Eating Behaviors*, *18*, 41–47.  
<https://doi.org/10.1016/j.eatbeh.2015.03.007>
- Duncan, A. E., Ziobrowski, H. N., & Nicol, G. (2017). The Prevalence of Past 12-Month and Lifetime DSM-IV Eating Disorders by BMI Category in US Men and Women. *European Eating Disorders Review*, *25*(3), 165–171.  
<https://doi.org/10.1002/erv.2503>
- Eisenberg, M. H., & Fitz, C. C. (2014). “Drunkorexia”: Exploring the who and why of a disturbing trend in college students’ eating and drinking behaviors. *Journal of American College Health*, *62*(8), 570–577.  
<https://doi.org/10.1080/07448481.2014.947991>
- Elosua, P., & López-Jáuregui, A. (2012). Internal structure of the spanish adaptation of the eating disorder inventory-3. *European Journal of Psychological Assessment*, *28*(1), 25–31. <https://doi.org/10.1027/1015-5759/a000087>
- Escrivá-Martínez, T., Galiana, L., Rodríguez-Arias, M., & Baños, R. M. (2019). The binge eating scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Frontiers in Psychology*, *10*, 530.  
<https://doi.org/10.3389/fpsyg.2019.00530>
- Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: a “transdiagnostic” theory and treatment. *Behaviour Research and Therapy*, *41*(5), 509–528. [https://doi.org/10.1016/S0005-7967\(02\)00088-8](https://doi.org/10.1016/S0005-7967(02)00088-8)

- Fazzino, T. L., Fleming, K., Sher, K. J., Sullivan, D. K., & Befort, C. (2017). Heavy Drinking in Young Adulthood Increases Risk of Transitioning to Obesity. *American Journal of Preventive Medicine*, *53*(2), 169–175.  
<https://doi.org/10.1016/j.amepre.2017.02.007>
- Ferriter, C., & Ray, L. A. (2011). Binge eating and binge drinking: An integrative review. *Eating Behaviors*, *12*(2), 99–107.  
<https://doi.org/10.1016/j.eatbeh.2011.01.001>
- Field, A. E., Sonneville, K. R., Micali, N., Crosby, R. D., Swanson, S. A., Laird, N. M., Treasure, J., Solmi, F., & Horton, N. J. (2012). Prospective association of common eating disorders and adverse outcomes. *Pediatrics*, *130*(2), e289-295.  
<https://doi.org/10.1542/peds.2011-3663>
- Fischer, S., Anderson, K. G., & Smith, G. T. (2004). Coping with distress by eating or drinking: Role of trait urgency and expectancies. *Psychology of Addictive Behaviors*, *18*(3), 269–274. <https://doi.org/10.1037/0893-164X.18.3.269>
- Fleming-Milici, F., & Harris, J. L. (2020). Adolescents' engagement with unhealthy food and beverage brands on social media. *Appetite*, *146*, 104501.  
<https://doi.org/10.1016/j.appet.2019.104501>
- Fletcher, P. C., & Kenny, P. J. (2018). Food addiction: a valid concept? *Neuropsychopharmacology*, *43*(13), 2506–2513. <https://doi.org/10.1038/s41386-018-0203-9>
- Flint, A. J., Gearhardt, A. N., Corbin, W. R., Brownell, K. D., Field, A. E., & Rimm, E. B. (2014). Food-addiction scale measurement in 2 cohorts of middleaged and older women. *99*(3), 578–586. <https://doi.org/10.3945/ajcn.113.068965>
- Fouladi, F., Mitchell, J. E., Crosby, R. D., Engel, S. G., Crow, S., Hill, L., Le Grange, D., Powers, P., & Steffen, K. J. (2015). Prevalence of Alcohol and Other Substance Use in Patients with Eating Disorders. *European Eating Disorders Review*, *23*(6), 531–536. <https://doi.org/10.1002/erv.2410>
- Freitas, S., Lopes, C. S., Appolinario, J. C., & Coutinho, W. (2006). The assessment of binge eating disorder in obese women: A comparison of the binge eating

scale with the structured clinical interview for the DSM-IV. *Eating Behaviors*, 7(3), 282–289. <https://doi.org/10.1016/J.EATBEH.2005.09.002>

Galán, I., González, M. J., & Valencia-Martín, J. L. (2014). Alcohol drinking patterns in Spain: a country in transition. *Revista Española de Salud Pública*, 88(4), 529–540. [http://www.scielosp.org/scielo.php?script=sci\\_arttext&pid=S1135-57272014000400007&lang=pt%0Ahttp://www.scielosp.org/pdf/resp/v88n4/07\\_original1.pdf](http://www.scielosp.org/scielo.php?script=sci_arttext&pid=S1135-57272014000400007&lang=pt%0Ahttp://www.scielosp.org/pdf/resp/v88n4/07_original1.pdf)

Galicia-Paredes, M. Á., Alonso, J. R., & Nogué Xarau, S. (2017). Intoxicaciones por drogas de abuso: sustancias emergentes en el siglo XXI. *Emergencias*, 26, 472–480. <http://diposit.ub.edu/dspace/handle/2445/109364>

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2016). Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*, 30(1), 113–121. <https://doi.org/10.1037/adb0000136>

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009a). Food addiction: An examination of the diagnostic criteria for dependence. *Journal of Addiction Medicine*, 3(1), 1–7. <https://doi.org/10.1097/ADM.0b013e318193c993>

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009b). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, 52(2), 430–436. <https://doi.org/10.1016/j.appet.2008.12.003>

Gearhardt, A. N., Davis, C., Kushner, R., & Brownell, K. D. (2011). The Addiction Potential of Hyperpalatable Foods. *Current Drug Abuse Reviewse*, 4(3), 140–145. <https://doi.org/10.2174/1874473711104030140>

Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An examination of the food addiction construct in obese patients with binge eating disorder. *International Journal of Eating Disorders*, 45(5), 657–663. <https://doi.org/10.1002/eat.20957>

Gearhardt, A. N., White, M. A., & Potenza, M. N. (2011). Binge eating disorder and food addiction. *Current Drug Abuse Reviews*, 4(3), 201–207. <https://doi.org/10.2174/1874473711104030201>



- Goldschmidt, A. B., Wall, M. M., Loth, K. A., Bucchianeri, M. M., & Neumark-Sztainer, D. (2014). The course of binge eating from adolescence to young adulthood. *Health Psychology, 33*(5), 457–460. <https://doi.org/10.1037/a0033508>
- Gordon, K. H., Holm-Denoma, J. M., Troop-Gordon, W., & Sand, E. (2012). Rumination and body dissatisfaction interact to predict concurrent binge eating. *Body Image, 9*(3), 352–357. <https://doi.org/10.1016/j.bodyim.2012.04.001>
- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*(1), 47–55. [https://doi.org/10.1016/0306-4603\(82\)90024-7](https://doi.org/10.1016/0306-4603(82)90024-7)
- Grant, B. F., Stinson, F. S., & Harford, T. C. (2001). Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: A 12-year follow-up. *Journal of Substance Abuse, 13*(4), 493–504. [https://doi.org/10.1016/S0899-3289\(01\)00096-7](https://doi.org/10.1016/S0899-3289(01)00096-7)
- Grupski, A. E., Hood, M. M., Hall, B. J., Azarbad, L., Fitzpatrick, S. L., & Corsica, J. A. (2013). Examining the Binge Eating Scale in Screening for Binge Eating Disorder in Bariatric Surgery Candidates. *Obesity Surgery, 23*(1), 1–6. <https://doi.org/10.1007/s11695-011-0537-4>
- Guitart, A. M., Espelt, A., Castellano, Y., Bartroli, M., Villalbí, J. R., Domingo-Salvany, A., & Brugal, M. T. (2011). Impacto del trastorno por consumo de alcohol en la mortalidad: ¿hay diferencias según la edad y el sexo? *Gaceta Sanitaria, 25*(5), 385–390. <https://doi.org/10.1016/j.gaceta.2011.03.019>
- Hadad, N. A., & Knackstedt, L. A. (2014). Addicted to palatable foods: Comparing the neurobiology of Bulimia Nervosa to that of drug addiction. *Psychopharmacology, 231*(9), 1897–1912. <https://doi.org/10.1007/s00213-014-3461-1>
- Haddad, C., Zakhour, M., Siddik, G., Haddad, R., Sacre, H., & Salameh, P. (2021). COVID- 19 outbreak: Does confinement have any impact on weight change perception? *Nutrition Clinique et Métabolisme, 35*(2), 137–143. <https://doi.org/10.1016/j.nupar.2021.02.003>
- Halmi, K. A. (2001). Physiology of anorexia and bulimia nervosa. In C. G. Fairburn &

- K. D. Brownell (Eds.), *Eating disorders and obesity* (pp. 267–271). Guilford Press.
- Harris, J. L., & Fleming-Milici, F. (2019). Food marketing to adolescents and young adults : Skeptical but still under the influence. *The Psychology of Food Marketing and (Over)Eating*, 25–43. <https://doi.org/10.4324/9780429274404-3>
- Harrop, E. N., & Marlatt, G. A. (2010). The comorbidity of substance use disorders and eating disorders in women: Prevalence, etiology, and treatment. *Addictive Behaviors*, 35(5), 392–398. <https://doi.org/10.1016/j.addbeh.2009.12.016>
- Hauck, C., Weiß, A., Schulte, E. ., Meule, A., & Ellrott, T. (2017). Prevalence of “Food Addiction” as Measured with the Yale Food Addiction Scale 2.0 in a Representative German Sample and Its Association with Sex, Age and Weight Categories. *Obesity Facts*, 10(1), 12–24. <https://doi.org/10.1159/000456013>
- Hemmingsson, E. (2014). A new model of the role of psychological and emotional distress in promoting obesity: conceptual review with implications for treatment and prevention. *Obesity Reviews*, 15(9), 769–779. <https://doi.org/10.1111/obr.12197>
- Herrero, J., & Meneses, J. (2006). Short Web-based versions of the perceived stress (PSS) and Center for Epidemiological Studies-Depression (CESD) Scales: A comparison to pencil and paper responses among Internet users. *Computers in Human Behavior*, 22(5), 830–846. <https://doi.org/10.1016/j.chb.2004.03.007>
- Hood, M. M., Grupski, A. E., Hall, B. J., Ivan, I., & Corsica, J. (2013). Factor structure and predictive utility of the Binge Eating Scale in bariatric surgery candidates. *Surgery for Obesity and Related Diseases*, 9(6), 942–948. <https://doi.org/10.1016/j.soard.2012.06.013>
- Hou, F., Xu, S., Zhao, Y., Lu, Q., Zhang, S., Zu, P., Sun, Y., Su, P., & Tao, F. (2013). Effects of emotional symptoms and life stress on eating behaviors among adolescents. *Appetite*, 68, 63–68. <https://doi.org/10.1016/j.appet.2013.04.010>
- Hudson, J. I., Hiripi, E., Pope, H. G., Kessler, R. C., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey

- Replication. *Biological Psychiatry*, 61(3), 348–358.  
<https://doi.org/10.1016/j.biopsych.2006.03.040>
- Hunt, T. K., & Forbush, K. T. (2016). Is “drunkorexia” an eating disorder, substance use disorder, or both? *Eating Behaviors*, 22, 40–45.  
<https://doi.org/10.1016/j.eatbeh.2016.03.034>
- Inchley, J., Currie, D., Vieno, A., Torsheim, T., Ferreira-Borges, C., Weber, M. M., Barnekow, V., & Breda, J. (2018). *Adolescent alcohol-related behaviours: trends and inequalities in the WHO European Region, 2002-2014*. WHO Regional Office for Europe. <https://research-repository.st-andrews.ac.uk/handle/10023/18833>
- Isorna, M., Fariña, F., Sierra, J. C., & Vallejo-Medina, P. (2015). Binge drinking: Risky sexual behaviors and drug facilitated sexual assault in spanish youths. *Suma Psicológica*, 22(1), 1–8. <https://doi.org/10.1016/j.sumpsi.2015.05.001>
- Jin-Yi, J., Kye-Hyun, K., Hee-Yeon, W., Dong-Won, S., Young-Chul, S., Kang-Seob, O., Eun-Hee, S., & Se-Won, L. (2017). Binge eating is associated with trait anxiety in Korean adolescent girls: a cross sectional study. *BMC Women’s Health*, 17(1), 8. <https://doi.org/10.1186/s12905-017-0364-4>
- Jones, S. A., Lueras, J. M., & Nagel, B. J. (2018). Effects of Binge Drinking on the Developing Brain. *Alcohol Research: Current Reviews*, 39(1), 87–96.  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6104956/>
- Kakoschke, N., Kemps, E., & Tiggemann, M. (2015). External eating mediates the relationship between impulsivity and unhealthy food intake. *Physiology and Behavior*, 147, 117–121. <https://doi.org/10.1016/j.physbeh.2015.04.030>
- Kalkan-Uğurlu, Y., Mataracı-Değirmenci, D., Durgun, H., & Gök-Uğur, H. (2021). The examination of the relationship between nursing students’ depression, anxiety and stress levels and restrictive, emotional, and external eating behaviors in COVID-19 social isolation process. *Perspectives in Psychiatric Care*, 57(2), 507–516. <https://doi.org/10.1111/ppc.12703>
- Kampov-Polevoy, A., Lange, L., Bobashev, G., Eggleston, B., Root, T., & Garbutt, J.

- C. (2014). Sweet-Liking Is Associated with Transformation of Heavy Drinking into Alcohol-Related Problems in Young Adults with High Novelty Seeking. *Alcoholism: Clinical and Experimental Research*, 38(7), 2119–2126.  
<https://doi.org/10.1111/acer.12458>
- Kanny, D., Naimi, T. S., Liu, Y., Lu, H., & Brewer, R. D. (2018). Annual Total Binge Drinks Consumed by U.S. Adults, 2015. *American Journal of Preventive Medicine*, 54(4), 486–496. <https://doi.org/10.1016/j.amepre.2017.12.021>
- Kelly-Weeder, S. (2011). Binge drinking and disordered eating in college students. *Journal of the American Academy of Nurse Practitioners*, 23(1), 33–41.  
<https://doi.org/10.1111/j.1745-7599.2010.00568.x>
- Kenney, S. R., Lac, A., Labrie, J. W., Hummer, J. F., & Pham, A. (2013). Mental health, sleep quality, drinking motives, and alcohol-related consequences: A path-analytic model. *Journal of Studies on Alcohol and Drugs*, 74(6), 841–851.  
<https://doi.org/10.15288/jsad.2013.74.841>
- Keski-Rahkonen, A., & Mustelin, L. (2016). Epidemiology of eating disorders in Europe: Prevalence, incidence, comorbidity, course, consequences, and risk factors. *Current Opinion in Psychiatry*, 29(6), 340–345.  
<https://doi.org/10.1097/YCO.0000000000000278>
- Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M. C., Benjet, C., Bruffaerts, R., de Girolamo, G., De Graaf, R., Maria Haro, J., Kovess-Masfety, V., O'Neill, S., Posada-Villa, J., Sasu, C., Scott, K., ... Xavier, M. (2013). The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry*, 73(9), 904–914.  
<https://doi.org/10.1016/j.biopsych.2012.11.020>
- Kim, Y. R., Hwang, B. I., Lee, G. Y., Kim, K. H., Kim, M., Kim, K. K., & Treasure, J. (2018). Determinants of binge eating disorder among normal weight and overweight female college students in Korea. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, 23(6), 849–860.  
<https://doi.org/10.1007/s40519-018-0574-2>

- Kleinman, N., Abouzaid, S., Andersen, L., Wang, Z., & Powers, A. (2014). Cohort Analysis Assessing Medical and Nonmedical Cost Associated With Obesity in the Workplace. *Journal of Occupational & Environmental Medicine*, 56(2), 161–170. <https://doi.org/10.1097/JOM.0000000000000099>
- Kline, R. B. (2015). *Principles and practices of structural equation modeling*. Guilford Publications.
- Kuntsche, E., Gmel, G., Wicki, M., Rehm, J., & Grichting, E. (2006). Disentangling gender and age effects on risky single occasion drinking during adolescence. *European Journal of Public Health*, 16(6), 670–675. <https://doi.org/10.1093/eurpub/ckl060>
- Kuntsche, E., Kuntsche, S., Thrul, J., & Gmel, G. (2017). Binge drinking: Health impact, prevalence, correlates and interventions. *Psychology & Health*, 32(8), 976–1017. <https://doi.org/10.1080/08870446.2017.1325889>
- Kuntsche, E., von Fischer, M., & Gmel, G. (2008). Personality factors and alcohol use: A mediator analysis of drinking motives. *Personality and Individual Differences*, 45(8), 796–800. <https://doi.org/10.1016/j.paid.2008.08.009>
- Laghi, F., Baiocco, R., Liga, F., Lonigro, A., & Baumgartner, E. (2014). Binge eating and binge drinking behaviors: Individual differences in adolescents' identity styles. *Journal of Health Psychology*, 19(3), 333–343. <https://doi.org/10.1177/1359105312470851>
- Laghi, F., D'Alessio, M., Pallini, S., & Baiocco, R. (2009). Attachment representations and time perspective in adolescence. *Social Indicators Research*, 90(2), 181–194. <https://doi.org/10.1007/s11205-008-9249-0>
- Laghi, F., Liga, F., Baumgartner, E., & Baiocco, R. (2012). Time perspective and psychosocial positive functioning among Italian adolescents who binge eat and drink. *Journal of Adolescence*, 35(5), 1277–1284. <https://doi.org/10.1016/j.adolescence.2012.04.014>
- Laghi, F., Pompili, S., Baumgartner, E., & Baiocco, R. (2015). The role of sensation seeking and motivations for eating in female and male adolescents who binge

eat. *Eating Behaviors*, 17, 119–124.  
<https://doi.org/10.1016/j.eatbeh.2015.01.011>

Lai, C. M., Mak, K. K., Pang, J. S., Fong, S. S. M., Ho, R. C. M., & Guldán, G. S. (2013). The associations of sociocultural attitudes towards appearance with body dissatisfaction and eating behaviors in Hong Kong adolescents. *Eating Behaviors*, 14(3), 320–324. <https://doi.org/10.1016/j.eatbeh.2013.05.004>

Lamis, D. A., Malone, P. S., Langhinrichsen-Rohling, J., & Ellis, T. E. (2010). Body investment, depression, and alcohol use as risk factors for suicide proneness in college students. *Crisis*, 31(3), 118–127. <https://doi.org/10.1027/0227-5910/a000012>

Langdon-Daly, J., & Serpell, L. (2017). Protective factors against disordered eating in family systems: a systematic review of research. *Journal of Eating Disorders*, 5(1), 12. <https://doi.org/10.1186/s40337-017-0141-7>

Lemeshow, A. R., Gearhardt, A. N., Genkinger, J. M., & Corbin, W. R. (2016). Assessing the psychometric properties of two food addiction scales. *Eating Behaviors*, 23, 110–114. <https://doi.org/10.1016/J.EATBEH.2016.08.005>

Li, K. K., Concepcion, R. Y., Lee, H., Cardinal, B. J., Ebbeck, V., Woekel, E., & Readdy, R. T. (2012). An Examination of Sex Differences in Relation to the Eating Habits and Nutrient Intakes of University Students. *Journal of Nutrition Education and Behavior*, 44(3), 246–250.  
<https://doi.org/10.1016/j.jneb.2010.10.002>

Liebman, M., Cameron, B. A., Carson, D. K., Brown, D. M., & Meyer, S. S. (2001). Dietary fat reduction behaviors in college students: Relationship to dieting status, gender and key psychosocial variables. *Appetite*, 36(1), 51–56.  
<https://doi.org/10.1006/appe.2000.0383>

Linardon, J., & Messer, M. (2019). Assessment of food addiction using the Yale Food Addiction Scale 2.0 in individuals with binge-eating disorder symptomatology: Factor structure, psychometric properties, and clinical significance. *Psychiatry Research*, 279, 216–221. <https://doi.org/10.1016/j.psychres.2019.03.003>

- Lipson, S. K., & Sonnevile, K. R. (2017). Eating disorder symptoms among undergraduate and graduate students at 12 U.S. colleges and universities. *Eating Behaviors, 24*, 81–88. <https://doi.org/10.1016/j.eatbeh.2016.12.003>
- Loxton, N. J., & Dawe, S. (2007). How do dysfunctional eating and hazardous drinking women perform on behavioural measures of reward and punishment sensitivity? *Personality and Individual Differences, 42*(6), 1163–1172. <https://doi.org/10.1016/j.paid.2006.09.031>
- Malesza, M., & Kaczmarek, M. C. (2021). One year reliability of the Dutch eating behavior questionnaire: an extension into clinical population. *Journal of Public Health: From Theory to Practice, 29*, 463–469. <https://doi.org/10.1007/s10389-019-01147-4>
- Mason, T. B., & Lewis, R. J. (2014). Profiles of Binge Eating: The Interaction of Depressive Symptoms, Eating Styles, and Body Mass Index. *Eating Disorders, 22*(5), 450–460. <https://doi.org/10.1080/10640266.2014.931766>
- Mason, T. B., Smith, K. E., Lavender, J. M., & Lewis, R. J. (2018). Independent and interactive associations of negative affect, restraint, and impulsivity in relation to binge eating among women. *Appetite, 121*, 147–153. <https://doi.org/10.1016/j.appet.2017.11.099>
- Maurage, P., Lannoy, S., Mange, J., Grynberg, D., Beaunieux, H., Banovic, I., Gierski, F., & Naassila, M. (2020). What we talk about when we talk about binge drinking: Towards an integrated conceptualization and evaluation. In *Alcohol and Alcoholism* (Vol. 55, Issue 5, pp. 468–479). Oxford University Press. <https://doi.org/10.1093/alcalc/agaa041>
- McDonald, R. P. (1999). *Test theory: A unified treatment*. Psychology Press.
- Measelle, J. R., Stice, E., & Hogansen, J. M. (2006). Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *Journal of Abnormal Psychology, 115*(3), 524–538. <https://doi.org/10.1037/0021-843X.115.3.524>
- Mehlig, K., Bogl, L. H., Hunsberger, M., Ahrens, W., De Henauw, S., Iguacel, I.,

- Jilani, H., Molnár, D., Pala, V., Russo, P., Tornaritis, M., Veidebaum, T., Kaprio, J., & Lissner, L. (2018). Children's propensity to consume sugar and fat predicts regular alcohol consumption in adolescence. *Public Health Nutrition*, *21*(17), 3202–3209. <https://doi.org/10.1017/S1368980018001829>
- Meiselman, H. L., Mastroianni, G., Buller, M., & Edwards, J. (1998). Longitudinal measurement of three eating behavior scales during a period of change. *Food Quality and Preference*, *10*(1), 1–8. [https://doi.org/10.1016/S0950-3293\(98\)00013-5](https://doi.org/10.1016/S0950-3293(98)00013-5)
- Melis, M., Diana, M., Enrico, P., Marinelli, M., & Brodie, M. S. (2009). Ethanol and acetaldehyde action on central dopamine systems: mechanisms, modulation, and relationship to stress. *Alcohol*, *43*(7), 531–539. <https://doi.org/10.1016/j.alcohol.2009.05.004>
- Meule, A. (2013). Impulsivity and overeating: A closer look at the subscales of the Barratt Impulsiveness Scale. *Frontiers in Psychology*, *4*, 177. <https://doi.org/10.3389/fpsyg.2013.00177>
- Meule, A. (2014). Are Certain Foods Addictive? *Frontiers in Psychiatry*, *5*. <https://doi.org/10.3389/fpsyt.2014.00038>
- Meule, A., von Rezori, V., & Blechert, J. (2014). Food addiction and bulimia nervosa. *European Eating Disorders Review*, *22*(5), 331–337. <https://doi.org/10.1002/ERV.2306>
- Micali, N., Solmi, F., Horton, N. J., Crosby, R. D., Eddy, K. T., Calzo, J. P., Sonnevile, K. R., Swanson, S. A., & Field, A. E. (2015). Adolescent Eating Disorders Predict Psychiatric, High-Risk Behaviors and Weight Outcomes in Young Adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *54*(8), 652–659. <https://doi.org/10.1016/j.jaac.2015.05.009>
- Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric Aspects of Impulsivity. *American Journal of Psychiatry*, *158*(11), 1783–1793. <https://doi.org/10.1176/appi.ajp.158.11.1783>
- Moral, M. V., & Ovejero, A. (2011). Consumo abusivo de alcohol en adolescentes



españoles: tendencias emergentes y percepciones de riesgo. *Universitas Psychologica*, 10(1), 71–87.

[http://www.scielo.org.co/scielo.php?script=sci\\_arttext&pid=S1657-92672011000100007&lng=en&nrm=iso&tlng=es](http://www.scielo.org.co/scielo.php?script=sci_arttext&pid=S1657-92672011000100007&lng=en&nrm=iso&tlng=es)

- Murphy, C. M., Stojek, M. K., & MacKillop, J. (2014). Interrelationships among impulsive personality traits, food addiction, and Body Mass Index. *Appetite*, 73, 45–50. <https://doi.org/10.1016/J.APPET.2013.10.008>
- Naish, K. R., Laliberte, M., MacKillop, J., & Balodis, I. M. (2018). Systematic review of the effects of acute stress in binge eating disorder. *European Journal of Neuroscience*, 50, 2415–2429. <https://doi.org/10.1111/ejn.14110>
- Navarro-Cruz, A. R., Vera, O., Munguia, P., Sosa-Sánchez, R. Á., Lazcano, M., Ochoa, C., & Hernández, P. (2017). Hábitos alimentarios en una población de jóvenes universitario (18-25 años) de la ciudad de Puebla. *Rev. Esp. Nutr. Comunitaria*, 23, 31–37. <https://doi.org/10.14642/RENC.2017.23.SUP2.5176>
- Nelson, M. C., Lust, K., Story, M., & Ehlinger, E. (2009). Alcohol use, eating patterns, and weight behaviors in a university population. *American Journal of Health Behavior*, 33(3), 227–237. <https://doi.org/10.5993/AJHB.33.3.1>
- Niedhammer, I., Bugel, I., Bonenfant, S., Goldberg, M., & Leclerc, A. (2000). Validity of self-reported weight and height in the French GAZEL cohort. *International Journal of Obesity*, 24(9), 1111–1118. <https://doi.org/10.1038/sj.ijo.0801375>
- Okumus, B., & Ozturk, A. B. (2021). The impact of perceived stress on US millennials' external and emotional eating behavior. *British Food Journal*, 123(1), 1–11. <https://doi.org/10.1108/BFJ-07-2019-0490/FULL/PDF>
- Orozco-Cabal, L., Rodríguez, M., Herin, D. V., Gempeler, J., & Uribe, M. (2010). Validity and Reliability of the Abbreviated Barratt Impulsiveness Scale in Spanish (BIS-15S)\*. *Revista Colombiana de Psiquiatría*, 39(1), 93–109. <https://pubmed.ncbi.nlm.nih.gov/21152412/>
- Paul, L. A., Grubaugh, A. L., Frueh, B. C., Ellis, C., & Egede, L. E. (2011). Associations between binge and heavy drinking and health behaviors in a

- nationally representative sample. *Addictive Behaviors*, 36(12), 1240–1245.  
<https://doi.org/10.1016/j.addbeh.2011.07.034>
- Pearlstein, T. (2002). Eating disorders and comorbidity. *Archives of Women's Mental Health*, 4(3), 67–78. <https://doi.org/10.1007/s007370200002>
- Pellegrini, M., Ponzo, V., Rosato, R., Scumaci, E., Goitre, I., Benso, A., Belcastro, S., Crespi, C., De Michieli, F., Ghigo, E., Broglio, F., & Bo, S. (2020). Changes in Weight and Nutritional Habits in Adults with Obesity during the “Lockdown” Period Caused by the COVID-19 Virus Emergency. *Nutrients*, 12(7), 2016. <https://doi.org/10.3390/NU12072016>
- Pepino, M., Stein, R., Eagon, J., Klein, S., Berenson, A. B., Laz, T. H., Pohlmeier, A. M., Rahman, M., & Cunningham, K. A. (2014). Bariatric surgery-induced weight loss causes remission of food addiction in extreme obesity. *Obesity*, 22(8), 1792–1798. <https://doi.org/10.1002/OBY.20797>
- Peveler, R., & Fairburn, C. (1990). Eating disorders in women who abuse alcohol. *Addiction*, 85(12), 1633–1638. <https://doi.org/10.1111/j.1360-0443.1990.tb01653.x>
- Phillipou, A., Meyer, D., Neill, E., Tan, E. J., Toh, W. L., Van Rheenen, T. E., & Rossell, S. L. (2020). Eating and exercise behaviors in eating disorders and the general population during the COVID-19 pandemic in Australia: Initial results from the COLLATE project. *International Journal of Eating Disorders*, 53(7), 1158–1165. <https://doi.org/10.1002/EAT.23317>
- Phillips, K. E., Kelly-Weeder, S., & Farrell, K. (2016). Binge eating behavior in college students: What is a binge? *Applied Nursing Research*, 30, 7–11. <https://doi.org/10.1016/j.apnr.2015.10.011>
- Pompili, S., & Laghi, F. (2017). Binge eating and binge drinking among adolescents: The role of drinking and eating motives. *Journal of Health Psychology*. <https://doi.org/10.1177/1359105317713359>
- Potenza, M. N., & Grilo, C. M. (2014). How Relevant is Food Craving to Obesity and Its Treatment? *Frontiers in Psychiatry*, 5, 164.

<https://doi.org/10.3389/fpsy.2014.00164>

Preti, A., de Girolamo, G., Vilagut, G., Alonso, J., de Graaf, R., Bruffaerts, R., Demyttenaere, K., Pinto-Meza, A., Haro, J. M., & Morosini, P. (2009). The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research, 43*(14), 1125–1132. <https://doi.org/10.1016/J.JPSYCHIRES.2009.04.003>

Puccio, F., Fuller-Tyszkiewicz, M., Ong, D., & Krug, I. (2016). A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *International Journal of Eating Disorders, 49*(5), 439–454. <https://doi.org/10.1002/eat.22506>

Pursey, K. M., Stanwell, P., Gearhardt, A. N., Collins, C. E., & Burrows, T. L. (2014). The prevalence of food addiction as assessed by the yale food addiction scale: A systematic review. *Nutrients, 6*(10), 4552–4590. <https://doi.org/10.3390/nu6104552>

Raben, A., Agerholm-Larsen, L., Flint, A., Holst, J. J., & Astrup, A. (2003). Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *American Journal of Clinical Nutrition, 77*(1), 91–100. <https://doi.org/10.1093/ajcn/77.1.91>

Racine, S. E., Burt, S. A., Iacono, W. G., McGue, M., & Klump, K. L. (2011). Dietary restraint moderates genetic risk for binge eating. *Journal of Abnormal Psychology, 120*(1), 119–128. <https://doi.org/10.1037/A0020895>

Raevuori, A., Suokas, J., Haukka, J., Gissler, M., Linna, M., Grainger, M., & Suvisaari, J. (2015). Highly increased risk of type 2 diabetes in patients with binge eating disorder and bulimia nervosa. *International Journal of Eating Disorders, 48*(6), 555–562. <https://doi.org/10.1002/eat.22334>

Rapuano, K. M., Zieselman, A. L., Kelley, W. M., Sargent, J. D., Heatherton, T. F., & Gilbert-Diamond, D. (2017). Genetic risk for obesity predicts nucleus accumbens size and responsivity to real-world food cues. *Proceedings of the National*

*Academy of Sciences of the United States of America*, 114(1), 160–165.  
<https://doi.org/10.1073/PNAS.1605548113>

Ribeiro-Silva, R. de C., Fiaccone, R. L., Conceição-Machado, M. E. P. da, Ruiz, A. S., Barreto, M. L., & Santana, M. L. P. (2018). Body image dissatisfaction and dietary patterns according to nutritional status in adolescents. *Jornal de Pediatria*, 94(2), 155–161. <https://doi.org/10.1016/j.jped.2017.05.005>

Ricca, V., Castellini, G., Lo Sauro, C., Ravaldi, C., Lapi, F., Mannucci, E., Rotella, C. M., & Faravelli, C. (2009). Correlations between binge eating and emotional eating in a sample of overweight subjects. *Appetite*, 53(3), 418–421.  
<https://doi.org/10.1016/j.appet.2009.07.008>

Ricca, V., Mannucci, E., Moretti, S., Di Bernardo, M., Zucchi, T., Cabras, P. L., & Rotella, C. M. (2000). Screening for binge eating disorder in obese outpatients. *Comprehensive Psychiatry*, 41(2), 111–115. [https://doi.org/10.1016/S0010-440X\(00\)90143-3](https://doi.org/10.1016/S0010-440X(00)90143-3)

Rippe, J. M., & Marcos, A. (2016). Controversies about sugars consumption: state of the science. *European Journal of Nutrition*, 55, 11–16.  
<https://doi.org/10.1007/s00394-016-1227-8>

Robinson, M., Burghardt, P., Patterson, C., Nobile, C., Akil, H., Watson, S., Berridge, K., & Ferrario, C. (2015). Individual Differences in Cue-Induced Motivation and Striatal Systems in Rats Susceptible to Diet-Induced Obesity. *Neuropsychopharmacology*, 40(9), 2113–2123.  
<https://doi.org/10.1038/NPP.2015.71>

Rodríguez-Pérez, C., Molina-Montes, E., Verardo, V., Artacho, R., García-Villanova, B., Guerra-Hernández, E. J., & Ruíz-López, M. D. (2020). Changes in Dietary Behaviours during the COVID-19 Outbreak Confinement in the Spanish COVIDiet Study. *Nutrients*, 12(6), 1730. <https://doi.org/10.3390/NU12061730>

Röjdmarm, S., Calissendorff, J., & Brismar, K. (2001). Alcohol ingestion decreases both diurnal and nocturnal secretion of leptin in healthy individuals. *Clinical Endocrinology*, 55(5), 639–647. <https://doi.org/10.1046/j.1365->

2265.2001.01401.x

- Rolls, B. J., Roe, L. S., & Meengs, J. S. (2007). The effect of large portion sizes on energy intake is sustained for 11 days. *Obesity*, *15*(6), 1535–1543.  
<https://doi.org/10.1038/oby.2007.182>
- Rosen, E. M., Primeaux, S. D., Simon, L., Welsh, D. A., Molina, P. E., & Ferguson, T. F. (2021). Associations of Binge Drinking and Heavy Alcohol Use on Sugar and Fat Intake in a Cohort of Southern People Living with HIV. *Alcohol and Alcoholism*, 1–9. <https://doi.org/10.1093/alcalc/agab066>
- Rostanzo, E., & Aloisi, A. M. (2021). Food addiction assessment in a nonclinical sample of the Italian population. *European Journal of Clinical Nutrition*.  
<https://doi.org/10.1038/S41430-021-00974-7>
- Rothman, R. B., Blough, B. E., & Baumann, M. H. (2008). Dual dopamine/serotonin releasers: Potential treatment agents for stimulant addiction. *Experimental and Clinical Psychopharmacology*, *16*(6), 458–474. <https://doi.org/10.1037/a0014103>
- Ruddock, H. K., Field, M., & Hardman, C. A. (2017). Exploring food reward and calorie intake in self-perceived food addicts. *Appetite*, *115*, 36–44.  
<https://doi.org/10.1016/j.appet.2016.12.003>
- Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*(2), 247–262. <https://doi.org/10.1037/0033-2909.99.2.247>
- Schiestl, E. T., & Gearhardt, A. N. (2018). Preliminary validation of the Yale Food Addiction Scale for Children 2.0: A dimensional approach to scoring. *European Eating Disorders Review*, *26*(6), 605–617. <https://doi.org/10.1002/ERV.2648>
- Schneider, F., Habel, U., Wagner, M., Franke, P., Salloum, J. B., Shah, N. J., Toni, I., Sulzbach, C., Hönig, K., Maier, W., Gaebel, W., & Zilles, K. (2001). Subcortical correlates of craving in recently abstinent alcoholic patients. *American Journal of Psychiatry*, *158*(7), 1075–1083.  
<https://doi.org/10.1176/APPI.AJP.158.7.1075/ASSET/IMAGES/LARGE/J413F4.JPEG>

- Schulte, E. M., Avena, N. M., & Gearhardt, A. N. (2015). Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PLoS ONE*, *10*(2), 1–18. <https://doi.org/10.1371/journal.pone.0117959>
- Schulte, E. M., & Gearhardt, A. N. (2017). Development of the Modified Yale Food Addiction Scale Version 2.0. *European Eating Disorders Review*, *25*(4), 302–308. <https://doi.org/10.1002/erv.2515>
- Schulte, E. M., Grilo, C. M., & Gearhardt, A. N. (2016). Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clinical Psychology Review*, *44*, 125–139. <https://doi.org/10.1016/j.cpr.2016.02.001>
- Schulte, E. M., Smeal, J. K., Lewis, J., & Gearhardt, A. N. (2018). Development of the Highly Processed Food Withdrawal Scale. *Appetite*, *131*, 148–154. <https://doi.org/10.1016/j.appet.2018.09.013>
- Selye, H. (1956). *The stress of life*. McGraw-Hill. <https://psycnet.apa.org/record/1957-08247-000>
- Sidor, A., & Rzymiski, P. (2020). Dietary Choices and Habits during COVID-19 Lockdown: Experience from Poland. *Nutrients*, *12*(6), 1657. <https://doi.org/10.3390/nu12061657>
- Simonnet, A., Chetboun, M., Poissy, J., Raverdy, V., Noulette, J., Duhamel, A., Labreuche, J., Mathieu, D., Pattou, F., Jourdain, M., Caizzo, R., Caplan, M., Cousin, N., Duburcq, T., Durand, A., El kalioubie, A., Favory, R., Garcia, B., Girardie, P., ... Verkindt, H. (2020). High Prevalence of Obesity in Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) Requiring Invasive Mechanical Ventilation. *Obesity*, *28*(7), 1195–1199. <https://doi.org/10.1002/oby.22831>
- Sinha, R. (2001). How does stress increase risk of drug abuse and relapse? *Psychopharmacology*, *158*(4), 343–359. <https://doi.org/10.1007/s002130100917>
- Sinha, R. (2008). Chronic Stress, Drug Use, and Vulnerability to Addiction. *Annals of the New York Academy of Sciences*, *1141*, 105–130. <https://doi.org/10.1016/j.cortex.2009.08.003>. Predictive

- Sinisterra-Loaiza, L. I., Vázquez, B. I., Miranda, J. M., Cepeda, A., & Cardelle-Cobas, A. (2021). Hábitos alimentarios en la población gallega durante el confinamiento por la COVID-19. *Nutricion Hospitalaria*, *37*(6), 1190–1196.  
<https://doi.org/10.20960/nh.03213>
- Smink, F. R. E., van Hoeken, D., Oldehinkel, A. J., & Hoek, H. W. (2014). Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *International Journal of Eating Disorders*, *47*(6), 610–619.  
<https://doi.org/10.1002/eat.22316>
- Smith, G. T., & Cyders, M. A. (2016). Integrating affect and impulsivity: The role of positive and negative urgency in substance use risk. *Drug and Alcohol Dependence*, *163*, S3–S12. <https://doi.org/10.1016/j.drugalcdep.2015.08.038>
- Society for Adolescent Health and Medicine. (2017). Young Adult Health and Well-Being: A Position Statement of the Society for Adolescent Health and Medicine. *Journal of Adolescent Health*, *60*(6), 758–759.  
<https://doi.org/10.1016/j.jadohealth.2017.03.021>
- Soler-Vila, H., Galán, I., Valencia-Martín, J. L., León-Muñoz, L. M., Guallar-Castillón, P., & Rodríguez-Artalejo, F. (2014). Binge drinking in Spain, 2008-2010. *Alcoholism: Clinical and Experimental Research*, *38*(3), 810–819.  
<https://doi.org/10.1111/acer.12275>
- Sonneville, K. R., Grilo, C. M., Richmond, T. K., Thurston, I. B., Jernigan, M., Gianini, L., & Field, A. E. (2015). Prospective association between overvaluation of weight and binge eating among overweight adolescent girls. *Journal of Adolescent Health*, *56*(1), 25–29.  
<https://doi.org/10.1016/j.jadohealth.2014.08.017>
- Sonneville, K. R., Horton, N. J., Micali, N., Crosby, R. D., Swanson, S. A., Solmi, F., & Field, A. E. (2013). Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: Does loss of control matter? *JAMA Pediatrics*, *167*(2), 149–155.  
<https://doi.org/10.1001/2013.jamapediatrics.12>

- Spinella, M. (2007). Normative data and a short form of the Barratt Impulsiveness Scale. *International Journal of Neuroscience*, 117(3), 359–368.  
<https://doi.org/10.1080/00207450600588881>
- Spoor, S. T. P., Bekker, M. H. J., Van Strien, T., & van Heck, G. L. (2007). Relations between negative affect, coping, and emotional eating. *Appetite*, 48(3), 368–376.  
<https://doi.org/10.1016/J.APPET.2006.10.005>
- Steele, E., Baraldi, L. G., Louzada, M. L. da C., Moubarac, J.-C., Mozaffarian, D., & Monteiro, C. A. (2016). Ultra-processed foods and added sugars in the US diet: evidence from a nationally representative cross-sectional study. *BMJ Open*, 6(3), e009892. <https://doi.org/10.1136/bmjopen-2015-009892>
- Steward, T., Mestre-Bach, G., Vintró-Alcaraz, C., Agüera, Z., Jiménez-Murcia, S., Granero, R., & Fernández-Aranda, F. (2017). Delay Discounting of Reward and Impulsivity in Eating Disorders: From Anorexia Nervosa to Binge Eating Disorder. *European Eating Disorders Review*, 25(6), 601–606.  
<https://doi.org/10.1002/erv.2543>
- Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action. *Clinical Psychology Review*, 14(7), 633–661. [https://doi.org/10.1016/0272-7358\(94\)90002-7](https://doi.org/10.1016/0272-7358(94)90002-7)
- Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *International Journal of Eating Disorders*, 27(2), 218–229. [https://doi.org/10.1002/\(SICI\)1098-108X\(200003\)27:2<218::AID-EAT10>3.0.CO;2-1](https://doi.org/10.1002/(SICI)1098-108X(200003)27:2<218::AID-EAT10>3.0.CO;2-1)
- Stice, E., Figlewicz, D. P., Gosnell, B. A., Levine, A. S., & Pratt, W. E. (2013). The contribution of brain reward circuits to the obesity epidemic. *Neuroscience and Biobehavioral Reviews*, 37(9 Pt A), 2047–2058.  
<https://doi.org/10.1016/J.NEUBIOREV.2012.12.001>
- Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset in adolescent girls: A 2-year prospective investigation. *Health Psychology*, 21(2), 131–138. <https://doi.org/10.1037/0278-6133.21.2.131>



- Striegel-Moore, R. H., & Franko, D. L. (2008). Should Binge Eating Disorder Be Included in the DSM-V? A Critical Review of the State of the Evidence. *Annual Review of Clinical Psychology, 4*(1), 305–324.  
<https://doi.org/10.1146/annurev.clinpsy.4.022007.141149>
- Sultson, H., Kukk, K., & Akkermann, K. (2017). Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. *Appetite, 116*, 423–430. <https://doi.org/10.1016/j.appet.2017.05.035>
- Swinburn, B. (1998). The determinants of fat intake in a multi-ethnic New Zealand population. Fletcher Challenge--University of Auckland Heart and Health Study Management Committee. *International Journal of Epidemiology, 27*(3), 416–421.  
<https://doi.org/10.1093/ije/27.3.416>
- Tapia-Rojas, C., Mira, R. G., Torres, A. K., Jara, C., Pérez, M. J., Vergara, E. H., Cerpa, W., & Quintanilla, R. A. (2017). Alcohol consumption during adolescence: A link between mitochondrial damage and ethanol brain intoxication. *Birth Defects Research, 109*(20), 1623–1639. <https://doi.org/10.1002/bdr2.1172>
- Touyz, S., Lacey, H., & Hay, P. (2020). Eating disorders in the time of COVID-19. *Journal of Eating Disorders, 8*(1), 19. <https://doi.org/10.1186/s40337-020-00295-3>
- Tremblay, A., Wouters, E., Wenker, M., St-Pierre, S., Bouchard, C., & Després, J. P. (1995). Alcohol and a high-fat diet: a combination favoring overfeeding. *The American Journal of Clinical Nutrition, 62*(3), 639–644.  
<https://doi.org/10.1093/ajcn/62.3.639>
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders, 5*(2), 295–315. [https://doi.org/10.1002/1098-108X\(198602\)5:2<295::AID-EAT2260050209>3.0.CO;2-T](https://doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T)
- van Strien, T., Herman, C. P., & Verheijden, M. W. (2009). Eating style, overeating,

and overweight in a representative Dutch sample. Does external eating play a role? *Appetite*, 52(2), 380–387. <https://doi.org/10.1016/j.appet.2008.11.010>

Ventura-Cots, M., Watts, A. E., & Bataller, R. (2017). Binge drinking as a risk factor for advanced alcoholic liver disease. *Liver International*, 37(9), 1281–1283. <https://doi.org/10.1111/LIV.13482>

Villarejo, C., Fernández-Aranda, F., Jiménez-Murcia, S., Peñas-Lledó, E., Granero, R., Penelo, E., Tinahones, F. J., Sancho, C., Vilarrasa, N., Montserrat-Gil de Bernabé, M., Casanueva, F. F., Fernández-Real, J. M., Frühbeck, G., De la Torre, R., Treasure, J., Botella, C., & Menchón, J. M. (2012). Lifetime Obesity in Patients with Eating Disorders: Increasing Prevalence, Clinical and Personality Correlates. *European Eating Disorders Review*, 20(3), 250–254. <https://doi.org/10.1002/erv.2166>

Vogeltanz-Holm, N. D., Wonderlich, S. A., Lewis, B. A., Wilsnack, S. C., Harris, T. R., Wilsnack, R. W., & Kristjanson, A. F. (2000). Longitudinal predictors of binge eating, intense dieting, and weight concerns in a national sample of women. *Behavior Therapy*, 31(2), 221–235. [https://doi.org/10.1016/S0005-7894\(00\)80013-1](https://doi.org/10.1016/S0005-7894(00)80013-1)

Volkow, N. D., Wang, G.-J., Fowler, J. S., & Telang, F. (2008). Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 363(1507), 3191–3200. <https://doi.org/10.1098/rstb.2008.0107>

Volkow, N. D., Wang, G. J., Fowler, J. S., Logan, J., Gatley, S. J., Wong, C., Hitzemann, R., & Pappas, N. R. (1999). Reinforcing effects of psychostimulants in humans are associated with increases in brain dopamine and occupancy of D(2) receptors. *The Journal of Pharmacology and Experimental Therapeutics*, 291(1), 409–415. <http://www.ncbi.nlm.nih.gov/pubmed/10490931>

von Ranson, K. M., McGue, M., & Iacono, W. G. (2003). Disordered eating and substance use in an epidemiological sample: II. Associations within families. *Psychology of Addictive Behaviors*, 17(3), 193–202. <https://doi.org/10.1037/0893-164X.17.3.193>

- Wang, L., Wang, H., Zhang, B., Popkin, B. M., & Du, S. (2020). Elevated fat intake increases body weight and the risk of overweight and obesity among Chinese adults: 1991–2015 trends. *Nutrients*, *12*(11), 1–13.  
<https://doi.org/10.3390/nu12113272>
- Wardle, J., Marsland, L., Sheikh, Y., Quinn, M., Fedoroff, I., & Ogden, J. (1992). Eating style and eating behaviour in adolescents. *Appetite*, *18*(3), 167–183.  
[https://doi.org/10.1016/0195-6663\(92\)90195-C](https://doi.org/10.1016/0195-6663(92)90195-C)
- Wechsler, H., & Nelson, T. F. (2008). What we have learned from the Harvard School of Public Health College Alcohol Study: Focusing attention on College Student Alcohol Consumption and the environmental conditions that promote it. *Journal of Studies on Alcohol and Drugs*, *69*(4), 481–490.  
<https://doi.org/10.15288/jsad.2008.69.481>
- Westerterp-Plantenga, M. S., & Verwegen, C. R. T. (1999). The appetizing effect of an aperitif in overweight and normal-weight humans. *American Journal of Clinical Nutrition*, *69*(2), 205–212. <https://doi.org/10.1093/ajcn/69.2.205>
- Wheeler, K., Greiner, P., & Boulton, M. (2005). Exploring alexithymia, depression, and binge eating in self-reported eating disorders in women. *Perspectives in Psychiatric Care*, *41*(3), 114–123. <https://doi.org/10.1111/j.1744-6163.2005.00022.x>
- Whiteside, S. P., & Lynam, D. R. (2001). The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*(4), 669–689. [https://doi.org/10.1016/S0191-8869\(00\)00064-7](https://doi.org/10.1016/S0191-8869(00)00064-7)
- Wiss, D., & Brewerton, T. (2020). Separating the Signal from the Noise: How Psychiatric Diagnoses Can Help Discern Food Addiction from Dietary Restraint. *Nutrients*, *12*(10), 2937. <https://doi.org/10.3390/NU12102937>
- Woods, A. M., Racine, S. E., & Klump, K. L. (2010). Examining the relationship between dietary restraint and binge eating: Differential effects of major and minor stressors. *Eating Behaviors*, *11*(4), 276–280.

<https://doi.org/10.1016/J.EATBEH.2010.08.001>

World Health Organization. (2000). *Obesity: preventing and managing the global epidemic: report of a WHO consultation*. World Health Organization.

<http://www.worldcat.org/title/obesity-preventing-and-managing-the-global-epidemic-report-of-a-who-consultation/oclc/48171257>

Yeomans, M. R. (2010). Alcohol, appetite and energy balance: Is alcohol intake a risk factor for obesity? *Physiology and Behavior*, *100*(1), 82–89.

<https://doi.org/10.1016/j.physbeh.2010.01.012>

Ziauddeen, H., Farooqi, I. S., & Fletcher, P. C. (2012). Obesity and the brain: how convincing is the addiction model? *Nature Reviews Neuroscience*, *13*(4), 279–286. <https://doi.org/10.1038/nrn3212>

Ziauddeen, H., & Fletcher, P. C. (2013). Is food addiction a valid and useful concept? *Obesity Reviews*, *14*(1), 19–28. <https://doi.org/10.1111/j.1467-789X.2012.01046.x>

Zúñiga, O., & Robles, R. (2006). Validez de constructo y consistencia interna del Cuestionario de Trastorno por Atracón en población mexicana con obesidad. *Psiquis*, *15*(5), 126–134. <https://www.imbiomed.com.mx/articulo.php?id=47533>

Zunker, C., Peterson, C. B., Crosby, R. D., Cao, L., Engel, S. G., Mitchell, J. E., & Wonderlich, S. A. (2011). Ecological momentary assessment of bulimia nervosa: Does dietary restriction predict binge eating? *Behaviour Research and Therapy*, *49*(10), 714–717. <https://doi.org/10.1016/j.brat.2011.06.006>

# **ANNEXES**



Annex 1: Study 1 article.

Binge eating and binge  
drinking: A two-way road?

An integrative review.





## REVIEW ARTICLE

**Binge Eating and Binge Drinking: A Two-Way Road? An Integrative Review**

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**Abstract:** Unhealthy diet and alcohol are serious health problems, especially in adolescents and young adults. "Binge" is defined as the excessive and uncontrolled consumption of food (binge eating) and alcohol (binge drinking). Both behaviors are frequent among young people and have a highly negative impact on health and quality of life. Several studies have explored the causes and risk factors of both behaviors, and the evidence concludes that there is a relationship between the two behaviors. In addition, some research postulates that binge eating is a precipitating factor in the onset and escalation of excessive alcohol consumption, while other studies suggest that alcohol consumption leads to excessive and uncontrollable food consumption. Given that no review has yet been published regarding the directionality between the two behaviors, we have set out to provide an up-to-date overview of binge eating and binge drinking problems, analyzing their commonalities and differences, and their uni- and bidirectional associations. In addition, we explore the reasons why young people tend to engage in both behaviors and consider directions for future research and clinical implications.

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**1. INTRODUCTION**

During the last two decades, an unhealthy diet (characterized by food with low nutritional quality and high energy intake) and alcohol consumption have been highly prevalent and now represent health risk factors for young people (range of 18-34 years) [1, 2]. The prevalence of these behaviors continues to increase alarmingly and contributes greatly to the high rates of obesity currently found in this population [3].

In many cases, the problem lies not only in the type of diet (e.g. ultra-processed foods rich in fats and sugar) but also in the pattern of consumption, namely the well-known "binge" phenomenon [4]. The term "binge" is used by the scientific community to describe a pattern of excessive and uncontrolled consumption, which can be applied to both food (Binge Eating - BE) and alcohol (Binge Drinking - BD). This binge pattern has become one of the most common ways of consuming food and alcohol in our society, and represents a dysfunctional intake with serious physical, psychological and social consequences [5, 6].

BE is characterized by the short-term intake (within approximately 2h) of an excessive amount of food (overeating) and the feeling of loss of control over that intake [4, 7]. BE can have serious consequences on daily functioning, causing deficiencies in social functioning and effecting productivity [8]. In addition, it can

produce medical problems and physical complications, including diabetes, metabolic syndrome, and celiac disease, and increases the risk of onset and maintenance of obesity [9]. BE is present in several eating disorders, such as BE disorder, bulimia nervosa, and binge/purge-type anorexia nervosa [4]. Additionally, it is observed in overweight and obese people, and in the general population [10].

Eating disorders are not frequent, but the rates of maladaptive eating behaviors are increasingly common among young people, especially college students, a high-risk group for the development of eating disorders [11]. Recent research indicates that BE is relatively stable in young adulthood [12], and the percentage of young adults who engage in BE ranges between 0.3 and 3.1% [13].

BD is a pattern of alcohol consumption that raises blood alcohol concentration to  $\geq 0.08$  g percent (more than 5 drinks in men or more than 4 drinks in women) within a 2-hour period [14]. Alcohol consumption is more frequent among young adults [1], and a common form of consumption among this age group is the binge form (consuming alcohol in large quantities, in a short period of time, and with a sense of losing control over intake) [15, 16]. Twenty-five percent of young adults report alcohol abuse [17], and 53 % report that they generally consume large amounts in a short time when they drink (five or more drinks in one session) [18]. This pattern of consumption is associated aggression, sexual disinhibition, stuttering, loss of control, poor academic performance [19, 20], impairment in cognitive functioning [21, 22], and short- and long-term problems such as dropping out of college, alcohol dependence after college, low job performance [23] and suicidal tendencies in the young [24]. It is also associated with serious health problems, such as development of cardiovascular disease, different types of cancer [25, 26], and liver injury, such as steatosis, steatohepatitis, hepatitis and alcoholic cirrhosis [27]. Excessive alcohol

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consumption is also related to a higher risk of traffic accidents, leading to high mortality among young people [28, 29]. In fact, Hingson et al. (2009) concluded that almost half of young Americans between 18 and 24 years who died in traffic accidents had consumed alcohol [30].

Given their high prevalence and the impact that both behaviors have on health and quality of life, many researchers have studied the co-occurrence between the two, as well as shared characteristics and common consequences [5, 31, 32]. Various theories have been put forward to explain the causes and risk factors of these behaviors, and the consensus in the literature is that there is a relationship between the two [5, 6]. However, no studies to date have explored the directionality between BE and BD behaviors.

The main purpose of this narrative review is to reflect in-depth on the problems of BE and BD. In particular, it aims to determine the common aspects and differences, and the possible directionality between the two behaviors: Could BE act as a risk factor for BD? Can BD precipitate BE? Under what circumstances do these bidirectional (relationships) occur? Finally, we will propose future lines of research based on our updated view of the state-of-the-art of the field.

## 2. COMMONALITIES BETWEEN BE AND BD

Research to date suggests that BE and BD share some characteristics, including: 1) nature and onset of the problem, 2) common negative consequences, 3) personal factors, and 4) social and cultural factors (See Fig. 1).

### 2.1. Nature and Onset of the Problem

BE and BD are characterized by the consumption of a certain substance (food and alcohol) in large quantities and over a short period of time [4]. In some ways, both behaviors can be understood as addictive behaviors [33], since they are characterized by a repetitive and uncontrollable response [5, 6] associated with negative consequences (psychological, social, personal, academic, etc.) [4, 5].

BE and BD usually begin in adolescence, and occur most frequently in young adulthood [10, 17]. Evidence shows that both are frequent high-risk behaviors [23, 34], especially among college students [5], with approximately 40% reporting consuming a greater amount of food than a normal person would in similar circumstances [35] and experiencing a loss of control [36], while the same percentage admit engaging in BD [5, 37].

Evidence suggests that both BE and BD are precursors of subsequent eating and substance use disorders [38]. Recent reviews point to the high comorbidity between the two types of disorders [31, 39]. Furthermore, BE and disordered eating behavior can predict alcohol and drug use problems in young adulthood [13, 40], in the same way that excessive alcohol consumption can predict disordered eating behavior [41]. Eating and substance abuse disorders can be aggravated by the presence of psychiatric comorbidities [42].

### 2.2. Common Negative Consequences

Research suggests that BE and BD produce serious physical, psychological, social, and academic consequences, such as affective disorders (depression and anxiety symptoms), increased risk of being overweight, obesity, diabetes, increased tendency to engage in risky behaviors, and poor academic performance [5, 19, 43, 44, 45]. The comorbidity between the two behaviors produces more negative experiences than the presence of only one and may increase the severity, chronicity, and resistance to treatment of other psychiatric disorders [46, 47].

These negative consequences are especially relevant in adolescence and young adulthood, a period of brain maturation in which important physiological, psychological, and synaptic plasticity

changes occur [48]. For this reason, adolescents and young adults are especially vulnerable to environmental threats, and are more vulnerable to experiencing problems with alcohol abuse or inappropriate eating habits [49]. Furthermore, young people who engage in BE and BD behaviors do not usually consider it a significant problem, and tend to minimize the associated damage, despite the short- and long-term negative consequences [50]. In this sense, both BE and BD are prevalent in the general young population, and both tend to be persistent, despite the associated negative consequences [5, 10, 51].

### 2.3. Personal Factors

Among the potential personal factors accounting for BE and BD comorbidity are stress levels, affect and emotions (negative affect, depression, low self-esteem and anxiety), and personality traits and dimensions (impulsivity, sensation-seeking, neuroticism and sensitivity to reward) [5, 47]. Gender differences in BE and BD are presented at the end of this section.

#### 2.3.1. Stress

Stress can be understood as a challenging and uncontrollable event that affects the adaptation processes necessary to recover homeostasis [52]. It has been extensively studied in relation to BD behavior, given its role as a risk factor in the development of alcohol use disorder [34]. Evidence indicates that college students who report higher levels of stress tend to drink alcohol in large amounts or report problems with alcohol use [53], and will participate in more BD episodes in the following two weeks [54].

Stress is also central to BE behavior. Recent research indicates that stress predicts BE episodes in college students [55] and may also be a common trigger for BE disorder [4, 56].

Although the mechanisms underlying the effects of stress on food and alcohol consumption are not yet clear, there seems to be an overlap of circuits and neurotransmitters both in the stress response and in the regulation of food intake and alcohol consumption; among them, cortisol, which is released in stressful situations and, in turn, plays a very important role as a precursor to the reinforcement of tasty foods and drugs of abuse [56, 57].

#### 2.3.2. Emotional or Affective Factors

Various studies have linked BE and BD with negative emotions [6, 58]. Recent studies indicate that depression influences impulsive intake, since most people with impulsive intake display symptoms of depression [59]. In addition, depressive symptoms and negative affect can act as predictors of BE [55, 60]. In line with this, McCabe & Vincent [61] found that both depression and anxiety predicted BE in adolescent women, while low self-esteem and anxiety predicted BE in adolescent men.

BD has also been associated with depressive symptoms [62]; depressive symptoms have been shown to predict excessive alcohol use [63], while excessive alcohol use can predict depressive symptoms [64]. In addition, research suggests a link between heavy drinking and depression; BD can affect recovery from depression [24], and increases the risk of suicide in young people [65].

Although the underlying psychological mechanism linking the two behaviors remains unknown, the current literature confirms the common goals of BE and BD; i.e. to regulate emotions and cope with distress and negative situations [6, 66, 67].

#### 2.3.3. Cognitive and Behavioral Factors

Young people who engage in BE and BD behaviors may experience negative consequences at a cognitive level, such as poor academic performance and a higher probability of academic failure [5, 68, 69].

Young adults with eating disorders, including BE, also display cognitive impairment, especially in visual perception skills and non-

verbal memory tests [70]. On the other hand, young people who consume alcohol in excess displayed a poorer performance in memory tests [71], and poorer performance in the medium and long term in tests of verbal learning and visual reproduction [72].

Among adolescents, findings suggest that both BE and BD are related to negative experiences in the past, a stronger inclination to fatalism, and a lower future orientation [50]. Additionally, this age group is more likely to report feelings of lower satisfaction with life and self-esteem [50], and individuals continue to binge (both food and alcohol) despite it interfering with other pleasurable activities [51].

There are factors that reduce the risk of both behaviors; in other words, protective factors. In relation to BE, a recent review indicates that the quality of family relationships and healthy family attitudes concerning food and weight can help prevent eating disorders in young people [73]. In relation to BD, high levels of perceived parental warmth and risk perception of alcohol consumption have been found to reduce the likelihood of young people participating in heavy drinking episodes [74]. Parental monitoring and family time can also help reduce the risk of heavy drinking in young people [75]. In this way, the family assumes a prominent role as a protective factor for both BE and BD. This evidence may help to develop future prevention and intervention strategies for BE and BD.

#### 2.3.4. Personality Factors

Impulsivity, sensation-seeking, neuroticism, and sensitivity to reward are the most studied personality constructs in research about BE and BD [76-80].

The negative urgency trait, which refers to the tendency to act precipitously or impulsively when distressed, has proven to be especially important in predicting BE and BD [81-83].

Sensitivity to reward - that is, a greater propensity to engage in impulsive behaviors and experiences of positive affection in situations marked by reward - is also a predictive factor for risk behaviors, and different investigations have highlighted that it is a shared characteristic among impulsive eaters and alcohol drinkers [84].

#### 2.3.5. Gender Differences in BE and BD

Studies investigating gender differences in BE among young people indicate that it can occur in both sexes [85, 86, 87], but is more frequent in women [86, 87], although one study reported a higher frequency in men [85]. In addition, young women show more loss of control during BE than young men [87, 88, 89], and more compensatory behavior [87, 88]. Women tend to induce vomiting after BE more often than men, and are more likely to use laxatives and exercise to lose weight [87].

Recently, some risk and protective factors for BE have been associated with gender. For instance, although concerns about weight predict BE in women rather than men, self-esteem tends to protect women from this behavior [90]. BE can lead to disability in adulthood in both sexes, although the rates are significantly higher in women [91]. These results suggest future interventions should be tailored to consider gender, with the aim of improving the efficiency of treatments.

Gender differences are also found in BD. While BD is more frequent among young men, this trend is changing, with rates of alcohol consumption increasing in young women. Furthermore, women start drinking earlier and present more BD episodes than men [92, 93, 94]. Young girls are more vulnerable than young boys to the problems associated with BD, and suffer more severe health and psychosocial consequences [95]. Women also progress more rapidly to addiction than men [96]. With regard to the variables that may influence BD, recent research indicates that women engage in BD because a higher level of reactivity to stress and greater vulnerability to affective problems such as depression and anxiety, while

men do so because of their higher levels of sensation-seeking and lower levels of inhibitory control [97]. These findings justify the need to provide measures that integrate gender differences in the prevention and treatment of BE and BD.

#### 2.4. Social and Cultural Factors

Social reasons also play an important role in the initiation and development of both behaviors.

Several studies have attempted to determine which sociocultural factors are determinants in the excessive consumption of food and alcohol. They have concluded that one of the strongest of these factors is peer pressure, with impulsive eaters and drinkers being more likely to indulge in both behaviors in order to abide by the group's norms, to engage in similar behaviors as the rest of the group and thus be socially accepted, and improve their social standing [50, 80, 98]. It would appear that another influencing factor is the media, which manipulates the perceptions of adolescents and can influence their decisions to become involved in both behaviors [50].

Another sociocultural factor related to both behaviors is the tendency of adolescents to compare themselves with their peer group. Young people who tend to compare themselves largely to others in terms of their opinions, thoughts, and performance tend to eat and drink more impulsively [50].

Perceived discrimination may also be a risk factor for the onset of impulsive BE and BD behaviors [99, 100]. The findings of research encourage an emphasis on prevention and intervention of socially disadvantaged groups, incorporating social support as a protective factor, with the aim of increasing adaptive coping responses [7].

### 3. DIFFERENCES BETWEEN BE AND BD

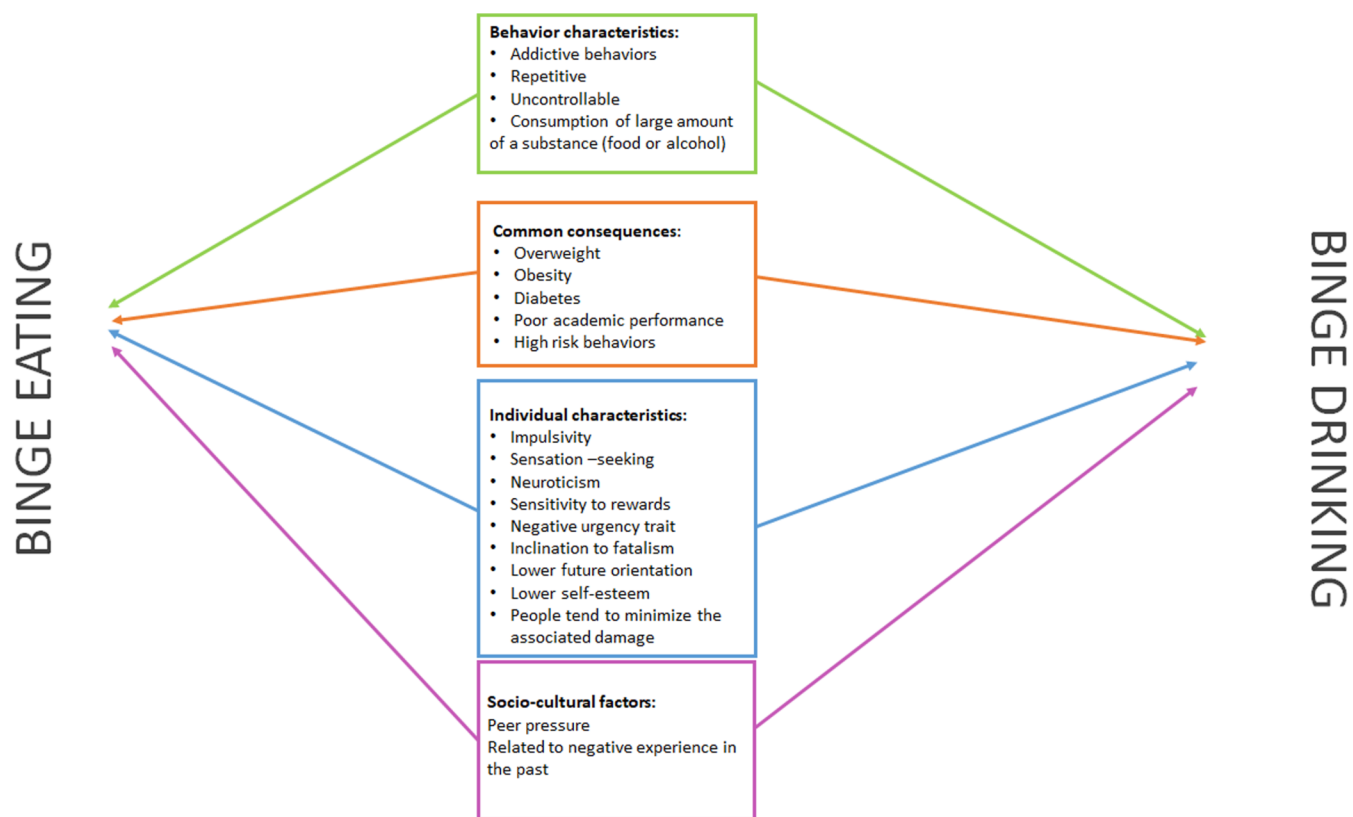
In addition to their large commonalities, BE and BD also have distinct and unique characteristics. These particularities could help to clarify the relationship between these two behaviors.

#### 3.1. Dietary Restriction and Body Dissatisfaction

Dietary restriction appears to be a key factor in BE [101]. According to research, negative urgency, the tendency to act precipitously in negative affective states [102], seems to moderate the relationship between dietary restriction and BE [103].

Another aspect that characterizes BE behavior is body dissatisfaction. A meta-analysis identified body dissatisfaction as the strongest risk factor for BE [104], although this relationship may be mediated by self-esteem and depression [105]. Overestimating one's shape and body weight may also predict BE [80], and both variables may be causal antecedents or consequences of BE [39]. The severity of BE has also been associated with increased concerns about body image and food [107].

Dietary restriction is an underlying phenomenon in BE behavior, and it is not absent in BD behavior. In this sense, some investigations have highlighted an association between dietary restriction and excessive alcohol consumption [108, 109], although the approach of the subject seems to be different. In particular, a recent global trend has been observed in university students who restrict their consumption of caloric foods to compensate for caloric intake related to alcohol consumption, a phenomenon conceptualized as "drunkorexia" [110, 111]. The incidence of this phenomenon among young people is high [82]; prevalence ranges between 14 and 46% [112], and it is more common among female college students [110], though it is also observed in young men [109]. This practice is deliberately carried out on drinking days, and the goals are to avoid the weight gain associated with alcohol consumption, to reach intoxication more quickly, and to enjoy the effects of this intoxication [112, 113]. Excessive drinking and dietary restriction can be very harmful. Research points to negative short-term conse-



**Fig. (1).** Commonalities between BE and BD. (A higher resolution / colour version of this figure is available in the electronic copy of the article).

quences, such as rapid alcohol intoxication, loss of consciousness, sexual exploitation, memory loss and injury, and long-term cognitive impairment, including brain damage, or liver cirrhosis [113].

### 3.2. The Importance of the Substance

In recent decades, the world has experienced a general change in the quality of diet, characterized by an increase in the consumption of ultra-processed foods containing saturated or trans fats, sodium, sugars, antioxidants, preservatives and colorants [2]. These foods have been designed to enhance the reinforcing and addictive properties of traditional foods, and have been compared to drugs of abuse, as they both activate the brain reward pathways that reinforce the rewarding properties of the substance in question [39]. This has fueled research in recent years about food addiction, based on evidence that certain foods, generally ultra-processed or highly palatable, have addictive potential, similar to drugs of abuse [114, 115]. These investigations propose that food addiction be included among recognized addictive disorders [89], while others point to the need for more research to confirm the theory's validity [116]. Currently, food addiction is not recognized as an addictive disorder by reference diagnostic manuals [4]; the diagnostic construct is controversial and remains under study [115].

In contrast, the concept of alcohol addiction is well established [4]. Alcohol is a psychoactive substance that is consumed for its positive reinforcing properties and has high addictive capacity. This makes people who consume alcohol with a certain frequency and quantity more susceptible to the addiction process [117, 118]. Recent research indicates that alcohol and other drugs have much more potent addictive effects than foods such as chocolate as a result of the neuroadaptive effects they produce at the cerebral level. These involve the gabaergic, opioid and dopaminergic circuits, which make the substance more appetizing and are critical in the development of drug addiction [119].

The risk of addiction to both food and alcohol is highly variable among individuals and depends on the interaction of many genetic, socioeconomic, cultural, temperamental and environmental factors [118, 120].

### 3.3. Withdrawal and Tolerance Phenomena

Withdrawal and tolerance phenomena are particularly relevant to alcohol consumption [39]. Individuals who consume alcohol excessively very often need to consume increasing amounts of alcohol in order to experience the desired effects, a process brought about by tolerance [4]. Furthermore, after withdrawal, individuals may experience physical or psychological symptoms in response to the absence of the substance (withdrawal phenomenon) [4], which together with BD, can determine a subsequent alcohol consumption disorder [121].

Highly processed foods (such as cakes, pizza, or French fries) may also be able to trigger withdrawal phenomenon [122]. However, as far as we are aware, there is as yet no published human study that has systematically examined the phenomenon of withdrawal from highly processed foods and BE, and withdrawal symptoms are not clearly defined in the context of food addiction [39, 122, 123].

Potential findings in this line may have clinical implications for intervening in both behaviors. Interventions in BD are aimed at reducing access to the substance and eliminating, as far as possible, its consumption [124], while in BE, the aim is to decrease dietary restriction and establish healthy eating patterns [125], since food is necessary for survival and cannot be eliminated, unlike alcohol.

In recent decades, new lines of research have aimed to prevent and treat different diseases using food products. This is the case of "nutraceuticals" which are defined as foods or parts of foods that provide health benefits and can prevent and treat different diseases

[126]. For example, these compounds can ameliorate the consequences derived from BE and BD, such as obesity, diabetes, dyslipidemia or heart disease [127, 128]. There are already reviews that highlight the role of nutraceuticals in the prevention and improvement of dyslipidemia, the main cardiovascular risk factor for heart diseases [128]. Recent research points to the role of ginger and avocado as an adjunct in the treatment or prevention of obesity, demonstrated through a decrease in inflammation and oxidative stress or a reduction in blood lipids [129]. Nevertheless, the exact role of nutraceuticals in the prevention and treatment of different diseases needs to be confirmed by future studies [128].

Beyond identifying and describing what similarities and differences underlie BE and BD, an issue that is beginning to interest researchers is the interrelationship between BE and BD [130, 131]. Previous studies have found an association between BE and BD [5], but no review to date has explored the directionality between the two behaviors. The following sections will attempt to broach this question.

#### 4. FROM BINGE DRINKING TO BINGE EATING

As explained above, the relationship between BE and BD has been extensively studied, mainly as a co-occurrence. The high correlation between the two behaviors is well known. Given the prevalence of both, they are becoming a recognized clinical concern. In the process of attempting to clarify the relation between BD and BE, many commonalities have been identified, but very little is known about the direction of this relation; that is, if bingeing on alcohol has an effect on the way people eat, or if the reverse is true, or both, and under which circumstances it occurs.

About the directional effects from one behavior to another, the evidence is scarce. Studies addressing the question about how drinking can have an effect on food intake have contemplated different characteristics of alcohol intake (quantity, type, pattern of intake, time in which consumption occurs) and its effect on food intake (type of food, quantity) (See Table 1).

In relation to the quantity of food eaten after acute alcohol consumption, evidence shows that alcohol stimulates short-term food intake when it is consumed before or with a meal [132, 133, 134, 135, 136]. The effect of the long-term intake of alcohol on food consumption has been studied through epidemiological approaches. In two large-scale cohort studies [137, 138], results revealed a positive relationship between alcohol consumption and energy intake, showing that more food is consumed on days when alcohol is ingested.

This effect has been hypothetically explained by different mechanisms. On one hand, it has been related to reduced satiety signaling after alcohol consumption [139, 140], while some studies have demonstrated that the consumption of alcohol induces loss of self-control [136, 141]. Researchers have indicated potential mechanisms of reward systems through which alcohol increases the rewarding value of food. In accordance with these studies, food reward comprises two components: "liking" and "wanting". Liking is the pleasantness of food and the pleasure derived from its taste, and is influenced by opioid, endocannabinoid and GABA neurotransmission, whereas wanting refers to the intrinsic motivation to eat, and depends on dopaminergic neurotransmission [142]. Alcohol consumption has been proven to stimulate both likings and wanting [143].

Researchers have also investigated the role of oral and gut sensory pathways in alcohol's effect on food reward [144]. Food intake after moderate consumption of alcohol or non-alcohol beverages was assessed, and results showed that alcohol consumption increased subsequent energy intake regardless of the food consumed after the beverage, a finding echoed by other studies [134, 136]. Overall, participants increase their food consumption after alcohol consumption, especially of high-fat savory food, in comparison to

subjects who do not consume alcohol. In addition, explicit liking of savory food has been shown to be more intense after alcohol consumption, although the role of oral and gut sensory signaling in alcohol's effect on food intake remains unclear [144]. In line with this, one study was conducted to explore how alcohol influences food intake by increasing the desire for food (appetite) [145]. The study tested the effects of alcohol delivered in two ways: bypassing the digestive system by infusing the alcohol directly into the blood stream, and by oral consumption. An increase in food intake was observed in both conditions, suggesting that this increased consumption of food is not due entirely to the interaction of alcohol and gut, but that it also involves the brain. The study in question also detected a different pattern among participants, formed of one group with a significantly higher increment in food intake and another group that ate less following alcohol consumption. The group that ate more also showed a differential hypothalamic activation.

Regarding the type of food consumed after alcohol intake, the study conducted by Tremblay et al [146] analyzed the relation between alcohol and a fatty diet. Alcohol had an impact on ad libitum food intake, showing that the energy content provided by alcohol was not compensated by a decrease in energy intake from food. These results demonstrated that a high alcohol intake has no inhibitory effect on lipid intake, a positive correlation with protein, and a negative correlation with carbohydrate intake. In the same line, Canton [133] found that alcohol did not differentially enhance intake of specific food items. The study in question focused on the effects of alcohol on the pleasantness of several tasty and bland foods. Results showed that alcohol did not promote intake by increasing the pleasantness of food or by stimulating appetite for these foods.

Another relevant variable is the amount of alcohol that has to be consumed to have an impact on food intake. Canton [132] found that administration of a high dose of alcohol (4 units) was associated with higher hunger levels and increased energy intake. The energy derived from alcohol was not compensated for. Low consumption of alcohol (1 unit) was below threshold values and did not elicit effects on food intake, thus confirming a dose-response effect.

In summary, studies targeting the effects of drinking behaviors on eating have focused on the characteristics of alcohol intake that exert an effect on food intake. Although there are hypotheses about the effects of BD on BE, no studies have yet been conducted to clarify the nature of this directionality or to throw light on the underlying characteristics.

#### 5. FROM BINGE EATING TO BINGE DRINKING

To our knowledge, only four studies have analyzed the longitudinal relationship between BE and BD with the aim of understanding their relation and their temporal interplay (See Table 1).

Measelle, Stice, & Hogansen [147] analyzed a sample of 493 adolescent females to examine whether initial elevations in depression, eating, antisocial, and substance abuse symptoms predicted future increases in other symptom domains. Potential eating pathologies were assessed with a structured psychiatric interview based on DSM-IV diagnostic criteria - the Eating Disorder Examination [148] - while substance abuse symptoms were evaluated by self-report items adapted from Stice et al. [148]. Adolescents were assessed annually over a five-year period (from 13 to 18 years old). Results showed that the girls' depression, eating disorder symptoms, and substance abuse scores increased on average over the five-year period, whereas their antisocial behavior decreased. The findings suggested that having an eating pathology predicts future growth in substance abuse, but not vice versa [147].

Similar results were reported by a study with 8,594 female participants of 9 to 15 years old [40]. Eating disorder behaviors were assessed by self-report-validated questions about purging behaviors,

**Table 1. Summary of results on relation from BD to BE.**

Author, Year	Purpose	Participants	Procedure	Outcomes
Caton, Ball, Ahern, & Hetherington 2004 [132]	To study the Dose-response effect of alcohol on appetite and food intake.	12 males.	Participants attended to the lab 3 times and were given breakfast, lunch, and dinner. Thirty minutes before lunch, they received 330 ml of no-alcohol lager (no-alcohol condition), the same amount of lager spiked with 1 unit of alcohol (1 UA condition) or 4 units of alcohol (4 UA condition). Ratings of appetite and mood were recorded before and after preloads and lunch, then hourly across the day.	Participants consumed more high-fat salty food items at lunch following 4 UA compared to the other preloads. Hunger was rated higher following 4 UA across the day in comparison to the other preloads, but fullness ratings not reflect any difference by condition. Energy intake at dinner was similar in all conditions and total energy intake across the day was significantly higher after 4 UA. Alcohol appears to stimulate appetite due to elevated levels of subjective hunger.
Caton, Marks, & Hetherington 2005 [133]	To study the effects of manipulating pleasantness on subsequent energy intake following a moderate dose of alcohol.	12 males.	Participants attended to the lab 4 times and were given breakfast, 4 h later were offered lunch. Twenty minutes before lunch participants received an alcohol or a no-alcohol containing beverage followed by a lunch of either bland or flavored foods. Ratings of appetite, mood and pleasantness of several bland and tasty foods were taken before and after preloads and lunch.	Ad libitum energy intake at lunch was greater following alcohol in both taste conditions in comparison to the no-alcohol conditions. No additive effect of palatability and alcohol was found; alcohol did not differentially affect intake of lunch items. Nor did alcohol increase the pleasantness of foods. Alcohol promoted food intake but this did not occur via increasing the pleasantness of the taste of foods.
Caton, Bate, & Hetherington, 2007 [134]	To compare the effects on appetite and energy intake within a meal and across 4 days of drinking wine either before (aperitif) or with (coingestion).	11 males.	Participants attended to the lab 3 times and were given breakfast. 3 h later returned for a two course lunch, then recorded food intake for the remainder of the day and the next 3 days. In the control condition, participants ate lunch ad libitum; in the aperitif condition red wine was consumed 20 min before lunch; and in the coingestion condition red wine was consumed with the starter and with the main course. Ratings of appetite and mood were administered before and after the meal.	Energy intake at lunch was greater when wine was consumed compared to control. In particular, intake of the starter was enhanced by wine consumption. Total Energy intake during the test days was significantly higher than during subsequent days revealing a tendency to overeat exacerbated by drinking wine before or with lunch.
Westerterp-Plantenga, & Verwegen, 1999 [135]	To assess the effects on energy intake of an aperitif compared with those of a water appetizer and 3 fruit juice appetizers.	52 participants (27 men and 27 women) with a body mass index between 20 and 32.	Participant was randomly given alcohol, fat, protein, carbohydrate, water, or no preload 30 min before an ad libitum lunch.	Energy intake and eating rate were higher, meal duration was longer, satiation started to increase later, and eating was prolonged after maximum satiation after an aperitif than after the other conditions.
Yeomans, 2010[136]	To review the evidence regarding the incidence of obesity and its relation with an excess energy intake, including alcohol.		Brief Review.	Research shows that energy consumed as alcohol is additive to that from other dietary sources, leading to over-consumption of energy when alcohol is consumed. In addition, alcohol consumed before or with meals tends to increase food intake. Higher intakes of alcohol (binge drinking) in the absence of alcohol dependence may increase the risk of obesity.
De Castro, & Orozco, 1990 [137]	To study alcohol effects on eating.	92 participants (23 male, 69 female).	Participants were asked to report in a diary their intakes the time of ingestion, their subjective state at the time of ingestion, during 7 days.	Alcohol appear to be ingested in addition to other nutrients not in replace. Therefore, the total calories intakes of drinkers compared with not drinkers are not different for carbohydrate, fat or protein intakes but are larger because of the calories from alcohol.
De Castro, & Orozco, 2009 [138]	To study the associations of different types of foods ingested at various times of day with total daily and macro-nutrient intakes.	1009 participants (388 male and 621 female).	Participants were asked to report in a diary their intakes during 7 days. They were asked to reported in a detail way, every possible item that they either ate or drank, the time and the amount.	Foods as ice cream, beef, potatoes, pastry, nuts, snacks, condiments, alcohol and soda, among others were significantly associated with higher total intake over the day. Dietary energy density appeared to mediate the associations between particular foods and beverages and overall energy intake.

(Table 1) Contd....

Author, Year	Purpose	Participants	Procedure	Outcomes
Raben Agerholm-Larsen, Flint, Holst, & Astrup, 2003[139]	To investigate the effects on appetite, energy intake and expenditure, and substrate metabolism of meals rich in 1 of the 4 macronutrients.	19 participants (9 women and 10 men).	Participants were measured on subjective appetite sensations, ad libitum food intake, energy expenditure, substrate metabolism, and hormone concentrations, 5 h after breakfast- Breakfast had similar energy density and fiber contents but rich in either protein, carbohydrate, fat, or alcohol.	No significant differences were found after the 4 meals, in hunger or satiety sensations or in ad libitum energy intake. After the alcohol meal, fat oxidation and leptin concentrations were greatly suppressed. Intake of an alcohol-rich meal stimulates energy expenditure but suppresses fat oxidation and leptin more than in the other conditions. Satiety and ad libitum energy intake were not significantly different between meals.
Röjdmarm, Calissendorff, & Brismar, 2008 [140]	To determine the effects of ingestion of ethanol on leptin secretion in normal subjects.	14 healthy, non-obese participants (7 male, 7 female).	Participants attended to the lab 2 times. On one occasion alcohol was ingested, and on the other water was given. The experiments took place in random order, one week apart. In group I two experiment were performed during the day, while in group II the experiments were carried out during the night. Each alcoholic drink contained 0.45 g ethanol/kg. Venous blood samples were collected before, during and after the drinks over periods of 6h in group I and 14h in group II. Plasma glucose, and serum concentrations of leptin, insulin and ethanol were determined immediately before ingestion of the first dose of alcohol, and subsequently at time points.	Ingestion of moderate amounts of alcohol has an inhibitory effect on leptin secretion in normal subjects. The effect of alcohol might serve as an appetizer by decreasing leptin secretion.
Caton, Nolan, & Hetherington, 2015 [141]	To review the psychological role of alcohol in disrupting attempts to restrict food intake for body weight management and to consider whether the disinhibiting effects of alcohol contributes to the stimulation of overeating in dieters.		Literature review.	Alcohol is associated with increased short-term energy intake. Dieters and restrained eaters report increased alcohol intake and increased frequency of binge drinking. The interaction between alcohol, appetite and dietary restraint is complex and further work is needed to extend our knowledge at a basic level regarding how individual differences influence how much alcohol is consumed, the frequency and pattern of alcohol consumption and how alcohol intake affects both appetite control and body weight regulation.
Berridge, 2009 [142]	To explore the role of 'wanting' and 'liking' for food rewards.		Literature review.	Brain mechanisms for food reward and appetite evolved under pressures to protect us from scarcity. All eating patterns are controlled intimately by brain mechanisms of food reward.
Melis, Diana, Marinelli, & Brodie, 2009[143]	To explore the link between acetaldehyde and ethanol actions on brain reward pathways to reduce alcohol craving.		Literature review.	The findings reviewed point out the specific elements of ethanol action on dopamine VTA neurons. Both ethanol and acetaldehyde increase the spontaneous activity of dopamine VTA neurons. This effect is associated with the rewarding and reinforcing properties of drugs of abuse. Therefore can be important for ethanol-seeking behavior. Stress also increases the spontaneous discharge of dopamine VTA neurons.
Schrieke, et al., 2015 [144]	To investigate whether food reward plays a role in the stimulating effect of moderate alcohol consumption on subsequent food intake, and to explore the role of oral and gut sensory pathways in alcohol's effect on food reward	24 men	Participants were randomly assigned to either consumption of vodka/orange juice (20 g alcohol) or orange juice only, followed by consumption of cake, MSF of cake or no cake. Food reward was evaluated by actual food intake measured by an ad libitum lunch 45 min after alcohol ingestion and by behavioural indices of wanting and liking	Moderate alcohol consumption increased food intake during the ad libitum lunch. This effect was related to the higher food reward experienced for savoury foods Alcohol increased intake and explicit liking of high-fat savoury foods. Moderate alcohol consumption increased implicit wanting for savoury and decreased implicit wanting for sweet before the meal. The importance of oral and gut sensory signalling in alcohol's effect on food reward remains largely unclear.

(Table 1) Contd....

Author, Year	Purpose	Participants	Procedure	Outcomes
Eilet et al., 2015 [145]	To evaluate the blood oxygenation level dependent (BOLD) response to the food aromas following pharmacokinetically controlled intravenous infusion of alcohol.	35 non-obese women	BOLD activation to food aromas was evaluated once during intravenous infusion of alcohol, and once during infusion of saline. Ad libitum intake of roast beef with noodles or Italian meat sauce with pasta following imaging was recorded.	An alcohol pre-load increased food consumption and potentiated differences between food and non-food BOLD responses in the region of the hypothalamus.
Tremblay et al., 1995 [146]	To explore the effects of alcohol and dietary fat on spontaneous energy and macronutrient intakes (study 1) and to explore the effects of alcohol on energy and macronutrient intakes (study 2).	Study 1: 8 males Study 2: 711 participants (351 males and 360 females).	Study 1: Participants were randomly assigned to four conditions (low-fat, placebo; low-fat, alcohol; high-fat, placebo; and high-fat, alcohol) lasting 2 d each during which they were requested to eat ad libitum in the laboratory. During each of the three daily meals, they had free access to foods that were selected according to their tastes and preferences. Each session differed from the others in the fat content of ingested foods and alcohol intake. Study 2: Participants were asked to track their dietary for 3 days.	The high-fat diet was associated with a substantial increase in daily energy intake. Alcohol had no inhibitory effect on food intake and its energy content an additional increase in energy intake. High alcohol intake was associated with a high daily energy intake and had no inhibitory effect on lipid intake.
Measelle, Stice, & Hogansen, 2006 [147]	To examine the co-occurrence between symptomatology.	493 adolescent females.	Co-occurring symptomatology was examined in a community sample of who were followed annually from early to late adolescence.	Depression, eating disorder, and substance abuse symptoms increased over time, whereas antisocial behavior decreased. Initial depressive and antisocial symptoms predicted future increases in the other; substance abuse and antisocial behavior symptoms showed prospective reciprocal relations. Initial depression predicted increases in eating and substance abuse symptoms. Initial eating disorder symptoms predicted increases in substance abuse problems.
Field, et al., 2012 [40]	To evaluate whether Bulimia Nervosa and subtypes of eating disorder not otherwise specified are predictive of developing adverse outcomes.	8594 females.	Participants received questionnaires annually from 1996 through 2001, then biennially through 2007 and 2008.	Participants with Binge eating disorder were most likely to become overweight, obese, or to develop depressive symptoms, than non-disordered peers. Participants with Purging Disorder had a significantly increased risk of using drugs and to binge drink. Purging Disorder and Binge eating disorder is common and predict a range of adverse outcomes.
Sonneville et al., 2013 [13]	To investigate the association between overeating and binge eating and adverse outcomes.	16882 adolescents and young adults living throughout the United States.	Prospective cohort study where participants were assessed on overeating and binge eating every 12 to 24 months between 1996 and 2005.	Risk of becoming overweight or obese, starting to binge drink frequently, starting to use marijuana, starting to use other drugs, and developing high levels of depressive symptoms.
Stice, Barrera, & Chassin, 1998 [149]	To test the differential prediction model and to examine the possible mechanisms by which predictors as socialization or psychopathology factors may relate to consumption.	216 families (adolescents and parents). Half of the teens were at risk because of parental alcoholism.	It is a prospective study where teens and parents were interviewed using a computer-assisted interview. Family members were interviewed at two time points separated by a 1-year period. The interview was conducted separately to minimize contamination.	Externalizing symptoms, parental alcoholism, peer influences, and parental support were indirectly related to negative consequences through their effects on user level. Externalizing symptoms, internalizing symptoms, peer influences, and parental approval of use directly predicted consequences. Internalizing pathology potentiated the relation between consumption and consequences, whereas parental support and control mitigated this relation.
Micali et al., 2015 [150]	To investigate whether anorexia nervosa, bulimia nervosa, binge eating disorder, and other eating disorders.	6,140 youth at age 14 and 5,069 at age 16.	Eating and outcomes of depression, anxiety disorders, binge drinking, drug use, deliberate self-harm, weight status were measured using interviews and questionnaires about 2 years after predictors.	Adolescent eating disorders, including subthreshold presentations, predict negative outcomes. All eating disorders were predictive of later anxiety disorders. Anorexia nervosa, bulimia nervosa, binge eating disorder, and other eating disorders were prospectively associated with depression. All eating disorders but anorexia nervosa predicted drug use and deliberate self-harm.



frequency of eating a very large amount of food, feelings of loss of control during eating, and feelings of guilt after BE. Following DSM-5 cut-offs, participants were classified as having BE Disorder (BE at least once per weeks with no purging); Purging Disorder (weekly vomiting or use of laxatives to control weight and no bingeing); Bulimia Nervosa (weekly BE and purging); and Eating Disorder Not Otherwise Specified (monthly BE and/or purging or overeating episodes with no loss of control). BD was assessed with a question about the frequency, in the previous year, of drinking  $\geq 4$  drinks over a few hours. Participants who reported at least 6 episodes of BD in the previous year were classified as frequent binge drinkers [40]. Results showed that Eating Disorder Not Otherwise Specified was the most common disorder in this population. All types of eating disorders were associated with a significant increase in the risk of starting to binge drink frequently.

In contrast, a prospective study of 16,882 adolescents and young adults found that BE did not predict more frequent BD in the future [13], although it did predict future drug use, increased depressive symptoms, and psychological distress. In the study in question, BE was defined as weekly (at least) episodes of eating a large amount of food with a loss of control, based on the DSM-5 criteria. BD was assessed by a question asking about the frequency of drinking four or more drinks over a few hours in the previous year. Participants who reported at least 6 episodes of BD were classified as frequent binge drinkers. Results showed that BE was more common among females than males. Among females, the prevalence of BE tended to increase with age. Neither overeating nor BE was associated with frequent BD in the future in either age- and sex-adjusted or fully adjusted models, while both overeating and BE predicted future marijuana or other drug use [13].

Similar results were found by a large UK population-based study in youths from 14 to 16 years old [150]. BE Disorder was diagnosed using DSM-5 criteria: weekly BE (for sub-threshold: monthly BE), absence of purging, and at least three cognitive symptoms (e.g. eating faster than normal, eating large amounts when not hungry, feeling guilty about the amount eaten, etc.). BD was assessed using the Alcohol Use Disorders Identification Test (AUDIT), a short questionnaire used to screen for problematic drinking. BD was defined as drinking  $\geq 6$  units of alcohol on one occasion at least monthly over the previous year. BE disorder and subthreshold BE were not prospectively associated with BD. The authors had observed this relation in a previous study [40], but attributed it to cultural differences.

Studies by Measelle et al. [141] and Field et al. [40] detected a prospective, unidirectional relationship between an earlier eating pathology and a subsequent rise in substance abuse. Although these studies did not analyze the mechanisms that could explain this association, it was hypothesized that BE behaviors promote feelings of shame and guilt, and that substance use could be a response to these negative emotions [98]. However, recent studies by Sonnevile et al. [13] and Micali et al. [150] did not confirm this longitudinal predictive relationship between BE and BD. Therefore, the results are inconclusive, and direct comparison of studies that analyze the longitudinal relation between BE and BD are difficult due to differences in diagnostic criteria, populations and assessment methods.

## 6. DISCUSSION

The main purpose of this narrative review is to provide an updated overview of BE and BD, analyzing their commonalities and differences, as well as their uni or/and bi-directional relationship.

In short, BE and BD share a number of characteristics, such as the onset and nature of the problem, associated negative consequences, negative affect and emotions, various personality dimensions (e.g., impulsivity, sensation-seeking, neuroticism, sensitivity to reward) and socio-cultural factors (e.g., to abide by the norms of the group, to be socially accepted, to improve social appearance).

These commonalities point to the need of considering both behaviors when planning interventions addressed at adolescents and young people. Future interventions could benefit from identifying the role of personal and socio-cultural factors shared by BE and BD, and transdiagnostic interventions could be more beneficial in reducing both behaviors by focusing on their commonalities.

In terms of the mechanisms that explain these commonalities, there seems to be an overlap of circuits and neurotransmitters - for example, cortisol - in the regulation of food intake and alcohol consumption [56, 57]. Moreover, it is hypothesized that BE and BD are often themselves mechanisms to regulate emotions and cope with distress and negative situations [6, 66, 67].

When analyzing the differences between BE and BD, dietary restriction and body dissatisfaction are more related to the former than the latter. However, in recent years the phenomenon of drunkorexia has challenged such differences. In this context, both binge patterns can be interpreted as examples of impulsive behaviors, suggesting that alcohol is a component of the risk of weight gain that exists as a consequence of impulsive behavioral choices [136].

Another difference between BE and BD is related to addiction. Although ultra-processed or highly palatable food is thought to have the same addictive potential as drugs of abuse [114, 115], addiction to food is not considered an addictive disorder in diagnostic classifications. Future studies should address the validity of this consensus; for example, it should be assessed whether highly processed foods (such as cakes, pizza, or French fries) are able to trigger the withdrawal phenomenon [122]. There are yet no human studies that have systematically examined the phenomenon of withdrawal in highly processed foods and BE, and withdrawal symptoms are not clearly defined in the context of food addiction [39, 122, 123].

To date, while there is no doubt that BE and BD share significant features, it remains unclear if these bingeing behaviors are uni or bi-directionally related. With regard to the directional effects of one behavior on another, the evidence is scarce. Studies addressing the question of how drinking can effect food intake have explored different characteristics of alcohol intake (quantity, type, pattern of intake, duration of consumption) and its effect over food intake (types of food and quantity, among others). Evidence shows that alcohol stimulates short-term food intake when it is consumed before or with a meal. This effect has been hypothetically attributed to reduced satiety signaling after alcohol consumption [139, 140], an increase in the sense of loss of self-control [136, 141], and the fact that alcohol may increase the rewarding value of food [142, 151]. In this sense, there appears to be a unidirectional relation between BD and BE.

On the other hand, prospective studies analyzing BE as a predictor of BD have generated mixed results. Some researchers have observed this association in both human and animal models [40, 152], while others have failed to replicate this result [13, 150]. These inconclusive findings could be explained by problems in the definition of BE and BD. Some studies have operationalized eating pathology as an eating disorder diagnosis, while others have focused on eating pathology symptoms. Moreover, some studies operationalize the respective outcome measure as a categorical variable, while others use a more continuous measure. In this sense, a meta-analysis of the relation between eating pathologies and depression found that the classification of outcome variables as categorical or continuous moderated the effects of the former on the latter, and vice versa, suggesting that longitudinal effects were greater when the outcome was categorical [153]. In this sense, it could be relevant to analyze if the classification of BE and BD as continuous or categorical variables moderates the association between them. In terms of classification, it is important to take into account that some of the studies mentioned in the present review were published before the release of DSM-5. In this context, it is

necessary to unify criteria in order to determine the size and direction of effects between BD and other eating pathologies. Moreover, future studies that assess bi-directionality might benefit from employing cross-panel designs to test for both simultaneous (cross-sectional) and cross-lagged (longitudinal) relationships to ensure valid conclusions are drawn. Finally, researchers should determine the optimal time lags between assessment points to ensure that the conclusions of longitudinal studies about the relationship between eating pathology and BD are as accurate as possible.

## CONCLUSION

The objective of this review was to analyze the factors that are now known to be common to BE and BD, as well as the particularities of each behavior. Additionally, we set out to reflect what is currently known about the directionality between the two behaviors, given that many studies have related BE and BD but have not determined the direction of that relationship. The result highlights the importance of understanding the relationship between eating pathology and BD, especially in young people. We demonstrate that the evidence regarding the directionality of the two behaviors remains weak; some studies show that BE acts as a precipitating factor in the occurrence and escalation of BD, while others have not found such an association. This may be due to problems in the definitions of BE and BD, which have changed considerably in recent years, in addition to the fact that many studies were conducted before the appearance of DSM-5. The question of how alcohol can affect food intake has also been addressed; in this respect, alcohol has been shown to stimulate food intake, both when consumed before or at mealtimes. Thus, there appears to be a two-way relationship between BE and BD, though further studies are needed to prove this bi-directionality, as the evidence is somewhat limited. Together, these results have considerable clinical implications. In view of the high co-morbidity between the two behaviors, establishing which factors influence both could help determine a common conceptual framework to better understand the etiology, development and maintenance of both behaviors, and to identify subgroups at higher risk and thus improve prevention and early intervention strategies.

## LIST OF ABBREVIATIONS

BD = Binge Drinking

BE = Binge Eating

## CONSENT FOR PUBLICATION

Not applicable.

## CONSENT FOR PUBLICATION

Not applicable.

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## CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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## REFERENCES

- [1] Dawson DA, Goldstein RB, Saha TD, Grant BF. Changes in alcohol consumption: United States, 2001-2002 to 2012-2013. *Drug Alcohol Depend* 2015; 148: 56-61. <http://dx.doi.org/10.1016/j.drugalcdep.2014.12.016> PMID: 25620731
- [2] Monteiro CA, Cannon G, Moubarac JC, Levy RB, Louzada MLC, Jaime PC. The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutr* 2018; 21(1): 5-17. <http://dx.doi.org/10.1017/S1368980017000234> PMID: 28322183
- [3] Moodie R, Stuckler D, Monteiro C, *et al.* Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. *Lancet* 2013; 381(9867): 670-9. [http://dx.doi.org/10.1016/S0140-6736\(12\)62089-3](http://dx.doi.org/10.1016/S0140-6736(12)62089-3) PMID: 23410611
- [4] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: American Psychiatric Association 2013.
- [5] Ferriter C, Ray LA. Binge eating and binge drinking: an integrative review. *Eat Behav* 2011; 12(2): 99-107. <http://dx.doi.org/10.1016/j.eatbeh.2011.01.001> PMID: 21385638
- [6] Laghi F, Baiocco R, Liga F, Lonigro A, Baumgartner E. Binge eating and binge drinking behaviors: individual differences in adolescents' identity styles. *J Health Psychol* 2014; 19(3): 333-43. <http://dx.doi.org/10.1177/1359105312470851> PMID: 23405028
- [7] Simone M, Scodes J, Mason T, Loth K, Wall MM, Neumark-Sztainer D. Shared and non-shared risk and protective factors of binge eating and binge drinking from adolescence to young adulthood. *J Health Psychol* 2019.1359105319844588 <http://dx.doi.org/10.1177/1359105319844588> PMID: 31014132
- [8] Pawaskar M, Witt EA, Supina D, Herman BK, Wadden TA. Impact of binge eating disorder on functional impairment and work productivity in an adult community sample in the United States. *Int J Clin Pract* 2017; 71(7): 1-9. <http://dx.doi.org/10.1111/ijcp.12970> PMID: 28741812
- [9] Keski-Rahkonen A, Mustelin L. Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. *Curr Opin Psychiatry* 2016; 29(6): 340-5. <http://dx.doi.org/10.1097/YCO.0000000000000278> PMID: 27662598
- [10] Kessler RC, Berglund PA, Chiu WT, *et al.* The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biol Psychiatry* 2013; 73(9): 904-14. <http://dx.doi.org/10.1016/j.biopsych.2012.11.020> PMID: 23290497
- [11] White S, Reynolds-Malec JB, Cordero E. Disordered eating and the use of unhealthy weight control methods in college students: 1995, 2002, and 2008. *Eat Disord* 2011; 19(4): 323-34. <http://dx.doi.org/10.1080/10640266.2011.584805> PMID: 22352972
- [12] Goldschmidt AB, Wall MM, Loth KA, Bucchianeri MM, Neumark-Sztainer D. The course of binge eating from adolescence to young adulthood. *Health Psychol* 2014; 33(5): 457-60. <http://dx.doi.org/10.1037/a0033508> PMID: 23977873
- [13] Sonnevile KR, Horton NJ, Micali N, *et al.* Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: does loss of control matter? *JAMA Pediatr* 2013; 167(2): 149-55. <http://dx.doi.org/10.1001/2013.jamapediatrics.12> PMID: 23229786
- [14] Tapia-Rojas C, Mira RG, Torres AK, *et al.* Alcohol consumption during adolescence: A link between mitochondrial damage and ethanol brain intoxication. *Birth Defects Res* 2017; 109(20): 1623-39. <http://dx.doi.org/10.1002/bdr2.1172> PMID: 29251843
- [15] Galán I, González MJ, Valencia-Martín JL. [Alcohol drinking patterns in Spain: a country in transition]. *Rev Esp Salud Publica* 2014; 88(4): 529-40.

- PMID: 25090408
- [16] Soler-Vila H, Galán I, Valencia-Martín JL, León-Muñoz LM, Gual-lar-Castillón P, Rodríguez-Artalejo F. Binge drinking in Spain, 2008-2010. *Alcohol Clin Exp Res* 2014; 38(3): 810-9. <http://dx.doi.org/10.1111/acer.12275> PMID: 24164355
- [17] Kanny D, Naimi TS, Liu Y, Lu H, Brewer RD. Annual Total Binge Drinks Consumed by U.S. Adults, 2015. *Am J Prev Med* 2018; 54(4): 486-96. <http://dx.doi.org/10.1016/j.amepre.2017.12.021> PMID: 29555021
- [18] Wechsler H, Nelson TF. What we have learned from the Harvard School Of Public Health College Alcohol Study: focusing attention on college student alcohol consumption and the environmental conditions that promote it. *J Stud Alcohol Drugs* 2008; 69(4): 481-90. <http://dx.doi.org/10.15288/jsad.2008.69.481> PMID: 18612562
- [19] Kuntsche E, Kuntsche S, Thrul J, Gmel G. Binge drinking: Health impact, prevalence, correlates and interventions. *Psychol Health* 2017; 32(8): 976-1017. <http://dx.doi.org/10.1080/08870446.2017.1325889> PMID: 28513195
- [20] Martinotti G, Lupi M, Carlucci L, *et al.* Alcohol drinking patterns in young people: A survey-based study. *J Health Psychol* 2017; 22(14): 1889-96. <http://dx.doi.org/10.1177/1359105316667795> PMID: 27624615
- [21] Cservenka A, Brumback T. The burden of binge and heavy drinking on the brain: Effects on adolescent and young adult neural structure and function. *Front Psychol* 2017; 8: 1111. <http://dx.doi.org/10.3389/fpsyg.2017.01111> PMID: 28713313
- [22] Smith JL, Iredale JM, Mattick RP. Sex differences in the relationship between heavy alcohol use, inhibition and performance monitoring: Disconnect between behavioural and brain functional measures. *Psychiatry Res Neuroimaging* 2016; 254: 103-11. <http://dx.doi.org/10.1016/j.psychres.2016.06.012> PMID: 27399307
- [23] Jennison KM. The short-term effects and unintended long-term consequences of binge drinking in college: a 10-year follow-up study. *Am J Drug Alcohol Abuse* 2004; 30(3): 659-84. <http://dx.doi.org/10.1081/ADA-200032331> PMID: 15540499
- [24] Schaffer M, Jeglic EL, Stanley B. The relationship between suicidal behavior, ideation, and binge drinking among college students. *Arch Suicide Res* 2008; 12(2): 124-32. <http://dx.doi.org/10.1080/13811110701857111> PMID: 18340594
- [25] Scoccianti C, Straif K, Romieu I. Recent evidence on alcohol and cancer epidemiology. *Future Oncol* 2013; 9(9): 1315-22. <http://dx.doi.org/10.2217/fon.13.94> PMID: 23980679
- [26] Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology* 2013; 144(6): 1252-61. <http://dx.doi.org/10.1053/j.gastro.2013.01.068> PMID: 23622135
- [27] Ventura-Cots M, Watts AE, Bataller R. Binge drinking as a risk factor for advanced alcoholic liver disease. *Liver Int* 2017; 37(9): 1281-3. <http://dx.doi.org/10.1111/liv.13482> PMID: 28845617
- [28] Gicquel L, Ordonneau P, Blot E, Toillon C, Ingrand P, Romo L. Description of various factors contributing to traffic accidents in youth and measures proposed to alleviate recurrence. *Front Psychiatry* 2017; 8: 94. <http://dx.doi.org/10.3389/fpsyg.2017.00094> PMID: 28620324
- [29] Llerena S, Arias-Loste MT, Puente A, Cabezas J, Crespo J, Fábrega E. Binge drinking: Burden of liver disease and beyond. *World J Hepatol* 2015; 7(27): 2703-15. <http://dx.doi.org/10.4254/wjh.v7.i27.2703> PMID: 26644814
- [30] Hingson RW, Zha W, Weitzman ER. Magnitude of and trends in alcohol-related mortality and morbidity among US college students ages 18-24, 1998-2005. *J Stud Alcohol Drugs* 2009; 16(1): 12-20. <http://dx.doi.org/10.15288/jsads.2009.s16.12>
- [31] Bahji A, Mazhar MN, Hudson CC, Nadkarni P, MacNeil BA, Hawken E. Prevalence of substance use disorder comorbidity among individuals with eating disorders: A systematic review and meta-analysis. *Psychiatry Res* 2019; 273: 58-66. <http://dx.doi.org/10.1016/j.psychres.2019.01.007> PMID: 30640052
- [32] Davis C, Mackew L, Levitan RD, Kaplan AS, Carter JC, Kennedy JL. Binge Eating Disorder (BED) in Relation to addictive behaviors and personality risk factors. *Front Psychol* 2017; 8: 579. <http://dx.doi.org/10.3389/fpsyg.2017.00579> PMID: 28487663
- [33] Benjamin L, Wulfert E. Dispositional correlates of addictive behaviors in college women: binge eating and heavy drinking. *Eat Behav* 2005; 6(3): 197-209. <http://dx.doi.org/10.1016/j.eatbeh.2003.08.001> PMID: 15854866
- [34] Kuntsche E, Knibbe R, Gmel G, Engels R. Why do young people drink? A review of drinking motives. *Clin Psychol Rev* 2005; 25(7): 841-61. <http://dx.doi.org/10.1016/j.cpr.2005.06.002> PMID: 16095785
- [35] Kelly-Weeder S. Binge drinking and disordered eating in college students. *J Am Acad Nurse Pract* 2011; 23(1): 33-41. <http://dx.doi.org/10.1111/j.1745-7599.2010.00568.x> PMID: 21208332
- [36] Lipson SK, Sonnevile KR. Eating disorder symptoms among undergraduate and graduate students at 12 U.S. colleges and universities. *Eat Behav* 2017; 24: 81-8. <http://dx.doi.org/10.1016/j.eatbeh.2016.12.003> PMID: 28040637
- [37] Croteau V, Morrell JS. Prevalence of Binge Drinking Episodes Among Male and Female College Students (P18-008-19) *Curr Dev Nutr* 2019; 3(1)
- [38] Grant BF, Stinson FS, Harford TC. Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: a 12-year follow-up. *J Subst Abuse* 2001; 13(4): 493-504. [http://dx.doi.org/10.1016/S0899-3289\(01\)00096-7](http://dx.doi.org/10.1016/S0899-3289(01)00096-7) PMID: 11775078
- [39] Schulte EM, Grilo CM, Gearhardt AN. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clin Psychol Rev* 2016; 44: 125-39. <http://dx.doi.org/10.1016/j.cpr.2016.02.001> PMID: 26879210
- [40] Field AE, Sonnevile KR, Micali N, *et al.* Prospective association of common eating disorders and adverse outcomes. *Pediatrics* 2012; 130(2): e289-95. <http://dx.doi.org/10.1542/peds.2011-3663> PMID: 22802602
- [41] Nelson MC, Lust K, Story M, Ehlinger E. Alcohol use, eating patterns, and weight behaviors in a university population. *Am J Health Behav* 2009; 33(3): 227-37. <http://dx.doi.org/10.5993/AJHB.33.3.1> PMID: 19063644
- [42] Lozano ÓM, Rojas AJ, Fernández Calderón F. Psychiatric comorbidity and severity of dependence on substance users: how it impacts on their health-related quality of life? *J Ment Health* 2017; 26(2): 119-26. <http://dx.doi.org/10.1080/09638237.2016.1177771> PMID: 27128492
- [43] Fazzino TL, Fleming K, Sher KJ, Sullivan DK, Befort C. Heavy Drinking in Young Adulthood Increases Risk of Transitioning to Obesity. *Am J Prev Med* 2017; 53(2): 169-75. <http://dx.doi.org/10.1016/j.amepre.2017.02.007> PMID: 28365088
- [44] Raevuori A, Suokas J, Haukka J, *et al.* Highly increased risk of type 2 diabetes in patients with binge eating disorder and bulimia nervosa. *Int J Eat Disord* 2015; 48(6): 555-62. <http://dx.doi.org/10.1002/eat.22334> PMID: 25060427
- [45] Striegel-Moore RH, Franko DL. Should binge eating disorder be included in the DSM-V? A critical review of the state of the evidence. *Annu Rev Clin Psychol* 2008; 4(1): 305-24. <http://dx.doi.org/10.1146/annurev.clinpsy.4.022007.141149> PMID: 18370619
- [46] Franko DL, Dorer DJ, Keel PK, Jackson S, Manzo MP, Herzog DB. How do eating disorders and alcohol use disorder influence each other? *Int J Eat Disord* 2005; 38(3): 200-7. <http://dx.doi.org/10.1002/eat.20178> PMID: 16216020
- [47] Ortega RO, Chapela IB, Santoncini CU. Disordered eating behaviors and binge drinking in female high-school students: The role of impulsivity. *Salud Ment* 2012; 35(2): 83-9.
- [48] Fleming R. Does alcohol damage the adolescent brain? Neuro-anatomical and neuropsychological consequences of adolescent drinking. *Neurosci Neuroecon* 2015; 4: 51-60. <http://dx.doi.org/10.2147/NAN.S60983>
- [49] Reichelt AC. Adolescent maturational transitions in the prefrontal cortex and dopamine signaling as a risk factor for the development of obesity and high fat/high sugar diet induced cognitive deficits. *Front Behav Neurosci* 2016; 10: 189. <http://dx.doi.org/10.3389/fnbeh.2016.00189> PMID: 27790098
- [50] Laghi F, Liga F, Baumgartner E, Baiocco R. Time perspective and psychosocial positive functioning among Italian adolescents who binge eat and drink. *J Adolesc* 2012; 35(5): 1277-84.

- <http://dx.doi.org/10.1016/j.adolescence.2012.04.014> PMID: 22672793
- [51] Schreiber LRN, Odlaug BL, Grant JE. The overlap between binge eating disorder and substance use disorders: Diagnosis and neurobiology. *J Behav Addict* 2013; 2(4): 191-8. <http://dx.doi.org/10.1556/JBA.2.2013.015> PMID: 25215200
- [52] Sinha R. Chronic stress, drug use, and vulnerability to addiction. *Ann N Y Acad Sci* 2008; 1141: 105-30. <http://dx.doi.org/10.1196/annals.1441.030> PMID: 18991954
- [53] Kenney SR, Lac A, Labrie JW, Hummer JF, Pham A. Mental health, sleep quality, drinking motives, and alcohol-related consequences: a path-analytic model. *J Stud Alcohol Drugs* 2013; 74(6): 841-51. <http://dx.doi.org/10.15288/jsad.2013.74.841> PMID: 24172110
- [54] Chen Y, Feeley TH. Predicting binge drinking in college students: Rational beliefs, stress, or loneliness? *J Drug Educ* 2015; 45(3-4): 133-55. <http://dx.doi.org/10.1177/0047237916639812> PMID: 27075608
- [55] Phillips KE, Kelly-Weeder S, Farrell K. Binge eating behavior in college students: What is a binge? *Appl Nurs Res* 2016; 30: 7-11. <http://dx.doi.org/10.1016/j.apnr.2015.10.011> PMID: 27091245
- [56] Naish KR, Laliberte M, MacKillop J, Balodis IM. Systematic review of the effects of acute stress in binge eating disorder. *Eur J Neurosci* 2019; 50(3): 2415-29. <http://dx.doi.org/10.1111/ejn.14110> PMID: 30099796
- [57] Sinha R. How does stress increase risk of drug abuse and relapse? *Psychopharmacology (Berl)* 2001; 158(4): 343-59. <http://dx.doi.org/10.1007/s002130100917> PMID: 11797055
- [58] Rosenbaum DL, White KS. The relation of anxiety, depression, and stress to binge eating behavior. *J Health Psychol* 2015; 20(6): 887-98. <http://dx.doi.org/10.1177/1359105315580212> PMID: 26032804
- [59] Jung JY, Kim KH, Woo HY, *et al.* Binge eating is associated with trait anxiety in Korean adolescent girls: a cross sectional study. *BMC Womens Health* 2017; 17(1): 8. <http://dx.doi.org/10.1186/s12905-017-0364-4> PMID: 28109277
- [60] Wheeler K, Greiner P, Boulton M. Exploring alexithymia, depression, and binge eating in self-reported eating disorders in women. *Perspect Psychiatr Care* 2005; 41(3): 114-23. <http://dx.doi.org/10.1111/j.1744-6163.2005.00022.x> PMID: 16138820
- [61] McCabe MP, Vincent MA. The Role of Biodevelopmental and Psychological Males and Females. *Eur Eat Disord Rev* 2003; 328: 315-28. <http://dx.doi.org/10.1002/erv.500>
- [62] Paljärvi T, Koskenvuo M, Poikolainen K, Kauhanen J, Sillanmäki L, Mäkelä P. Binge drinking and depressive symptoms: a 5-year population-based cohort study. *Addiction* 2009; 104(7): 1168-78. <http://dx.doi.org/10.1111/j.1360-0443.2009.02577.x> PMID: 19438420
- [63] Lamis DA, Malone PS, Langhinrichsen-Rohling J, Ellis TE. Body investment, depression, and alcohol use as risk factors for suicide proneness in college students. *Crisis* 2010; 31(3): 118-27. <http://dx.doi.org/10.1027/0227-5910/a000012> PMID: 20573605
- [64] Pietraszek MH, Urano T, Sumiوشي K, *et al.* Alcohol-induced depression: involvement of serotonin. *Alcohol Alcohol* 1991; 26(2): 155-9. <http://dx.doi.org/10.1093/oxfordjournals.alcalc.a045096> PMID: 1878077
- [65] Tuisku V, Pelkonen M, Kiviruusu O, Karlsson L, Ruuttu T, Marttunen M. Factors associated with deliberate self-harm behaviour among depressed adolescent outpatients. *J Adolesc* 2009; 32(5): 1125-36. <http://dx.doi.org/10.1016/j.adolescence.2009.03.001> PMID: 19307015
- [66] Laghi F, D'Alessio M, Pallini S, Baiocco R. Attachment representations and time perspective in adolescence. *Soc Indic Res* 2009; 90(2): 181-94. <http://dx.doi.org/10.1007/s11205-008-9249-0>
- [67] Pompili S, Laghi F. Binge eating and binge drinking among adolescents: The role of drinking and eating motives. *J Health Psychol* 2019; 24(11): 1505-16. <http://dx.doi.org/10.1177/1359105317713359> PMID: 28810467
- [68] Nemer ASDA, Fausto MA, Silva-Fonseca VAD, Ciomei MH, Quintaes KD. Pattern of alcoholic beverage consumption and academic performance among college students. *Archives of Clinical Psychiatry (São Paulo)* 2013; 40(2): 65-70. <http://dx.doi.org/10.1590/S0101-60832013000200003>
- [69] Serra R, Kiekens G, Vanderlinden J, *et al.* Binge eating and purging in first-year college students: Prevalence, psychiatric comorbidity, and academic performance. *Int J Eat Disord* 2019; 1-10. <http://dx.doi.org/10.1002/eat.23211> PMID: 31868255
- [70] Grau A, Magallón-Neri E, Faus G, Feixas G. Cognitive impairment in eating disorder patients of short and long-term duration: a case-control study. *Neuropsychiatr Dis Treat* 2019; 15: 1329-41. <http://dx.doi.org/10.2147/NDT.S199927> PMID: 31190837
- [71] Sneider JT, Cohen-Gilbert JE, Crowley DJ, Paul MD, Silveri MM. Differential effects of binge drinking on learning and memory in emerging adults. *J Addict Res Ther* 2013; (7): (Suppl. 7). <http://dx.doi.org/10.4172/2155-6105.S7-006> PMID: 24404407
- [72] Brown SA, Tapert SF, Granholm E, Delis DC. Neurocognitive functioning of adolescents: effects of protracted alcohol use. *Alcohol Clin Exp Res* 2000; 24(2): 164-71. <http://dx.doi.org/10.1111/j.1530-0277.2000.tb04586.x> PMID: 10698367
- [73] Langdon-Daly J, Serpell L. Protective factors against disordered eating in family systems: a systematic review of research. *J Eat Disord* 2017; 5(1): 12. <http://dx.doi.org/10.1186/s40337-017-0141-7> PMID: 28360998
- [74] Cleveland MJ, Feinberg ME, Bontempo DE, Greenberg MT. The role of risk and protective factors in substance use across adolescence. *J Adolesc Health* 2008; 43(2): 157-64. <http://dx.doi.org/10.1016/j.jadohealth.2008.01.015> PMID: 18639789
- [75] Danielsson AK, Romelsjö A, Tengström A. Heavy episodic drinking in early adolescence: gender-specific risk and protective factors. *Subst Use Misuse* 2011; 46(5): 633-43. <http://dx.doi.org/10.3109/10826084.2010.528120> PMID: 20964532
- [76] Adams ZW, Kaiser AJ, Lynam DR, Charnigo RJ, Milich R. Drinking motives as mediators of the impulsivity-substance use relation: pathways for negative urgency, lack of premeditation, and sensation seeking. *Addict Behav* 2012; 37(7): 848-55. <http://dx.doi.org/10.1016/j.addbeh.2012.03.016> PMID: 22472524
- [77] Adan A, Forero DA, Navarro JF. Personality traits related to binge drinking: A systematic review. *Front Psychiatry* 2017; 8: 134. <http://dx.doi.org/10.3389/fpsy.2017.00134> PMID: 28804465
- [78] Coskunpinar A, Dir AL, Cyders MA, Cyders MA. Multidimensionality in impulsivity and alcohol use: a meta-analysis using the UPPS model of impulsivity. *Alcohol Clin Exp Res* 2013; 37(9): 1441-50. <http://dx.doi.org/10.1111/acer.12131> PMID: 23578176
- [79] Escrivá-Martínez T, Galiana L, Rodríguez-Arias M, Baños RM. The binge eating scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Front Psychol* 2019; 10: 530. <http://dx.doi.org/10.3389/fpsyg.2019.00530> PMID: 30967808
- [80] Laghi F, Pompili S, Baumgartner E, Baiocco R. The role of sensation seeking and motivations for eating in female and male adolescents who binge eat. *Eat Behav* 2015; 17: 119-24. <http://dx.doi.org/10.1016/j.eatbeh.2015.01.011> PMID: 25687232
- [81] Bø R, Billieux J, Landrø NI. Which facets of impulsivity predict binge drinking? *Addict Behav Rep* 2016; 3: 43-7. <http://dx.doi.org/10.1016/j.abrep.2016.03.001> PMID: 29531999
- [82] Smith GT, Cyders MA. Integrating affect and impulsivity: The role of positive and negative urgency in substance use risk. *Drug Alcohol Depend* 2016; 163(Suppl. 1): S3-S12. <http://dx.doi.org/10.1016/j.drugalcdep.2015.08.038> PMID: 27306729
- [83] Fischer S, Anderson KG, Smith GT. Coping with distress by eating or drinking: role of trait urgency and expectancies. *Psychol Addict Behav* 2004; 18(3): 269-74. <http://dx.doi.org/10.1037/0893-164X.18.3.269> PMID: 15482082
- [84] Loxton NJ, Dawe S. How do dysfunctional eating and hazardous drinking women perform on behavioural measures of reward and punishment sensitivity? *Pers Individ Dif* 2007; 42(6): 1163-72. <http://dx.doi.org/10.1016/j.paid.2006.09.031>

- [85] Saules KK, Collings AS, Hoodin F, *et al.* The contributions of weight problem perception, BMI, gender, mood, and smoking status to binge eating among college students. *Eat Behav* 2009; 10(1): 1-9. <http://dx.doi.org/10.1016/j.eatbeh.2008.07.010> PMID: 19171310
- [86] Heatherton TF, Nichols P, Mahamedi F, Keel P. Body weight, dieting, and eating disorder symptoms among college students, 1982 to 1992. *Am J Psychiatry* 1995; 152(11): 1623-9. <http://dx.doi.org/10.1176/ajp.152.11.1623> PMID: 7485625
- [87] Kelly-Weeder S, Jennings KM, Wolfe BE. Gender differences in binge eating and behavioral correlates among college students. *Eat Weight Disord* 2012; 17(3): e200-2. <http://dx.doi.org/10.1007/BF03325348> PMID: 23086256
- [88] Lynch WC, Everingham A, Dubitzky J, Hartman M, Kasser T. Does binge eating play a role in the self-regulation of moods? *Integr Physiol Behav Sci* 2000; 35(4): 298-313. <http://dx.doi.org/10.1007/BF02688792> PMID: 11330493
- [89] Striegel-Moore RH, Rosselli F, Perrin N, *et al.* Gender difference in the prevalence of eating disorder symptoms. *Int J Eat Disord* 2009; 42(5): 471-4. <http://dx.doi.org/10.1002/eat.20625> PMID: 19107833
- [90] Sehm M, Warschburger P. Prospective associations between binge eating and psychological risk factors in adolescence. *J Clin Child Adolesc Psychol* 2018; 47(5): 770-84. <http://dx.doi.org/10.1080/15374416.2016.1178124> PMID: 27399285
- [91] Striegel RH, Bedrosian R, Wang C, Schwartz S. Why men should be included in research on binge eating: results from a comparison of psychosocial impairment in men and women. *Int J Eat Disord* 2012; 45(2): 233-40. <http://dx.doi.org/10.1002/eat.20962> PMID: 22031213
- [92] Keyes KM, Grant BF, Hasin DS. Evidence for a closing gender gap in alcohol use, abuse, and dependence in the United States population. *Drug Alcohol Depend* 2008; 93(1-2): 21-9. <http://dx.doi.org/10.1016/j.drugaldep.2007.08.017> PMID: 17980512
- [93] Keyes KM, Martins SS, Blanco C, Hasin DS. Telescoping and gender differences in alcohol dependence: new evidence from two national surveys. *Am J Psychiatry* 2010; 167(8): 969-76. <http://dx.doi.org/10.1176/appi.ajp.2009.09081161> PMID: 20439391
- [94] Substance Abuse and Mental Health Services Administration. Substance Abuse Treatment: Addressing the Specific Needs of Women Knowledge Application Program (Keys) for Clinicians. Rockville: U.S. Department of Health and Human Services 2014.
- [95] Nolen-Hoeksema S, Hilt L. Possible contributors to the gender differences in alcohol use and problems. *J Gen Psychol* 2006; 133(4): 357-74. <http://dx.doi.org/10.3200/GENP.133.4.357-374> PMID: 17128956
- [96] Becker JB, Perry AN, Westenbroek C. Sex differences in the neural mechanisms mediating addiction: a new synthesis and hypothesis. *Biol Sex Differ* 2012; 3(1): 14. <http://dx.doi.org/10.1186/2042-6410-3-14> PMID: 22676718
- [97] Dir AL, Bell RL, Adams ZW, Hulvershorn LA. Gender differences in risk factors for adolescent binge drinking and implications for intervention and prevention. *Front Psychiatry* 2017; 8: 289. <http://dx.doi.org/10.3389/fpsy.2017.00289> PMID: 29312017
- [98] Lai CM, Mak KK, Pang JS, Fong SSM, Ho RCM, Guldan GS. The associations of sociocultural attitudes towards appearance with body dissatisfaction and eating behaviors in Hong Kong adolescents. *Eat Behav* 2013; 14(3): 320-4. <http://dx.doi.org/10.1016/j.eatbeh.2013.05.004> PMID: 23910774
- [99] Gilbert PA, Zemore SE. Discrimination and drinking: A systematic review of the evidence. *Soc Sci Med* 2016; 161: 178-94. <http://dx.doi.org/10.1016/j.socscimed.2016.06.009> PMID: 27315370
- [100] Hatzenbuehler ML, Keyes KM, Hasin DS. Associations between perceived weight discrimination and the prevalence of psychiatric disorders in the general population. *Obesity (Silver Spring)* 2009; 17(11): 2033-9. <http://dx.doi.org/10.1038/oby.2009.131> PMID: 19390520
- [101] Fairburn CG, Harrison PJ. Eating disorders. *Lancet* 2003; 361(9355): 407-16. [http://dx.doi.org/10.1016/S0140-6736\(03\)12378-1](http://dx.doi.org/10.1016/S0140-6736(03)12378-1) PMID: 12573387
- [102] Verdejo-García AJ, Perales JC, Pérez-García M. Cognitive impulsivity in cocaine and heroin polysubstance abusers. *Addict Behav* 2007; 32(5): 950-66. <http://dx.doi.org/10.1016/j.addbeh.2006.06.032> PMID: 16876962
- [103] Emery RL, King KM, Fischer SF, Davis KR. The moderating role of negative urgency on the prospective association between dietary restraint and binge eating. *Appetite* 2013; 71: 113-9. <http://dx.doi.org/10.1016/j.appet.2013.08.001> PMID: 23938953
- [104] Stice E. Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychol Bull* 2002; 128(5): 825-48. <http://dx.doi.org/10.1037/0033-2909.128.5.825> PMID: 12206196
- [105] Brechan I, Kvalem IL. Relationship between body dissatisfaction and disordered eating: mediating role of self-esteem and depression. *Eat Behav* 2015; 17: 49-58. <http://dx.doi.org/10.1016/j.eatbeh.2014.12.008> PMID: 25574864
- [106] Sonnevile KR, Grilo CM, Richmond TK, *et al.* Prospective association between overvaluation of weight and binge eating among overweight adolescent girls. *J Adolesc Health* 2015; 56(1): 25-9. <http://dx.doi.org/10.1016/j.jadohealth.2014.08.017> PMID: 25438968
- [107] Duarte C, Pinto-Gouveia J, Ferreira C. Expanding binge eating assessment: Validity and screening value of the Binge Eating Scale in women from the general population. *Eat Behav* 2015; 18: 41-7. <http://dx.doi.org/10.1016/j.eatbeh.2015.03.007> PMID: 25880043
- [108] Lupi M, Acciavatti T, Santacroce R, Cinosi E, Martinotti G, Di Giannantonio M. "Drunkorexia": a pilot study in an Italian sample. *Research in Advances in Psychiatric* 2015; 21: 28-32.
- [109] Lupi M, Martinotti G, Di Giannantonio M. Drunkorexia: an emerging trend in young adults. *Eat Weight Disord* 2017; 22(4): 619-22. <http://dx.doi.org/10.1007/s40519-017-0429-2> PMID: 28840571
- [110] Eisenberg MH, Fitz CC. "Drunkorexia": exploring the who and why of a disturbing trend in college students' eating and drinking behaviors. *J Am Coll Health* 2014; 62(8): 570-7. <http://dx.doi.org/10.1080/07448481.2014.947991> PMID: 25102366
- [111] Hunt TK, Forbush KT. Is "drunkorexia" an eating disorder, substance use disorder, or both? *Eat Behav* 2016; 22: 40-5. <http://dx.doi.org/10.1016/j.eatbeh.2016.03.034> PMID: 27085168
- [112] Roosen KM, Mills JS. Exploring the motives and mental health correlates of intentional food restriction prior to alcohol use in university students. *J Health Psychol* 2015; 20(6): 875-86. <http://dx.doi.org/10.1177/1359105315573436> PMID: 26032803
- [113] Giles SM, Champion H, Sutfin EL, McCoy TP, Wagoner K. Calorie restriction on drinking days: an examination of drinking consequences among college students. *J Am Coll Health* 2009; 57(6): 603-9. <http://dx.doi.org/10.3200/JACH.57.6.603-610> PMID: 19433398
- [114] Carter A, Hendrikse J, Lee N, *et al.* The Neurobiology of "Food Addiction" and Its Implications for Obesity Treatment and Policy. *Annu Rev Nutr* 2016; 36(1): 105-28. <http://dx.doi.org/10.1146/annurev-nutr-071715-050909> PMID: 27296500
- [115] Gordon EL, Ariel-Donges AH, Bauman V, Merlo LJ. What is the evidence for "Food Addiction"? A systematic review. *Nutrients* 2018; 10(4): 477. <http://dx.doi.org/10.3390/nu10040477> PMID: 29649120
- [116] Pursey KM, Davis C, Burrows TL. Nutritional Aspects of Food Addiction. *Curr Addict Rep* 2017; 4(2): 142-50. <http://dx.doi.org/10.1007/s40429-017-0139-x>
- [117] Corbit LH, Janak PH. Habitual alcohol seeking: neural bases and possible relations to alcohol use disorders. *Alcohol Clin Exp Res* 2016; 40(7): 1380-9. <http://dx.doi.org/10.1111/acer.13094> PMID: 27223341
- [118] Vengeliene V, Bilbao A, Molander A, Spanagel R. Neuropharmacology of alcohol addiction. *Br J Pharmacol* 2008; 154(2): 299-315. <http://dx.doi.org/10.1038/bjp.2008.30> PMID: 18311194
- [119] Koob GF, Volkow ND. Neurobiology of addiction: a neurocircuitry analysis. *Lancet Psychiatry* 2016; 3(8): 760-73. [http://dx.doi.org/10.1016/S2215-0366\(16\)00104-8](http://dx.doi.org/10.1016/S2215-0366(16)00104-8) PMID: 27475769
- [120] West R, Brown J. Theory of addiction. 2nd ed. Chichester, West Sussex, UK: Wiley Blackwell/Addiction Press 2014.
- [121] Hingson RW, Zha W. Age of drinking onset, alcohol use disorders, frequent heavy drinking, and unintentionally injuring oneself and others after drinking. *Pediatrics* 2009; 123(6): 1477-84.

- <http://dx.doi.org/10.1542/peds.2008-2176> PMID: 19482757
- [122] Schulte EM, Smeal JK, Lewis J, Gearhardt AN. Development of the Highly Processed Food Withdrawal Scale. *Appetite* 2018; 131: 148-54.  
<http://dx.doi.org/10.1016/j.appet.2018.09.013> PMID: 30227182
- [123] Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? *Obes Rev* 2013; 14(1): 19-28.  
<http://dx.doi.org/10.1111/j.1467-789X.2012.01046.x> PMID: 23057499
- [124] del Ciampo LA, del Ciampo IRL. Effects of Alcohol on the Developing Brain: The Adolescent at Risk. *J Adv Med Med Res* 2019; 30(4): 1-8.  
<http://dx.doi.org/10.9734/jammr/2019/v30i430186>
- [125] Iacovino JM, Gredysa DM, Altman M, Wilfley DE. Psychological Treatments for binge eating disorder 2012; 14(4): 432-6.  
<http://dx.doi.org/10.1007/s11920-012-0277-8>
- [126] Kalra EK. Nutraceutical--definition and introduction. *AAPS PharmSci* 2003; 5(3)E25  
<http://dx.doi.org/10.1208/ps050325> PMID: 14621960
- [127] Blüher M. Adipose tissue dysfunction contributes to obesity related metabolic diseases. *Best Pract Res Clin Endocrinol Metab* 2013; 27(2): 163-77.  
<http://dx.doi.org/10.1016/j.beem.2013.02.005> PMID: 23731879
- [128] Scicchitano P, Cameli M, Maiello M, Modesti PA, Muiesan ML, Novo S, *et al.* Nutraceuticals and dyslipidaemia: beyond the common therapeutics. *J Funct Foods* 2014; 6: 11-32.  
<http://dx.doi.org/10.1016/j.jff.2013.12.006>
- [129] Tramontin NDS, Luciano TF, Marques SO, de Souza CT, Muller AP. Ginger and avocado as nutraceuticals for obesity and its comorbidities. *Phytother Res* 2020; 1-9.  
<http://dx.doi.org/10.1002/ptr.6619> PMID: 31989713
- [130] Blanco-Gandía MC, Cantacors L, Aracil-Fernández A, *et al.* Effects of bingeing on fat during adolescence on the reinforcing effects of cocaine in adult male mice. *Neuropharmacology* 2017; 113(Pt A): 31-44.  
<http://dx.doi.org/10.1016/j.neuropharm.2016.09.020> PMID: 27666001
- [131] Rolland B, Naassila M, Duffau C, Houchi H, Gierski F, André J. Binge eating, but not other disordered eating symptoms, is a significant contributor of binge drinking severity: Findings from a cross-sectional study among french students. *Front Psychol* 2017; 8: 1878.  
<http://dx.doi.org/10.3389/fpsyg.2017.01878> PMID: 29163267
- [132] Caton SJ, Ball M, Ahern A, Hetherington MM. Dose-dependent effects of alcohol on appetite and food intake. *Physiol Behav* 2004; 81(1): 51-8.  
<http://dx.doi.org/10.1016/j.physbeh.2003.12.017> PMID: 15059684
- [133] Caton SJ, Marks JE, Hetherington MM. Pleasure and alcohol: manipulating pleasantness and the acute effects of alcohol on food intake. *Physiol Behav* 2005; 84(3): 371-7.  
<http://dx.doi.org/10.1016/j.physbeh.2004.12.013> PMID: 15763574
- [134] Caton SJ, Bate L, Hetherington MM. Acute effects of an alcoholic drink on food intake: aperitif versus co-ingestion. *Physiol Behav* 2007; 90(2-3): 368-75.  
<http://dx.doi.org/10.1016/j.physbeh.2006.09.028> PMID: 17107693
- [135] Westertep-Plantenga MS, Verwegen CRT. The appetizing effect of an aperitif in overweight and normal-weight humans. *Am J Clin Nutr* 1999; 69(2): 205-12.  
<http://dx.doi.org/10.1093/ajcn/69.2.205> PMID: 9989681
- [136] Yeomans MR. Alcohol, appetite and energy balance: is alcohol intake a risk factor for obesity? *Physiol Behav* 2010; 100(1): 82-9.  
<http://dx.doi.org/10.1016/j.physbeh.2010.01.012> PMID: 20096714
- [137] de Castro JM, Orozco S. Moderate alcohol intake and spontaneous eating patterns of humans: evidence of unregulated supplementation. *Am J Clin Nutr* 1990; 52(2): 246-53.  
<http://dx.doi.org/10.1093/ajcn/52.2.246> PMID: 2375290
- [138] de Castro JM. When, how much and what foods are eaten are related to total daily food intake. *Br J Nutr* 2009; 102(8): 1228-37.  
<http://dx.doi.org/10.1017/S0007114509371640> PMID: 19650955
- [139] Raben A, Agerholm-Larsen L, Flint A, Holst JJ, Astrup A. Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *Am J Clin Nutr* 2003; 77(1): 91-100.  
<http://dx.doi.org/10.1093/ajcn/77.1.91> PMID: 12499328
- [140] Rödmark S, Calissendorff J, Brismar K. Alcohol ingestion decreases both diurnal and nocturnal secretion of leptin in healthy individuals. *Clin Endocrinol (Oxf)* 2001; 55(5): 639-47.  
<http://dx.doi.org/10.1046/j.1365-2265.2001.01401.x> PMID: 11894976
- [141] Caton SJ, Nolan LJ, Hetherington MM. Alcohol, Appetite and Loss of Restraint. *Curr Obes Rep* 2015; 4(1): 99-105.  
<http://dx.doi.org/10.1007/s13679-014-0130-y> PMID: 26627094
- [142] Berridge KC. 'Liking' and 'wanting' food rewards: brain substrates and roles in eating disorders. *Physiol Behav* 2009; 97(5): 537-50.  
<http://dx.doi.org/10.1016/j.physbeh.2009.02.044> PMID: 19336238
- [143] Melis M, Diana M, Enrico P, Marinelli M, Brodie MS. Ethanol and acetaldehyde action on central dopamine systems: mechanisms, modulation, and relationship to stress. *Alcohol* 2009; 43(7): 531-9.  
<http://dx.doi.org/10.1016/j.alcohol.2009.05.004> PMID: 19913196
- [144] Schrieks IC, Staffeu A, Griffioen-Roose S, *et al.* Moderate alcohol consumption stimulates food intake and food reward of savoury foods. *Appetite* 2015; 89: 77-83.  
<http://dx.doi.org/10.1016/j.appet.2015.01.021> PMID: 25636235
- [145] Eiler WJA II, Džemidžić M, Case KR, *et al.* The aperitif effect: Alcohol's effects on the brain's response to food aromas in women. *Obesity (Silver Spring)* 2015; 23(7): 1386-93.  
<http://dx.doi.org/10.1002/oby.21109> PMID: 26110891
- [146] Tremblay A, Wouters E, Wenker M, St-Pierre S, Bouchard C, Després JP. Alcohol and a high-fat diet: a combination favoring overfeeding. *Am J Clin Nutr* 1995; 62(3): 639-44.  
<http://dx.doi.org/10.1093/ajcn/62.3.639> PMID: 7661127
- [147] Measelle JR, Stice E, Hogansen JM. Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *J Abnorm Psychol* 2006; 115(3): 524-38.  
<http://dx.doi.org/10.1037/0021-843X.115.3.524> PMID: 16866592
- [148] Fairburn CG, Cooper Z. *The eating disorder examination. Binge eating: Nature, assessment and treatment.* 12th ed. New York: Guilford Press 1993; pp. 317-60.
- [149] Stice E, Barrera M Jr, Chassin L. Prospective differential prediction of adolescent alcohol use and problem use: examining the mechanisms of effect. *J Abnorm Psychol* 1998; 107(4): 616-28.  
<http://dx.doi.org/10.1037/0021-843X.107.4.616> PMID: 9830249
- [150] Micali N, Solmi F, Horton NJ, *et al.* Adolescent Eating Disorders Predict Psychiatric, High-Risk Behaviors and Weight Outcomes in Young Adulthood. *J Am Acad Child Adolesc Psychiatry* 2015; 54(8): 652-659.e1.  
<http://dx.doi.org/10.1016/j.jaac.2015.05.009> PMID: 26210334
- [151] Cooper SJ. Palatability-dependent appetite and benzodiazepines: new directions from the pharmacology of GABA(A) receptor subtypes. *Appetite* 2005; 44(2): 133-50.  
<http://dx.doi.org/10.1016/j.appet.2005.01.003> PMID: 15808888
- [152] Blanco-Gandía MC, Ledesma JC, Aracil-Fernández A, *et al.* The rewarding effects of ethanol are modulated by binge eating of a high-fat diet during adolescence. *Neuropharmacology* 2017; 121: 219-30.  
<http://dx.doi.org/10.1016/j.neuropharm.2017.04.040> PMID: 28457972
- [153] Puccio F, Fuller-Tyszkiewicz M, Ong D, Krug I. A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *Int J Eat Disord* 2016; 49(5): 439-54.  
<http://dx.doi.org/10.1002/eat.22506> PMID: 26841006

Annex 2: Study 2 article. The Binge Eating Scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index.







# The Binge Eating Scale: Structural Equation Competitive Models, Invariance Measurement Between Sexes, and Relationships With Food Addiction, Impulsivity, Binge Drinking, and Body Mass Index

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**Introduction:** The Binge Eating Scale (BES) is a widely used self-report questionnaire to identify compulsive eaters. However, research on the dimensions and psychometric properties of the BES is limited.

**Objective:** The aim of this study was to examine the properties of the Spanish version of the BES.

**Methods:** Confirmatory Factor Analyses (CFAs) were carried out to verify the BES factor structure in a sample of Spanish college students ( $N = 428$ , 75.7% women; age range = 18–30). An invariance measurement routine was carried out across sexes, the latent means were compared, and estimates of reliability and convergent and discriminant validity were presented.

**Results:** A one-factor model fit the data best and was also equivalent between sexes. The scalar invariance model showed statistically significant differences across sexes, with a higher prevalence in women. Regarding reliability, the results were excellent. Finally, high statistically significant correlations were obtained with other measures of binge eating (BE), food addiction, impulsivity, binge drinking, and body mass index (BMI).

**Conclusion:** The Spanish 16-item BES is a valid and reliable scale to evaluate BE in the youth population.

**Keywords:** binge eating, psychometric properties, confirmatory factor analysis, college students, sexes, convergent validity

**Abbreviations:** BE, binge eating; BES, Binge Eating Scale; BIS-15S, the Barratt Impulsiveness Scale-15S; BMI, body mass index; CFAs, confirmatory factor analyses; CFI, comparative fit index; EDI-3, Eating Disorder Inventory-3; FIML, full information maximum likelihood; mYFAS, Modified Yale Food Addiction Scale; RMSEA, root mean squared error of approximation; WLSMV, weighted least squares means and variances; YFAS, Yale Food Addiction Scale.

## INTRODUCTION

Binge eating has become a serious problem worldwide with numerous consequences for general and clinical populations (Kessler et al., 2013). BE is characterized by the appearance of episodes of compulsive eating (BE) with two essential aspects, the ingestion of an excessive amount of food in a short time and a feeling of loss of control over eating. BE is also accompanied by emotional distress and the absence of compensatory behaviors (which are characteristic of bulimia nervosa) (American Psychiatric Association, 2013).

Binge eating is also common in the community population, and its prevalence has increased over time (de Freitas et al., 2008; Smink et al., 2014). In fact, international research reports very high BE prevalence rates in adolescents and young adults (Ribeiro et al., 2014; Goldschmidt et al., 2015), with higher prevalence rates in women than in men (Preti et al., 2009; Kessler et al., 2013). Croll et al. (2002) reported that 26% of young women and 13% of young men have experienced an episode of BE once in the past year. In turn, Smink et al. (2014) recently showed two peaks in the onset of BE, the first one immediately after puberty at an average age of 14, and the second one at the end of adolescence (19–24 years) (Stice et al., 2013).

Several studies offer evidence suggesting that the BE disorder is associated with psychiatric comorbidities. Thus, a large amount of research has established a well-defined relationship between BE and addiction disorders. In particular, recent research has found a positive correlation between BE (BES) and food addiction (YFAS) in both clinical (Imperatorii et al., 2014) and non-clinical populations (Burrows et al., 2017). In addition, recent research also indicates the presence of comorbidity between BE and the use of alcohol and binge drinking in young people (Kessler et al., 2013; Laghi et al., 2014; Fouladi et al., 2015). Impulsivity has also been positively associated with BE (Steward et al., 2017; Mason et al., 2018) and could play an important role in the comorbidity between disordered eating behavior and excessive alcohol consumption in young students (Ocampo et al., 2012). Finally, a significant positive association between BE and BMI and obesity has consistently been observed. Evidence to date suggests that the relationship between BE and BMI and obesity occurs in both clinical and non-clinical populations (Hudson et al., 2007; Villarejo et al., 2012; Kessler et al., 2013; Duarte et al., 2015; Duncan et al., 2017; Mustelin et al., 2017). Psychiatric comorbidity with BE can increase its severity, chronicity, and resistance to any type of psychiatric treatment, and it has been associated with numerous medical conditions, a deterioration in quality of life, a greater risk of weight gain and obesity, and increased medical mortality (Ocampo et al., 2012; Kessler et al., 2013; Thornton et al., 2017).

The high prevalence of BE and the problems it causes have led to a need to establish instruments for its measurement. In this regard, several self-report questionnaires have been developed. Gormally et al. (1982) developed the BES, Yanovski (1993) developed the Questionnaire on Eating and Weight Patterns-R (QEWP-R), and Fairburn and Beglin (1994) developed the Eating Disorder Examination Questionnaire (EDE-Q). These instruments have been widely used to assess BE.

The BES (Gormally et al., 1982) was designed as a measure of severity (vs. diagnosis) of BE, with the additional property of evaluating its affective, cognitive, and behavioral manifestations. Research on the BES scale stems from its outstanding role as a screening measure in clinical (Freitas et al., 2006) and non-clinical populations (Duarte et al., 2015) to evaluate BE severity and intervention outcomes (Telch et al., 2001; Katterman et al., 2014). Studies carried out in the past decade, mainly with obese patients, have shown that the BES is very sensitive and specific in distinguishing between compulsive and normal eaters (Freitas et al., 2006; Grupski et al., 2013). In addition, a large number of investigations have confirmed that the BES shows good validity in both general (Meno et al., 2008; Gordon et al., 2012; Duarte et al., 2015) and clinical populations (Zúñiga and Robles, 2006; Dezhkam et al., 2009; Hood et al., 2013).

Despite the relevance of the BES in eating-disorder research, its factor structure is still controversial. Gormally et al. (1982) originally proposed a two-factor structure, dividing the items into cognitive and behavioral BE. Since then, the scale has been validated in French, Portuguese, English, Italian, Malay, and Spanish (Mexican) (Gormally et al., 1982; Ricca et al., 2000; Freitas et al., 2006; Zúñiga and Robles, 2006; Robert et al., 2013; Duarte et al., 2015; Brunault et al., 2016). Of these studies, only four have studied the factorial structure of the BES, with differing results. In the Mexican study, the only one carried out in a Spanish-speaking sample, the authors found a two-factor structure through exploratory means (Zúñiga and Robles, 2006). This structure, however, presented some problems because several items showed important cross-loadings and/or higher loadings in a different factor from the expected one. Similar results for the internal structure were presented in the Malay version (Robert et al., 2013). The authors tested the BES factorial structure with an exploratory factor analysis and posited a two-factor structure as the best solution for the data. Major problems of this study included retaining a factor that explained only 8.15% of the variance and, again, using varimax rotation. However, when the authors studied the sensitivity, specificity, and reliability of the BES, they found a unidimensional structure. More recently, Duarte et al. (2015) and Brunault et al. (2016) provided evidence of a one-factor structure in a sample of Portuguese women from the general population and in French non-clinical and clinical populations, respectively. Using CFA, Duarte et al. (2015) found a good fit for a one-factor structure of the BES, with appropriate reliability estimates for both the scale and the items, and good convergent validity. The Portuguese version of the BES was found to have high test-retest reliability. Brunault et al. (2016) also provided evidence of a one-factor structure by means of exploratory factor analysis. Again, reliability estimates were adequate. Previous studies carried out by Ricca et al. (2000) and Freitas et al. (2006), although not focused on the factorial structure, studied the sensitivity and specificity of the BES in Italian and Portuguese samples, respectively, with appropriate results when used as a unidimensional diagnostic instrument. Other recent studies focused on the BES factorial structure are presented by Imperatori et al. (2016) and Marek et al. (2016). The study by Marek et al. (2016) reported a

good fit for a bifactorial solution, based on previous results found by Hood et al. (2013). However, these authors found a lack of incremental validity for the behavioral manifestation factor, and modest evidence for the feelings/cognitions factor. Therefore, Marek et al. (2016) defended the use of the BES as a unidimensional measure of BE severity. Imperatori et al. (2016), in the same direction, tested both the one-factor and competing two-factor models and found a comparable fit to the data. Therefore, they defended the one-factor model as the most parsimonious one.

The BES questionnaire has been widely used, but research on the factors and properties of the BES in the general population is still quite limited. In particular, no study has examined the psychometric properties of the BES in general populations of young men and women, and specifically, in the Spanish general population. In addition, results on its dimensionality are contradictory, and most research has examined its validity in specific samples, such as clinical samples or samples of women, especially obese women who undergo bariatric surgery to lose weight (Hood et al., 2013; Marek et al., 2016), or obese and overweight patients seeking weight loss treatment (Imperatori et al., 2016). There is a need for more evidence about the factorial structure of the scale and additional psychometric properties in non-clinical samples.

## Aims

The aim of this study is to examine the psychometric properties of the BES in a large sample of Spanish university students, following several steps: (1) to study the factor structure by means of competitive structural equation models, specifically CFA; (2) to test the measurement invariance of the BES between sexes; (3) to offer evidence of its reliability; and (4) to investigate its convergent and discriminant validity by describing its relationships with variables that have been associated with the BES, such as BE behavior, food addiction, impulsivity, use of alcohol/binge drinking, and BMI, as found in other studies (Fouladi et al., 2015; Burrows et al., 2017; Mustelin et al., 2017; Mason et al., 2018).

## MATERIALS AND METHODS

### Sample

The sample consisted of 428 Spanish university students who voluntarily took part in the present study. The sample comprised female university students ( $n = 324$ , 75.7%), with an average age of 21.04 years ( $SD = 4.22$ ), and male university students ( $n = 104$ ; 24.3%), with an average age of 22.27 years ( $SD = 5.39$ ). Thirty-four participants (7.9%) were underweight ( $BMI < 18.5$ ), 330 (77.1%) had normal weight ( $18.5 \leq BMI \leq 24.99$ ), 55 (12.9%) were overweight ( $25 \leq BMI \leq 29.99$ ), and 11 (2.1%) were obese ( $BMI \geq 30$ ), according to the World Health Organization [WHO] (2000). Two participants did not respond. Female students had a mean BMI of 21.91 ( $SD = 2.98$ ), and male students had a mean BMI of 23.46 ( $SD = 2.84$ ). More information can be found in **Table 1**.

**TABLE 1 |** Sociodemographic and clinical data.

	Female		Male	
	N	%	N	%
Sex	324	75.7	104	24.3
	M	SD	M	SD
Age	21.04	4.22	22.27	5.39
Weight (in kg)	58.57	8.91	73.76	9.28
Height (in cm)	1.63	0.06	1.77	0.07
BES	8.35	6.57	22.27	5.39
BE disorder	0.65	0.89	0.78	1.00
Food addiction	5.81	4.56	5.30	4.94
Motor impulsiveness	4.46	4.01	5.37	4.40
Non-planning	7.78	4.73	9.16	5.06
Attention	7.28	4.37	8.44	4.46
Binge drinking	0.46	0.69	0.52	0.66
BMI	21.91	2.98	23.46	2.84

*M*, mean; *SD*, standard deviation; *BES*, Binge Eating Scale score; *BE disorder*, binge eating disorder as surveyed in EDI-3: "Have you ever engaged in binge eating (eaten a lot of food and felt like you couldn't stop eating)?" ; *BMI*, body mass index.

## Measures

### The Binge Eating Scale (BES; Gormally et al., 1982)

The BES is a self-administered questionnaire composed of 16 items: eight items that describe behavioral manifestations (for example, eating fast or consuming large amounts of food) and eight items on associated feelings and cognitions (for example, fear of not stopping eating). Each item has a response range from 0 to 3 points (0 = no severity of the BES symptoms, 3 = serious problems on the BES symptoms). Marcus et al. (1988) created a range of scores for the BES from 0 to 46 points: a score of less than 17 points indicates minimal BE problems; a score between 18 and 26 points indicates moderate BE problems, and a score of more than 27 points indicates severe BE problems. Psychometric properties of the BES in the Spanish population are considered in this study.

In this investigation, the Mexican version of the BES validated in the Spanish language was used (Zúñiga and Robles, 2006). This version of the scale was subjected to a rigorous cultural adaptation procedure. First, a Spanish–English bilingual translator who was not familiar with the questionnaire reviewed the translation. Second, a native Spanish speaker who knew the purpose of the study reviewed the translated BES elements. Later, we evaluated whether the scale items were understood properly by administering the BES to forty Spanish university students. The objective was to confirm that it was a simple scale for the general young population to understand. The Spanish version of the BES was an exact translation of the original English version; therefore, the decision was made to use the same scale. The final version of the BES and its instructions are contained in the **Annex**.

### The Eating Disorder Inventory-3 (EDI-3; Clausen et al., 2011)

The EDI-3 consists of a brief self-report questionnaire designed to evaluate the risk variables and other variables associated with

eating behavior disorders. It consists of 91 items grouped in 12 subscales. Participants completed all 26 items on the scale (drive for thinness, bulimia, body dissatisfaction, and BE disorder). In the present study, only item 26, measuring BE behavior, was used in this validation: “*Have you ever engaged in binge eating (eaten a lot of food and felt like you couldn’t stop eating)?*” The participants responded to the items on a 6-point Likert scale (0 = never; 6 = always). In this study, the validated Spanish version of the EDI-3 was used (Elosua and López-Jáuregui, 2012). The alphas for the complete scale in this sample were 0.903 for drive for thinness, 0.813 for bulimia, and 0.747 for body dissatisfaction.

### The Modified Yale Food Addiction Scale (mYFAS; Flint et al., 2014)

The mYFAS (short version of the YFAS) is a brief, self-administered instrument designed to assess the signs of addictive eating behavior. It is composed of nine items, seven that evaluate the diagnostic criteria for food addiction, and two that assess clinically significant deterioration and distress. The questionnaire uses a Likert rating scale (0 = never; 4 = four or more times or daily). The YFAS had good psychometric properties in the general population (Gearhardt et al., 2009; Pedram et al., 2013). In this study, the nine items from the validated Spanish version of the YFAS (Granero et al., 2014) were extracted. The internal consistency coefficient found for the Spanish version of the mYFAS in this study was  $\alpha = 0.769$ .

### Barratt Impulsiveness Scale-15S (BIS-15S; Spinella, 2007)

The BIS-15 is a brief, self-administered scale consisting of 15 items subdivided in three dimensions (motor, non-planning, and attention) that evaluate impulsivity. Items are scored on a 4-point Likert scale (0 = rarely, 4 = always or almost always). In the present study, the validated Spanish version (BIS-15S) was used (Orozco-Cabal et al., 2010), which contains good psychometric qualities in terms of internal consistency, temporal stability, and internal structure. Estimates of internal consistency in the present study were 0.783 for motor impulsivity, 0.756 for non-planned impulsivity, and 0.701 for attentional impulsivity.

### Binge Drinking

We used the most internationally supported measure to assess binge drinking. The participants answered the following question: “*Taking into account all types of alcoholic beverages, did you consume five or more drinks in a row (four if you are a woman) on at least one occasion in the past month? How many times in the past month?*” The participants responded on a 4-point Likert scale (0 = never; 4 = four or more times a week) (Kuntsche et al., 2006; Paul et al., 2011).

### Body Mass Index (BMI)

The BMI was calculated by dividing the weight in kilograms by the square of the height in meters ( $BMI = \text{weight [kg]} / \text{height [m}^2\text{]}$ ) (World Health Organization [WHO], 2000).

### Additional Information

Participants were required to provide data about their sex, age, education level, country of residence, weight (in kg), and height (in cm), along with filling out the other questionnaires.

### Procedure

Most of the participants were recruited in the classrooms of the University of Valencia, and some of them by email and social networks. The participants were informed about the study design, the voluntary nature of their participation, and the confidentiality of the data obtained. Thus, they were informed that the questionnaires would be answered online through a computer or mobile phone. Once they had received this information, the participants delivered their informed consent on paper and, subsequently, were provided with a link to access the online survey. Participants were asked to answer the online survey. The survey was conducted using the Lime Survey web platform<sup>1</sup>, where the participants provided demographic data and answered the measures mentioned in the previous section. Later, a blind evaluator analyzed the self-reported responses. The data collection followed the ethical standards. The study was approved by the Ethics Committee of the University of Valencia and was carried out in accordance with the ethical standards of the 1964 Declaration of Helsinki (Procedure number: H1513854038939).

### Statistical Analyses

The first step consisted of the study of factorial validity using CFA. The two structures found in the literature were tested: a one-factor model (BE) and a model with two correlated factors (behavioral and emotional/cognitive BE). Model fit was assessed using the chi square statistic, the CFI, with values of more than 0.90 (ideally 0.95) indicating good fit, and the RMSEA, with values of 0.08 or less for an excellent fit (Hu and Bentler, 1999). For model fit comparison, CFI differences were estimated. Whereas Little (1997) argued that CFI differences of 0.05 could be considered negligible, Cheung and Rensvold (2002) recommended a more restrictive cut-off point of 0.01. Both contributions were taken into account in the current research.

Once the best fitting model had been retained, the structure was tested separately in samples of women and men. Because the model fitted both sets of data adequately, the invariance routine was developed. Measurement invariance of factor loadings, intercepts, and means were tested, as recommended by Thompson and Green (2006) and van de Schoot et al. (2012). First, we tested the configural model, in which a model with a one-factor structure was estimated in the two samples, women and men. This model is also called the baseline model because its fit is used as the baseline fit with which the other models are compared. Second, we evaluated weak or metric invariance. In this model, factor loadings are constrained across samples; that is, they become the same for men and women. When metric invariance holds, it means that both women and men attribute the same meaning to BE. Third, we tested strong or scalar invariance. In the scalar invariance model, the intercepts are constrained across samples. If the model is tenable, it means

<sup>1</sup>encuestas.uv.es



that the meaning of BE (the factor loadings) and the intercepts are equal across groups. Finally, because scalar invariance was found, we constrained latent means across samples. If this last model fits, it means that levels of BE are equal across sexes. The models were compared using both chi-square differences and CFI differences.

Due to the ordinal nature of the BES items, we employed WLSMV as the estimation method, as recommended by Muthén and Muthén (1998), Flora and Curran (2004), and Brown (2006).

Evidence of reliability of the proposed structure was also gathered: Cronbach's alpha, McDonald's omega, and the items' homogeneity were estimated for both samples.

Finally, evidence of external validity was gathered by calculating the Pearson correlation between the total score on the BES and other related variables, such as an indicator of BE obtained from the EDI-3 (specifically, item number 26, "Have you binge eaten (eaten a lot of food and felt like you couldn't stop eating?"), food addiction, impulsivity, binge drinking, and BMI.

Analyses were performed using Mplus version 8 (Muthén and Muthén, 1998) and SPSS version 24 software. Missing data were dealt with using FIML, which is the most recommended method for structural equation modeling (Finney and DiStefano, 2013).

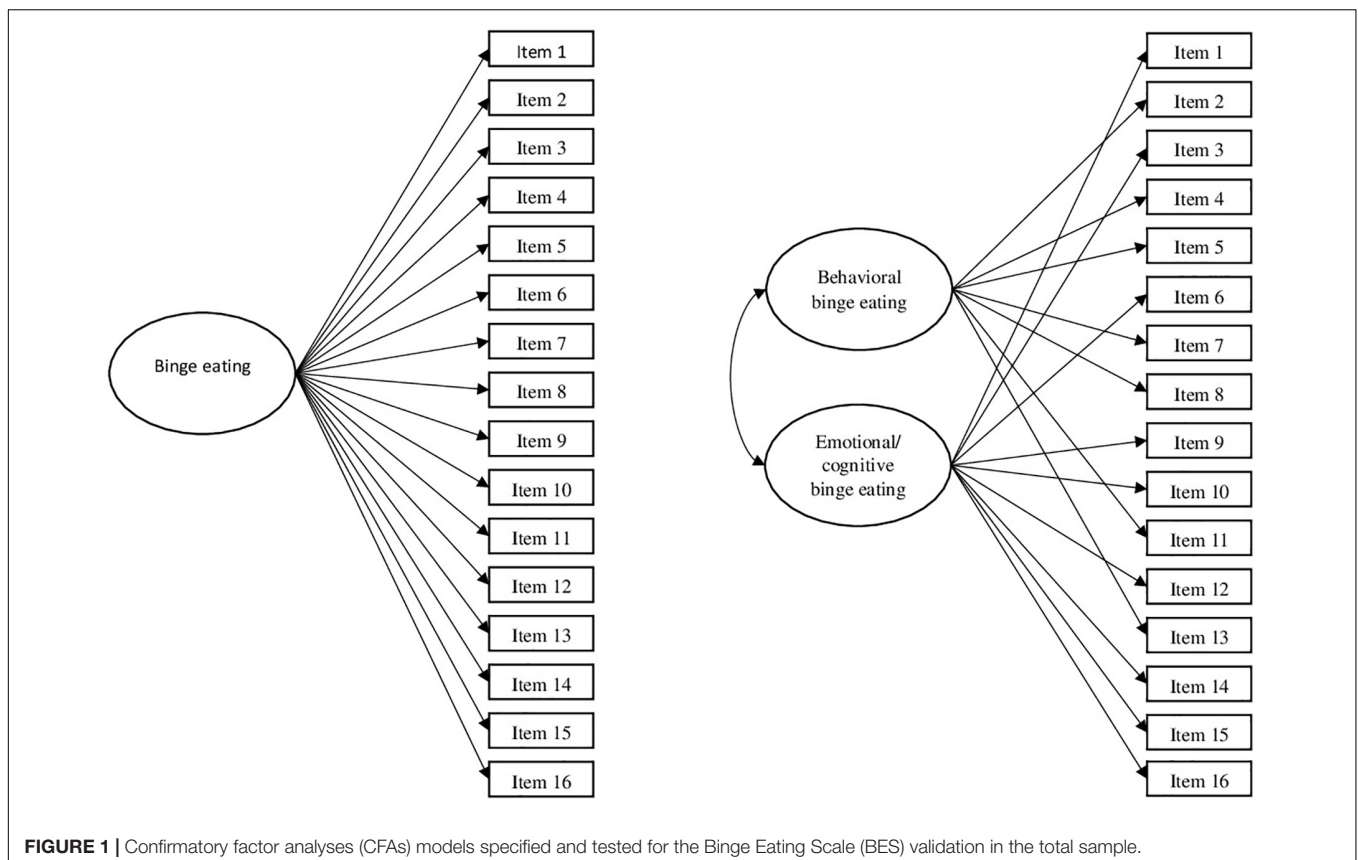
## RESULTS

Two competitive CFA, with the structures shown in **Figure 1**, were specified, estimated, and tested in the total sample. **Table 2**

shows the fit indexes for these models. Fit was excellent for both models (see **Table 2**). However, the correlation found between the two factors in the two-factor model was extremely high ( $r = 0.925 [0.912, 0.938]$ ), and thus showed no discriminant validity. The CFI difference was not large ( $\Delta CFI = 0.004$ ); it was smaller than the one recommended by Little (1997) and the more restrictive one proposed by Cheung and Rensvold (2002). Taking all of this information into account, the one-factor solution, the most parsimonious one, was retained as the best representation of the data.

Once the one-factor model had been retained, the structure was tested separately in samples of women and men. The CFA of the BES tested in the samples of women and men separately showed an adequate fit (see **Table 2**).

Regarding the invariance routine results, the configural model fitted the data adequately (see **Table 2**). Thus, it was retained as the baseline model. When metric invariance was tested, statistically significant differences were found between the chi-squares, but the CFI and the RMSEA improved. Taking into account that in large samples the power of the chi square statistic to detect minor deviations is high (Fan et al., 1999), and the rest of the indices improved when factor loadings were constrained in the men's sample, the metric invariance model was retained. Then, scalar invariance was tested, and again statistically significant differences were found between the chi-squares, but with a trivial decrease in the CFI (0.001) and an improvement in the RMSEA. Once again, the evidence guaranteed scalar



**TABLE 2 |** Confirmatory factor analyses and set of nested models to test for measurement invariance.

	$\chi^2$	df	p	CFI	RMSEA	RMSEA CI	$\Delta$ CFI	$\Delta\chi^2$	$\Delta$ df	p
One-factor model in the total sample	374.349	104	< 0.001	0.939	0.076	[0.067, 0.084]	–	–	–	–
Two-factor model in the total sample	359.458	103	< 0.001	0.943	0.074	[0.066, 0.082]	0.004	–	–	–
One-factor model in women's sample	321.494	104	< 0.001	0.935	0.080	[0.070, 0.090]	–	–	–	–
One-factor model in men's sample	134.841	104	0.022	0.975	0.053	[0.021, 0.077]	–	–	–	–
Configural invariance	430.278	208	< 0.001	0.947	0.071	[0.061, 0.080]	–	–	–	–
Metric invariance	433.573	223	< 0.001	0.950	0.066	[0.057, 0.076]	0.003	35.993	15	0.001
Scalar invariance	485.085	270	< 0.001	0.949	0.061	[0.052, 0.070]	0.001	76.228	47	0.004
Scalar invariance with constrained latent means	547.693	271	< 0.001	0.934	0.069	[0.061, 0.077]	–0.015	16.186	1	< 0.001

CFI, comparative fit index; RMSEA, root mean squared error of approximation; RMSEA CI, RMSEA 90% confidence interval.

invariance. Given that the BES has been found to be metric invariant, mean comparisons are meaningful, and they can be made at the latent level. When men's mean on the BE factor was constrained to women's, significant differences between the chi-squares were found, along with a significant decrease in the CFI (0.015) and an increase in the RMSEA. Thus, the scalar invariance model was retained as the most parsimonious model, and its factor loadings and intercepts are presented in **Table 3**. Regarding the level of BE, women showed higher levels: mean difference = 0.548, standard error = 0.144,  $p < 0.001$ , Cohen's  $d = 0.548$ .

Evidence of the reliability and internal consistency of the Spanish version of the BES was provided at scale and item levels. Cronbach's alpha was 0.869, and McDonalds' omega was 0.915, indicating appropriate reliability estimates for the scale. Descriptive statistics, item homogeneity, alpha if item-deleted,

and inter-item correlations for the unidimensional model in the total sample are presented in **Table 4**.

Finally, the results pointed to adequate convergent validity of the scale in the current sample, with a positive, high, and statistically significant correlation between the BES and the BE indicator from the EDI-3 ( $r = 0.621, p < 0.001$ ). Regarding other related variables, relations were also positive and statistically significant with the dimensions of impulsivity: motor ( $r = 0.202, p < 0.001$ ), non-planned ( $r = 0.164, p = 0.001$ ), and attentional ( $r = 0.284, p < 0.001$ ); food addiction ( $r = 0.761, p < 0.001$ ); the binge-drinking indicator ( $r = 0.139, p = 0.023$ ); and BMI ( $r = 0.243, p < 0.001$ ).

## DISCUSSION

Binge eating is a problematic clinical condition in young people. Studies have shown that youth is a critical stage for the onset of eating disorders, with the highest prevalence of the BE disorder at the beginning of young adulthood. The BES scale is one of the most widely used measures for screening and evaluating BE in both clinical and non-clinical samples. However, the factorial structure of the BES in the general population of men and women has not yet been evaluated.

The present study aimed to examine the psychometric properties of the Spanish version of the BES in young populations of men and women using several approaches. First, evidence of its factorial structure was gathered, following a competitive models approach. Second, and once the BES internal structure had been established, its measurement invariance was tested across sexes. Third, estimates of reliability were calculated. Finally, evidence of both convergent and discriminant validity was provided. These steps will guide the discussion.

Regarding evidence of the factorial structure, previous scientific evidence was taken into account, and both the one- and two-dimensional structures of the BES were tested. Our results supported the unidimensionality of the scale, with the best fitting model being the simplest structure. This result is consistent with recent results by Duarte et al. (2015) and Brunault et al. (2016), who also found a one-factor structure in the Portuguese and French versions of the scale. However, the previous study in the Spanish language had pointed to a structure with two-correlated factors (Zúñiga and Robles, 2006),

**TABLE 3 |** Unstandardized and standardized factor loadings and intercept thresholds.

BES items	Factor loadings			Intercepts		
	UN	ST women	ST men	$\nu_1$	$\nu_2$	$\nu_3$
		sample	sample			
1	0.638	0.538	0.615	–0.386	0.944	2.755
2	0.567	0.492	0.570	0.020	0.514	2.094
3	1.162	0.758	0.818	0.576	1.929	3.732
4	0.680	0.562	0.640	–1.086	0.776	2.145
5	0.936	0.683	0.753	–0.253	1.823	3.326
6	0.845	0.646	0.719	0.187	1.921	3.318
7	1.240	0.778	0.835	1.409	2.533	3.551
8	0.863	0.653	0.726	–0.711	1.148	2.961
9	0.795	0.622	0.697	0.508	1.472	2.225
10	1.525	0.836	0.881	0.508	2.627	4.100
11	1.124	0.747	0.809	–0.048	2.431	3.834
12	0.646	0.543	0.620	0.802	1.748	2.635
13	0.380	0.355	0.422	0.148	1.391	1.729
14	1.086	0.736	0.799	–0.153	1.393	3.024
15	0.746	0.598	0.674	–0.285	1.099	2.141
16	0.637	0.537	0.615	0.665	1.835	2.992

UN, factor loading unstandardized estimates (constrained to equality across samples); ST, factor loading standardized estimates;  $\nu$ , intercept threshold.

**TABLE 4** | Means, standard deviations, item-adjusted total correlations, and alpha if item deleted for the one-factor model of the BES.

BES items	Total sample		Women sample				Men sample				$r_{it}$	$\alpha_{id}$
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	Skewness	Kurtosis	<i>M</i>	<i>SD</i>	Skewness	Kurtosis		
1	1.82	0.78	1.88	0.77	0.31	-0.92	1.53	0.68	1.07	0.71	0.470	0.863
2	1.80	0.94	1.81	0.94	0.51	-1.36	1.78	0.95	0.85	-0.48	0.389	0.870
3	1.43	0.68	1.45	0.69	1.33	0.78	1.35	0.63	1.82	3.00	0.625	0.856
4	2.07	0.74	2.14	0.72	0.42	0.23	1.81	0.77	0.58	-0.30	0.482	0.863
5	1.63	0.64	1.69	0.65	0.55	-0.07	1.43	0.61	1.38	2.03	0.570	0.859
6	1.48	0.63	1.55	0.63	0.80	-0.05	1.25	0.55	2.41	6.48	0.493	0.862
7	1.23	0.57	1.24	0.56	2.51	6.37	1.20	0.59	3.24	10.43	0.551	0.860
8	1.86	0.72	1.87	0.71	0.39	-0.30	1.81	0.74	0.45	-0.60	0.565	0.859
9	1.46	0.78	1.48	0.80	1.71	2.22	1.45	0.76	1.57	1.56	0.487	0.863
10	1.42	0.64	1.47	0.67	1.34	1.41	1.32	0.56	1.87	4.29	0.661	0.855
11	1.53	0.60	1.55	0.59	0.66	-0.03	1.47	0.62	1.21	1.62	0.612	0.858
12	1.31	0.62	1.33	0.62	1.93	3.38	1.25	0.63	2.75	7.35	0.445	0.864
13	1.55	0.77	1.58	0.78	1.45	1.85	1.50	0.80	1.82	3.09	0.309	0.871
14	1.69	0.78	1.76	0.78	0.71	-0.20	1.41	0.71	1.58	1.48	0.614	0.856
15	1.75	0.82	1.84	0.82	0.73	-0.09	1.55	0.79	1.44	1.57	0.511	0.862
16	1.33	0.59	1.35	0.59	1.55	1.78	1.25	0.58	2.53	6.31	0.432	0.865

*M*, mean; *SD*, standard deviation;  $r_{it}$ , item-adjusted total correlation;  $\alpha_{id}$ , alpha if item deleted.

also found in the Malay version (Robert et al., 2013). Although this was the original authors' approach (Gormally et al., 1982), when tested here the results showed a high correlation between behavioral and cognitive BE. Thus, taking into account the trivial differences found in the models' fit, we defend the one-dimensional structure.

Once the factor structure had been established, an invariance measurement routine was carried out in order to test whether the scale was invariant across sexes. The best fitting and more parsimonious model was the scalar invariant model. Thus, our results provide evidence of the absence of measurement bias when groups of women and men are compared. In the context of eating disorders, where women and men are usually viewed as different populations, measurement invariance becomes a core issue in making group comparisons (Kline, 2015). However, this condition is assumed, rather than tested, in most of the research carried out with the BES. In the original work, for instance, Gormally et al. (1982) tested mean differences across two samples (one with only females, and the other with females and males), but without offering evidence of BES invariance. In the same direction, Ricca et al. (2000) compared men's and women's scores on the BES, but with no previous test of measurement invariance, that is, with no guarantee of the absence of measurement bias.

The next step in the research, because scalar invariance held, was to compare latent means, and this was done by testing an additional model in which latent means were constrained. The model fit significantly decreased, and, thus, the results pointed to the existence of differences between means. Indeed, the scalar invariance model showed statistically significant differences between women and men, with a medium-sized difference, favoring the group of women. This result agrees with previous research that has revealed higher prevalence and mean scores on binge behavior in women (Ricca et al., 2000; Preti et al., 2009;

Kessler et al., 2013). However, this is the first time this model has been tested in a latent mean context, that is, in an error-free measurement context.

Finally, estimates of reliability and convergent and discriminant validity are also provided. Regarding reliability, the results were excellent. In the case of convergent and discriminant validity, our evidence matched previous findings perfectly, with high and statistically significant correlations between the BES scores and the BE indicator from the EDI-3 and food addiction, and statistically significant but lower correlation values between the BES and the dimensions of impulsivity, binge drinking, and BMI (Freitas et al., 2006; Villarejo et al., 2012; Duncan et al., 2017; Steward et al., 2017; Mason et al., 2018). Our findings are also consistent with those presented by Imperatori et al. (2014) and Burrows et al. (2017) suggesting a strong association between BE and food addiction, and they corroborate previous evidence on the association between BE and alcohol/binge drinking (Kessler et al., 2013; Laghi et al., 2014; Fouladi et al., 2015). These results show the important clinical implications of understanding the relationship between BE and addictive behaviors, such as knowing what mechanisms underlie the appearance and development of these behaviors.

In summary, our findings indicate that the BES shows adequate psychometric properties when used in samples of Spanish females and males from a youth population. Indeed, this is the first time two competitive models have been tested for the internal structure of the BES, with evidence suggesting a one-dimensional structure, consistent with DSM-5 criteria (BE is defined by the rapid ingestion of an excessive amount of food and the loss of control over that ingestion, with discomfort with regard to BE and the absence of compensatory behaviors; American Psychiatric Association, 2013). As far as we know, this is the first study to evaluate the psychometric properties of the

BES in the general youth population, including both males and females. To date, the only study that analyzed the BES factorial structure and psychometric properties in the general population was conducted by Duarte et al. (2015), but it only included a sample of Portuguese women. In addition, our study is the first one to test the scale's measurement invariance across samples, in this case, across sexes.

These findings demonstrate that the Spanish version of the BES is a valid and reliable scale for the assessment of BE in a youth sample. This brief, easy-to-administer, self-report questionnaire consists of 16 items on one scale. It provides relevant information about clinically significant symptoms of BE, and it may be especially useful in prevention programs and community interventions for disordered eating behaviors.

This study contributes to a relevant line of research in the field of eating disorder evaluation and, specifically, BE. The present study confirms the unifactorial structure of the BES in a young community population of men and women. The BES is one of the most widely used scales worldwide in the detection of BE in both clinical and non-clinical samples. Its validation in a young general sample can help us to detect cases of BE in order to prevent a possible eating disorder and its associated medical comorbidities. In addition, this study provides data about the relationship between BE and other comorbid variables, helping us to better understand this prevalent problem in today's society and, especially, in young people.

## Limitations and Future Research

Several limitations should be considered. First, only self-applied measures were used, and the participants may have suffered from social desirability bias. Thus, future research should include a semi-structured interview to obtain better reliability and specificity. Second, self-reported height and weight were used to calculate BMI. Several studies suggest that self-reported measures of weight and height should be viewed with caution because middle-aged men and women are more likely to exhibit biases; in particular, weight tends to be underestimated and height overestimated (Niedhammer et al., 2000), and these biases lead to underestimation of the BMI value. Third, it would have been interesting to test both versions of the questionnaire (English/Spanish) in bilingual students to obtain a stronger validation of the questionnaire. Fourth, all the participants were recruited from the University; therefore, the findings cannot be generalized to clinical settings. Several studies support the BES as a valid screening measure of BE in a non-clinical population (Duarte et al., 2015). In addition, there is a high prevalence of BE in the young general population, and so it was considered important to know the psychometric properties of the instrument in young people, in order to have a validated screening measure of BE in this population. However, future lines of research could

perform invariance routines in clinical and non-clinical samples to verify whether the Spanish version of the BES has similar reliable results in clinical and non-clinical populations. Fifth, although the samples of women and men were unbalanced, the total size of the men's sample did not allow us to adjust group size. This limitation will be taken into account in subsequent studies. In addition, although this study has shown that women have higher levels of BE than men, future studies should replicate this finding with a larger sample of men, in order to increase the accuracy of the groups' means. Sixth, due to problems of sample availability, the test-retest reliability could not be verified. Future studies should take this limitation into account. Finally, with regard to the one-dimensionality of the scale, future studies should explore whether the single dimension of the scale obtained in this study varies in other types of samples, for example, samples of older men and women, given the high prevalence of BE in this population (Hudson et al., 2007).

## DATA AVAILABILITY

The datasets generated for this study are available on request to the corresponding author.

## AUTHOR CONTRIBUTIONS

TE-M contributed to preparing the measures and the protocol, researched the literature, recruited the participants and collected the data, and wrote the manuscript. LG contributed to the literature research, analyzed the data, and wrote the manuscript. MR-A and RB contributed to the design of the study and wrote the manuscript. All the authors contributed to manuscript revision and read and approved the submitted version.

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## REFERENCES

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edn. Washington, DC: American Psychiatric Association. doi: 10.1176/appi.books.9780890425596

Brown, T. A. (2006). *Confirmatory Factor Analysis for Applied Research*. New York, NY: Guilford Press.

Brunault, P., Gaillard, P., Ballon, N., Couet, C., Isnard, P., Cook, S., et al. (2016). Validation de la version française de la Binge Eating Scale: étude de sa structure factorielle, de sa consistance interne et de sa validité de construit en population



- clinique et non clinique. *Encephale* 42, 426–433. doi: 10.1016/j.encep.2016.02.009
- Burrows, T., Skinner, J., McKenna, R., and Rollo, M. (2017). Food addiction, binge eating disorder, and obesity: is there a relationship? *Behav. Sci.* 7:54. doi: 10.3390/bs7030054
- Cheung, G. W., and Rensvold, R. B. (2002). Evaluating goodness-of-fit indexes for testing measurement invariance. *Struct. Equ. Model.* 9, 233–255. doi: 10.1207/S15328007SEM0902\_5
- Clausen, L., Rosenvinge, J. H., Friberg, O., and Rokkedal, K. (2011). Validating the eating disorder inventory-3 (EDI-3): a comparison between 561 female eating disorders patients and 878 females from the general population. *J. Psychopathol. Behav. Assess.* 33, 101–110. doi: 10.1007/s10862-010-9207-4
- Croll, J., Neumark-Sztainer, D., Story, M., and Ireland, M. (2002). Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. *J. Adolesc. Health* 31, 166–175. doi: 10.1016/S1054-139X(02)00368-3
- de Freitas, S. R., Appolinario, J. C., de Moura Souza, A., and Sichieri, R. (2008). Prevalence of binge eating and associated factors in a Brazilian probability sample of midlife women. *Int. J. Eat. Disord.* 41, 471–478. doi: 10.1002/eat.20530
- Dezhkam, M., Moloodi, R., Mootabi, F., and Omidvar, N. (2009). Standardization of the binge eating scale among Iranian obese population. *Iranian J. Psychiatry* 4, 143–146.
- Duarte, C., Pinto-Gouveia, J., and Ferreira, C. (2015). Expanding binge eating assessment: validity and screening value of the Binge Eating Scale in women from the general population. *Eat. Behav.* 18, 41–47. doi: 10.1016/j.eatbeh.2015.03.007
- Duncan, A. E., Ziobrowski, H. N., and Nicol, G. (2017). The prevalence of past 12-Month and lifetime DSM-IV eating disorders by BMI category in US men and women. *Eur. Eat. Disord. Rev.* 25, 165–171. doi: 10.1002/erv.2503
- Elosua, P., and López-Jáuregui, A. (2012). Internal structure of the Spanish adaptation of the eating disorder inventory-3. *Eur. J. Psychol. Assess.* 28, 25–31. doi: 10.1027/1015-5759/a000087
- Fairburn, C. G., and Beglin, S. J. (1994). Assessment of eating disorders: interview or self-report questionnaire? *Int. J. Eat. Disord.* 16, 363–370.
- Fan, X., Thompson, B., and Wang, L. (1999). Effects of sample size, estimation methods, and model specification on structural equation modeling fit indexes. *Struct. Equ. Model.* 6, 56–83. doi: 10.1080/10705519909540119
- Finney, S. J., and DiStefano, C. (2013). “Nonnormal and categorical data in structural equation modeling,” in *Quantitative Methods in Education and the Behavioral Sciences: Issues, Research, and Teaching. Structural Equation Modeling: A Second Course*, eds G. R. Hancock and R. O. Mueller (Charlotte, NC: IAP Information Age Publishing), 439–492.
- Flint, A. J., Gearhardt, A. N., Corbin, W. R., Brownell, K. D., Field, A. E., and Rimm, E. B. (2014). Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *Am. J. Clin. Nutr.* 99, 578–586. doi: 10.3945/ajcn.113.068965
- Flora, D. B., and Curran, P. J. (2004). An empirical evaluation of alternative methods of estimation for confirmatory factor analysis with ordinal data. *Psychol. Methods* 9, 466–491. doi: 10.1037/1082-989X.9.4.466
- Fouladi, F., Mitchell, J. E., Crosby, R. D., Engel, S. G., Crow, S., Hill, L., et al. (2015). Prevalence of alcohol and other substance use in patients with eating disorders. *Eur. Eat. Disord. Rev.* 23, 531–536. doi: 10.1002/erv.2410
- Freitas, S., Lopes, C. S., Appolinario, J. C., and Coutinho, W. (2006). The assessment of binge eating disorder in obese women: a comparison of the binge eating scale with the structured clinical interview for the DSM-IV. *Eat. Behav.* 7, 282–289. doi: 10.1016/j.eatbeh.2005.09.002
- Gearhardt, A. N., Corbin, W. R., and Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite* 52, 430–436. doi: 10.1016/j.appet.2008.12.003
- Goldschmidt, A. B., Loth, K. A., MacLehose, R. F., Pisetsky, E. M., Berge, J. M., and Neumark-Sztainer, D. (2015). Overeating with and without loss of control: associations with weight status, weight-related characteristics, and psychosocial health. *Int. J. Eat. Disord.* 48, 1150–1157. doi: 10.1002/eat.22465
- Gordon, K. H., Holm-Denoma, J. M., Troop-Gordon, W., and Sand, E. (2012). Rumination and body dissatisfaction interact to predict concurrent binge eating. *Body Image* 9, 352–357. doi: 10.1016/j.bodyim.2012.04.001
- Gormally, J., Black, S., Daston, S., and Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addict. Behav.* 7, 47–55. doi: 10.1016/0306-4603(82)90024-7
- Granero, R., Hilker, I., Agüera, Z., Jiménez-Murcia, S., Sauchelli, S., Islam, M. A., et al. (2014). Food addiction in a Spanish sample of eating disorders: DSM-5 diagnostic subtype differentiation and validation data. *Eur. Eat. Disord. Rev.* 22, 389–396. doi: 10.1002/erv.2311
- Grupski, A. E., Hood, M. M., Hall, B. J., Azarbad, L., Fitzpatrick, S. L., and Corsica, J. A. (2013). Examining the binge eating scale in screening for binge eating disorder in bariatric surgery candidates. *Obes. Surg.* 23, 1–6. doi: 10.1007/s11695-011-0537-4
- Hood, M. M., Grupski, A. E., Hall, B. J., Ivan, I., and Corsica, J. (2013). Factor structure and predictive utility of the Binge Eating Scale in bariatric surgery candidates. *Surg. Obes. Relat. Dis.* 9, 942–948. doi: 10.1016/j.soard.2012.06.013
- Hu, L., and Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct. Equ. Model.* 6, 1–55. doi: 10.1080/10705519909540118
- Hudson, J. I., Hiripi, E., Pope, H. G. Jr., and Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol. Psychiatry* 61, 348–358. doi: 10.1016/j.biopsych.2006.03.040
- Imperatori, C., Innamorati, M., Contardi, A., Continisio, M., Tamburello, S., Lamis, D. A., et al. (2014). The association among food addiction, binge eating severity and psychopathology in obese and overweight patients attending low-energy-diet therapy. *Compr. Psychiatry* 55, 1358–1362. doi: 10.1016/j.comppsy.2014.04.023
- Imperatori, C., Innamorati, M., Lamis, D. A., Contardi, A., Continisio, M., Castelnovo, G., et al. (2016). Factor structure of the binge eating scale in a large sample of obese and overweight patients attending low energy diet therapy. *Eur. Eat. Disord. Rev.* 24, 174–178. doi: 10.1002/erv.2384
- Katterman, S. N., Kleinman, B. M., Hood, M. M., Nackers, L. M., and Corsica, J. A. (2014). Mindfulness meditation as an intervention for binge eating, emotional eating, and weight loss: a systematic review. *Eat. Behav.* 15, 197–204. doi: 10.1016/j.eatbeh.2014.01.005
- Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., et al. (2013). The prevalence and correlates of binge eating disorder in the world health organization world mental health surveys. *Biol. Psychiatry* 73, 904–914. doi: 10.1016/J.BIOPSYCH.2012.11.020
- Kline, R. B. (2015). *Principles and Practice of Structural Equation Modeling*. New York, NY: Guilford Publications.
- Kuntsche, E., Gmel, G., Wicki, M., Rehm, J., and Grichting, E. (2006). Disentangling gender and age effects on risky single occasion drinking during adolescence. *Eur. J. Public Health* 16, 670–675. doi: 10.1093/eurpub/ckl060
- Laghi, F., Baiocco, R., Liga, F., Lonigro, A., and Baumgartner, E. (2014). Binge eating and binge drinking behaviors: individual differences in adolescents’ identity styles. *J. Health Psychol.* 19, 333–343. doi: 10.1177/1359105312470851
- Little, T. D. (1997). Mean and Covariance Structures (MACS) analyses of cross-cultural data: practical and theoretical issues. *Multivar. Behav. Res.* 32, 53–76. doi: 10.1207/s15327906mbr3201\_3
- Marcus, M. D., Wing, R. R., and Hopkins, J. (1988). Obese binge eaters: affect, cognitions, and response to behavioral weight control. *J. Consult. Clin. Psychol.* 56, 433–439. doi: 10.1037/0022-006X.56.3.433
- Marek, R. J., Tarescavage, A. M., Ben-Porath, Y. S., Ashton, K., and Heinberg, L. J. (2016). Replication and evaluation of a proposed two-factor Binge Eating Scale (BES) structure in a sample of bariatric surgery candidates. *Surg. Obes. Relat. Dis.* 11, 659–665. doi: 10.1016/j.soard.2014.09.015
- Mason, T. B., Smith, K. E., Lavender, J. M., and Lewis, R. J. (2018). Independent and interactive associations of negative affect, restraint, and impulsivity in relation to binge eating among women. *Appetite* 121, 147–153. doi: 10.1016/j.appet.2017.11.099
- Meno, C. A., Hannum, J. W., Espelage, D. E., and Low, K. S. D. (2008). Familial and individual variables as predictors of dieting concerns and binge eating in college females. *Eat. Behav.* 9, 91–101. doi: 10.1016/j.eatbeh.2007.06.002
- Mustelin, L., Bulik, C. M., Kaprio, J., and Keski-Rahkonen, A. (2017). Prevalence and correlates of binge eating disorder related features in the community. *Appetite* 109, 165–171. doi: 10.1016/j.appet.2016.11.032
- Muthén, L. K., and Muthén, B. O. (1998). *Mplus User’s Guide*, 8th Edn. Los Angeles, CA: Muthén & Muthén.

- Niedhammer, I., Bugel, I., Bonenfant, S., Goldberg, M., and Leclerc, A. (2000). Validity of self-reported weight and height in the French GAZEL cohort. *Int. J. Obes.* 24, 1111–1118. doi: 10.1038/sj.ijo.0801375
- Ocampo, R., Bojorquez, L., and Unikel, C. (2012). Disordered eating behaviors and binge drinking in female high-school students: the role of impulsivity. *Salud Mental* 35, 83–89.
- Orozco-Cabal, L., Rodríguez, M., Herin, D. V., Gempeler, J., and Uribe, M. (2010). Validity and reliability of the abbreviated barratt impulsiveness scale in Spanish (BIS-15S)\*. *Rev. Colomb. Psiquiatr.* 39, 93–109. doi: 10.1016/S0034-7450(14)60239-0
- Paul, L. A., Grubaugh, A. L., Frueh, B. C., Ellis, C., and Egede, L. E. (2011). Associations between binge and heavy drinking and health behaviors in a nationally representative sample. *Addict. Behav.* 36, 1240–1245. doi: 10.1016/j.addbeh.2011.07.034
- Pedram, P., Wadden, D., Amini, P., Gulliver, W., Randell, E., Cahill, F., et al. (2013). Food addiction: its prevalence and significant association with obesity in the general population. *PLoS One* 8:e74832. doi: 10.1371/journal.pone.0074832
- Preti, A., de Girolamo, G., Vilagut, G., Alonso, J., de Graaf, R., Bruffaerts, R., et al. (2009). The epidemiology of eating disorders in six European countries: results of the ESEMED-WMH project. *J. Psychiatr. Res.* 43, 1125–1132. doi: 10.1016/j.jpsychires.2009.04.003
- Ribeiro, M., Conceição, E., Vaz, A. R., and Machado, P. P. (2014). The prevalence of binge eating disorder in a sample of college students in the North of Portugal. *Eur. Eat. Disord. Rev.* 22, 185–190. doi: 10.1002/erv.2283
- Ricca, V., Mannucci, E., Moretti, S., Di Bernardo, M., Zucchi, T., Cabras, P. L., et al. (2000). Screening for binge eating disorder in obese outpatients. *Compr. Psychiatry* 41, 111–115. doi: 10.1016/S0010-440X(00)90143-3
- Robert, S. A., Rohana, A. G., Suehazlyn, Z., Maniam, T., Azhar, S. S., and Azmi, K. N. (2013). The validation of the malay version of binge eating scale: a comparison with the structured clinical interview for the DSM-IV. *J. Eat. Disord.* 1:28. doi: 10.1186/2050-2974-1-28
- Smink, F. R. E., van Hoeken, D., Oldehinkel, A. J., and Hoek, H. W. (2014). Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *Int. J. Eat. Disord.* 47, 610–619. doi: 10.1002/eat.22316
- Spinella, M. (2007). Normative data and a short form of the Barrat Impulsiveness Scale. *Int. J. Neurosci.* 117, 359–368. doi: 10.1080/00207450600588881
- Steward, T., Mestre-Bach, G., Vintró-Alcaraz, C., Agüera, Z., Jiménez-Murcia, S., Granero, R., et al. (2017). Delay discounting of reward and impulsivity in eating disorders: from anorexia nervosa to binge eating disorder. *Eur. Eat. Disord. Rev.* 25, 601–606. doi: 10.1002/erv.2543
- Stice, E., Marti, C. N., and Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *J. Abnormal Psychol.* 122, 445–457. doi: 10.1037/a0030679
- Telch, C. F., Agras, W. S., and Linehan, M. M. (2001). Dialectical behavior therapy for binge eating disorder. *J. Consult. Clin. Psychol.* 69, 1061–1065. doi: 10.1037/0022-006X.69.6.1061
- Thompson, M. S., and Green, S. B. (2006). “Evaluating between-group differences in latent variable means,” in *Structural Equation Modeling: A Second Course, Quantitative Methods in Education and the Behavioral Sciences: Issues, Research, and Teaching*, eds G. R. Hancock and R. O. Mueller (Charlotte, NC: IAP Information Age Publishing), 119–169.
- Thornton, L. M., Watson, H. J., Jangmo, A., Welch, E., Wiklund, C., von Hausswolff-Juhlin, Y., et al. (2017). Binge-eating disorder in the Swedish national registers: somatic comorbidity. *Int. J. Eat. Disord.* 50, 58–65. doi: 10.1002/eat.22624
- van de Schoot, R., Lugtig, P., and Hox, J. (2012). A checklist for testing measurement invariance. *Eur. J. Dev. Psychol.* 9, 486–492. doi: 10.1080/17405629.2012.686740
- Villarejo, C., Fernández-Aranda, F., Jiménez-Murcia, S., Peñas-Lledó, E., Granero, R., Penelo, E., et al. (2012). Lifetime obesity in patients with eating disorders: increasing prevalence, clinical and personality correlates. *Eur. Eat. Disord. Rev.* 20, 250–254. doi: 10.1002/erv.2166
- World Health Organization [WHO] (2000). *Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation*. Geneva: World Health Organization.
- Yanovski, S. (1993). Binge eating disorder: current knowledge and future directions. *Obes. Res.* 1, 306–324. doi: 10.1002/j.1550-8528.1993.tb00626.x
- Zúñiga, O., and Robles, R. (2006). Validez de constructo y consistencia interna del Cuestionario de Trastorno por Atracón en población mexicana con obesidad. *Psiquis* 15, 126–134.

**Conflict of Interest Statement:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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## ANNEX: CUESTIONARIO DE TRASTORNO POR ATRACÓN

Instrucciones: A continuación, se encuentran 16 grupos de tres o cuatro oraciones. Lea con cuidado cada una de las oraciones de cada grupo y marque con una «X» (cruz) la que mejor describa como se siente con respecto a los problemas que ha tenido para controlar su forma de comer.

### GRUPO 1

1. No me siento preocupado(a) de mi peso o mi talla cuando estoy con otros.
2. Me siento preocupado(a) de cómo luzco para los demás, pero normalmente esto no me hace sentirme decepcionado(a) de mí mismo(a).
3. Me siento preocupado(a) acerca de mi apariencia y peso y esto me hace sentir decepcionado(a) de mí mismo(a).
4. Me siento muy preocupado(a) acerca de mi peso y frecuentemente siento una pena intensa y disgusto por mí mismo(a). Trato de evitar contactos sociales debido a mi preocupación por mi apariencia.

### GRUPO 2

1. No tengo ninguna dificultad para comer lentamente y de manera adecuada.
2. Aunque parece que «devoro» la comida no termino sintiéndome «lleno(a)» por haber comido mucho.
3. Algunas veces como muy rápido y después me siento incómodamente lleno(a).
4. Siempre o casi siempre me paso la comida sin masticarla y cuando esto pasa me siento incómodamente «lleno(a)» porque he comido demasiado.

### GRUPO 3

1. Me siento capaz de controlar mis ganas de comer cuando yo quiero.
2. Siento que he fallado en controlar mi alimentación más que una persona promedio.
3. Me siento incapaz de controlar mis ganas de comer.
4. Me siento desesperado(a) porque no soy capaz de controlar mi manera de comer.

### GRUPO 4

1. No acostumbro comer cuando estoy aburrido(a).
2. ALGUNAS VECES me pongo a comer cuando estoy aburrido(a), pero SIEMPRE O CASI SIEMPRE soy capaz de mantenerme ocupado(a) y dejar de pensar en la comida.
3. SIEMPRE O CASI SIEMPRE me pongo a comer cuando estoy aburrido(a), pero ALGUNAS VECES puedo hacer otra actividad para dejar de pensar en la comida.
4. SIEMPRE O CASI SIEMPRE me pongo a comer cuando estoy aburrido(a) y nada parece ayudarme a romper este hábito.

### GRUPO 5

1. GENERALMENTE como cuando me siento físicamente con hambre.

2. ALGUNAS VECES como algo impulsivamente a pesar de que no tengo hambre.
3. MUCHAS VECES como cosas que realmente no disfruto para satisfacer mi sensación de hambre a pesar saber que físicamente no necesito comer en ese momento.
4. A pesar de que físicamente no tengo hambre, tengo una sensación de hambre en mi boca que sólo es satisfecha comiendo cosas que me llenan la boca, como un sándwich. Cuando hago esto, algunas veces escupo la comida para no engordar.

### GRUPO 6

1. No siento culpa ni me odio después de comer de más.
2. A VECES siento culpa o me odio después de comer de más.
3. SIEMPRE O CASI SIEMPRE siento culpa o me odio después de comer de más.

### GRUPO 7

1. Cuando hago dieta y la rompo porque como de más, puedo volver a controlar mi forma de comer.
2. Cuando hago dieta y la rompo comiendo algo «prohibido», ALGUNAS VECES siento que «me equivoqué» y como aún más.
3. Cuando hago dieta y la rompo porque como de más, MUCHAS VECES siento que «cometí un error» y como aún más.
4. SIEMPRE O CASI SIEMPRE hago dieta y la rompo porque tengo un atracón. Parece que mi vida transcurriera entre «atracones» y «tener hambre».

### GRUPO 8

1. MUY POCAS VECES O NUNCA como tanto como para sentirme incómodo(a).
2. ALGUNAS VECES, aproximadamente 1 vez al mes, como tanto que al final termino sintiéndome muy «lleno(a)».
3. MUCHAS VECES durante el mes tengo periodos donde como grandes cantidades de comida, ya sea a la hora de la comida o entre comidas.
4. SIEMPRE O CASI SIEMPRE como tanta comida que me siento incomodo(a) al acabar de comer, y a veces hasta con un poco de náuseas.

### GRUPO 9

1. Mi ingesta de calorías no es muy baja ni muy alta.
2. ALGUNAS VECES después de comer demasiado, trato de disminuir mi ingesta de calorías casi a cero para compensar el exceso que había comido.
3. SIEMPRE O CASI SIEMPRE como demasiado durante la noche. Generalmente no tengo hambre durante el día pero como demasiado en la noche.
4. Durante mi vida adulta hay semanas en que tengo largos periodos donde prácticamente me mato de hambre y luego como de más. Parece ser que mi vida transcurre entre «atracones» y «hambre».

**GRUPO 10**

1. Normalmente soy capaz de parar de comer cuando yo quiero. Sé cuándo: «suficiente es suficiente».
2. ALGUNAS VECES me da compulsión de comer y parece que no puedo controlar mi manera de comer.
3. MUCHAS VECES me da una urgencia por comer y pareciera que no la puedo controlar, pero otras veces si la puedo controlar.
4. SIEMPRE O CASI SIEMPRE me siento incapaz de controlar mi urgencia por comer. Tengo miedo de no poder parar de comer cuando yo quiera.

**GRUPO 11**

1. No tengo ningún problema para parar de comer cuando me siento «lleno».
2. Puedo parar de comer cuando me siento lleno, pero A VECES como demasiado y me siento «muy lleno».
3. MUCHAS VECES tengo el problema para parar de comer y me siento incómodamente «lleno».
4. SIEMPRE O CASI SIEMPRE soy incapaz de parar de comer cuando yo quiero y algunas veces ha sido necesario inducirme el vómito, usar laxantes o diuréticos para aliviar mi sensación de estar «muy lleno».

**GRUPO 12**

1. Como lo mismo cuando estoy con otros (familia, reunión social) que cuando estoy solo.
2. ALGUNAS VECES cuando estoy con otras personas no como tanto como quisiera porque me siento preocupado(a) acerca de mi forma de comer.
3. MUCHAS VECES como pequeñas cantidades de comida cuando hay gente a mí alrededor porque me siento avergonzado de mi forma de comer.
4. SIEMPRE O CASI SIEMPRE me siento muy avergonzado(a) por comer de más y elijo tiempos para comer de más cuando sé que nadie podría verme. Me siento como «un tragón de closet».

**GRUPO 13**

1. Hago tres comidas al día y SÓLO ALGUNAS VECES como bocadillos entre comidas.
2. Hago tres comidas al día, pero MUCHAS VECES como bocadillos entre éstas.
3. Cuando como muchos bocadillos me salto las comidas regulares.
4. Hay periodos que parece que estuviera comiendo todo el tiempo, sin ninguna comida planeada.

**GRUPO 14**

1. No pienso mucho acerca de tratar de controlar comer cosas que en realidad no deseo.
2. ALGUNAS VECES pienso acerca de tratar de controlar mi urgencia por comer.
3. MUCHAS VECES paso mucho tiempo pensando acerca de cuanto comí o acerca de tratar de no comer más.
4. SIEMPRE O CASI SIEMPRE estoy pensando en «comer o no comer». Siento que vivo para comer.

**GRUPO 15**

1. No pienso que la comida sea «lo más importante».
2. Tengo antojos fuertes de comida, pero sólo por periodos cortos de tiempo.
3. HAY DÍAS que parece que no puedo pensar en otra cosa que no sea la comida.
4. LA MAYORÍA DE LOS DÍAS estoy preocupado(a) acerca de la comida. Siento como si viviera para comer.

**GRUPO 16**

1. SIEMPRE O CASI SIEMPRE puedo distinguir si estoy físicamente hambriento(a) o no. Como lo suficiente como para satisfacerme.
2. A VECES me siento inseguro(a) de saber si estoy físicamente hambriento o no. Cuando pasa esto me es difícil saber qué tanto debo comer para satisfacerme.
3. Aunque pienso que debería saber cuántas calorías debo comer, no tengo ni la menor idea cual es la cantidad «normal» de comida para mí.

Annex 3: Study 3 article.  
Understanding the Influence  
of Eating Patterns on Binge  
Drinking: A Mediation  
Model.





Article

# Understanding the Influence of Eating Patterns on Binge Drinking: A Mediation Model

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**Abstract:** Background: Binge drinking is an important health problem, and it has been related to binge eating and fat intake in animal models, but this relationship has not been tested in humans. The first objective of this study was to analyze whether binge eating and fat intake are related to binge drinking in a youth sample. The second objective was to analyze whether binge eating and fat intake mediate the relationship between individual factors associated with binge eating and fat intake (sex, body mass index (BMI), drive for thinness, body dissatisfaction, eating styles, impulsivity, and food addiction) and binge drinking. Methods: A sample of 428 undergraduate students filled out several questionnaires on binge drinking, binge eating, fat intake, drive for thinness, body dissatisfaction, eating styles, food addiction, and impulsivity. Results: Results showed an excellent model fit:  $\chi^2(25) = 30.342$  ( $p = 0.212$ ), comparative fit index (CFI) = 0.992, root mean squared error of approximation (RMSEA) = 0.022 [90% CI = 0.000, 0.047]. Binge eating and fat intake were positively related to binge drinking. Furthermore, emotional eating, external eating, and food addiction showed positive and statistically significant indirect relationships with binge drinking, whereas the relationship with restrained eating was negative. Conclusions: These findings point to the need to use a broader approach in understanding and preventing binge drinking in the youth population by showing the influence of the eating pattern on this problem. This information could be helpful in preventing future behaviors and improving interventions that address health risk behaviors.

**Keywords:** binge drinking; binge eating; fat intake; youth; undergraduate students

## 1. Introduction

The consumption of alcohol by young people has risen sharply in recent decades, and the risk of binge drinking in this population has increasingly been recognized [1]. In Spain, 25% of young people are considered binge drinkers [2]. Commonly, binge drinking is defined as the consumption of five or more drinks in men or four or more drinks in women on a single occasion (about 2 h) [3]. This consumption pattern has become one of the main public health problems because it is associated with multiple adverse consequences, such as low quality of life, unsafe driving, aggressiveness, risky sexual behaviors, cognitive impairment, and emotional and relationship problems [4–7]. In addition, the high prevalence



of binge drinking is especially worrisome in this developmental period, especially in young people, given that they are particularly vulnerable to the neurotoxic effects of alcohol, due to the functional and structural brain changes that occur at this stage [8]. Therefore, it is necessary to identify the risk factors associated with binge drinking in order to understand and prevent this behavior.

Currently, we have a large number of studies in the literature that point to a high comorbidity between eating disorders and alcohol consumption, including empirical studies [9], narrative reviews [10,11], systematic reviews, and meta-analyses [12,13]. However, recently, the emphasis has begun to be placed not only on eating disorders as factors that can help to explain excessive alcohol consumption but also on intake and dietary patterns, such as fat intake and binge eating. In this sense, fat intake is understood as the frequency of consumption of “unhealthy fats”, i.e., saturated fats and trans fats [14], and it has been associated with excessive alcohol consumption [15,16]. Along this line, studies in animal models show a greater preference for fats by rats previously injected with ethanol [15]. In addition, rats that consume more fat show a preference for consuming alcohol over water, a relationship that is not observed in rats that consume carbohydrates [17]. Moreover, evidence in humans also shows higher alcohol consumption by people with high fat consumption [16,18,19]. In this sense, it is hypothesized that this relationship can be explained by the activation of common pathways: fat intake activates the dopaminergic pathway, which is also involved in the processes of reward and motivation of alcohol consumption [20].

In addition, it has been shown that it is not only the type of diet (i.e., frequency of fat intake) but also the type of intake behavior (i.e., binge eating) that seems to contribute to explaining binge drinking [21]. Along this line, binge eating has been defined as the excessive intake of food (fat, carbohydrate, vegetables . . . ) over a short-term period and the lack of control over this intake behavior [22,23]. According to different studies, binge eating and binge drinking share commonalities, regarding the pattern of behavior (impulsivity, loss of control), nature and onset of the problem, and prevalence, among others [24–28]. Given this, some authors explored whether binge eating is related to binge drinking and have concluded that both are associated [9,24,25,29–31]. This relation has also been seen in animal models [21].

Regarding binge eating and fat intake, it is well known that there are a variety of individual factors that can underlie high levels of binge eating and fat intake, such as sex, BMI, drive for thinness, body dissatisfaction, eating styles, among others [22,32–36]. Therefore, we aimed to study whether these factors can be indirectly related to binge drinking through their relationship with binge eating and/or fat intake. For example, it is known that people show different tendencies in their food behavior depending on their sex. In this case, literature has shown that there is a difference in prevalence between men and women. While women are more likely to binge [22], men are more likely to have a higher-fat diet in early adulthood [37]. On the other hand, individuals with a higher body mass index (BMI) report a greater number of binges [34] and a higher fat intake [38]. There is also a relationship between personality characteristics and these behaviors. In this sense, impulsivity may lie beneath binge eating and fat intake behaviors. Impulsivity can be defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions [39]. It can be understood that is composed of three components: motor impulsivity (acting without thinking), unplanned impulsivity (a lack of future orientation or forethought), and intentional impulsivity (inability to focus attention or concentrate) [40]. Literature showed that motor and intentional impulsivity seem to underlie both binge eating [41] and unhealthy food consumption [32]. Finally, eating styles may also be at the base of binge eating and fat intake behaviors. Van Strien et al. [42] distinguished three eating styles: emotional eating (eating in response to negative emotions), external eating (eating in response to external cues, such as sight or smell), and restrictive eating (deliberately restricting food to decrease or maintain weight). Studies indicate that both emotional and external eating are positively associated with increased fat intake [35,43], while restrictive eating is associated with decreased fat intake [44]. Furthermore, all three eating styles are associated with binge eating [34], and may act as strong predictors of binge eating [36,45].



Along the same line, the food addiction construct may act as a predictor of the severity of binge eating [33], and those who are addicted to food are also more likely than non-addicts to consume high-fat food [46]. Other variables that predispose to binge eating and fat intake are body dissatisfaction and drive for thinness, as they positively predict binge eating [47] and are negatively associated with fat intake [44,48].

So far, literature has indicated that binge drinking can predict an increase in binge eating and fat intake in young people [24,31]. However, a line of animal research has attempted to study the inverse relationship, suggesting that binge eating and fat intake may also play a role in the development of binge drinking [21]. These studies have explored the relationship, showing that both binge eating and fat intake predict an increase in subsequent ethanol overconsumption in adolescent mice [21]. However, the directionality of this relationship has hardly been studied, and the results are inconclusive [24]. The aforementioned evidence has raised the question of whether binge eating and fat intake might be influencing binge drinking [21], and to our knowledge, no research has focused on evaluating the relation among binge eating, fat intake, and binge drinking in this young population.

Therefore, the first aim of the present study was to explore the relationship between binge eating, fat intake, and binge drinking. Specifically, this model hypothesizes that binge eating and fat intake will positively influence binge drinking. Moreover, taking into account that there are individual factors that may be at the base of binge eating and fat intake, it is hypothesized that those factors can be indirectly related to binge drinking. In this sense, the second objective for this study was to analyze whether binge eating and fat intake mediate the relationship between these individual factors (sex, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity and food addiction) and binge drinking. With that in mind, we hypothesized that these individual factors may be acting on binge drinking through binge eating and fat intake. To accomplish these objectives, a structural equation model will be tested to study the relationships of binge drinking in the young population.

## 2. Materials and Methods

### 2.1. Participants

The sample of the present study was composed of undergraduate students living in the province of Valencia (Spain). A total of 428 undergraduate students, 324 females (75.7%; mean age 21.04; SD = 4.22) and 104 males (24.3%; mean age 22.27; SD = 5.39), took part voluntarily in the present study.

### 2.2. Design and Procedure

The study has a cross-sectional design, with data collection carried out at one time point. Data collection followed ethical requirements, and all participants were fully informed about the voluntary nature of their participation and the confidentiality of the collected data. They provided their informed consent prior to being included in the study. Participants were recruited through e-mail, social networks, and word of mouth, and they were directed to a dedicated online survey. The survey was carried out using the Lime Survey web platform, where participants provided demographic data and answered the questionnaires. The study was approved by the Valencia Ethics Committee and performed in accordance with the ethical standards of the 1964 Declaration of Helsinki (Procedure number: H1513854038939).

### 2.3. Measures

The structural model included several exogenous, mediation, and dependent variables. The exogenous variables were:

Sex, with two categories: men and women.

Body mass index (BMI). BMI was calculated by dividing self-reported current weight (in kilograms) by height squared (in meters) [49].

Eating disorders, measured with the Eating Disorder Inventory-3 (EDI-3; [50], in its Spanish version [51]). The EDI-3 is a self-report questionnaire consisting of 91 items grouped in 12 subscales designed to assess eating disorder psychopathology and the associated psychological symptoms. In the present study, only the eating disorder risk composite factor was used. The factor consists of 25 items rated on a 6-point Likert scale, and is composed of three scales that measure the risk of having an eating disorder: drive for thinness, bulimia, and body dissatisfaction. The bulimia scale is not used in the present study as it overlapped with the binge eating mediator variable. In this study, Cronbach's alpha was 0.903 for drive for thinness, and 0.747 for body dissatisfaction.

Eating styles, using the Dutch Eating Behavior Questionnaire (DEBQ; [42,52]). This questionnaire includes 33 items that measure emotional eating, external eating, and restrained eating. The questionnaire showed adequate levels of internal consistency, with a Cronbach's alpha value of 0.952 for emotional eating, 0.884 for external eating, and 0.926 for restrained eating.

Impulsivity. This variable was assessed using the Barratt Impulsiveness Scale-15 (BIS-15; [53]; Spanish version: [54]). It is a brief, self-administered scale composed of 15 items grouped in three factors: motor, non-planning, and attentional impulsivity. All the items evaluate impulsivity in different facets. Internal consistency estimates were 0.781 for motor impulsivity, 0.760 for non-planning impulsivity, and 0.690 for attentional impulsivity.

Food addiction, using the modified Yale Food Addiction Scale (mYFAS; [55]). The mYFAS evaluates signs of addictive-like eating behavior. It is composed of nine items consisting of one question from each of the symptom groups that make up the seven diagnostic criteria, plus two individual items that assess the presence of clinically significant impairment and distress. The internal consistency coefficient in this study, measured with Cronbach's alpha, was 0.769.

As regards the mediation variables, they included:

Binge eating. This variable was assessed using the total score on the Binge Eating Scale (BES; [56]; Spanish version: [57]). The BES is a 16-item self-report questionnaire designed to identify the symptoms associated with binge eating (eating large amounts of food in a short time and feeling loss of control). Internal consistency in this sample was 0.869.

Fat intake. Fat intake consisted of the total score on the Short Fat Questionnaire (SFQ; [14]). This scale is a 17-item self-report questionnaire that assesses the weekly frequency of fat intake, such as fried food consumption, consumption of sauces or creams, use of fats for cooking, consumption of processed meat, cakes, ice cream, degree of cooking of food, and consumption of milk (with or without fat). For instance: "How many times a week do you eat French fries?" "And chocolate?", The response scale ranges from never to six times or more. Scores ranged from zero to 62, with a higher score indicating higher frequency of fat intake. This measure has been widely used in the young population to measure fat intake and it has been related to overweight, obesity, and substance use, among others [58,59].

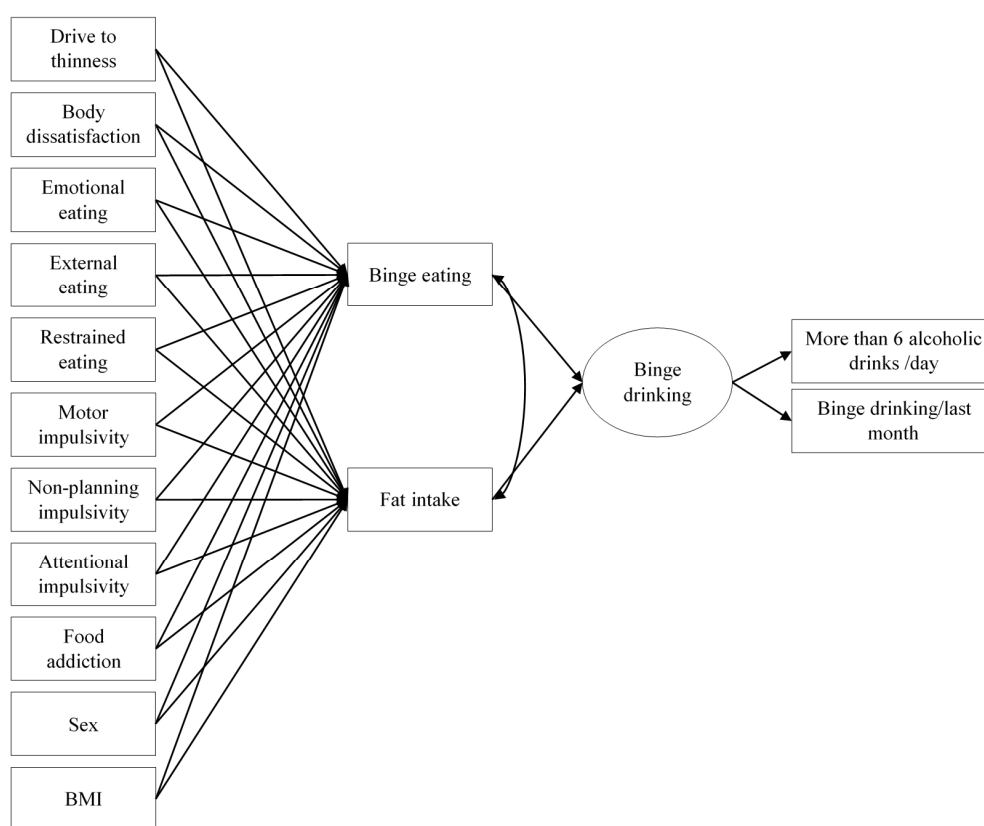
This questionnaire had to undergo a rigorous cultural adaptation procedure. It was translated into English by a Spanish-English translator, and subsequently several Spanish reviewers adapted the translated items. The Spanish version of the SFQ is quite similar to the original validation [14]. Internal consistency in this sample, estimated with Cronbach's alpha, was 0.796.

Finally, the dependent variable in the model was binge drinking. This variable was measured with the main tools to assess binge drinking: item 3 from the AUDIT scale [60] ("How often do you have six or more drinks on one occasion?"), ranging from 0 (never) to 4 (daily or almost daily); and an indicator of binge drinking ("Considering all types of alcoholic beverages, did you ever have five or more drinks (four if you are female) in a two-hour period (one time) in the past month? How many times in the past month?"), ranging from 0 (never) to 4 (4 or more times per week) [61,62]. Internal consistency was 0.735.

#### 2.4. Statistical Analyses

Analyses included descriptive statistics and a multiple indicators multiple causes (MIMIC) structural model. The MIMIC model allows us to study the relationships of binge drinking in a context free of measurement error in the main outcome under study, while considering unique relations with specific binge drinking indicators or items.

The model hypothesized, estimated, and tested a direct relationship between binge eating (measured with the Binge Eating Scale) and fat intake (measured with the Short Fat Questionnaire) and binge drinking, modeled as a latent factor. In addition, several variables related to binge eating and fat intake were included in the model to test its indirect effects (through binge eating and/or fat intake) on binge drinking. These variables were: sex, BMI, drive for thinness, body dissatisfaction, emotional eating, external eating, restrained eating, motor impulsivity, non-planning impulsivity, attentional impulsivity, and food addiction. The model can be consulted in Figure 1.



**Figure 1.** Multiple indicators multiple causes (MIMIC) model to study binge drinking; Notes: For the sake of clarity, standard errors are not shown.

The plausibility of the model was assessed using general fit criteria, in addition to examining the analytical fit. Regarding the general model fit, the fit statistics and indices recommended in the literature were used [63,64]: (a) Chi square statistic [65]; (b) comparative fit index (CFI), with values above 0.90 indicating a good representation of the data and, ideally, 0.95; [63]; and (c) root mean squared error of approximation (RMSEA), with values of 0.08 considered a reasonable fit [65], and its 90% confidence interval (CI).

In the case of the analytical fit, factor loadings for the measurement part of the model were examined, along with the effects of the different variables involved. Indirect effects were also calculated, and the CI around the estimate of the effects was also estimated using a bootstrap resampling method. This procedure has been recommended as the best method to generate the required sampling distributions for testing indirect effects [66].

The model was estimated using maximum likelihood with robust standard errors (MLR), using Mplus version 8 [67].

### 3. Results

First, descriptive statistics were calculated for all the variables included in the model. As Table 1 shows, the scores on all the scales were in line with the average in the original validation, showing non-clinical symptomatology for binge eating, food addiction, and impulsivity. Specifically, in relation to the question “How often do you have 6 or more drinks in a single day?”, only 0.7% of the participants reported having had six or more drinks in one day daily or almost daily; 4.0% did this weekly; 22.8%, monthly; 37.8% less than once a month; and 34.6%, never. Regarding the question “Considering all types of alcoholic beverages, did you have five or more drinks (four if you are female) in a two-hour period (one time) in the past month? How many times in the past month?”, only 0.4% of the participants did this twice or three times a week; 9.8%, two to four times a month; 27.4%, once or less than once a month; and 62.4%, never. These values were expected because the sample was drawn from the general population.

**Table 1.** Descriptive statistics for the variables under study.

Variables	Mean	Standard Deviation
BMI	22.29	3.02
Drive for thinness	7.68	6.94
Body dissatisfaction	11.26	6.50
Emotional eating	25.99	10.70
External eating	30.27	7.33
Restrained eating	21.79	8.70
Motor impulsivity	4.66	4.11
Non-planning impulsivity	8.09	4.41
Attentional impulsivity	7.54	4.65
Food addiction	5.69	4.65
Binge Eating Scale	7.80	6.63
Short Fat Questionnaire	22.47	7.92
More than 6 alcoholic drinks/day	0.99	0.89
Binge drinking/past month	0.48	0.68

Regarding the study of the relationships of binge drinking, a structural MIMIC model was hypothesized to relate the aforementioned variables to binge drinking. This initial or theoretical model (Figure 1) fitted the data perfectly:  $\chi^2(23) = 28.025$  ( $p = 0.214$ ), CFI = 0.992, RMSEA = 0.023 [90% CI = 0.000, 0.048]. The measurement part of the model showed a strong link between the indicators and their corresponding latent variable (binge drinking), with a factor loading of 0.855 ( $p < 0.001$ ) for more than 6 alcoholic drinks/day (item 3 on the AUDIT scale), and 0.663 ( $p < 0.001$ ) for binge drinking episodes in the past month.

In terms of the relations with binge drinking, both binge eating and fat intake showed a positive and statistically significant relationship. Binge eating showed a positive effect of 0.226 ( $p < 0.001$ ), and fat intake had a positive effect of 0.217 ( $p < 0.001$ ). The correlation between these two indicators was, however, not statistically significant ( $r = -0.021$ ;  $p = 0.684$ ).

The exogenous variables that were related with binge eating were: drive for thinness ( $\beta = 0.284$ ,  $p < 0.001$ ), body dissatisfaction ( $\beta = 0.108$ ,  $p = 0.019$ ), emotional eating ( $\beta = 0.174$ ,  $p < 0.001$ ), external eating ( $\beta = 0.119$ ,  $p = 0.004$ ), restrained eating ( $\beta = -0.122$ ,  $p = 0.026$ ), and food addiction ( $\beta = 0.443$ ,  $p < 0.001$ ). In the case of fat intake, the statistically significant related variables were: drive for thinness ( $\beta = -0.184$ ,  $p = 0.018$ ), external eating ( $\beta = 0.375$ ,  $p < 0.001$ ), restrained eating ( $\beta = -0.259$ ,  $p < 0.001$ ), motor impulsivity ( $\beta = 0.132$ ,  $p = 0.013$ ), and food addiction ( $\beta = 0.177$ ,  $p = 0.008$ ). Correlations among the exogenous variables were in the expected direction and can be consulted in Table 2.

**Table 2.** Correlations among the exogenous variables in the MIMIC model.

Variables	1	2	3	4	5	6	7	8	9	10
1. Drive for thinness	–									
2. Body dissatisfaction	0.608 ***	–								
3. Emotional eating	0.377 ***	0.383 ***	–							
4. External eating	0.175 **	0.177 **	0.497 ***	–						
5. Restrained eating	0.797 ***	0.504 ***	0.405 ***	0.226 ***	–					
6. Motor impulsivity	0.169 **	0.197 **	0.138 *	0.163 **	0.190 **	–				
7. Non-planning impulsivity	0.001 n.s.	0.071 n.s.	0.104 n.s.	0.103 n.s.	0.020 n.s.	0.327 ***	–			
8. Attentional impulsivity	0.132 *	0.264 ***	0.230 ***	0.214 ***	0.101 n.s.	0.284 ***	0.266 ***	–		
9. Food addiction	0.558 ***	0.512 ***	0.613 ***	0.403 ***	0.561 ***	0.299 ***	0.092 n.s.	0.225 ***	–	
10. Sex	0.247 ***	0.147 **	0.210 ***	0.143 **	0.213 ***	−0.093 n.s.	−0.117 *	−0.107 *	0.062 n.s.	–
11. BMI	0.260 ***	0.317 ***	0.159 **	−0.018 n.s.	0.242 ***	0.062 n.s.	0.086 n.s.	0.105 *	0.184 **	−0.220 ***

Notes: Sex was coded as: 0 = men, 1 = women; positive correlations indicate higher levels for women, whereas negative correlations indicate higher levels for men; n.s.  $p > 0.050$ ; \*  $p < 0.050$ ; \*\*  $p < 0.010$ ; \*\*\*  $p < 0.001$ .

Finally, indirect effects of the exogenous variables on the binge drinking factor were examined. As shown in Table 3, emotional eating, external eating, and food addiction showed positive, statistically significant relations with binge drinking, whereas the relationship with restrained eating was negative.

**Table 3.** Indirect effects of the exogenous variables on the binge drinking factor in the MIMIC model.

Variables	Effect through Binge Eating [95% CI]	Effect through Fat Intake [95% CI]	Total Effect [95% CI]
Drive for thinness	0.064 ** [0.029, 0.120]	−0.040 * [−0.096, −0.008]	0.024 n.s. [−0.031, 0.085]
Body dissatisfaction	0.024 * [0.005, 0.054]	0.006 n.s. [−0.019, 0.036]	0.031 n.s. [−0.004, 0.076]
Emotional eating	0.039 ** [0.016, 0.076]	0.002 n.s. [−0.025, 0.032]	0.042 * [0.006, 0.089]
External eating	0.027 * [0.009, 0.057]	0.081 ** [0.035, 0.143]	0.108 *** [0.055, 0.175]
Restrained eating	−0.028 n.s. [−0.064, 0.005]	−0.056 ** [−0.117, −0.020]	−0.084 ** [−0.151, −0.038]
Motor impulsivity	−0.011 n.s. [−0.037, 0.007]	0.029 * [0.006, 0.068]	0.017 n.s. [−0.013, −0.057]
Non-planning impulsivity	0.019 n.s. [−0.003, 0.042]	−0.017 n.s. [−0.047, 0.001]	0.002 n.s. [−0.028, 0.029]
Attentional impulsivity	0.004 n.s. [−0.011, 0.023]	−0.006 n.s. [−0.034, 0.015]	−0.002 n.s. [−0.033, 0.026]
Food addiction	0.100 *** [0.045, 0.166]	0.038 * [0.010, 0.087]	0.139 *** [0.072, 0.218]
Sex	0.005 n.s. [−0.008, 0.023]	−0.004 n.s. [−0.029, 0.017]	0.002 n.s. [−0.026, 0.027]
BMI	0.013 n.s. [0.000, 0.032]	−0.011 n.s. [−0.036, 0.007]	0.002 n.s. [−0.024, 0.028]

Notes: Sex was coded as: 0 = men, 1 = women; positive correlations indicate higher levels for women, whereas negative correlations indicate higher levels for men; n.s.  $p > 0.050$ ; \*  $p < 0.050$ ; \*\*  $p < 0.010$ ; \*\*\*  $p < 0.001$ .

Overall, 68.3% of binge eating ( $R^2 = 0.683$ ,  $p < 0.001$ ), 27.0% of fat intake ( $R^2 = 0.270$ ,  $p < 0.001$ ), and 10.4% of binge drinking ( $R^2 = 0.104$ ,  $p = 0.015$ ) were explained.

#### 4. Discussion

Firstly, the current study examined whether binge eating and fat intake are related to binge drinking in a youth sample. As it was hypothesized, binge eating and fat intake are related to binge drinking, meaning that higher scores on binge eating and higher fat intake behaviors are associated with higher scores on binge drinking behaviors. These findings are in line with recent studies in animal models that showed a causal relationship between a high-fat diet and binge eating behaviors with higher consumption of ethanol [21]. Along the same line, other authors have also proposed a relationship between fat intake and binge drinking [15–19], and between binge eating and binge drinking [24,25]. Furthermore, prospective studies have found that binge eating predicts the risk of frequent drinking and substance abuse [68,69] or drug consumption [13,70]. It has been hypothesized that binge eating induces emotions of guilt and shame, and alcohol and substance use could be used to regulate these negative emotions [71].

In addition, there are other mechanisms that may be at the base of binge eating and fat intake, such as eating styles [34,44,45] or food addiction [33,46], among others, that could help explain these relationships.

Taking this into account, secondly, the mediating role of binge eating and fat intake between various individual factors (sex, BMI, drive for thinness, body dissatisfaction, eating styles, impulsivity and food addiction) and binge drinking was examined. Therefore, it was hypothesized that these individual factors may be acting on binge drinking through binge eating and fat intake. As hypothesized, binge drinking is indirectly related to other relevant variables. Specifically, emotional eating, external eating, and food addiction showed positive and statistically significant indirect relationships with binge drinking, whereas the relationship with restrained eating was negative.

First, our results showed that emotional eating was positively related to binge drinking through its relationship with binge eating. These findings replicate some results obtained in previous research. There is evidence that emotional eating can act as a predictor of binge eating [34,36], but it was not yet known whether binge eating could also act as a mediator between emotional eating and binge drinking. In these results, we can conclude that people who eat more in response to negative emotions may be more likely to have higher binge eating scores, and in turn, engaging in binge eating may contribute to higher alcohol consumption.



Second, our results showed that external eating was positively related to drinking behaviors through its relationship with binge eating and fat intake. There is evidence in the literature about the positive relationship between external eating and binge eating [34,45], and between external eating and fat intake [35,43], but our study is the first to analyze the mediating role of binge eating and fat intake in the relationship between external eating and binge drinking.

Third, our results showed that restrained eating was associated with lower binge drinking behaviors, and this relationship was mediated by the decrease in fat intake. Although the literature has found that restrained eating often leads to higher alcohol consumption, our data could suggest the role of fat intake in better understanding this relationship. That is, people who restrict food and eat less fat will drink less alcohol, possibly because of the calories in both substances. Moreover, a recent study pointed out that alcohol consumption and restrictive behaviors are weakly related [72], and a potential explanatory hypothesis would be that fat intake acts as a mediator between these variables.

Fourth, food addiction was related to binge eating and fat intake, and through these behaviors, it could lead to an increase in alcohol consumption in the form of binge drinking. Although the literature has pointed out the relationship between food addiction and alcohol [73,74], our study suggests that binge eating and fat intake could act as mediating factors in this relationship.

Several limitations must be highlighted in the present study. The sample was limited to undergraduate students. Therefore, these findings cannot be generalized to other demographic groups such as young people with a low-medium educational status or adults, in whom the prevalence of binge drinking has also been found to be very high. Thus, further studies are required. In the present study, binge drinking was measured with two indicators commonly used in the literature: frequency of consumption of high alcohol doses from the AUDIT-3, and the presence of a binge drinking episode in the past. Both indicators showed an adequate factorial saturation indicating that they worked well as measures of binge drinking, and this result is consistent with the literature [60,62]. Although this is the most common measure used, the research still does not have a standardized measure for binge drinking, and future studies are needed in this field to create a comprehensive and standardized measure. Moreover, other future lines may include testing the model invariance in different subgroups, such as women and men, to detect possible different paths for different populations. Another limitation is the cross-sectional nature of the study, which calls for a cautious interpretation of the results, with other alternative models being possible [75]. Therefore, future research is needed to examine the interactions among these variables in a longitudinal design that makes it possible to assess the temporal relationships between them. In addition, the study was carried out with self-report measures, which may be subject to self-report bias and yield different results compared to clinical interviews or semi-structured assessments [76]. Furthermore, it should also be noted that the explained variance of binge drinking in the present model is small. For future research, it would be necessary to include other relevant factors traditionally related to binge drinking, as well as a sample with a wider variety of characteristics (such as age or the presence of risk factors related to health), to improve the explanatory capability of the current model. Finally, it should be noted the importance for future studies to explore the role of other nutrients, such as carbohydrates, since they can also play an important role in alcohol consumption. In the present study, only the role of high-fat foods has been studied, given its current prevalence of consumption, its negative health consequences, as well as its relationship with alcohol consumption in rodents. This is the first study to explore the relationship between fat intake and binge drinking in young adults, but future research should explore how different nutrients (fats and carbohydrates) separately influence binge drinking.

## 5. Conclusions

This study provides evidence of a direct relationship between binge eating and fat intake and binge drinking, and an indirect relationship between different eating patterns (emotional, restrictive and external eating, and food addiction) and binge drinking, mediated through binge eating and fat

intake. The present study is the first to transfer the previous results in mice to humans and assess the influence of eating patterns on binge drinking.

The results of this study can have several implications for health professionals and researchers interested in promoting health and preventing risk behaviors in young people, such as binge drinking, by helping to improve interventions that aim to prevent or decrease binge drinking in young populations. Prevention and intervention strategies could target young people with high scores on these eating patterns to weaken the association between these eating patterns and binge drinking. More specifically, it should be noted that binge eating and fat intake could be a gateway to the initiation and escalation of binge drinking. If we focus on improving these eating patterns in young populations, this can have a dampening effect on the level of alcohol consumption among young people and its consequences. In addition, it should be considered that eating styles (emotional, external, and restrained eating) and food addiction may also precipitate binge drinking through the relationship with binge eating and fat intake. Taking into account this study, it would be appropriate to focus on those young people who have high scores in those eating styles, and who show high scores in food addiction, in order to try to improve those eating patterns as much as possible and thus reduce the binge drinking pattern.

Despite some limitations, these findings provide a unique contribution to the current understanding of the relationship between eating patterns and binge drinking in young people. All these eating patterns are variables to be considered for future interventions and prevention of binge drinking among young people, given their association with binge drinking; however, further research is needed to determine whether these eating patterns can actually explain the high prevalence of binge drinking.

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## Abbreviations

CFI	Comparative Fit Index
CI	Confidence Interval
MIMIC	Multiple Indicators Multiple Causes
MLR	Maximum Likelihood with Robust standard errors
RMSEA	Root Mean Squared Error of Approximation

## References

1. World Health Organization. *Global Status Report on Alcohol and Health*; World Health Organization: Geneva, Switzerland, 2014; Available online: [http://www.who.int/substance\\_abuse/publications/global\\_alcohol\\_report/en/](http://www.who.int/substance_abuse/publications/global_alcohol_report/en/) (accessed on 10 October 2020).
2. Ministerio de Sanidad, Consumo y Bienestar Social, Plan Nacional sobre Drogas. Encuesta EDADES 2017: Encuesta Sobre Alcohol y otras drogas en España (EDADES), 1995–2017. Available online: [http://www.pnsd.mscbs.gob.es/profesionales/sistemasInformacion/sistemaInformacion/pdf/EDADES\\_2017\\_Informe.pdf](http://www.pnsd.mscbs.gob.es/profesionales/sistemasInformacion/sistemaInformacion/pdf/EDADES_2017_Informe.pdf) (accessed on 15 May 2020).
3. National Institute of Alcohol Abuse and Alcoholism. NIAAA Council Approves Definition of Binge Drinking. Available online: <https://cutt.ly/EwQHD2G> (accessed on 26 November 2020).



4. Cservenka, A.; Brumback, T. The burden of binge and heavy drinking on the brain: Effects on adolescent and young adult neural structure and function. *Front Psychol.* **2017**, *8*, 1111. [[CrossRef](#)] [[PubMed](#)]
5. Kuntsche, E.; Kuntsche, S.; Thrul, J.; Gmel, G. Binge drinking: Health impact, prevalence, correlates and interventions. *Psychol. Health* **2017**, *32*, 976–1017. [[CrossRef](#)] [[PubMed](#)]
6. Martinotti, G.; Lupi, M.; Carlucci, L.; Santacroce, R.; Cinosi, E.; Acciavatti, T.; Sarchione, F.; Verrastro, V.; Diotaiuti, P.; Petruccielli, I.; et al. Alcohol drinking patterns in young people: A survey-based study. *J. Health Psychol.* **2017**, *22*, 1889–1896. [[CrossRef](#)] [[PubMed](#)]
7. Patrick, M.E.; Terry-McElrath, Y.M.; Evans-Polce, R.J.; Schulenberg, J.E. Negative alcohol-related consequences experienced by young adults in the past 12 months: Differences by college attendance, living situation, binge drinking, and sex. *Addict. Behav.* **2020**, *105*, 106320. [[CrossRef](#)]
8. Jones, S.A.; Lueras, J.M.; Nagel, B.J. Effects of binge drinking on the developing brain: Studies in humans. *Alcohol Res. Curr. Rev.* **2018**, *39*, 87–96.
9. Bahji, A.; Mazhar, M.N.; Hudson, C.C.; Nadkarni, P.; MacNeil, B.A.; Hawken, E. Prevalence of substance use disorder comorbidity among individuals with eating disorders: A systematic review and meta-analysis. *Psychiatry Res.* **2019**, *273*, 58–66. [[CrossRef](#)]
10. Conason, A.H.; Sher, L. Alcohol use in adolescents with eating disorders. *Int. J. Adolesc. Med. Health* **2006**, *18*, 31–36. [[CrossRef](#)]
11. Gadalla, T.; Piran, N. Co-occurrence of eating disorders and alcohol use disorders in women: A meta-analysis. *Arch. Women Ment. Health* **2007**, *10*, 133–140. [[CrossRef](#)]
12. Schulte, E.M.; Grilo, C.M.; Gearhardt, A.N. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clin. Psychol. Rev.* **2016**, *44*, 125–139. [[CrossRef](#)]
13. Sonnevile, K.R.; Horton, N.J.; Micali, N.; Crosby, R.D.; Swanson, S.A.; Solmi, F.; Field, A.E. Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: Does loss of control matter? *JAMA Pediatr.* **2013**, *167*, 149–155. [[CrossRef](#)]
14. Dobson, A.J.; Blijlevens, R.; Alexander, H.M.; Croce, N.; Heller, R.F.; Higginbotham, N.; Pike, G.; Plotnikoff, R.; Russell, A.; Walker, R. Short fat questionnaire: A self-administered measure of fat-intake behaviour. *Aust. J. Public Health* **1993**, *17*, 144–149. [[CrossRef](#)]
15. Barson, J.R.; Karatayev, O.; Chang, G.Q.; Johnson, D.F.; Bocarsly, M.E.; Hoebel, B.G.; Leibowitz, S.F. Positive relationship between dietary fat, ethanol intake, triglycerides, and hypothalamic peptides: Counteraction by lipid-lowering drugs. *Alcohol* **2009**, *43*, 433–441. [[CrossRef](#)]
16. Stickley, A.; Koyanagi, A.; Kuposov, R.; McKee, M.; Murphy, A.; Ruchkin, V. Binge drinking and eating problems in Russian adolescents. *Alcohol. Clin. Exp. Res.* **2015**, *39*, 540–547. [[CrossRef](#)]
17. Krahn, D.D.; Gosnell, B.A. Fat-preferring rats consume more alcohol than carbohydrate-preferring rats. *Alcohol* **1991**, *8*, 313–316. [[CrossRef](#)]
18. Swinburn, B.A.; Walter, L.; Ricketts, H.; Whitlock, G.; Law, B.; Norton, R.; Jackson, R.; MacMahon, S. The determinants of fat intake in a multi-ethnic New Zealand population. *Int. J. Epidemiol.* **1998**, *27*, 416–421. [[CrossRef](#)] [[PubMed](#)]
19. Kesse, E.; Clavel-Chapelon, F.; Slimani, N.; Van Liere, M. Do eating habits differ according to alcohol consumption? Results of a study of the French cohort of the European Prospective Investigation into Cancer and Nutrition (E3N-EPIC). *Am. J. Clin. Nutr.* **2001**, *74*, 322–327. [[CrossRef](#)] [[PubMed](#)]
20. Valdivia, S.; Cornejo, M.P.; Reynaldo, M.; De Francesco, P.N.; Perello, M. Escalation in high fat intake in a binge eating model differentially engages dopamine neurons of the ventral tegmental area and requires ghrelin signaling. *Psychoneuroendocrinology* **2015**, *60*, 206–216. [[CrossRef](#)] [[PubMed](#)]
21. Blanco-Gandía, M.C.; Ledesma, J.C.; Aracil-Fernández, A.; Navarrete, F.; Montagud-Romero, S.; Aguilar, M.A.; Manzanares, J.; Miñarro, J.; Rodríguez-Arias, M. The rewarding effects of ethanol are modulated by binge eating of a high-fat diet during adolescence. *Neuropharmacology* **2017**, *121*, 219–230. [[CrossRef](#)]
22. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*, 5th ed.; American Psychiatric Association: Arlington, VA, USA, 2013.
23. Fitzgibbon, M.L.; Blackman, L.R. Binge eating disorder and bulimia nervosa: Differences in the quality and quantity of binge eating episodes. *Int. J. Eat. Disord.* **2000**, *27*, 238–243. [[CrossRef](#)]
24. Escrivá-Martínez, T.; Herrero, R.; Molinari, G.; Rodríguez-Arias, M.; Verdejo-García, A.; Baños, R.M. Binge Eating and Binge Drinking: A Two-Way Road? An Integrative Review. *Curr. Pharm. Des.* **2020**, *26*, 2402–2415. [[CrossRef](#)]

25. Ferriter, C.; Ray, L.A. Binge eating and binge drinking: An integrative review. *Eat Behav.* **2011**, *12*, 99–107. [[CrossRef](#)] [[PubMed](#)]
26. Patrick, M.E.; Terry-McElrath, Y.M.; Lanza, S.T.; Jager, J.; Schulenberg, J.E.; O'Malley, P.M. Shifting age of peak binge drinking prevalence: Historical changes in normative trajectories among young adults aged 18 to 30. *Alcohol. Clin. Exp. Res.* **2019**, *43*, 287–298. [[CrossRef](#)] [[PubMed](#)]
27. Stice, E.; Marti, C.N.; Rohde, P. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *J. Abnorm. Psychol.* **2013**, *122*, 445–457. [[CrossRef](#)]
28. Croll, J.K.; Neumark-Sztainer, D.; Story, M.; Ireland, M. Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: Relationship to gender and ethnicity. *J. Adolesc. Health* **2002**, *31*, 166–175. [[CrossRef](#)]
29. Davis, C.; Mackew, L.; Levitan, R.D.; Kaplan, A.S.; Carter, J.C.; Kennedy, J.L. Binge Eating Disorder (BED) in Relation to addictive behaviors and personality risk factors. *Front. Psychol.* **2017**, *8*, 579. [[CrossRef](#)] [[PubMed](#)]
30. Laghi, F.; Baiocco, R.; Liga, F.; Lonigro, A.; Baumgartner, E. Binge eating and binge drinking behaviors: Individual differences in adolescents' identity styles. *J. Health Psychol.* **2014**, *19*, 333–343. [[CrossRef](#)] [[PubMed](#)]
31. Vogeltanz-Holm, N.D.; Wonderlich, S.A.; Lewis, B.A.; Wilsnack, S.C.; Harris, T.R.; Wilsnack, R.W.; Kristjanson, A.F. Longitudinal predictors of binge eating, intense dieting, and weight concerns in a national sample of women. *Behav. Ther.* **2000**, *31*, 221–235. [[CrossRef](#)]
32. Kakoschke, N.; Kemps, E.; Tiggemann, M. External eating mediates the relationship between impulsivity and unhealthy food intake. *Physiol. Behav.* **2015**, *147*, 117–121. [[CrossRef](#)]
33. Linardon, J.; Messer, M. Assessment of food addiction using the Yale Food Addiction Scale 2.0 in individuals with binge-eating disorder symptomatology: Factor structure, psychometric properties, and clinical significance. *Psychiatry Res.* **2019**, *279*, 216–221. [[CrossRef](#)]
34. Mason, T.B.; Lewis, R.J. Profiles of binge eating: The interaction of depressive symptoms, eating styles, and body mass index. *Eat. Disord.* **2014**, *22*, 450–460. [[CrossRef](#)]
35. Camilleri, G.M.; Méjean, C.; Kesse-Guyot, E.; Andreeva, V.A.; Bellisle, F.; Hercberg, S.; Péneau, S. The associations between emotional eating and consumption of energy-dense snack foods are modified by sex and depressive symptomatology. *J. Nutr.* **2014**, *144*, 1264–1273. [[CrossRef](#)]
36. Sultson, H.; Kukk, K.; Akkermann, K. Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. *Appetite* **2017**, *116*, 423–430. [[CrossRef](#)] [[PubMed](#)]
37. Li, K.K.; Concepcion, R.Y.; Lee, H.; Cardinal, B.J.; Ebbeck, V.; Woekel, E.; Readdy, R.T. An examination of sex differences in relation to the eating habits and nutrient intakes of university students. *J. Nutr. Educ. Behav.* **2012**, *44*, 246–250. [[CrossRef](#)] [[PubMed](#)]
38. Wang, L.; Wang, H.; Zhang, B.; Popkin, B.M.; Du, S. Elevated Fat Intake Increases Body Weight and the Risk of Overweight and Obesity among Chinese Adults: 1991–2015 Trends. *Nutrients* **2020**, *12*, 3272. [[CrossRef](#)] [[PubMed](#)]
39. Moeller, F.G.; Barratt, E.S.; Dougherty, D.M.; Schmitz, J.M.; Swann, A.C. Psychiatric aspects of impulsivity. *Am. J. Psychiatry.* **2001**, *158*, 1783–1793. [[CrossRef](#)] [[PubMed](#)]
40. Barratt, E.S. Impulsiveness subtraits: Arousal and information processing. In *Motivation, Emotion and Personality*; Spence, J.T., Izard, C.E., Eds.; Elsevier Science Publishers: Amsterdam, The Netherlands, 1985; pp. 137–146.
41. Meule, A. Impulsivity and overeating: A closer look at the subscales of the Barratt Impulsiveness Scale. *Front. Psychol.* **2013**, *4*, 177. [[CrossRef](#)]
42. Van Strien, T.; Frijters, J.E.; Bergers, G.P.; Defares, P.B. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *Int. J. Eat. Disord.* **1986**, *5*, 295–315. [[CrossRef](#)]
43. Anschutz, D.J.; Van Strien, T.; Van De Ven, M.O.; Engels, R.C. Eating styles and energy intake in young women. *Appetite* **2009**, *53*, 119–122. [[CrossRef](#)]
44. Liebman, M.; Cameron, B.A.; Carson, D.K.; Brown, D.M.; Meyer, S.S. Dietary fat reduction behaviors in college students: Relationship to dieting status, gender and key psychosocial variables. *Appetite* **2001**, *36*, 51–56. [[CrossRef](#)]

45. Burton, P.; Smit, H.J.; Lightowler, H.J. The influence of restrained and external eating patterns on overeating. *Appetite* **2007**, *49*, 191–197. [CrossRef]
46. Ruddock, H.K.; Field, M.; Hardman, C.A. Exploring food reward and calorie intake in self-perceived food addicts. *Appetite* **2017**, *115*, 36–44. [CrossRef]
47. Gordon, K.H.; Holm-Denoma, J.M.; Troop-Gordon, W.; Sand, E. Rumination and body dissatisfaction interact to predict concurrent binge eating. *Body Image* **2012**, *9*, 352–357. [CrossRef] [PubMed]
48. Ribeiro-Silva, R.D.C.; Fiaccone, R.L.; Conceição-Machado, M.E.P.D.; Ruiz, A.S.; Barreto, M.L.; Santana, M.L.P. Body image dissatisfaction and dietary patterns according to nutritional status in adolescents. *J. Pediatr.* **2018**, *94*, 155–161. [CrossRef] [PubMed]
49. World Health Organization. *Obesity: Preventing and Managing the Global Epidemic*; World Health Organization: Geneva, Switzerland, 2000; Available online: <http://www.worldcat.org/title/obesity-preventing-and-managing-the-global-epidemic-report-of-a-who-consultation/oclc/48171257> (accessed on 10 October 2020).
50. Clausen, L.; Rosenvinge, J.H.; Friberg, O.; Rokkedal, K. Validating the Eating Disorder Inventory-3 (EDI-3): A Comparison between 561 Female Eating Disorders Patients and 878 Females from the General Population. *J. Psychopathol. Behav. Assess.* **2011**, *33*, 101–110. [CrossRef]
51. Elosua, P.; López-Jáuregui, A. Internal Structure of the Spanish Adaptation of the Eating Disorder Inventory-3. *Eur. J. Psychol. Assess.* **2012**, *28*, 25–31. [CrossRef]
52. Cebolla, A.; Barrada, J.R.; Van Strien, T.; Oliver, E.; Baños, R. Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite* **2014**, *73*, 58–64. [CrossRef]
53. Spinella, M. Normative data and a short form of the Barrat Impulsiveness Scale. *Int. J. Neurosci.* **2007**, *117*, 359–368. [CrossRef] [PubMed]
54. Orozco-Cabal, L.; Rodríguez, M.; Herin, D.V.; Gempeler, J.; Uribe, M. Validity and Reliability of the Abbreviated Barratt Impulsiveness Scale in Spanish (BIS-15S)\*. *Rev. Colomb. Psiquiatr.* **2010**, *39*, 93–109. [CrossRef]
55. Flint, A.J.; Gearhardt, A.N.; Corbin, W.R.; Brownell, K.D.; Field, A.E.; Rimm, E.B. Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *Am. J. Clin. Nutr.* **2014**, *99*, 578–586. [CrossRef]
56. Gormally, J.; Black, S.; Daston, S.; Rardin, D. The assessment of binge eating severity among obese persons. *Addict. Behav.* **1982**, *7*, 47–55. [CrossRef]
57. Escrivá-Martínez, T.; Galiana, L.; Rodríguez-Arias, M.; Baños, R.M. The binge eating scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Front. Psychol.* **2019**, *10*, 530. [CrossRef] [PubMed]
58. Hayatbakhsh, M.R.; O’Callaghan, M.J.; Mamun, A.A.; Williams, G.M.; Clavarino, A.; Najman, J.M. Cannabis use and obesity and young adults. *Am. J. Drug Alcohol Abus.* **2010**, *36*, 350–356. [CrossRef] [PubMed]
59. O’Connor, J.; Steinbeck, K.; Hill, A.; Booth, M.; Kohn, M.; Shah, S.; Baur, L. Evaluation of a community-based weight management program for overweight and obese adolescents: The Loozit study. *Nutr. Diet.* **2008**, *65*, 121–127. [CrossRef]
60. Maurage, P.; Lannoy, S.; Mange, J.; Grynberg, D.; Beaunieux, H.; Banovic, I.; Gierski, F.; Naassila, M. What we talk about when we talk about binge drinking: Towards an integrated conceptualization and evaluation. *Alcohol Alcohol.* **2020**, *55*, 468–479. [CrossRef] [PubMed]
61. Kuntsche, E.; Gmel, G.; Wicki, M.; Rehm, J.; Grichting, E. Disentangling gender and age effects on risky single occasion drinking during adolescence. *Eur. J. Public Health* **2006**, *16*, 670–675. [CrossRef]
62. Paul, L.A.; Grubaugh, A.L.; Frueh, B.C.; Ellis, C.; Egede, L.E. Associations between binge and heavy drinking and health behaviors in a nationally representative sample. *Addict. Behav.* **2011**, *36*, 1240–1245. [CrossRef]
63. Hu, L.; Bentler, P.M. Cut-off criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Struct. Equ. Modeling* **1999**, *6*, 1–55. [CrossRef]
64. Tanaka, J.S. Multifaceted conceptions of fit in structural equation models. In *Testing Structural Equation Models*; Bollen, K.A., Long, J.S., Eds.; Sage: Newbury Park, CA, USA, 1993; pp. 10–39.
65. Kline, R.B. *Principles and Practices of Structural Equation Modeling*, 3rd ed.; Guilford Press: New York, NY, USA, 2015.
66. MacKinnon, D.P.; Fairchild, A.J.; Fritz, M.S. Mediation analysis. *Annu. Rev. Psychol.* **2007**, *58*, 593–614. [CrossRef]
67. Muthén, L.K.; Muthén, B.O. Confirmatory factor analysis and structural equation modeling. In *Mplus User’s Guide*, 8th ed.; Muthén & Muthén: Los Angeles, CA, USA, 2017; pp. 55–112.

68. Field, A.E.; Sonneville, K.R.; Micali, N.; Crosby, R.D.; Swanson, S.A.; Laird, N.M.; Treasure, J.; Solmi, F.; Horton, N.J. Prospective association of common eating disorders and adverse outcomes. *Pediatrics* **2012**, *130*, e289–e295. [[CrossRef](#)]
69. Measelle, J.R.; Stice, E.; Hogansen, J.M. Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *J. Abnorm. Psychol.* **2006**, *115*, 524–538. [[CrossRef](#)]
70. Micali, N.; Solmi, F.; Horton, N.J.; Crosby, R.D.; Eddy, K.T.; Calzo, J.P.; Sonneville, K.R.; Swanson, S.A.; Field, A.E. Adolescent Eating Disorders Predict Psychiatric, High-Risk Behaviors and Weight Outcomes in Young Adulthood. *J. Am. Acad. Child. Adolesc. Psychiatry* **2015**, *54*, 652–659. [[CrossRef](#)] [[PubMed](#)]
71. Caton, S.J.; Nolan, L.J.; Hetherington, M.M. Alcohol, Appetite and Loss of Restraint. *Curr. Obes. Rep.* **2015**, *4*, 99–105. [[CrossRef](#)] [[PubMed](#)]
72. Baker, J.H.; Munn-Chernoff, M.A.; Lichtenstein, P.; Larsson, H.; Maes, H.; Kendler, K.S. Shared familial risk between bulimic symptoms and alcohol involvement during adolescence. *J. Abnorm. Psychol.* **2017**, *126*, 506–518. [[CrossRef](#)] [[PubMed](#)]
73. Gearhardt, A.N.; Corbin, W.R.; Brownell, K.D. Preliminary validation of the Yale food addiction scale. *Appetite* **2009**, *52*, 430–436. [[CrossRef](#)]
74. Gearhardt, A.N.; Corbin, W.R.; Brownell, K.D. Food addiction: An examination of the diagnostic criteria for dependence. *J. Addict. Med.* **2009**, *3*, 1–7. [[CrossRef](#)]
75. MacCallum, R.C.; Austin, J.T. Applications of structural equation modeling in psychological research. *Annu. Rev. Psychol.* **2000**, *51*, 201–226. [[CrossRef](#)]
76. Berg, K.C.; Peterson, C.B.; Frazier, P.; Crow, S.J. Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *Int. J. Eat. Disord.* **2012**, *45*, 428–438. [[CrossRef](#)]

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Annex 4: Study 4 article.  
Food addiction and their  
relationships with other  
eating behaviors in a college  
sample.



**Title:** Food addiction and their relationships with other eating behaviors in a college sample

## **Abstract**

**Introduction:** Food Addiction (FA) is defined by the excessive and dysregulated intake of high-calorie foods. The modified Yale Food Addiction Scale 2.0 (mYFAS 2.0) is a widely used instrument to assess FA.

**Objectives:** To examine the psychometric properties of the Spanish mYFAS 2.0; to analyze the relationships between FA with other eating behaviors, sociodemographic variables, and BMI; and to test the predictive power of relevant dietary variables related to FA.

**Method:** The sample consisted of 400 college students ( $M_{age} = 24.16$ ,  $SD_{age} = 6.12$ ; 51% female), who completed the mYFAS 2.0 and measures of eating-related constructs.

**Results:** A confirmatory factor analysis supported the one-factor structure of the mYFAS 2.0, showing excellent fit indices. The scale showed adequate internal consistency ( $\alpha = .828$ ), and good convergent validity with the mYFAS. FA was related to emotional eating, external eating, restrained eating, binge eating, and bulimia. No differences were found between gender and BMI, and younger scored higher on FA. The predictive model explained a 56.6% of the variance in FA.

**Conclusions:** The mYFAS 2.0 is a valid and reliable scale to assess FA in Spanish population. The predictive power of dietary variables (BMI, emotional eating, external eating, restrained eating, binge eating, and bulimia) was demonstrated. All these variables should be considered to prevent FA in young people.

**Keywords:** Food addiction; Modified Yale Food Addiction Scale 2.0; psychometric properties; Spanish adaptation; convergent validity; predictive model.



## **Introduction**

The construct of "food addiction" (FA) is controversial and assumes a certain correspondence with that of substance abuse. In this sense, FA has been defined as a dysregulated and excessive intake of high-calorie foods (i.e., foods high in fat and/or sugars) (Schulte et al., 2015), and it has been suggested that it may share several characteristics with drug abuse that may reflect common neural (e.g., brain reward pathways) (Fletcher & Kenny, 2018) and psychological factors (e.g., drug/food preoccupation) (Carter et al., 2016). In fact, the most widely used instrument to assess FA translates the DSM criteria for substance abuse to eating behavior. Specifically, the original Yale Food Addiction Scale (YFAS) (Gearhardt et al., 2009) translated the DSM-IV-TR diagnostic criteria for substance dependence (APA, 2000), and the new version of this instrument (YFAS 2.0; Gearhardt et al., 2016) reflects the changes in the DSM-5 diagnostic criteria for substance use disorder (APA, 2013). There is a short version (mYFAS 2.0; Schulte & Gearhardt, 2017) which has evidenced good psychometric properties (Schulte & Gearhardt, 2017), and has been adapted in some languages (Italian, Portuguese/Brazilian, French, Arabic, or Czech), but not into Spanish.

Recently, interest on FA research has been growing, especially because it has been associated with higher rates of eating disorders (in particular, binge eating disorder), and overweight or obesity (Imperator et al., 2016). Some data point that around 60% of people with binge eating disorder report clinical symptoms of FA (Long et al., 2015), and 25% of people with obesity meet significant criteria for FA (Pursey et al., 2014). In addition, the literature indicates that FA can be also present in normal-weight individuals without eating disorders history, with some studies indicating 11% of normal-weight individuals reporting these symptoms (Pursey et al., 2014).

Research on the prevalence of FA in non-clinical individuals has examined the role of age and gender, rising contradictory results. The most supported hypothesis is that young people are more likely to show FA than older individuals (Hauck et al., 2017; Schulte & Gearhardt, 2017), and this could be explained because youth involves many physiological and psychological changes that may increase vulnerability to environmental threats (such as addictions or inappropriate eating habits) (Bava & Tapert, 2010; Harris & Fleming-Milici, 2019). However, one study has found an increased likelihood of FA in adulthood (Pursey et al., 2014), suggesting that older people are more exposed to unhealthy foods, and this repeated exposure may reduce the sensitivity of the brain reward circuitry, which could lead them to consume more food to feel good (Stice et al., 2013). In relation to gender, the results are contradictory as well, with some studies supporting higher FA in women (Gearhardt et al., 2016; Pursey et al., 2014), while others find no differences (Ahmed & Sayed, 2017; Schulte & Gearhardt, 2017). Given the high prevalence of FA in the non-clinical sample and the inconsistency of the results, it is necessary to further research to identify vulnerable individuals in order to better design the prevention and treatment of this problem.

Research has been also interested in identifying which factors may contribute to the onset and development of FA. Several studies point to a relationship between FA and dietary variables, suggesting that some eating behaviors and eating styles could be related to this problematic food intake. For example, FA has been linked to emotional eating (Schulte & Gearhardt, 2017), external eating (Pepino et al., 2014), restrained eating (Schiestl & Gearhardt, 2018), binge eating (Fauconnier et al., 2020), bulimia (El Archi et al., 2020), and also Body Mass Index (BMI) (Schulte & Gearhardt, 2017). All these eating behaviors are related to the consumption of unhealthy foods, and such repeated consumption over time may eventually lead to FA. It is therefore important to understand the specific

relationships between all these variables in order to better explain the FA problem, especially in non-clinical population.

### **Aims**

This study has several objectives. First, it aims to examine the psychometric properties of the Spanish translation of mYFAS 2.0 in a sample of the Spanish non-clinical population, assessing the factorial structure through structural equation modelling, specifically CFA, and providing evidence of its reliability. This study also is aimed to analyze the relationship of FA with other eating behavior variables (emotional eating, external eating, restrained eating, binge eating, and bulimia), with sociodemographic characteristics (age, gender), and BMI. Finally, this study will test the predictive power of eating variables related to FA (emotional eating, external eating, restrained eating, binge eating, bulimia, and BMI) through structural equation modelling.

## **Methods**

### **Participants**

The sample consisted of college students from the province of Valencia (Spain). A total of 400 participants took part in the study, 204 females (51%; mean age  $23.35 \pm 4.65$ ) and 196 males (49%; mean age  $24.99 \pm 7.26$ ).

Regarding the BMI and in accordance to the World Health Organization (WHO, 2020), 24 participants (6%) were underweight ( $BMI < 18.5$ ), 296 (74%) had normal weight ( $18.5 \leq BMI \leq 24.99$ ), 64 (16%) were overweight ( $25 \leq BMI \leq 29.99$ ), and 15 (3,8%) were obese ( $BMI \geq 30$ ). One participant did not respond. Female students had a mean BMI of 21.90 (SD = 3.22), and male students had a mean BMI of 23.87 (SD = 4.23).

### **Design and procedure**

A cross-sectional design was used in the present study with one-time point data collection. Participants were invited to participate by announcing the study in the classrooms of the University, and online. All the surveys were administered through the University of Valencia's Lime Survey Platform, and no compensation were given to participants.

### **Ethical considerations**

The study was conducted following the ethical standards of the Declaration of Helsinki in 1964 and was approved by the Ethics Committee of the University of Valencia (Registration number: H151385403893939). All participants were informed of the confidentiality of the data and voluntarily collaborated in the study. All of them gave their informed consent prior to their inclusion in this study.

## **Measurements**

The study included the recollection of sociodemographic information (i.e., gender, with two categories: female and male, and age). Body Mass Index (BMI) was calculated by dividing self-reported current weight by height squared (kilograms/meters<sup>2</sup>) (WHO, 2020). The questionnaires used in the study were:

### **Modified Yale Food Addiction Scale 2.0**

The mYFAS 2.0 is a self-report questionnaire that measures FA (Schulte & Gearhardt, 2017). It is the short form of the YFAS 2.0 scale (Gearhardt et al., 2016). It consists of 13 items that are presented on an 8-point Likert-type scale (0 = never, 7 = every day), and that specifically measures addictive eating behaviors (e.g., "I avoided work, school, or social activities because I was afraid of overeating there.", "My overeating prevented me from taking care of my family or doing household chores.").

The mYFAS 2.0 provides three scoring options: an original score, a symptom count, and a diagnostic score based on the DSM-V criteria for substance use disorder. For the original score, the mean of the 11 items is scored without the two items of clinical significance (impairment and distress). For the symptom count scoring option, each item is scored 0 or 1 depending on whether it meets the established criteria or not; finally, the items are summed and a score between 0 and 11 (0 to 11 symptoms) is obtained. Clinical significance is not added to the score. For the diagnostic scoring option, the symptom count score is obtained, and the clinical significance criterion (impairment or distress) is also added, obtaining a score that divides the sample as follows: does not meet FA criteria (one or fewer symptoms plus does not meet impairment or distress), mild FA (two to three

symptoms plus impairment or distress), moderate FA (four or five symptoms plus impairment or distress), or severe FA (six or more symptoms plus impairment or distress).

The Spanish translation of this questionnaire underwent a rigorous procedure. First, the questionnaire was translated from English into Spanish by a bilingual translator. Second, three Spanish reviewers familiar with the study reviewed the translated items. Third, the study researchers evaluated the scale for correct understanding and administered it to 20 students to corroborate that it was understandable. The Spanish version was an exact translation of the original version (Schulte & Gearhardt, 2017). The final version of the Spanish mYFAS 2.0 and its instructions are available in the Appendix 1. Internal consistency in this study is reported in the Results section.

### **Modified Yale Food Addiction Scale**

The mYFAS is a brief 9-item scale that measures FA (Flint et al., 2014). It was developed from the YFAS scale (Gearhardt et al., 2009). Both scales were developed following the diagnostic criteria for substance use according to the DSM-IV-R. This scale consists of 9 items, of which the first 7 measure diagnostic criteria and the last two items assess the presence of clinically significant impairment and distress. Internal consistency in this sample, estimated with Cronbach's alpha, was .766.

### **Dutch Eating Behavior Questionnaire**

The Dutch Eating Behavior Questionnaire (DEBQ; van Strien et al., 1986) is a self-report instrument composed of 33 items that assess three types of eating behavior: 13 items for emotional eating (eating in response to negative emotions, such as anger or anxiety), 10 items for external eating (eating in response to external food-related stimuli), and 10 items

for restrained eating (voluntary restriction of eating to reduce or maintain weight). It uses a five-point Likert-type scale (1= never, 5= very often). In this study, we used the Spanish version (Cebolla et al., 2014). The Cronbach's alphas for this study were for emotional eating: .952, for external eating: .873, and for restrained eating: .900.

### **Binge Eating Scale**

The Binge Eating Scale (BES; Gormally et al., 1982) is a self-administered questionnaire composed of 16 items that manifest behavioral and cognitive disturbances of food (e.g., eating large amounts of food or worrying about food). Items are answered on a 4-point Likert-type scale (0= no severity of binge eating symptoms; 3= severity of binge eating symptoms). The scores range from 0 to 46, with higher scores indicating binge eating severity. For the present study, the Spanish validation of the BES was used (Escrivá-Martínez et al., 2019). The Cronbach's alpha for the present sample was .873.

### **Eating Disorder Inventory-3 Referral Form**

The Eating Disorders Inventory-3 referral form (EDI-3-RF) is derived from the Eating Disorder Inventory-3 (Clausen et al., 2011). The EDI-3-RF is a brief self-report questionnaire composed of 25 items grouped in three scales that measure the risk of having an eating disorder (drive for thinness, bulimia, and body dissatisfaction). The items are rated on a 6-point Likert scale (1 = always, 6 = never). We only use the bulimia scale in this study (eg., "I think about vomiting to lose weight"), using the Spanish validation of the EDI-3-RF (Elosua & López-Jáuregui, 2012). The Cronbach's alpha for this sample was .836.

## **Analyses**

Analyses include two confirmatory factor analyses (CFA), which hypothesized a one-factor structure, for both the original scores and recorded scores for symptom count of the 11 items of the mYFAS 2.0. Weighted Least Square Mean and Variance Adjusted Estimators (WLSMV) was used, given the multivariate non-normality of the data. In order to assess model fit, the fit criteria used were: the chi-square, the Comparative Fit Index (CFI), the Standardized Root Mean Square Residual (SRMR), and the Root Mean Square Error of Approximation (RMSEA). CFI above .90 (better more than .95) and SRMR or RMSEA below .08 (better below .05) indicate good fit.

Reliability estimates were also calculated, and included both Cronbach's alpha and Omega, for both types of scores of the mYFAS 2.0.

Once the internal structure was studied, and adequate reliability of the scale was evidenced, descriptive statistics for the scale were calculated, using means and standard deviations for the quantitative score, and frequency distributions and percentages for the symptom count scores.

To test for convergent validity, two CFA were estimated. In both, a general factor of FA explained the 11 items of the mYFAS 2.0 together with the 7 items of the mYFAS; each CFA included the original and the symptom count scores, respectively.

Additionally, the relationships between FA (measured with the mYFAS 2.0 original scores and symptom count scores) and emotional eating, external eating, restrained eating, binge eating, and bulimia were analyzed using Pearson correlations and ANOVAs.

To analyze potential differences and relationships between mYFAS 2.0 and age, gender, and BMI, we applied chi square test, t-test, analysis of variance (ANOVA), and Pearson correlations.



Finally, we tested the predictive power of the variables related to FA in a structural equation modeling context. For this purpose, two structural equation models were hypothesized, estimated, and tested, in which the aforementioned variables and their relationship with FA were simultaneously tested, using both mYFAS 2.0 original and symptom count scores. Specifically, two multiple indicators and multiple causes (MIMIC) models were computed, modelling FA as the single latent variable in each of them. Weighted Least Square Mean and Variance Adjusted Estimators (WLSMV) was used. To assess model fit, we used the fit criteria reported before. All the analyses were performed using IBM SPSS Statistics for Windows, Version 24.0 (IBM Corp, 2016), and Mplus, Version 8 (Muthén & Muthén, 2017).

## Results

### Modified Yale Food Addiction Scale 2.0 factor structure and reliability

A confirmatory factor analysis, based on the structure found by FA Schulte and Gearhardt (2017), with a one-factor solution was conducted, with the first 11 items with the original scoring of the mYFAS 2.0. The model showed an excellent fit:  $\chi^2(44) = 116.934$  ( $p < .001$ ); CFI = .975; SRMR = .047; RMSEA = .064[.050,.079]. All factor loadings were statistically significant ( $p < .001$ ), with values of .527 or higher. Details on this matter, together with 'items' descriptive statistics, can be consulted in Table 1.

Additionally, and to study the internal structure of the mYFAS 2.0 when items were recoded for symptom count, a second CFA was conducted, again with excellent fit:  $\chi^2(44) = 59.343$  ( $p = .061$ ); CFI = .989; SRMR = .067; RMSEA = .030 [.000,.047]. Factor loadings were, again, statistically significant ( $p < .001$ ), with values of .580 or higher (see Table 1).

[INSERT TABLE 1 HERE]

As regards mYFAS 2.0 reliability, the scale exhibited good reliability, either when used with the original score or with the symptom count. When reliability estimates were calculated with the original score, Cronbach's alpha was .828 and Omega was .916. When calculated with the symptom count scores, Cronbach's alpha was .783, and Omega .921.

Mean using the quantitative scores was 8.23 (SD = 8.96), with a minimum score of 0 and a maximum of 44. Regarding the symptom count scoring method, mean score was 3.58 (SD = 2.62), with a minimum score of 0 and a maximum score of 11. Using the diagnostic threshold scoring method, 12.5% met mild FA ( $n = 50$ ); 11.3% met moderate FA ( $n = 45$ ); and 20.6% met severe FA. The diagnosis of FA was made if they presented more than three symptoms plus impairment or distress. Thus, 31.9% met FA criteria.

Convergent validity was studied using the mYFAS. For this purpose and considering that both mYFAS 2.0 and mYFAS assess FA, a confirmatory factor analysis in which one factor of FA explained the 11 items of the mYFAS 2.0 together with the 7 items of the mYFAS was conducted, both for the original and the symptom count scores of the two measures. The two models showed adequate fit:  $\chi^2(153) = 320.448$  ( $p < .001$ ); CFI = .959; SRMR = .062; RMSEA = .059 [.050,.067], for the model in which items with the original score were used; and  $\chi^2(153) = 187.683$  ( $p = .001$ ); CFI = .961; SRMR = .126; RMSEA = .031 [.020,.041], for the model in which symptom count scores were used. As shown in Figure 1, all factor loadings were statistically significant except for item 8 of the mYFAS, which resulted non-statistically significant in both scoring methods.

[INSERT FIGURE 1 HERE]

### **Relationships among FA and other variables**

FA, as measured with the mYFAS 2.0 original scores and symptom count scores, was related to emotional eating, external eating, restrained eating, binge eating, and bulimia. All correlations were statistically significant ( $p < .001$ ) (Table 2).

[INSERT TABLE 2 HERE]

When using the diagnostic scores, differences in emotional eating were also found:  $F(3, 395) = 58.124$ ,  $p < .001$ ,  $\eta^2 = .306$ , with post hoc differences between those showing no FA and the rest of the groups ( $p < .050$ ), with lower emotional eating for the former; also between those with severe FA and the rest of the groups ( $p < .001$ ), being this the group with higher levels of emotional eating; however, no differences in emotional eating were found between those with mild and moderate FA ( $p > .050$ ). Similar results were found

for external eating,  $F(3, 395) = 16.787, p < .001, \eta^2 = .113$ , although the relationship found was of smaller value. Post hoc differences were found between participants with severe FA and the rest of the subgroups ( $p < .010$ ), with higher levels of external eating for those with severe FA. Regarding restrained eating, there were also differences between mYFAS 2.0 diagnostic scores:  $F(3,395) = 28.580, p < .001, \eta^2 = .178$ . Post hoc comparisons pointed statistically significant differences between no FA and mild FA ( $p < .001$ ), moderate FA ( $p = .002$ ) and severe FA ( $p < .001$ ), with lower restrained levels for those with no FA; and statistically significant differences between severe FA and mild FA, with higher levels of restrained for the former ( $p = .022$ ) (see Table 3).

[INSERT TABLE 3 HERE]

Additionally, the relationship of FA and binge eating was studied. Binge eating showed the strongest association with FA:  $F(3, 395) = 96.634, p < .001, \eta^2 = .423$ , with statistically significant post hoc differences between participants with no FA and those with moderate ( $p < .001$ ) or severe ( $p < .001$ ) FA; also, between mild FA and severe FA ( $p < .001$ ); and between moderate FA and severe FA ( $p < .001$ ). In all the cases, the higher the level of FA, the higher binge eating scores (Table 3).

Finally, the relationship between FA and bulimia was studied. This relationship was also found when using the mYFAS 2.0 diagnostic scores:  $F(3,395) = 50.188, p < .001, \eta^2 = .276$ . Post hoc tests showed statistically significant differences in bulimia scores between those with no FA and those with moderate ( $p = .004$ ) and severe ( $p < .001$ ) FA; those with mild and severe FA ( $p < .001$ ); and those with moderate and severe FA ( $p < .001$ ), with higher levels of bulimia for higher FA. Please, see details in Table 3.

### **FA measures with demographics and BMI**

Regarding age, correlations were used to study the relationship with mYFAS 2.0. Both when studied with the original or with symptom count score, the relationship was negative and statistically significant ( $r = -.127, p = .011$ ; and  $r = -.163, p = .001$ ; respectively), with younger individuals reporting higher addictive-like eating behaviors (Table 2). No significant association was found between age and the diagnostic scoring method:  $F(3, 395) = 1.680, p = .171, \eta^2 = .013$  (see Table 3).

As for gender, there were not differences for the original score:  $t(397) = -0.107, p = .915$ ; the symptom count:  $t(397) = -0.615, p = .539$ ; or diagnostic score:  $\chi^2(3) = 0.350, p = .950$ ,  $V$  de Cramer = .030.

Concerning BMI, there were no association with the original or symptom count score ( $r = .075, p = .133$ ; and  $r = .075, p = .137$ ) (Table 2). In addition, no association with mYFAS 2.0 diagnostic categories was found:  $F(3, 395) = 2.195, p = .088, \eta^2 = .016$  (Table 3).

### **Structural equation modeling predicting FA**

To test the predictive power of the variables related to FA in a multivariate context, two structural equation models were hypothesized, estimated, and tested, in which the aforementioned variables and their relationship to FA were simultaneously tested, using both mYFAS 2.0 original and symptom count scores. The models had a clear rationale; as literature has shown, several variables have been related to FA, and therefore may be potential predictors of this problem. The variables included in the models were those previously related to FA (Meule et al., 2014; Schiestl & Gearhardt, 2018; Schulte & Gearhardt, 2017; Wiss & Brewerton, 2020), and included: BMI, emotional eating, external eating, restrained eating, binge eating, and bulimia. All these variables were

hypothesized to directly impact on FA, as presented in Figure 2. These variables were interrelated, with estimated covariances in the structural model among all the exogenous variables with significant product–moment correlations. Correlations are given in Table 4.

[INSERT FIGURE 2 HERE]

[INSERT TABLE 4 HERE]

The two models (Figure 2) fitted the data well:  $\chi^2(104) = 284.089$  ( $p < .001$ ); CFI = .952; SRMR = .051; RMSEA = .066 [.057,.075], for the model in which items with the original score were used; and  $\chi^2(104) = 207.344$  ( $p < .001$ ); CFI = .959; SRMR = .067; RMSEA = .050 [.040,.060], for the model in which symptom count scores were used. All factor loadings were statistically significant ( $p < .001$ ) and ranged from .536 (item 4) to .824 (item 7) in the original scores model, and from .532 (item 3) to .861 (item 7) in the symptom count scores model.

In the case of the model for mYFAS 2.0 original scores, all the predictive variables resulted statistically significant, with binge eating being the one with the strongest relationship with FA (see Figure 2). In all, almost 45% of FA variance was explained ( $R^2 = .445, p < .001$ ). In the case of the model for mYFAS 2.0 symptom count scores, external eating and restrained eating showed no statistically significant predictive power over FA, and, again, binge eating showed the strongest relation. In this case, more than 56% of FA variance was explained ( $R^2 = .566, p < .001$ ).

## **Discussion**

The aim of this study was to examine the psychometric properties of the Spanish version of the mYFAS 2.0 in a sample of young Spaniards, to analyze the relationships between FA with other eating behaviours, sociodemographic variables and BMI, and to test the predictive power of eating behavior variables related to FA using structural equation modeling.

The factor structure of the scale was assessed using the CFA. In both its original scoring method and its recoded version for symptom count, the scale provided evidence of a single dimension of FA, which adequately explained all 11 mYFAS 2.0 items. This result is consistent with Schulte and Gearhardt (2017), who also suggested a good fit for a single-factor model ( $CFI \geq 0.95$ ;  $TLI \geq 0.95$ ). In addition, all items in the original validation had factor loadings for a single factor of 0.73 or higher. Therefore, given the goodness-of-fit parameters and good factor loadings for each item, the authors maintained a single-factor solution for the mYFAS 2.0 (Schulte & Gearhardt, 2017). As for the reliability of the mYFAS 2.0, Cronbach's alpha and Omega were calculated, again using both the original scoring method and in its recoded version for symptom counts. The results were excellent, pointing to the accuracy of the instrument for assessing FA in the Spanish population.

In our study, the frequency of FA revealed that 31.9% of participants met FA criteria. This frequency is higher than that described in Granero et al. (2014) study, who reported 3.3 % FA in young Spanish women using YFAS 2.0. Other studies conducted with college sample showed higher prevalence, ranging from 11.4% to 25% (Gearhardt et al., 2009; Murphy et al., 2014; Rostanzo & Aloisi, 2021). Beyond the potential differences that may arise from using different versions of YFAS2.0 (we use mYFAS2.0), the FA frequency in our study is high and points out the need to analyze in depth the role of specific

variables (age, sex, pathology, BMI, etc.) to have a clearer understanding of their FA presence in the general population.

The present study also analyzed the adequacy of a general factor of FA for both versions, the mYFAS 2.0 and the mYFAS. Results of CFA pointed out that both scales measure, indeed, the same construct. The models were expected to fit adequately, as both scales have good psychometric properties, and the symptoms and scoring form are very similar (Lemeshow et al., 2016; Schulte & Gearhardt, 2017). This is the first time that the relationship between the two measures has been studied.

The relationships between FA and other relevant eating behavior variables (emotional eating, external eating, restrained eating, binge eating, and bulimia) were analyzed. The YFAS 2.0 scale was found to be related to all of them. Regarding emotional and external eating, results are in line with previous literature, ( Berenson et al., 2014; Davis et al., 2011; Gearhardt et al., 2009) That is, a greater tendency to eat in the face of emotional (e.g., anxiety or depression) and/or environmental triggers (e.g., in the presence of delicious food) is related to higher FA. Emotional eaters tend to eat more and eat sweet foods after negative moods (Van Strien et al., 2013), suggesting that food can play a role in calming negative mental states, which may lead to increased food consumption in order to feel better (Dingemans et al., 2009), and this may perhaps contribute to FA. Similarly, external eaters tend to report higher energy intake (Wardle et al., 1992). This overeating may lead to a reduction in the response of the brain reward system (Stice et al., 2013), which may be related to the consumption of larger amounts of food to feel good.

Our data also show a positive relationship between restrained eating and FA, like previous studies (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020). This relationship might be mediated by episodic binge eating, since it has been suggested that dieting can precipitate binge eating (Schiestl & Gearhardt, 2018; Wiss & Brewerton, 2020).



However, this association could come from the reverse direction, in the sense that young people who exhibit FA behaviors may try to restrict eating in order to maintain or lose weight. Future non-correlational studies are needed to clarify how this relationship between dietary intake restriction and FA is established.

Regarding binge eating, a strong positive relationship with FA was observed, being the strongest relationship among all variables, in the same direction than previous studies (Escrivá-Martínez et al., 2019; Gearhardt et al., 2012, 2016). It could be speculated that elevated food consumption may sensitize the dopamine-mesolimbic reward system, resulting in an excessive increase in wanting to continue consuming food, as occurs with drugs (Robinson et al., 2015). In line with this result, a positive relationship between bulimia and FA was also observed. Meule et al. (2014) also noted that bulimia was positively and strongly associated with FA. Both bulimia and FA share several commonalities, such as high food intake or discomfort after eating behavior. In addition, binge eating is a major component of bulimia, and as mentioned, this behavior is strongly and positively related to FA (Escrivá-Martínez et al., 2019; Gearhardt et al., 2012, 2016).

The present study has also analyzed the association between FA and sociodemographic variables (age and gender). In relation to age, higher FA scores were observed in younger participants. Previous studies have also found that younger individuals score higher on FA symptomatology than older individuals (Hauck et al., 2017; Schulte & Gearhardt, 2017). This could be seen as part of a higher vulnerability to addiction in younger people, who can be more susceptible to environmental effects. Their ability to assess risks can be more limited and they may have more difficulties in control to resist the consumption of high-fat/sugar foods. In relation to gender, no significant differences were observed. There is evidence confirming no relationship between addictive-type eating and gender (Ahmed & Sayed, 2017; Hauck et al., 2017; Schulte & Gearhardt, 2017). However some

studies have found that women reported a higher number of FA symptoms than men (Gearhardt et al., 2016; Pursey et al., 2014), although the samples of these studies is usually composed of overweight or obese women (Pursey et al., 2014). Our participants are young people of both sexes, with a normal BMI distribution, so this sample composition could explain the absence of differences.

Regarding BMI, no significant correlation with FA was observed. The original study by Schulte and Gearhardt (2017) found a relationship, although the effect size was small. Other subsequent studies with non-clinical samples also did not find this relationship (Ahmed & Sayed, 2017; Berenson et al., 2015). Again, the characteristics of the samples could explain these differences, and specifically the BMI scores. Participants from the original study showed a high BMI (mean about 27 points), whereas our sample showed a lower BMI (mean 23 points). More data are needed to confirm whether this relationship between BMI and FA occurs or not in non-clinical normoweight samples.

Finally, our last objective was to analyze the predictive power of the dietary variables related to FA (BMI, emotional eating, external eating, restrained eating, binge eating, and bulimia) using structural equation modeling. Our results indicate that BMI predict FA. That is, although our data do not show a significant correlation between these two variables in our sample, results indicate that higher BMI predicts AF. As mentioned, the literature is inconclusive regarding the relationships between these two variables (Meule, 2011; Minhas et al., 2021), but it has been proposed that individuals with genetic risk for obesity may be more predisposed to eating behaviors unhealthy (Gearhardt et al., 2011) as these individuals are more responsive to food stimuli (Rapuano et al., 2017). Longitudinal studies are needed to better understand this association.

The predictive power of eating styles (emotional eating, external eating, and restrained eating) on FA was also observed. However, it is noteworthy that both external and

restrained eating did not predict FA in the model of the mYFAS 2.0 symptom count scores. This discrepancy points out that the different ways of scoring can have different implications. The symptom count measure can have clinical relevance, but for research purposes it is recommended to assess FA with the original score. These results also suggest that interventions focused on healthy eating behaviors could be beneficial to prevent FA. Specifically, interventions focused on emotional regulation, environmental stimulus control, avoiding restrictive diets, and nutrition education, may be useful in this field.

Another relevant finding is the high predictive power of binge eating in FA. In this line, bulimia also predicted FA. The pattern of bulimia (binge eating and food restriction) shares many neurobiological characteristics with drug addiction, for example, the dopaminergic, glutamatergic, and opioid systems are known to play similar roles in both (Hadad & Knackstedt, 2014). In addition, the foods that are often present in binge eating can have a psychoactive effect (through the release of serotonin and tryptophan), which can lead to addiction (Soria-Salas, 2011). It is likely that the behavioral act of binge eating may become an addiction for some individuals, It would be desirable that future studies can corroborate through more ecological and longitudinal and experimental designs whether binge eating and /or bulimia can act as a risk factor for FA.

In short, more than 50% of the variance of FA was explained by these eating behaviors variables. To our best knowledge, this is the first study to demonstrate the predictive relationship of all these dietary variables with FA, thus pointing out its relevance to prevent this problem in young people.

Some limitations of the study should be noted. First, self-reported height and weight were used to calculate BMI. These data should be viewed with caution because weight tends to be underestimated and height to be overestimated (Niedhammer et al., 2000). Second,

since these are university students, the results cannot be generalized to other populations or to a clinical setting. Finally, third, the data were cross-sectional, so no firm conclusions can be drawn about the direction of the associations obtained. More longitudinal designed studies with large and heterogeneous samples are needed to deeper explore these relationships.

## **Conclusion**

In conclusion, the Spanish version of the mYFAS 2.0 showed adequate psychometric properties to assess FA in a nonclinical Spanish sample. Studies could benefit from this scale to identify participants with FA. Furthermore, the predictive power of dietary variables related to FA (BMI, emotional eating, external eating, restrained eating, binge eating, and bulimia) was demonstrated. This is the first study to point out the predictive power of these variables on FA, all together explaining 56.6% of the variance of FA. All these variables can be considered to prevent FA in young people.

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### **Conflict of interest disclosure**

The authors declare no conflict of interest.

### **Ethics approval statement:**

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of University of Valencia (Spain) (March 1, 2018; H151385403893939).

### **Patient consent statement**

All participants signed the informed consent before answering the questionnaires.

## References

- Ahmed, A. Y., & Sayed, A. M. (2017). Prevalence of food addiction and its relationship to body mass index. *Egyptian Journal of Medical Human Genetics*, *18*(3), 257–260. <https://doi.org/10.1016/j.ejmhg.2016.10.002>
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed.). American Psychiatric Association.  
<https://www.cppm.org.ar/wp-content/uploads/2015/06/DSMIV.pdf>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th Ed.). American Psychiatric Association.  
<https://doi.org/10.1176/appi.books.9780890425596>
- Bava, S., & Tapert, S. F. (2010). Adolescent Brain Development and the Risk for Alcohol and Other Drug Problems. *Neuropsychol Rev*, *20*, 398–413.  
<https://doi.org/10.1007/s11065-010-9146-6>
- Berenson, A. B., Laz, T. H., Pohlmeier, A. M., Rahman, M., & Cunningham, K. A. (2014). Bariatric surgery-induced weight loss causes remission of food addiction in extreme obesity. *Obesity*, *22*(8), 1792–1798.  
<https://doi.org/10.1002/OBY.20797>
- Berenson, A. B., Laz, T. H., Pohlmeier, A. M., Rahman, M., & Cunningham, K. A. (2015). Prevalence of Food Addiction Among Low-Income Reproductive-Aged Women. *Journal of Women's Health*, *24*(9), 740–744.  
<https://doi.org/10.1089/JWH.2014.5182>
- Burrows, T., Skinner, J., McKenna, R., & Rollo, M. (2017). Food Addiction, Binge Eating Disorder, and Obesity: Is There a Relationship? *Behavioral Sciences*, *7*(3), 54. <https://doi.org/10.3390/BS7030054>

- Carter, A., Hendrikse, J., Lee, N., Yücel, M., Verdejo-Garcia, A., Andrews, Z., & Hall, W. (2016). The Neurobiology of "food Addiction" and Its Implications for Obesity Treatment and Policy. *Annual Review of Nutrition*, *36*, 105–128.  
<https://doi.org/10.1146/annurev-nutr-071715-050909>
- Cebolla, A., Barrada, J. R., van Strien, T., Oliver, E., & Baños, R. (2014). Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite*, *73*, 58–64. <https://doi.org/10.1016/j.appet.2013.10.014>
- Clark, S., & Saules, K. (2013). Validation of the Yale Food Addiction Scale among a weight-loss surgery population. *Eating Behaviors*, *14*(2), 216–219.  
<https://doi.org/10.1016/J.EATBEH.2013.01.002>
- Clausen, L., Rosenvinge, J. H., Friborg, O., & Rokkedal, K. (2011). Validating the eating disorder inventory-3 (EDI-3): A comparison between 561 female eating disorders patients and 878 females from the general population. *Journal of Psychopathology and Behavioral Assessment*, *33*(1), 101–110.  
<https://doi.org/10.1007/s10862-010-9207-4>
- Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that "food addiction" is a valid phenotype of obesity. *Appetite*, *57*(3), 711–717. <https://doi.org/10.1016/J.APPET.2011.08.017>
- Dingemans, A. E., Martijn, C., Jansen, A. T. M., & van Furth, E. F. (2009). The effect of suppressing negative emotions on eating behavior in binge eating disorder. *Appetite*, *52*(1), 51–57. <https://doi.org/10.1016/j.appet.2008.08.004>
- El Archi, S., Brunault, P., Ballon, N., Réveillère, C., & Barrault, S. (2020). Differential association between food craving, food addiction and eating-related characteristics in persons at risk for eating disorders. *European Review of*

*Applied Psychology*, 70(2), 100513.

<https://doi.org/10.1016/J.ERAP.2019.100513>

Elosua, P., & López-Jáuregui, A. (2012). Internal structure of the spanish adaptation of the eating disorder inventory-3. *European Journal of Psychological Assessment*, 28(1), 25–31. <https://doi.org/10.1027/1015-5759/a000087>

Escrivá-Martínez, T., Galiana, L., Rodríguez-Arias, M., & Baños, R. M. (2019). The binge eating scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Frontiers in Psychology*, 10, 530. <https://doi.org/10.3389/fpsyg.2019.00530>

Fauconnier, M., Rousselet, M., Brunault, P., Thiabaud, E., Lambert, S., Rocher, B., Challet-Bouju, G., & Grall-Bronnec, M. (2020). Food Addiction among Female Patients Seeking Treatment for an Eating Disorder: Prevalence and Associated Factors. *Nutrients*, 12(6), 1897. <https://doi.org/10.3390/NU12061897>

Fletcher, P. C., & Kenny, P. J. (2018). Food addiction: a valid concept? *Neuropsychopharmacology*, 43(13), 2506–2513. <https://doi.org/10.1038/s41386-018-0203-9>

Flint, A. J., Gearhardt, A. N., Corbin, W. R., Brownell, K. D., Field, A. E., & Rimm, E. B. (2014). Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *American Journal of Clinical Nutrition*, 99(3), 578–586. <https://doi.org/10.3945/ajcn.113.068965>

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, 52(2), 430–436. <https://doi.org/10.1016/j.appet.2008.12.003>



- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2016). Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors, 30*(1), 113–121. <https://doi.org/10.1037/adb0000136>
- Gearhardt, A., White, M., & Potenza, M. (2011). Binge Eating Disorder and Food Addiction. *Current Drug Abuse Reviews, 4*(3), 201–207. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3671377/>
- Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An examination of the food addiction construct in obese patients with binge eating disorder. *International Journal of Eating Disorders, 45*(5), 657–663. <https://doi.org/10.1002/eat.20957>
- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*(1), 47–55. [https://doi.org/10.1016/0306-4603\(82\)90024-7](https://doi.org/10.1016/0306-4603(82)90024-7)
- Granero, R., Jiménez-Murcia, S., Gerhardt, A. N., Agüera, Z., Aymamí, N., Gómez-Peña, M., Lozano-Madrid, M., Mallorquí-Bagué, N., Mestre-Bach, G., Neto-Antao, M. I., Riesco, N., Sánchez, I., Steward, T., Soriano-Mas, C., Vintró-Alcaraz, C., Menchón, J. M., Casanueva, F. F., Diéguez, C., & Fernández-Aranda, F. (2018). Validation of the Spanish version of the Yale Food Addiction Scale 2.0 (YFAS 2.0) and clinical correlates in a sample of eating disorder, gambling disorder, and healthy control participants. *Frontiers in Psychiatry, 9*, 208. <https://doi.org/10.3389/fpsy.2018.00208>
- Hadad, N. A., & Knackstedt, L. A. (2014). Addicted to palatable foods: comparing the neurobiology of Bulimia Nervosa to that of drug addiction. *Psychopharmacology, 231*(9), 1897-1912.

<https://doi.org/10.1007/s00213-014-3461-1>

- Harris, J. L., & Fleming-Milici, F. (2019). Food marketing to adolescents and young adults: Skeptical but still under the influence. En F. Folkvord (Ed.). *The Psychology of Food Marketing and OverEating* (pp. 25–43). Routledge.  
<https://doi.org/10.4324/9780429274404-3>
- Hauck, C., Weiß, A., Schulte, E. ., Meule, A., & Ellrott, T. (2017). Prevalence of "Food Addiction" as Measured with the Yale Food Addiction Scale 2.0 in a Representative German Sample and Its Association with Sex, Age and Weight Categories. *Obesity Facts*, *10*(1), 12–24. <https://doi.org/10.1159/000456013>
- IBM Corp. (2016). *IBM SPSS Statistics for Windows, Version 24.0*. IBM Corp.
- Imperator, C., Fabbriatore, M., Vumbaca, V., Innamorati, M., Contardi, A., & Farina, B. (2016). Food Addiction: Definition, measurement and prevalence in healthy subjects and in patients with eating disorders. *Rivista Di Psichiatria*, *51*(2), 60–65. <https://doi.org/10.1708/2246.24196>
- Lemeshow, A. R., Gearhardt, A. N., Genkinger, J. M., & Corbin, W. R. (2016). Assessing the psychometric properties of two food addiction scales. *Eating Behaviors*, *23*, 110–114. <https://doi.org/10.1016/J.EATBEH.2016.08.005>
- Long, C. G., Blundell, J. E., & Finlayson, G. (2015). A Systematic Review of the Application and Correlates of YFAS-Diagnosed "Food Addiction" in Humans: Are Eating-Related "Addictions" a Cause for Concern or Empty Concepts?. *Obesity Facts*, *8*(6), 386–401. <https://doi.org/10.1159/000442403>
- Meule, A. (2011). How prevalent is "food addiction"? *Frontiers in Psychiatry*, *2*, 2009–2012. <https://doi.org/10.3389/fpsy.2011.00061>
- Meule, A. (2012). Food addiction and body-mass-index: A non-linear relationship.

*Medical Hypotheses*, 79(4), 508–511.

<https://doi.org/10.1016/J.MEHY.2012.07.005>

Meule, A., von Rezori, V., & Blechert, J. (2014). Food addiction and bulimia nervosa.

*European Eating Disorders Review*, 22(5), 331–337.

<https://doi.org/10.1002/ERV.2306>

Minhas, M., Murphy, C. M., Balodis, I. M., Acuff, S. F., Buscemi, J., Murphy, J. G., &

MacKillop, J. (2021). Multidimensional elements of impulsivity as shared and unique risk factors for food addiction and alcohol misuse. *Appetite*, 159, 105052.

<https://doi.org/10.1016/J.APPET.2020.105052>

Murphy, C. M., Stojek, M. K., & MacKillop, J. (2014). Interrelationships among

impulsive personality traits, food addiction, and Body Mass Index. *Appetite*, 73,

45–50. <https://doi.org/10.1016/J.APPET.2013.10.008>

Muthén, L. K., & Muthén, B. O. (1998-2017). *Mplus user's guide*. Eight edition.

Muthén & Muthén.

Niedhammer, I., Bugel, I., Bonenfant, S., Goldberg, M., & Leclerc, A. (2000). Validity

of self-reported weight and height in the French GAZEL cohort. *International*

*Journal of Obesity*, 24(9), 1111–1118. <https://doi.org/10.1038/sj.ijo.0801375>

Pedram, P., Wadden, D., Amini, P., Gulliver, W., Randell, E., Cahill, F., Vasdev, S.,

Goodridge, A., Carter, J. C., Zhai, G., Ji, Y., & Sun, G. (2013). Food Addiction:

Its Prevalence and Significant Association with Obesity in the General

Population. *PLoS ONE*, 8(9), e74832.

<https://doi.org/10.1371/journal.pone.0074832>

Pepino, M., Stein, R., Eagon, J., & Klein, S. (2014). Bariatric surgery-induced weight

loss causes remission of food addiction in extreme obesity. *Obesity*, 22(8),

1792–1798. <https://doi.org/10.1002/OBY.20797>

Pursey, K. M., Stanwell, P., Gearhardt, A. N., Collins, C. E., & Burrows, T. L. (2014). The prevalence of food addiction as assessed by the Yale Food Addiction Scale: A systematic review. *Nutrients*, *6*(10), 4552–4590.  
<https://doi.org/10.3390/nu6104552>

Rapuano, K. M., Zieselman, A. L., Kelley, W. M., Sargent, J. D., Heatherton, T. F., & Gilbert-Diamond, D. (2017). Genetic risk for obesity predicts nucleus accumbens size and responsivity to real-world food cues. *Proceedings of the National Academy of Sciences of the United States of America*, *114*(1), 160–165.  
<https://doi.org/10.1073/PNAS.1605548113>

Robinson, M. J., Burghardt, P. R., Patterson, C. M., Nobile, C. W., Akil, H., Watson, S. J., Berridge, K. C., & Ferrario, C. R. (2015). Individual differences in cue-induced motivation and striatal systems in rats susceptible to diet-induced obesity. *Neuropsychopharmacology*, *40*(9), 2113–2123.  
<https://doi.org/10.1038/npp.2015.71>

Rostanzo, E., & Aloisi, A. (2021). Food addiction assessment in a nonclinical sample of the Italian population. *European Journal of Clinical Nutrition*, *1-5*.  
<https://doi.org/10.1038/S41430-021-00974-7>

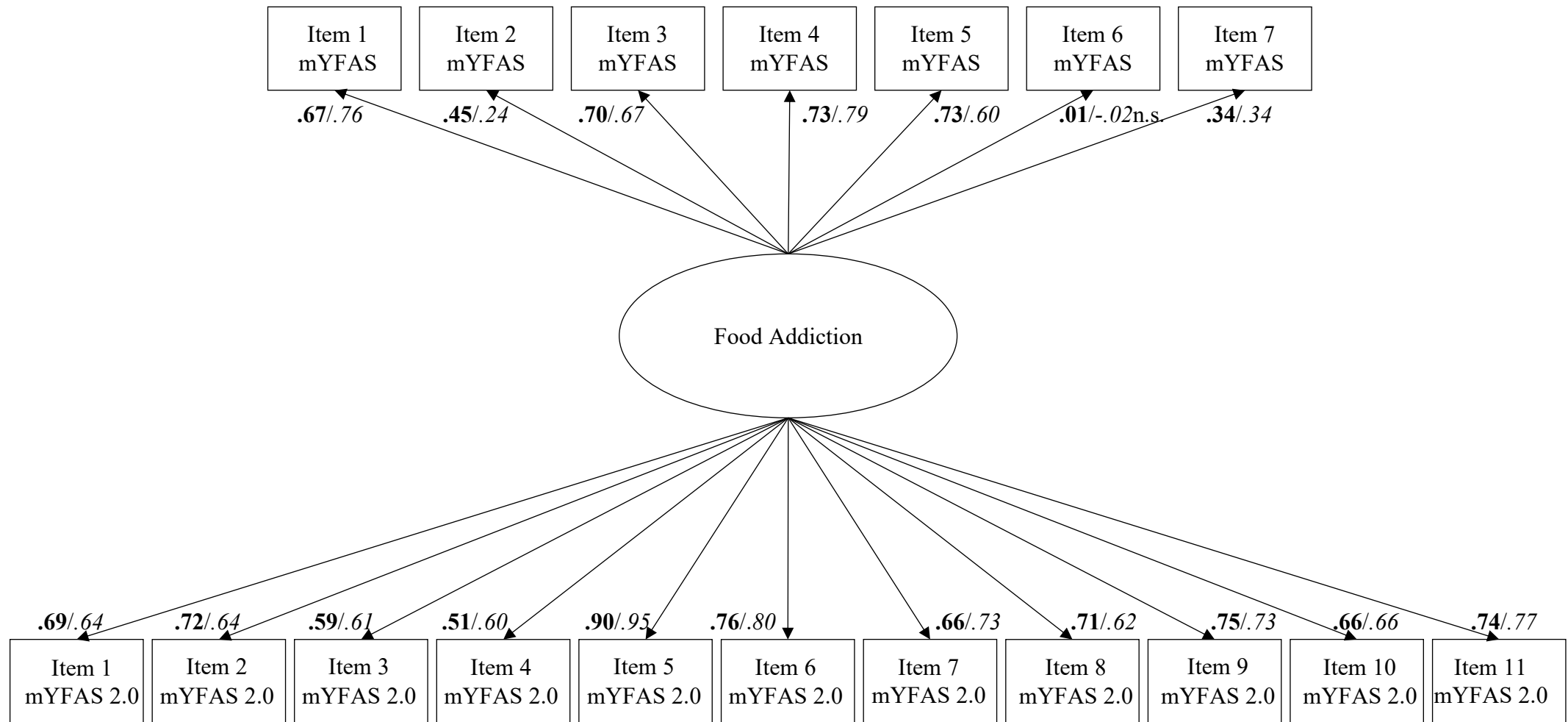
Saules, K. K., Collings, A. S., Hoodin, F., Angelella, N. E., Alschuler, K., Ivezaj, V., Saunders-Scott, D., & Wiedemann, A. A. (2009). The contributions of weight problem perception, BMI, gender, mood, and smoking status to binge eating among college students. *Eating Behaviors*, *10*(1), 1–9.  
<https://doi.org/10.1016/j.eatbeh.2008.07.010>

Schiestl, E. T., & Gearhardt, A. N. (2018). Preliminary validation of the Yale Food

- Addiction Scale for Children 2.0: A dimensional approach to scoring. *European Eating Disorders Review*, 26(6), 605–617. <https://doi.org/10.1002/ERV.2648>
- Schulte, E. M., Avena, N. M., & Gearhardt, A. N. (2015). Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PLoS ONE*, 10(2), 1–18. <https://doi.org/10.1371/journal.pone.0117959>
- Schulte, E. M., & Gearhardt, A. N. (2017). Development of the Modified Yale Food Addiction Scale Version 2.0. *European Eating Disorders Review*, 25(4), 302–308. <https://doi.org/10.1002/erv.2515>
- Soria-Salas, I. (2011). Trastornos de la conducta alimentaria. *Revista Digital de Medicina Psicosomática y Psicoterapia*, 1, 1–24. [https://www.psicociencias.org/pdf\\_noticias/trastornos\\_de\\_la\\_conduca\\_alimentar\\_ia.pdf](https://www.psicociencias.org/pdf_noticias/trastornos_de_la_conduca_alimentar_ia.pdf)
- Stice, E., Figlewicz, D., Gosnell, B., Levine, A., & Pratt, W. (2013). The contribution of brain reward circuits to the obesity epidemic. *Neuroscience and Biobehavioral Reviews*, 37(9 Pt A), 2047–2058. <https://doi.org/10.1016/J.NEUBIOREV.2012.12.001>
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, 5(2), 295–315. [https://doi.org/10.1002/1098-108X\(198602\)5:2<295::AID-EAT2260050209>3.0.CO;2-T](https://doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T)
- van Strien, T., Cebolla, A., Etchemendy, E., Gutierrez-Maldonado, J., Ferrer-Garcia, M., Botella, C., & Baños, R. (2013). Emotional eating and food intake after sadness and joy. *Appetite*, 66, 20–25. <https://doi.org/10.1016/j.appet.2013.02.016>

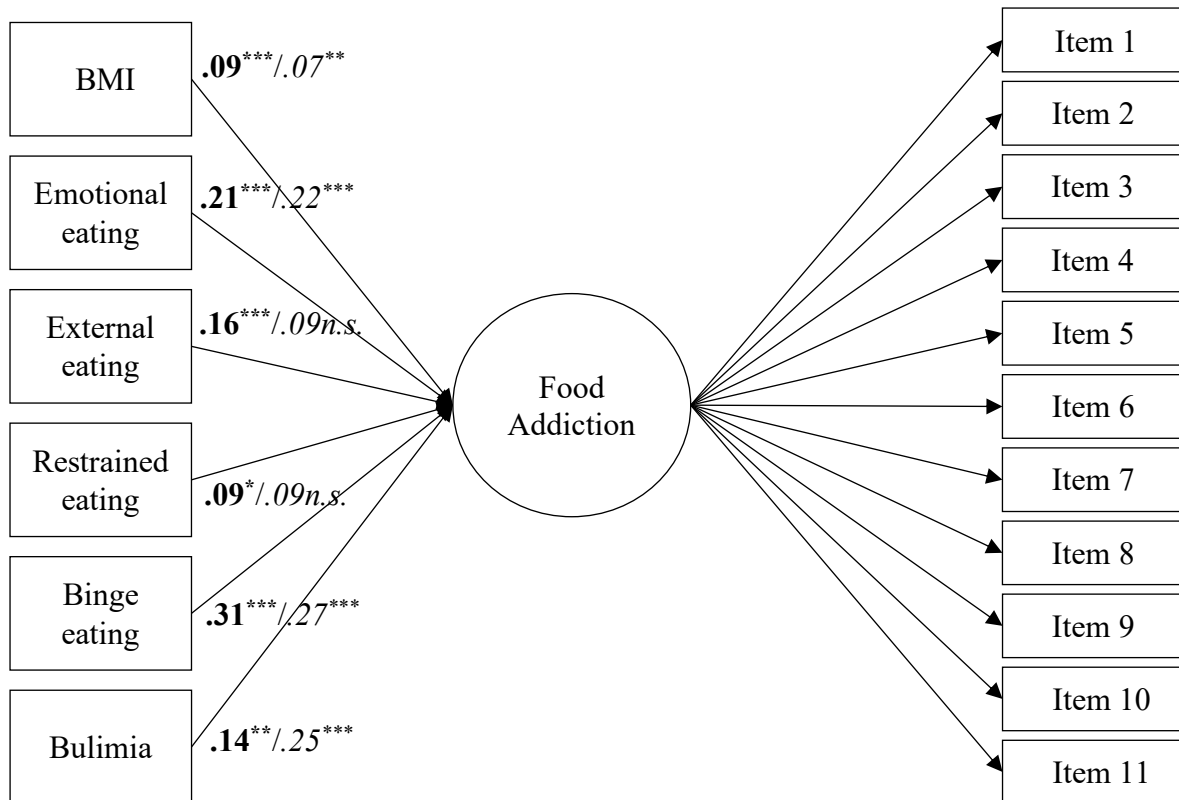
- Volkow, N., Wang, G., Fowler, J., Logan, J., Schlyer, D., Hitzemann, R., Lieberman, J., Angrist, B., Pappas, N., & MacGregor, R. (1994). Imaging endogenous dopamine competition with [<sup>11</sup>C]raclopride in the human brain. *Synapse*, *16*(4), 255–262. <https://doi.org/10.1002/SYN.890160402>
- Wardle, J., Marsland, L., Sheikh, Y., Quinn, M., Fedoroff, I., & Ogden, J. (1992). Eating style and eating behaviour in adolescents. *Appetite*, *18*(3), 167–183. [https://doi.org/10.1016/0195-6663\(92\)90195-C](https://doi.org/10.1016/0195-6663(92)90195-C)
- World Health Organization. Obesity: Preventing and Managing the Global Epidemic; World Health Organization: 2000. <http://www.worldcat.org/title/obesity-preventing-andmanaging-the-global-epidemic-report-of-a-who-consul>
- Wiss, D., & Brewerton, T. (2020). Separating the Signal from the Noise: How Psychiatric Diagnoses Can Help Discern Food Addiction from Dietary Restraint. *Nutrients*, *12*(10), 2937. <https://doi.org/10.3390/NU12102937>

Figure 1. Results of the confirmatory factor analyses, including mYFAS 2.0 and mYFAS scores.



Notes: All factor loadings were statistically significant ( $p < .050$ ), except for those marked n.s. Bold values correspond to the original mYFAS 2.0 and mYFAS scores, whereas italic values correspond to symptom count mYFAS 2.0 and mYFAS scores.

Figure 2. Results of MIMIC models predicting food addiction.



Notes: \*  $p < .050$ ; \*\*  $p < .010$ ; \*\*\*  $p < .001$ ; n.s. non statistically significant. Bold values correspond to the original mYFAS 2.0 scores, whereas italic values correspond to symptom count mYFAS 2.0 scores. Correlations between exogenous variables can be consulted in Table 4.



Table 1. Descriptive statistics and factor loadings for mYFAS 2.0 original and symptom count scores.

Item number	Item content	Original score			Symptom count score		
		M	SD	$\lambda$	M	SD	$\lambda$
1	I ate to the point where I felt physically ill.	0.83	1.28	.704	0.46	0.49	.644
2	I tried and failed to cut down on or stop eating certain foods.	1.27	1.75	.747	0.48	0.50	.748
3	I spent more time feeling sluggish or tired from overeating.	1.25	1.38	.697	0.63	0.48	.662
4	I avoided work, school or social activities because I was afraid I would overeat there.	0.24	0.87	.616	0.10	0.30	.580
5	I kept eating in the same way even though my eating caused emotional problems.	0.69	1.52	.763	0.27	0.44	.801
6	Eating the same amount of food did not give me as much enjoyment as it used to.	0.72	1.40	.683	0.31	0.46	.737
7	If I had emotional problems because I had not eaten certain foods, I would eat those foods to feel better.	1.33	1.90	.527	0.46	0.49	.594

8	My friends or family were worried about how much I overate.	0.36	1.08	.736	0.14	0.35	.748
9	My overeating got in the way of me taking care of my family or doing household chores.	0.08	0.43	.911	0.04	0.20	.994
10	I was so distracted by eating that I could have been hurt (e.g. when driving a car, crossing the street and operating machinery).	0.18	0.69	.637	0.09	0.29	.685
11	I had such strong urges to eat certain foods that I could not think of anything else.	1.29	1.63	.721	0.55	0.49	.637

Table 2. Correlations between mYFAS 2.0 original scores, mYFAS 2.0 symptom count scores, emotional eating, external eating, restrained eating, binge eating, bulimia, age, and BMI.

	<b>mYFAS 2.0 original scores</b>	<b>mYFAS 2.0 symptom count</b>
Emotional eating	r = .596 (p < .001)	r = .588 (p < .001)
External eating	r = .468 (p < .001)	r = .428 (p < .001)
Restrained eating	r = .371 (p < .001)	r = .397 (p < .001)
Binge eating	r = .724 (p < .001)	r = .647 (p < .001)
Bulimia	r = .714 (p < .001)	r = .588 (p < .001)
Age	r = -.127 (p = .011)	r = -.163 (p = .001)
BMI	r = .075 (p = .133)	r = .075 (p = .137)

Table 3. Descriptive statistics for emotional eating, external eating, restrained eating, binge eating, bulimia, age, and BMI, for the different FA diagnostic groups.

	<b>No food addiction</b>		<b>Mild food addiction</b>		<b>Moderate food addiction</b>		<b>Severe food addiction</b>	
	<b>M</b>	<b>SD</b>	<b>M</b>	<b>SD</b>	<b>M</b>	<b>SD</b>	<b>M</b>	<b>SD</b>
Emotional eating	22.44	8.11	26.64	8.56	29.22	10.06	38.27	12.15
External eating	28.77	7.04	29.36	6.24	30.36	6.97	35.13	7.44
Restrained eating	19.77	6.75	24.92	9.13	24.20	7.71	28.15	7.77
Binge eating	3.90	3.27	5.84	4.53	7.62	5.32	14.54	7.64
Bulimia	1.12	2.08	1.42	1.90	3.09	3.67	6.63	6.33
Age	24.60	6.46	24.78	7.80	23.24	3.45	23.12	5.00
BMI	22.55	3.71	22.73	3.85	22.86	3.07	23.82	4.59

Table 4. Correlations between exogenous variables in the structural equation models.

	<b>BMI</b>	<b>Emotional eating</b>	<b>External eating</b>	<b>Restrained eating</b>	<b>Binge eating</b>
BMI	--				
Emotional eating	<b>r = .040 (p = .422)</b> <i>r = .040 (p = .420)</i>	--			
External eating	<b>r = -.021 (p = .643)</b> <i>r = -.022 (p = .640)</i>	<b>r = .512 (p &lt; .001)</b> <i>r = .513 (p &lt; .001)</i>	--		
Restrained eating	<b>r = .141 (p &lt; .001)</b> <i>r = .141 (p &lt; .001)</i>	<b>r = .419 (p &lt; .001)</b> <i>r = .420 (p &lt; .001)</i>	<b>r = .221 (p &lt; .001)</b> <i>r = .220 (p &lt; .001)</i>	--	
Binge eating	<b>r = .137 (p &lt; .001)</b> <i>r = .137 (p &lt; .001)</i>	<b>r = .643 (p &lt; .001)</b> <i>r = .644 (p &lt; .001)</i>	<b>r = .482 (p &lt; .001)</b> <i>r = .482 (p &lt; .001)</i>	<b>r = .544 (p &lt; .001)</b> <i>r = .544 (p &lt; .001)</i>	--
Bulimia	<b>r = .070 (p = .063)</b> <i>r = .070 (p = .063)</i>	<b>r = .646 (p &lt; .001)</b> <i>r = .647 (p &lt; .001)</i>	<b>r = .465 (p &lt; .001)</b> <i>r = .466 (p &lt; .001)</i>	<b>r = .347 (p &lt; .001)</b> <i>r = .347 (p &lt; .001)</i>	<b>r = .714 (p &lt; .001)</b> <i>r = .714 (p &lt; .001)</i>

Notes: Bold values correspond to the original mYFAS 2.0 scores, whereas italic values correspond to symptom count mYFAS 2.0 scores.

## Appendix 1

### Escala Yale de adicción a los alimentos modificada versión 2.0 (mYFAS 2.0)

Esta encuesta pregunta sobre tus hábitos alimentarios del año pasado. Las personas a veces tienen dificultad para controlar la cantidad que comen de ciertos alimentos, como, por ejemplo:

- Dulces como helado, chocolate, donuts, galletas, pastel, caramelos
- Almidones como pan blanco, panecillos, pasta y arroz
- Aperitivos salados como papas fritas, rosquilletas y galletas saladas
- Alimentos ricos en grasa como bistec, tocino, hamburguesas, pizza y papas fritas
- Bebidas azucaradas como gaseosas, refrescos, bebidas para deportistas y bebidas energéticas

Cuando las siguientes preguntas se refieran a "DETERMINADOS ALIMENTOS", por favor piensa en CUALQUIER alimento o bebida similar a los enumerados en los grupos de alimentos o bebidas anteriores o CUALQUIER OTRO alimento con el que hayas tenido dificultad en el último año.

EN LOS ÚLTIMOS 12 MESES:	Nunca	Menos de una vez al mes	Una vez al mes	2-3 veces al mes	Una vez a la semana	2-3 veces a la semana	4-6 veces a la semana	A diario
1. He comido hasta el punto de sentirme físicamente enfermo.	0	1	2	3	4	5	6	7
2. Lo intenté y no pude reducir o dejar de comer ciertos alimentos.	0	1	2	3	4	5	6	7
3. He pasado mucho tiempo sintiéndome lento o cansado por comer en exceso.	0	1	2	3	4	5	6	7
4. He evitado el trabajo, la escuela o las actividades sociales porque tenía miedo de comer en exceso allí.	0	1	2	3	4	5	6	7
5. Seguí comiendo de la misma manera a pesar de que mi alimentación me ha causado problemas emocionales.	0	1	2	3	4	5	6	7

6. Comer la misma cantidad de comida no me ha dado tanto placer como solía hacerlo.	0	1	2	3	4	5	6	7
7. Si tuviera problemas emocionales por no comer ciertos alimentos, los comería para sentirme mejor.	0	1	2	3	4	5	6	7
8. Mis amigos y familiares estaban preocupados por lo mucho que yo comía.	0	1	2	3	4	5	6	7
9. Mi exceso de comida me impidió cuidar de mi familia o hacer tareas domésticas.	0	1	2	3	4	5	6	7
10. Estaba tan distraído al comer que podría haberme herido (por ejemplo, al conducir un coche, al cruzar la calle o al operar con maquinaria).	0	1	2	3	4	5	6	7
11. Tenía tantas ganas de comer ciertos alimentos que no podía pensar en otra cosa.	0	1	2	3	4	5	6	7
12. Tuve problemas significativos en mi vida debido a la comida y al comer. Estos pueden haber sido problemas con mi rutina diaria, trabajo, escuela, amigos, familia, o salud.	0	1	2	3	4	5	6	7
13. Mi comportamiento alimentario me ha causado mucho malestar.	0	1	2	3	4	5	6	7





Annex 5: Study 5 article.  
Eating behaviors, eating  
styles and body mass index  
during COVID-19  
confinement in a college  
sample: a predictive model.



**Title:** Eating behaviors, eating styles and body mass index during COVID-19 confinement in a college sample: a predictive model

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**Title:** Eating behaviors, eating styles and body mass index during COVID-19 confinement in a college sample: a predictive model

### **Abstract**

**Introduction:** COVID-19 confinement affected lifestyles. There is inconclusive evidence about changes in eating patterns, and there are few studies on the impact on body mass index (BMI), the occurrence of dysfunctional behaviors (binge eating, fat intake), and the predictive role of maladaptive eating styles (emotional, external, and restrained eating).

**Purposes:** 1) To analyze the differences in binge eating, fat intake, BMI, and maladaptive eating styles before and during COVID-19 confinement, and 2) to analyze whether maladaptive eating styles (before confinement) predicted binge eating, fat intake, and BMI during confinement.

**Methods:** One hundred forty-six Spanish college students (71.2% female;  $M_{age}=22.93$ ,  $SD_{age}=3.33$ ) completed several dietary measures and BMI twice: before COVID-19 confinement (T1, November 2019) and during COVID-19 confinement (T2, April 2020).

**Results:** BMI and maladaptive eating styles did not change in T2 (vs T1). However, binge eating and fat intake decreased in T2. Emotional eating at T1 positively predicted BMI and binge eating at T2. External eating at T1 positively (and marginally) predicted fat intake at T2. Restrained eating at T1 positively predicted binge eating at T2, and negatively (and marginally) predicted BMI and fat intake at T2. The model explained 80.5% of the variance in BMI, 41.5% of the variance in binge eating, and 25.8% of the variance in fat intake during COVID-19 confinement.

**Conclusion:** The COVID-19 confinement had a positive impact on some eating behaviors. Future policies should focus part of their prevention on maladaptive eating styles to curb dysfunctional eating behaviors and BMI problems in times of stress.

**Keywords:** COVID-19 confinement; maladaptive eating styles; binge eating; fat intake; body mass index; dysfunctional eating behaviors.

### **Level of evidence**

Level IV, Evidence obtained from multiple time series without intervention

## Introduction

The lockdown due to COVID-19 helped in reducing infection rates, but it also involved a change in the population's living habits, especially affecting lifestyles. Research that analyzed how the COVID-19 confinement and the pandemic situation influenced eating behaviors in general population have produced mixed results.

Several studies have shown that social isolation increases negative emotions, and food intake could be a coping strategy to deal with these unpleasant emotions (NIH US National Library of Medicine, 2020). Specifically, it has been observed that feelings of loneliness and boredom can lead individuals to increase food consumption (Moynihan et al., 2015), leading in turn to weight gain if they do not engage in any physical activity (Rodríguez et al., 2020). Being at home and having large amounts of food available 24 hours a day during this period may also have increased food intake. Along these lines, several studies highlighted an increase in the consumption of unhealthy foods (rich in fats and sugars) and an increase in the Body Mass Index (BMI) during confinement (Ammar et al., 2021; Di Renzo et al., 2020; Pellegrini et al., 2020; Sidor & Rzymiski, 2020), although the increase in BMI occurred especially in overweight or obese individuals. In this regard, binge eating behavior -understood as the consumption of a large amount of food in a short time and loss of control during intake- (American Psychiatric Association, 2013) also increased during confinement (Phillipou et al., 2020). Another line of studies pointed out that there was an increase in healthy food consumption and a decrease in fast food intake during the confinement, as individuals had more time to cook and take care of eating and physical exercise behaviors, especially in young people (Di Renzo et al., 2020; Rodríguez-Pérez et al., 2020). In fact, general population reported no changes in their BMI (Di Renzo et al., 2020; Haddad et al., 2021).

In addition to understanding whether eating behaviors changed, it seems highly important to knowing the risk factors that may predict binge eating, unhealthy food consumption (e.g., fat intake), and BMI during confinement. Knowing the risk factors may help us improve their prevention and treatment in future stressful situations. In this regard, maladaptive eating styles stand out as important risk factors: emotional eating (eating in response to negative emotions), external eating (eating in response to external cues, such as smelling delicious food), and restrained eating (eating restrictively to lose or control weight) (van Strien et al., 1986). There is strong evidence suggesting associations between maladaptive eating styles, binge eating and unhealthy food consumption (Mason & Lewis, 2014; Racine et al., 2011; Rolls et al., 2007; Stice et al., 2002).

Individuals with higher emotional eating show greater dysregulation of their emotions and may use food to cope with those negative emotions (Spoor et al., 2007). In fact, emotional eating may also directly affect BMI (Czeczor-Bernat & Brytek-Matera, 2021). Studies conducted during confinement indicate that the way of responding emotionally to COVID-19 may have also increased the risk of dysfunctional eating behaviors. For instance, emotional eaters may have increased food consumption with the goal of seeking emotional reward in food during confinement, overriding any hunger and satiety signals from the body (Cecchetto et al., 2021).

In addition, being an external eater could also have an influence on food consumption during confinement, as perceived stress may influence external eating (Okumus & Ozturk, 2021). Besides, a positive relationship has been observed between external eating and consuming higher amounts of energy and unhealthy food during regular periods (Anschutz et al., 2009). In this regard, considering that large amounts of food were stored at home during confinement (Romeo-Arroyo et al., 2020), and that external eaters tend to eat more in response to food properties, including contextual ones (van Strien et al., 1986), it could be expected that external eaters would eat more -specially, unhealthy- food during confinement. In turn, restrained eaters could also have overeaten in the face of the confinement situation, as stress may increase the risk of binge eating in individuals who exhibit restrained eating behaviors (Woods et al., 2010).

To date, the effect of confinement on eating behaviors remain unclear as conclusions are heterogeneous depending on the study sample (e.g., overweight may be risk factor) (Pellegrini et al., 2020; Sidor & Rzymiski, 2020). However, it should be noted that most studies only measured eating behaviors and BMI at one point in time (during confinement) and asked about eating behavior retrospectively (Ammar et al., 2021; Di Renzo et al., 2020; Haddad et al., 2021; Sidor & Rzymiski, 2020), which could lead to biases. To our best knowledge, there are no studies assessing changes in the eating behaviors and BMI during confinement and comparing to data obtained before confinement. Therefore, the main objective of this study was to analyze the differences in binge eating, fat intake, BMI, and maladaptive eating styles before and during COVID-19 confinement in a sample of college students. Furthermore, considering the literature mentioned, it can be assumed that maladaptive eating styles could influence binge eating behavior, fat intake, and BMI during confinement. However, the predictive role of maladaptive traits of eating styles in these variables has not been studied. Therefore, the secondary objective of this study was to analyze whether maladaptive eating styles (emotional, external, and

restrained eating) assessed before confinement predicted binge eating, fat intake, and BMI during the confinement. We hypothesized changes in binge eating, fat intake and BMI during the confinement, and we expected that these variables would be affected by the maladaptive eating styles. However, we did not specify a hypothesis in any direction due to the exploratory nature of this study.

## Method

### Participants

The sample of the present study consisted of 146 college Spanish students, divided in 104 women (71,2%;  $M_{age}=22.20$ ;  $SD=2.97$ ) and 42 males (28,8%;  $M_{age}=24.74$ ;  $SD=3.53$ ). Before confinement, the mean BMI<sup>1</sup> was 22.66 ( $SD=2.92$ ), and 6.9% were underweight, 75.17% were normal, 16.55% were overweight, and 1.38% were obese. During confinement, the mean BMI was 22.92 ( $SD=3.35$ ), and 6.9% were underweight, 71.72% were normal, 18.62% were overweight, and 2.76% were obese.

Inclusion criteria were: age between 18 and 30 years old, living in Spain during the confinement (April, 2021), and being enrolled in the study conducted in November, 2019 (XXXXXX et al., 2020). Exclusion criteria were: having an eating disorder, having a medical condition that may affect eating behavior or mood, and having a diagnosis of severe mental disorder.

### Measures

***Socio-demographic and anthropometric characteristics.*** Participants included information about their sex, age, marital status, weight, and height.

***Eating styles: Dutch Eating Behavior Questionnaire (DEBQ;*** Cebolla et al., 2014; van Strien et al., 1986). This questionnaire assessed three different eating styles: emotional eating, external eating, and restrained eating. Higher scores mean greater scores in emotional, external, and restrained eating behavior. In this study, the internal consistency was adequate ( $\alpha=.953$  for emotional eating,  $\alpha=.885$  for external eating, and  $\alpha=.920$  for restrained eating).

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<sup>1</sup> BMI was calculated following WHO guidelines (World Health Organization, 2000).



**Binge eating: Binge Eating Scale (BES;** Escrivá-Martínez et al., 2019; Gormally et al., 1982). This questionnaire identifies the symptoms associated with binge eating (eating large amounts of food in a short time and the feel of losing control). Higher scores indicate greater binge eating. In this study, the internal consistency was adequate ( $\alpha=.868$  for before confinement, and  $\alpha=.854$  during confinement).

**Fat intake: Short Fat Questionnaire (SFQ;** Dobson et al., 1993). This scale evaluates the weekly frequency of intake of high-fat food. Higher scores indicate a higher frequency of fat food intake. In this study, the internal consistency was adequate ( $\alpha=.794$  before confinement, and  $\alpha=.826$  during confinement).

### **Design and procedure**

Participants belonging to a previous study (XXXXXX et al., 2020) were contacted via email and were invited to participate. Those who agreed to participate in the current study signed an informed consent, in which also agreed to retrieve their previous information. The first assessment was completed in November 2019 (T1; non-pandemic period in Spain), and the second assessment was completed in April 2020 (T2; strict confinement period in Spain). All the study was conducted using the Lime Survey platform of the University of Valencia.

### **Ethical considerations**

The study was performed under the ethical standards of the Declaration of Helsinki (World Medical Association, 2013) and approved by the ethical committee of the University of XXXXXX (Number: 1821046).

### **Data analyses**

To decide which statistical test to compute to satisfy the first aim of the study, normality assumptions were tested on the difference of pairs in each of the variables of interest using Shapiro-Wilk tests. If the assumption was met, a paired t-test was conducted. Otherwise, a robust paired t-test was carried out, concretely Yuen's test on trimmed means for dependent samples with a 0.2 level of trim (Wilcox, 2013). Effect sizes were computed in the form of Cohen's  $d$  ( $d$ ; (Cohen, 1988)) when

assumptions were met, and in the form of robust Cohen's  $d$  ( $d_R$ ; (Algina et al., 2005)) in case they were not.

Regarding the second aim of the study, a Structural Equation Model (SEM) was tested to examine whether maladaptive eating styles before confinement (T1) had an effect on binge eating, fat intake, and BMI during confinement (T2). A crossed-lagged autoregressive panel model was proposed. The theoretical model is shown in Figure 1. The Maximum Likelihood Robust (MLR) method of estimation was used. Model fit was examined using chi-square statistic ( $\chi^2$ ), Comparative Fit Index (CFI), Weighted Least Squares Mean and Variance corrected (WLSMV), and Standardized Root Mean Square Residual (SRMR) (Hu & Bentler, 1999).

\*INSERT ABOUT HERE FIGURE 1\*

Bivariate analyses were performed using R (R Core Team, 2021). Additional to the system library, R packages employed in the analyses were: WRS2 (Mair & Wilcox, 2020) and LRS (Navarro, 2015). Multivariate analyses were performed using MPlus 8.6 (Muthén & Muthén, 1998).

## Results

### Differences in BMI, binge eating, fat intake, and eating styles, before and during COVID-19 confinement

The Shapiro-Wilk test showed the score difference between T1 and T2 did not follow a normal distribution for BMI, binge eating and fat intake; however, the distribution for all three eating styles were normal (see Table 1).

\*INSERT ABOUT HERE TABLE 1\*

Due to the non-normality for the difference of pairs in binge eating, fat intake and BMI, Yuen's test for dependent samples was performed in these cases. Regarding BMI, no differences were found between T1 ( $M=22.66$ ,  $SD=2.92$ ) and T2 ( $M=22.92$ ,  $SD=3.35$ ),  $t(87)=-0.68$ ,  $p=0.499$ ,  $d_R=0.21$ . However, regarding binge eating and fat intake, individuals exhibited a reduction in their binge eating habits during confinement by COVID-19 compared to the time before the confinement (T1:  $M=7.67$ ,  $SD=6.33$ ; T2:  $M=6.53$ ,  $SD=5.82$ ;  $t(87)=2.07$ ,  $p=.041$ ,  $d_R=0.23$ ), and also decreased their fat intake during confinement (T1:  $M=20.39$ ,  $SD=8.17$ ; T2:  $M=22.21$ ,  $SD=7.70$ ;  $t(87)=2.75$ ,  $p=.007$ ,  $d_R=0.36$ ).

Regarding maladaptive eating styles, t-tests were computed to examine differences in emotional, external, and restrained eating before and during the COVID-19 confinement. Results showed no statistically significant differences for emotional eating before and during the restrictions (T1:  $M=25.27$ ,  $SD=9.90$ ; T2:  $M=25.42$ ,  $SD=10.44$ ;  $t(143)=-0.42$ ,  $p=.672$ ,  $d=0.035$ ). For external eating, no statistically significant differences emerged (T1:  $M=29.40$ ,  $SD=7.20$ ; T2:  $M=28.77$ ,  $SD=7.17$ ;  $t(143)=1.20$ ,  $p=.231$ ,  $d=0.10$ ). This was also the case for restrained eating (T1:  $M=21.31$ ,  $SD=7.88$ ; T2:  $M=21.71$ ,  $SD=7.93$ ;  $t(143)=-0.83$ ,  $p=.408$ ,  $d=.069$ ).

### **The predicting role of maladaptive eating styles (before confinement) in BMI, binge eating, fat intake, and eating styles (during confinement).**

The theoretical model shown in Figure 1 was established and tested. Model results indicated an excellent fit of model to the data:  $\chi^2(21)=22.04$ ,  $p=.397$ , CFI=.99, RMSEA=.018 (90% CI .000 - .073), SRMR=.036. Standardized model effects are shown in Table 2. Correlations among exogenous variables can be shown in Table 3 and correlations among dependent variables are displayed in Table 4.

\*INSERT TABLE 2, 3 and 4\*

Regarding the effects of eating styles before confinement (T1) on binge eating, fat intake, and BMI during confinement (T2), results showed that emotional eating in T1 had statistically significant effects on both BMI ( $\beta=.105$ ,  $p=.036$ ) and binge eating ( $\beta=.159$ ,  $p=.041$ ) at T2. Restrained eating in T1 also displayed a statistically significant effect on binge eating ( $\beta=.165$ ,  $p=.004$ ) in T2 and a marginally significant effect on BMI ( $\beta=-.080$ ,  $p=.071$ ) in T2. Lastly, both restrained eating ( $\beta=-.102$ ,  $p=.055$ ) and external eating ( $\beta=.138$ ,  $p=.090$ ) before confinement had marginally significant effects on fat intake at T2.

The model explained an 80.5% of variance in BMI, a 41.5% of binge eating, and a 25.8% of fat intake during COVID-19 confinement. Moreover, eating styles scores before confinement explained the variance of eating styles during confinement to different extents: 47.9% for emotional eating, 40.8% for external eating, and 50.3% for restrained eating.

## **Discussion**

The current longitudinal study compares binge eating, fat intake, BMI, and maladaptive eating behaviors before and during the strict COVID-19 confinement in a sample of Spanish college students. Furthermore, the predictive role of maladaptive eating styles (emotional, external, and restrained eating) before the confinement was also analyzed.

The first objective was focused on analyzing the differences in binge eating, fat intake, BMI, and maladaptive eating behaviors between before and during COVID-19 confinement. Regarding BMI, no significant changes were observed. This stability in BMI could be related to the fact that our sample adopted healthy lifestyle habits during confinement. Individuals decreased their binge eating and fat intake during the lockdown. Although this finding could be surprising -since it could be reasonable to think that people eat worse in very stressful times- (Romeo-Arroyo et al., 2020), this is in line with other studies that showed that people adopted healthy habits during this COVID-19 confinement (Romero-Blanco et al., 2020). One possible explanation is that the consumed food was healthier since it was homemade, and there was less access to restaurants and ultra-processed foods (Di Renzo et al., 2020; Sinisterra-Loaiza et al., 2021). In addition, some evidence shows that younger people adhered better to healthy eating patterns than older people (Di Renzo et al., 2020). Furthermore, most of the participants had a normal weight condition (75%) before confinement, and maybe, we can speculate that these dysfunctional eating behaviors only occurred in overweight or obese young people. Along this lines, Di Renzo et al. (Di Renzo et al., 2020) pointed out that people with normal weight have greater adherence to healthy eating guidelines than overweight people.

As regards the changes in emotional, external, and restrained eating, no changes we found before and during confinement. The literature supports these results, as eating styles tend to be considered as a trait rather than state factors (Malesza & Kaczmarek, 2021) and often show temporal stability in the young population despite adverse circumstances (Meiselman et al., 1998), as well as in the clinical population (Malesza & Kaczmarek, 2021). Therefore, our result reinforces the idea that eating attitudes have a stable character, even after experiencing stressful events, such as COVID-19 confinement.

The second objective was aimed at analyzing whether the maladaptive eating styles assessed before COVID-19 confinement influenced binge eating, fat intake, and BMI during confinement. The scores revealed that emotional eating positively predicted BMI and binge eating but did not predict fat intake. This result can be explained by the classical psychosomatic theory, in which people who exhibit emotional eating cannot differentiate hunger from physiological signals accompanying negative emotions

(Bruch, 1964). Therefore, individuals respond to negative emotions by eating more (Hemmingsson, 2014). The emotional eating also predicted the increase in BMI, confirming longitudinally the strong relationship found in the literature (Koenders & van Strien, 2011; Kontinen et al., 2019).

In the present study, the previous emotional eating influenced only binge eating and not fat intake. It can be possible that unhealthy food consumption did not occur in response to negative emotions in this case (Adriaanse et al., 2011). Another hypothesis could be that emotional eating may be associated with impulsivity behavior and loss of control over food intake in the face of negative emotions, using this behavior to regulate the emotions. In this case, it might not influence what someone eats (e.g., unhealthy food) but how much it is eaten (e.g., binge eating). Future studies are needed to further analyze which personal factors predispose to emotional eating.

Our results also indicated that external eating positively and marginally predicted fat intake during confinement, although it did not predict binge eating and BMI. The first result supports the idea of other studies, which indicate that external eating is often associated with higher food intake, principally high-fat foods (Anschutz et al., 2009). However, external eating did not predict binge eating during confinement. Although external eating style may serve as a predisposing factor for binge eating, this seems to occur in combination with high BMI and depressive problems (Mason & Lewis, 2014). Regarding the non-significant effect of external eating on BMI, this was consistent with the results of another study (Anschutz et al., 2009). Hence, this finding imply that an increase in BMI in our sample may be determined to a greater extent by emotional eating than by the response to environmental cues (van Strien et al., 2009). However, considering that most of our participants had a normal weight and the sample was nonclinical, it seems plausible that this relation was no significant. Moreover, it should be noted that the BMI variability of our sample is limited, with underrepresented BMI ranges, which could be biasing this relationship. Furthermore, the relationship between external eating and BMI could have been mediated by the craving for fatty food and fast food (Burton et al., 2007). Future studies should analyze whether the craving influences this relationship during confinement since only the direct relationship between external eating and BMI was studied in our study.

Finally, our results showed that restrained eating positively predicted binge eating, and negatively predicted fat intake and BMI during confinement, although the latter result was marginal. These results are consistent with the fact that restrained eaters want to lose or maintain weight, leading them to eat less fat to accomplish this goal (Anschutz et al., 2009). However, calorie deprivation may

precipitate binge eating, because of the loss of cognitive control and the inability to maintain the diet (Fairburn et al., 2003; Stice et al., 2000).

Our findings suggest that interventions focused on reducing dysfunctional eating behaviors in youth at times of high stress should focus on providing cues to control emotional hunger in youth (e.g., mindful eating techniques) and on controlling or reducing any external or restrained behaviors that may lead to the increase of binge eating, fat intake or BMI. Understanding the causal relationship between different eating behaviors is necessary to prevent and treat obesity and health problems in youth.

### **Strengths and limitations**

This study has some limitations. One weakness is that body weight was self-reported, which could lead to underestimating or overestimating the data. Besides, measures of eating behaviors were also self-reported, which may lead to biased results compared to more structured clinical assessments or interviews (Berg et al., 2012). Additionally, the sample's representativeness is narrow, as the study was limited to college students. Future studies should include other populations, such as older adults, or clinical populations. Despite these limitations, our study also presents several strengths. It is a longitudinal study that examines dietary variables and BMI before and during confinement; so, the present study is not biased by the common limitations of the retrospective studies. In addition, it is the first study to assess the predictive role of maladaptive eating styles on binge eating, fat intake, and BMI in a highly stressful context.

### **Conclusion**

COVID-19 confinement may have a positive impact on youths' eating behaviors. In addition, it has been shown that emotional, external, and restrained eating can directly influence binge eating, fat intake, and BMI during the COVID-19 confinement. Most people experienced confinement as a stressful situation, which implied a sort of characteristics that are specific for this situation (e.g., the movement restriction), but with some commonalities with other stressful situations (e.g., the uncertainty about the future, the emotional dysregulation). In this sense, results obtained in the study could be extended to other situations and could lead to future studies, where preventing or treating maladaptive eating styles may help preventing eating and weight related problems.

**What is already known on this subject?**

Previous cross-sectional or retrospective studies have assessed the changes in dysfunctional eating behaviors and body mass index (BMI) during confinement, but no studies have assessed longitudinally these variables before and during confinement. Evidence has also demonstrated associations between maladaptive eating styles (e.g., emotional eating, external eating, or restrained eating) with binge eating, fat intake, and BMI. However, the previous findings show the limitations of the retrospective studies (e.g., recall bias) and cross-sectional studies (e.g., difficulties in verifying the cause-and-effect relationships).

### **What does this study add?**

This longitudinal study shows the changes in binge eating, fat intake, BMI, and maladaptive eating styles (before and during confinement in Spain), and the predictive role of maladaptive eating styles (before the confinement) on binge eating, fat intake, BMI (during the confinement) in a sample of college students.

### **Statements and Declarations**

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#### **Competing interests**

The authors have no relevant financial or non-financial interests to disclose.

#### **Author Contributions**

Conceptualization, T.E.-M., M.R.-A. and R.M.B.; methodology, T.E.-M. and R.H.; software, T.E.-M. and R.H.; formal analysis, T.E.-M. and R.H.; resources, M.R.-A. and R.M.B.; data curation, T.E.-M. and R.H.; writing—original draft preparation, T.E.-M. and R.H.; writing—review and editing, all authors; supervision, M.R.-A. and R.M.B.; funding acquisition, M.R.-A. and R.M.B. All authors have read and agreed to the published version of the manuscript.

### **Data availability**

The datasets generated during and/or analyzed during the current study are available in the OSF repository, [https://osf.io/qfc7d/?view\\_only=85911ed7b2e24a5ab16447a887b90520](https://osf.io/qfc7d/?view_only=85911ed7b2e24a5ab16447a887b90520)

### **Ethics approval**

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the University of Valencia (Number: 1821046).

### **Consent to participate**

Informed consent was obtained from all individual participants included in the study.

### **References**

- Adriaanse, M. A., de Ridder, D. T. D., & Evers, C. (2011). Emotional eating: Eating when emotional or emotional about eating? *Psychology & Health, 26*(1), 23–39.  
<https://doi.org/10.1080/08870440903207627>
- Algina, J., Keselman, H. J., & Penfield, R. D. (2005). An alternative to Cohen's standardized mean difference effect size: A robust parameter and confidence interval in the two independent groups case. *Psychological Methods, 10*(3), 317–328. <https://doi.org/10.1037/1082-989X.10.3.317>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). American Psychiatric Association.
- Ammar, A., Trabelsi, K., Brach, M., Chtourou, H., Boukhris, O., Masmoudi, L., Bouaziz, B., Bentlage, E., How, D., Ahmed, M., Mueller, P., Mueller, N., Hammouda, O., Paineiras-Domingos, L. L.,



- Braakman-Jansen, A., Wrede, C., Bastoni, S., Pernambuco, C. S., Mataruna, L., ... Hoekelmann, A. (2021). Effects of home confinement on mental health and lifestyle behaviours during the COVID-19 outbreak: insights from the ECLB-COVID19 multicentre study. *Biology of Sport*, *38*(1), 9–21. <https://doi.org/10.5114/BIOLSPORT.2020.96857>
- Anschutz, D. J., Van Strien, T., Van De Ven, M. O. M., & Engels, R. C. M. E. (2009). Eating styles and energy intake in young women. *Appetite*, *53*(1), 119–122. <https://doi.org/10.1016/j.appet.2009.03.016>
- Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2012). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *International Journal of Eating Disorders*, *45*(3), 428–438. <https://doi.org/10.1002/eat.20931>
- Bruch, H. (1964). Psychological Aspects of Overeating And Obesity. *Psychosomatics*, *5*(5), 269–274. [https://doi.org/10.1016/S0033-3182\(64\)72385-7](https://doi.org/10.1016/S0033-3182(64)72385-7)
- Burton, P., Smit, H. J., & Lightowler, H. J. (2007). The influence of restrained and external eating patterns on overeating. *Appetite*, *49*(1), 191–197. <https://doi.org/10.1016/j.appet.2007.01.007>
- Cebolla, A., Barrada, J. R., van Strien, T., Oliver, E., & Baños, R. (2014). Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite*, *73*, 58–64. <https://doi.org/10.1016/j.appet.2013.10.014>
- Cecchetto, C., Aiello, M., Gentili, C., Ionta, S., & Osimo, S. A. (2021). Increased emotional eating during COVID-19 associated with lockdown, psychological and social distress. *Appetite*, *160*, 105122. <https://doi.org/10.1016/j.appet.2021.105122>
- Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). Academic Press.
- Czeczor-Bernat, K., & Brytek-Matera, A. (2021). The impact of food-related behaviours and emotional functioning on body mass index in an adult sample. *Eating and Weight Disorders*, *26*(1), 323–329. <https://doi.org/10.1007/S40519-020-00853-3>
- Di Renzo, L., Gualtieri, P., Pivari, F., Soldati, L., Attinà, A., Cinelli, G., Cinelli, G., Leggeri, C., Caparello, G., Barrea, L., Scerbo, F., Esposito, E., & De Lorenzo, A. (2020). Eating habits and lifestyle changes during COVID-19 lockdown: An Italian survey. *Journal of Translational Medicine*, *18*(1), 229. <https://doi.org/10.1186/s12967-020-02399-5>
- Dobson, A. J., Blijlevens, R., Alexander, H. M., Croce, N., Heller, R. F., Higginbotham, N., Pike, G.,

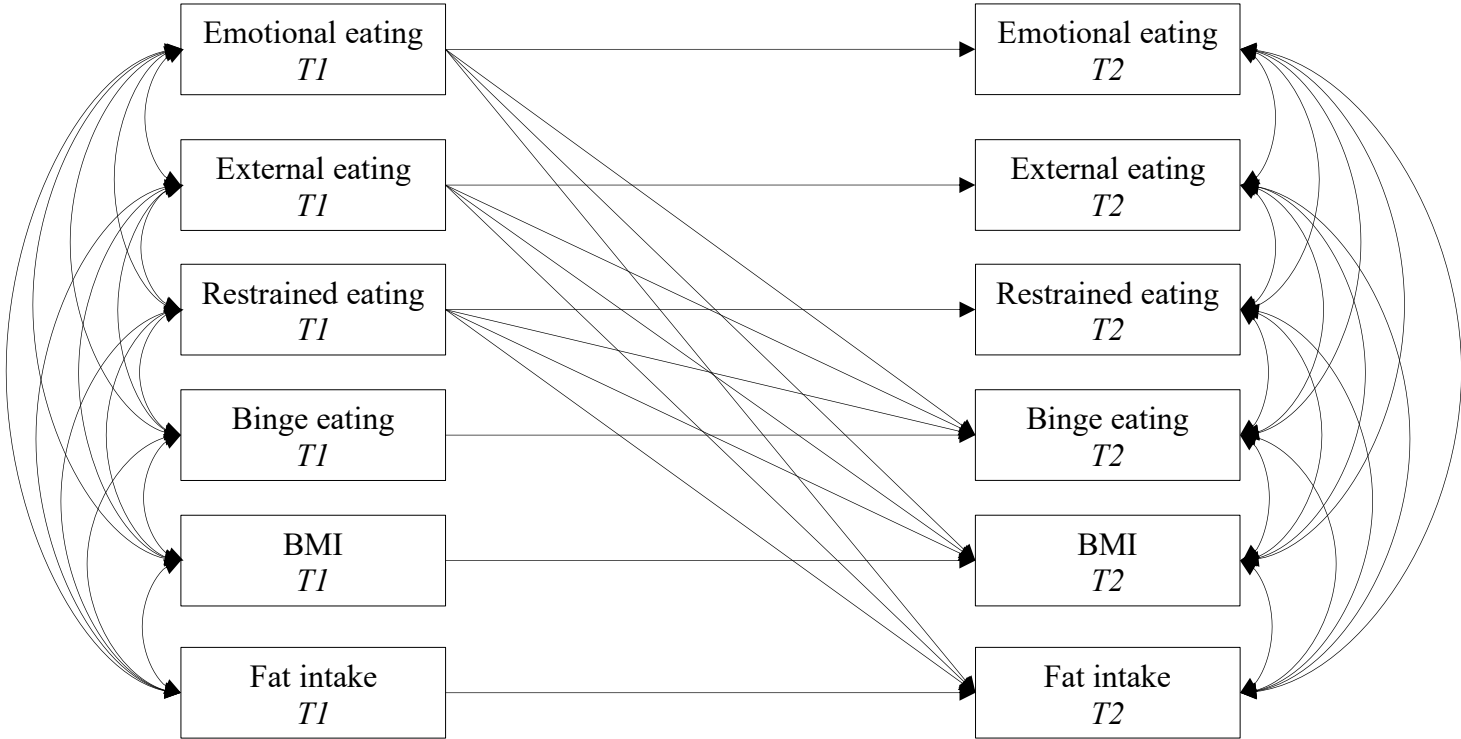
- Plotnikoff, R., Russell, A., & Walker, R. (1993). Short fat questionnaire: a self-administered measure of fat-intake behaviour. *Australian Journal of Public Health, 17*(2), 144–149.  
<https://doi.org/10.1111/j.1753-6405.1993.tb00123.x>
- Escrivá-Martínez, T., Galiana, L., Rodríguez-Arias, M., & Baños, R. M. (2019). The binge eating scale: Structural equation competitive models, invariance measurement between sexes, and relationships with food addiction, impulsivity, binge drinking, and body mass index. *Frontiers in Psychology, 10*, 530. <https://doi.org/10.3389/fpsyg.2019.00530>
- Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: a “transdiagnostic” theory and treatment. *Behaviour Research and Therapy, 41*(5), 509–528.  
[https://doi.org/10.1016/S0005-7967\(02\)00088-8](https://doi.org/10.1016/S0005-7967(02)00088-8)
- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*(1), 47–55. [https://doi.org/10.1016/0306-4603\(82\)90024-7](https://doi.org/10.1016/0306-4603(82)90024-7)
- Haddad, C., Zakhour, M., Siddik, G., Haddad, R., Sacre, H., & Salameh, P. (2021). COVID- 19 outbreak: Does confinement have any impact on weight change perception? *Nutrition Clinique et Métabolisme, 35*(2), 137–143. <https://doi.org/10.1016/j.nupar.2021.02.003>
- Hemmingsson, E. (2014). A new model of the role of psychological and emotional distress in promoting obesity: conceptual review with implications for treatment and prevention. *Obesity Reviews, 15*(9), 769–779. <https://doi.org/10.1111/obr.12197>
- Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*(1), 1–55.  
<https://doi.org/10.1080/10705519909540118>
- Koenders, P. G., & van Strien, T. (2011). Emotional Eating, Rather Than Lifestyle Behavior, Drives Weight Gain in a Prospective Study in 1562 Employees. *Journal of Occupational & Environmental Medicine, 53*(11), 1287–1293. <https://doi.org/10.1097/JOM.0b013e31823078a2>
- Kontinen, H., van Strien, T., Männistö, S., Jousilahti, P., & Haukkala, A. (2019). Depression, emotional eating and long-term weight changes: a population-based prospective study. *International Journal of Behavioral Nutrition and Physical Activity, 16*(1), 28. <https://doi.org/10.1186/s12966-019-0791-8>
- Mair, P., & Wilcox, R. (2020). Robust statistical methods in R using the WRS2 package. *Behavior Research Methods, 52*(2), 464–488. <https://doi.org/10.3758/s13428-019-01246-w>

- Malesza, M., & Kaczmarek, M. C. (2021). One year reliability of the Dutch eating behavior questionnaire: an extension into clinical population. *Journal of Public Health: From Theory to Practice*, 29, 463–469. <https://doi.org/10.1007/s10389-019-01147-4>
- Mason, T. B., & Lewis, R. J. (2014). Profiles of Binge Eating: The Interaction of Depressive Symptoms, Eating Styles, and Body Mass Index. *Eating Disorders*, 22(5), 450–460. <https://doi.org/10.1080/10640266.2014.931766>
- Meiselman, H. L., Mastroianni, G., Buller, M., & Edwards, J. (1998). Longitudinal measurement of three eating behavior scales during a period of change. *Food Quality and Preference*, 10(1), 1–8. [https://doi.org/10.1016/S0950-3293\(98\)00013-5](https://doi.org/10.1016/S0950-3293(98)00013-5)
- Moynihan, A. B., van Tilburg, W. A. P., Igou, E. R., Wisman, A., Donnelly, A. E., & Mulcaire, J. B. (2015). Eaten up by boredom: Consuming food to escape awareness of the bored self. *Frontiers in Psychology*, 6(APR), 369. <https://doi.org/10.3389/FPSYG.2015.00369>
- Muthén, L. K., & Muthén, B. O. (1998). *Mplus User's Guide*. (8th Ed.). Muthén & Muthén.
- Navarro, D. J. (2015). *Learning statistics with R: A tutorial for psychology students and other beginners*. (V. 0.6 (ed.)). University of New South Wales.
- NIH US National Library of Medicine. (2020). *Eating Habits of Adults During the Quarantine*. *Clinical trials*. <https://clinicaltrials.gov/ct2/show/NCT04339842>
- Okumus, B., & Ozturk, A. B. (2021). The impact of perceived stress on US millennials' external and emotional eating behavior. *British Food Journal*, 123(1), 1–11. <https://doi.org/10.1108/BFJ-07-2019-0490/FULL/PDF>
- Pellegrini, M., Ponzio, V., Rosato, R., Scumaci, E., Goitre, I., Benso, A., Belcastro, S., Crespi, C., De Michieli, F., Ghigo, E., Broglio, F., & Bo, S. (2020). Changes in Weight and Nutritional Habits in Adults with Obesity during the “Lockdown” Period Caused by the COVID-19 Virus Emergency. *Nutrients*, 12(7), 2016. <https://doi.org/10.3390/NU12072016>
- Phillipou, A., Meyer, D., Neill, E., Tan, E. J., Toh, W. L., Van Rheenen, T. E., & Rossell, S. L. (2020). Eating and exercise behaviors in eating disorders and the general population during the COVID-19 pandemic in Australia: Initial results from the COLLATE project. *International Journal of Eating Disorders*, 53(7), 1158–1165. <https://doi.org/10.1002/EAT.23317>
- R Core Team. (2021). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing. <https://www.r-project.org/>.

- Racine, S. E., Burt, S. A., Iacono, W. G., McGue, M., & Klump, K. L. (2011). Dietary restraint moderates genetic risk for binge eating. *Journal of Abnormal Psychology, 120*(1), 119–128.  
<https://doi.org/10.1037/A0020895>
- Rodríguez-Pérez, C., Molina-Montes, E., Verardo, V., Artacho, R., García-Villanova, B., Guerra-Hernández, E. J., & Ruíz-López, M. D. (2020). Changes in Dietary Behaviours during the COVID-19 Outbreak Confinement in the Spanish COVIDiet Study. *Nutrients, 12*(6), 1730.  
<https://doi.org/10.3390/NU12061730>
- Rodríguez, M. Á., Crespo, I., & Olmedillas, H. (2020). Exercising in times of COVID-19: what do experts recommend doing within four walls? *Revista Espanola De Cardiologia (English Ed.), 73*(7), 527–529. <https://doi.org/10.1016/J.REC.2020.04.001>
- Rolls, B. J., Roe, L. S., & Meengs, J. S. (2007). The effect of large portion sizes on energy intake is sustained for 11 days. *Obesity, 15*(6), 1535–1543. <https://doi.org/10.1038/oby.2007.182>
- Romeo-Arroyo, E., Mora, M., & Vázquez-Araújo, L. (2020). Consumer behavior in confinement times: Food choice and cooking attitudes in Spain. *International Journal of Gastronomy and Food Science, 21*, 100226. <https://doi.org/10.1016/J.IJGFS.2020.100226>
- Romero-Blanco, C., Rodríguez-Almagro, J., Onieva-Zafra, M. D., Parra-Fernández, M. L., Prado-Laguna, M. D. C., & Hernández-Martínez, A. (2020). Physical activity and sedentary lifestyle in university students: Changes during confinement due to the covid-19 pandemic. *International Journal of Environmental Research and Public Health, 17*(18), 1–13.  
<https://doi.org/10.3390/IJERPH17186567>
- Sidor, A., & Rzymiski, P. (2020). Dietary Choices and Habits during COVID-19 Lockdown: Experience from Poland. *Nutrients, 12*(6), 1657. <https://doi.org/10.3390/nu12061657>
- Sinisterra-Loaiza, L. I., Vázquez, B. I., Miranda, J. M., Cepeda, A., & Cardelle-Cobas, A. (2021). Hábitos alimentarios en la población gallega durante el confinamiento por la COVID-19. *Nutricion Hospitalaria, 37*(6), 1190–1196. <https://doi.org/10.20960/nh.03213>
- Spoor, S. T. P., Bekker, M. H. J., Van Strien, T., & van Heck, G. L. (2007). Relations between negative affect, coping, and emotional eating. *Appetite, 48*(3), 368–376.  
<https://doi.org/10.1016/J.APPET.2006.10.005>
- Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *International Journal of Eating Disorders, 27*(2), 218–229.

- [https://doi.org/10.1002/\(SICI\)1098-108X\(200003\)27:2<218::AID-EAT10>3.0.CO;2-1](https://doi.org/10.1002/(SICI)1098-108X(200003)27:2<218::AID-EAT10>3.0.CO;2-1)
- Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset in adolescent girls: A 2-year prospective investigation. *Health Psychology, 21*(2), 131–138. <https://doi.org/10.1037/0278-6133.21.2.131>
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders, 5*(2), 295–315. [https://doi.org/10.1002/1098-108X\(198602\)5:2<295::AID-EAT2260050209>3.0.CO;2-T](https://doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T)
- van Strien, T., Herman, C. P., & Verheijden, M. W. (2009). Eating style, overeating, and overweight in a representative Dutch sample. Does external eating play a role? *Appetite, 52*(2), 380–387. <https://doi.org/10.1016/j.appet.2008.11.010>
- Wilcox, R. (2013). *Introduction to Robust Estimation and Hypothesis Testing* (3rd ed.). Elsevier.
- Woods, A. M., Racine, S. E., & Klump, K. L. (2010). Examining the relationship between dietary restraint and binge eating: Differential effects of major and minor stressors. *Eating Behaviors, 11*(4), 276–280. <https://doi.org/10.1016/J.EATBEH.2010.08.001>
- World Health Organization. (2000). *Obesity: preventing and managing the global epidemic: report of a WHO consultation*. World Health Organization. <http://www.worldcat.org/title/obesity-preventing-and-managing-the-global-epidemic-report-of-a-who-consultation/oclc/48171257>
- World Medical Association. (2013). World Medical Association declaration of Helsinki: Ethical principles for medical research involving human subjects. *JAMA - Journal of the American Medical Association, 310*(20), 2191–2194. <https://doi.org/10.1001/jama.2013.281053>

Figure 1. Theoretical proposed model.



**Table 1.** Results of the Shapiro-Wilk normality tests.

Variable	<i>W</i>	<i>p</i>
Emotional eating	.989	.347
External eating	.987	.217
Restrained eating	.985	.125
Body Mass Index	.922	<.001
Binge eating	.976	.013
Fat intake	.917	<.001

**Table 2.** Coefficients, standard errors, and p-values of the tested model.

	Standardized coefficient	Standard error	p-value
<i>Emotional eating T2</i>			
Emotional eating T1	.692	.046	<.001
<i>External eating T2</i>			
External eating T1	.639	.052	<.001
<i>Restrained eating T2</i>			
Restrained eating T1	.709	.044	<.001
<i>BMI T2</i>			
BMI T1	.897	.024	<.001
Emotional eating T1	.105	.050	.036
External eating T1	-.014	.048	.766
Restrained eating T1	-.080	.044	.071
<i>Binge eating T2</i>			
Binge eating T1	.418	.082	<.001
Emotional eating T1	.159	.078	.041
External eating T1	.046	.070	.512
Restrained eating T1	.165	.057	.004
<i>Fat intake T2</i>			
Fat intake T1	.425	.095	<.001
Emotional eating T1	-.094	.059	.107
External eating T1	.138	.082	.090
Restrained eating T1	-.102	.053	.055

**Table 3.** Correlation coefficients and standard errors among exogenous variables.

		(1)	(2)	(3)	(4)	(5)
(1) Emotional eating T1		1				
(2) External eating T1	Coefficient	.485**	1			
	Standard error	.067				
(3) Restrained eating T1	Coefficient	.315**	.153	1		
	Standard error	.082	.084			
(4) BMI T1	Coefficient	.052	-.008	.152*	1	
	Standard error	.093	.094	.073		
(5) Binge eating T1	Coefficient	.587**	.433**	.472**	.245*	1
	Standard error	.059	.075	.062	.087	
(6) Fat intake T1	Coefficient	.126	.370**	-.190*	-.013	-.007
	Standard error	.095	.074	.078	.089	.092

\*\*  $p < .001$ ; \*  $p < .05$

**Table 4.** Correlation coefficients and standard errors among dependent variables.

		(1)	(2)	(3)	(4)	(5)
(7) Emotional eating T2		1				
(8) External eating T2	Coefficient	.529**	1			
	Standard error	.067				
(9) Restrained eating T2	Coefficient	.336**	.271*	1		
	Standard error	.081	.085			
(10) BMI T2	Coefficient	-.008	-.031	.109	1	
	Standard error	.071	.075	.073		
(11) Binge eating T2	Coefficient	.553**	.513**	.344**	.015	1
	Standard error	.062	.066	.079	.080	
(12) Fat intake T2	Coefficient	.134	.505**	-.104	.002	.224*
	Standard error	.092	.082	.068	.065	.080

\*\*  $p < .001$ ; \*  $p < .05$



Annex 6: Study 6 article.  
Eating styles moderate the  
relationship between stress  
and binge eating: A study  
conducted during COVID-19  
lockdown



# Eating styles moderate the relationship between stress and binge eating: A study conducted during COVID-19 lockdown

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**Abstract:** Stress plays an important role in binge eating (BE). However, the moderating role of other variables as eating styles (emotional, external, and restrictive eating) has not been sufficiently explored. COVID-19 lockdown was a worldwide emergency, experienced as stressful by a wide number of people. The purpose of this study was to examine the moderating role of eating styles on the relationship between perceived stress and BE during the lockdown. The sample consisted of 114 college students (73.5% female;  $M_{age} = 20.5$  years). All participants completed their socio-demographic profile and answered the Dutch Eating Behavior Questionnaire (DEBQ) to measure eating styles. In addition, they participated in a 7-day Ecological Momentary Assessment (EMA) using a mobile app, which measured perceived stress via the Perceived Stress Scale-4 (PSS-4) and the number of binge eating's episodes (BE) each day. Data collection took place between April 22 and 30, 2020, when the lockdown was more restrictive. All three eating styles moderated the relationship between perceived stress and BE during the lockdown. These eating patterns constitute an important risk factor for binge eating in stressful situations, such as the COVID-19 pandemic. The clinical implications of the study are discussed.

**Keywords:** stress; eating styles; binge eating; COVID-19 lockdown; Ecological Momentary Assessment.

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## 1. Introduction

Binge Eating (BE) is defined as the objective consumption of a large amount of food in a short time, accompanied by the feeling of lack of control, and usually goes with psychological distress, such as guilt or shame [1]. There is a high lifetime prevalence for Binge Eating Disorder (1,6%-2,8%) [2-4], concretely it is observed a 1.12% lifetime prevalence in Spain [5]. The frequency of this behavior has shown an increment in recent decades [2,6,7], with a higher prevalence in early adulthood [8]. Additionally, BE episodes have a wide variety of consequences, such as increased morbid obesity, diabetes, hypertension, and mood and anxiety disorders [2,3]. Given the rising prevalence of the associated consequences of BE, it is important to know which factors can help predict BE and prevent its apparition.

Several factors have been associated with BE, such as negative feelings or depression, availability of a large amount of food on hand, engaging in a restraint diet [9–12]. Various studies have pointed to a close relationship between stress and BE. For example, individuals with BE are likely to have a lower stress tolerance and rate everyday events as more stressful [9]. Interpersonal stress and a loneliness feeling induced experimentally has also been associated with BE [13].

Since many of the population have experienced the restriction periods imposed by the COVID-19 pandemic as a stressful period, it is interesting to explore whether perceived stress during lockdown was related to a higher frequency of BE. In this line, it has been suggested that the uncertainty generated by the pandemic could increase stress levels, which could trigger episodes of BE as a coping strategy to deal with psychological distress [14]. In fact, some studies have observed that people used food to calm stress during lockdown [15,16].

Still, not everyone who experiences stress shows BE behaviors or an equal level of vulnerability for this behavior. Different psychological, environmental and genetic factors (e.g., impulsiveness, alterations in the dopaminergic system features or bullying) have been proposed to be associated with BE [17]. For instance, coping styles have been proposed to mediate the relationship between stress and BE [18]. Furthermore, people who engage in BE tend to have maladaptive eating styles compared to those who do not engage in such behavior [11]. This variable has been used in previous research as a moderator in order to predict overeating and BE [19,20]. Therefore, perceived stress could be not the only factor that makes individuals binge eat more, but problematic eating styles may increase the feeling of loss of control and increased BE.

In the context of these findings, we propose that eating styles may play an important role in the relationship between stress and BE, as a moderating factor. Three dysfunctional eating styles (emotional, external, and restrictive eating) were widely studied [21,22]. These styles involve different dysfunctional eating behaviors, that for one or another reason neglect the feelings of hunger and satiety. Instead, the consumption of food is in response to emotional stimuli, such as depression or anxiety (emotional eating), in response to the availability of delicious food (external eating), or as consequence of the adoption of food restriction behaviors to reduce or maintain weight (restrictive eating) [23]. Evidence has shown that these three maladaptive eating styles may increase the likelihood of BE [24]. For instance, nutritional deprivation due to caloric restriction and cognitive variables (e.g., all-or-nothing thinking) carried out in a restrictive style can precipitate an episode of binge eating [12], especially in a stressful moment. Moreover, people with emotional eating may perform BE to mitigate negative emotions [25]. However, this relationship has not been analyzed during periods of high stress. That is, can eating styles impact the relationship between perceived stress and BE?

Therefore, this study aimed to analyze the moderating role of maladaptive eating styles (emotional eating, external eating, and restrictive eating) in the relationship between perceived stress and BE during COVID-19 lockdown. It is hypothesized that high scores on the three dysfunctional eating styles (emotional eating, external eating, and restrictive eating) will moderate the relationship between perceived stress and BE episodes during confinement.

## 2. Materials and Methods

### 2.1 Participants

The sample consisted of 114 college students from the University of Valencia (Spain). The participants were 73.5% female ( $n = 83$ ) and 26.5% ( $n = 30$ ) male ( $M_{\text{age}} = 20.5$ ;  $SD = 5.24$ ). The 54.4% ( $n = 62$ ) were single and the 44.8% had a partner. 19.3% of the participants worked or teleworked during lockdown, and 71.1% were students. Finally, the mean on Body Mass Index (BMI) was 22.63 ( $SD = 3.32$ ) (For more details, see Table 1).

**Table 1***Participants' characteristics*

	% (n) or M (SD)
<b>Sex</b>	
Men	26.5% (n = 30)
Women	73.5% (n = 83)
<b>Age</b> (range = 20 to 53 years old)	24.5 (5.24)
<b>BMI</b> (Body Mass Index)	22.63 (3.32)
<b>Marital status</b>	
Single	54.4% (n = 62)
In a relationship (not married)	43.9% (n = 50)
In a relationship (married)	0.9% (n = 1)
Divorced	0.9% (n = 1)
<b>Occupational status before the lockdown</b>	
Worker	26.3% (n = 30)
Unemployed	3.5% (n = 4)
Student	68.4% (n = 78)
Other options	1.8% (n = 2)
<b>Occupational status during the lockdown</b>	
Working/Teleworking	19.3% (n = 22)
Studying	71.1% (n = 81)
I have been out of work during the coronavirus crisis.	5.3% (n = 6)
Another option	4.4% (n = 5)

**Note.** BMI = Body Mass Index; M = Mean; SD = Standard Deviation; n = Number of participants.

## 2.2 Measures

### *Socio-demographic data.*

Participants provided information regarding sex, age, weight, height, marital status, and occupational status before and during lockdown.

### *Body Mass Index (BMI).*

BMI was calculated using the formula stated by the World Health Organization meaning, dividing weight by the square of height [26].

### *Eating Styles (Dutch Eating Behavior Questionnaire; DEBQ).*

Eating styles were evaluated with the Dutch Eating Behavior Questionnaire [23,27]. DEBQ is a self-report instrument composed of 33 items that assess three eating styles: emotional eating (eating in response to negative emotions, such as sadness or anxiety), external eating (eating after seeing appetizing foods), and restricted eating (restricting food to maintain desirable weight). The scale is responses in Likert format, ranging from 1 (Never) to 5 (Very often). In this study, high reliability scores were obtained for emotional eating ( $\alpha = .95$ ), external eating ( $\alpha = .87$ ) and restrictive eating ( $\alpha = .90$ ).

### *Ecological Momentary Assessment (EMA)*

The present study has assessed stress and BE using Ecological Momentary Assessment (EMA). This methodology avoids recall bias and improves ecological validity, as it is answered when participants are in their natural environment. In addition, it allows monitoring changes through repeated assessments of behaviors or experiences in real-time [28].

### *Stress (Perceived Stress Scale-4; PSS-4).*

Stress was assessed with the Spanish version of PSS-4 scale [29]. The PSS-4 is a self-report scale composed of four items (e.g.: How often have you been able to control difficulties in your life?) that assess the perceived stress at a specific time, generally, over a week. It uses a five-point Likert-type scale ranging from 1 (Never) to 5 (Very often). For the study, this questionnaire was assessed at the end of each day. Moderate to high reliability was obtained for the scale in this sample ( $\alpha$  between .72 and .89).

### *Binge eating (BE).*

Based on previous research [30,31], BE was assessed through two questions after each meal (breakfast, lunch, snack, and dinner):

First, a question regarding the presence of an overeating behavior was asked: "Have you overeaten (eaten more than would be reasonable for a short period)?" If they answered negatively, no further questions were asked, but if they answered affirmatively, they were asked to report how excessive they considered the episode, using a scale ranging from 1 = "not at all" to 5 = "extremely".

Second, participants were asked regarding the loss of control: "During this episode of overeating, did you feel like you were losing control or couldn't stop eating?". If they answered no, no further questions were asked, but if they answered yes, they were asked again in Likert format (1 = "not at all" and 5 = "extremely") about how intense the loss of control was.

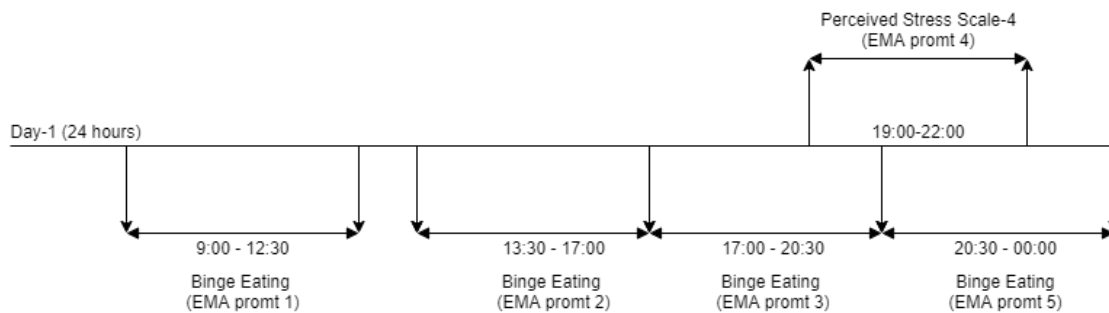
To determine whether an eating episode constituted a BE episode or not, the criteria stated by Mason et al. [30] and Smith et al. [31] was used. According to the authors, a BE episode is considered when a mean score of 3 or more is obtained on the two questions.

## **2.3 Procedure**

Participants were recruited from the University of Valencia (Spain) through social networks and instant messaging services. Being over 18 years and having an Android smartphone were the inclusion criteria for the study. Responding participants signed the informed consent prior answer the online survey. After, they complete the baseline questionnaires collecting socio-demographic and eating styles.

The survey was created using the Lime Survey platform. The data was collected between April 22 and 30, 2020. After completing the baseline questionnaire, participants were asked to download a mobile app on Google Play called "Foodbook" (<https://play.google.com/store/apps/details?id=labpsitec.registrocomida&hl=es&gl=US>). Through the APP, participants also received four daily notifications for one week assessing the food consumed (breakfast, lunch, snack, and dinner) and BE after each meal. The application sent a push notification randomly between 19:00 and 22:00 every day for a week to assess perceived stress. After finishing the recollection, means were calculated. We only consider participants who answered for at least four days. Responding to each notification took around 2 to 5 minutes (for details, see Figure 1).

This study was conducted following the Declaration of the Helsinki Principles and was approved by the Ethics Committee of the University of Valencia (procedure number: 1821046). Finally, two evaluators analyzed the self-reports following the ethical data collection standards and performed the statistical analyses.

**Figure 1.** Timeline of the Ecological Momentary Assessment with the information evaluated in one day.

**Note.** A total of five push notifications (5 prompts per day) were sent daily. Participation was for seven calendar days from the time people downloaded the application.

## 2.4 Statistical analysis

The analyses were carried out using the SPSS 25.0 statistical package. First, the descriptive data and frequencies of the participants were obtained about the demographic information and the questionnaires evaluated. Next, correlations were carried out between the subscales of the DEBQ (emotional eating, external eating, and restrictive eating), mean weekly stress (PSS-4), and the total of BE throughout the week. Finally, and based on the results found in the correlations, the PROCESS 4.0 tool was used to examine whether the different eating styles moderate the relationship between stress and BE during the lockdown. These analyses were performed using the procedure described by [32] the macro PROCESS (version 4.0), choosing model 1 with a 95% as confidence interval and 5000 simple bootstraps. For these analyses, the values of stress were transformed into low level (-1SD), moderate level (mean), and high level (+1SD), and all the variables defining the product were centered around the mean. Interactions were considered significant if  $p < 0.05$ . The independent variable was the perceived stress, and the dependent variable was the number of weekly BE episodes in all models. Three models were tested with three different moderating variables: emotional eating, external eating, and restrictive eating. The multicollinearity of the moderation models was explored using the variance inflation factor (VIF). Multicollinearity was not found since there was no coefficient greater than 4 [33].

## 3. Results

### 3.1 Scores of the eating styles, stress, and binge eating

Table 2 shows the results of the means (M) and standard deviations (SD) obtained by the participants in the DEBQ, PSS-4, and BE.

Overall, participants showed mean scores for external eating ( $M = 2.94$ ;  $SD = .71$ ), emotional eating ( $M = 1.96$ ;  $SD = .78$ ), and restrictive eating ( $M = 2.17$ ;  $SD = .76$ ). In terms of stress, participants showed mean levels of rated stress ( $M = 1.96$ ;  $SD = .63$ ). Finally, a low BE score was observed ( $M = .16$ ;  $SD = .58$ ).

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**Table 2**

*Descriptive analysis of the Dutch Eating Behavior Questionnaire, Stress, and Binge eating.*

	M (SD)
215 <b>DEBQ</b>	
216 Emotional eating (range = 1-5)	1.96 (1.96)
217 External eating (range = 1-5)	2.94 (.71)
217 Restrictive eating (range = 1-5)	2.17 (.76)
218 <b>Binge eating</b>	.16 (.58)
218 <b>Stress</b> (range = 1-5)	1.96 (.63)

219 **Note.** DEBQ = Dutch Eating Behavior Questionnaire; Binge eating = Total of binge  
 eating episodes during the week; Stress = Mean of the Perceived Stress Scale-4  
 during the week. M = Mean; SD = Standard Deviation; n = Number of participants.  
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**3.2 Bivariate correlations of the eating styles, stress, and binge eating**

Table 3 shows the relationships between the eating styles, stress, and BE, and their level of significance.

Emotional eating correlated positively with external eating ( $r = .44^{***}$ ) and restrictive eating ( $r = .33^{***}$ ). External eating correlated with restrictive eating ( $r = .26^{**}$ ). In addition, stress correlated with emotional eating ( $r = .32^{**}$ ) and external eating ( $r = .23^*$ ); however, stress did not correlate with restrictive eating ( $r = -.02$ ). BE correlated significantly with emotional eating ( $r = .44^{***}$ ) and with external eating ( $r = .26^*$ ), but not with restrictive eating ( $r = .16$ ). Finally, stress and BE are also correlated ( $r = .29^*$ ).

**Table 3**

*Correlations between the subscales of the Dutch Eating Behavior Questionnaire, Stress, and Binge eating.*

	1	2	3	4
<b>1. Emotional eating</b>	—			
<b>2. External eating</b>	.44***	—		
<b>3. Restrictive eating</b>	.33***	.26**	—	
<b>4. Stress</b>	.32**	.23*	-.02	—
<b>5. Binge eating</b>	.44***	.26*	.16	.29*

**Note.** Emotional eating = Dutch Eating Behavior Questionnaire - Emotional eating; External eating = Dutch Eating Behavior Questionnaire - External eating; Restrictive eating = Dutch Eating Behavior Questionnaire - Restrictive eating; Stress = Mean of the Perceived Stress Scale-4 during the week; Binge eating = Total of binge eating episodes during the week. \*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$

**3.3 Stress moderation in the relationship between the eating styles and binge eating**

The moderation analyses presented in Table 4 showed the influence of eating styles on the relationship between stress and BE.



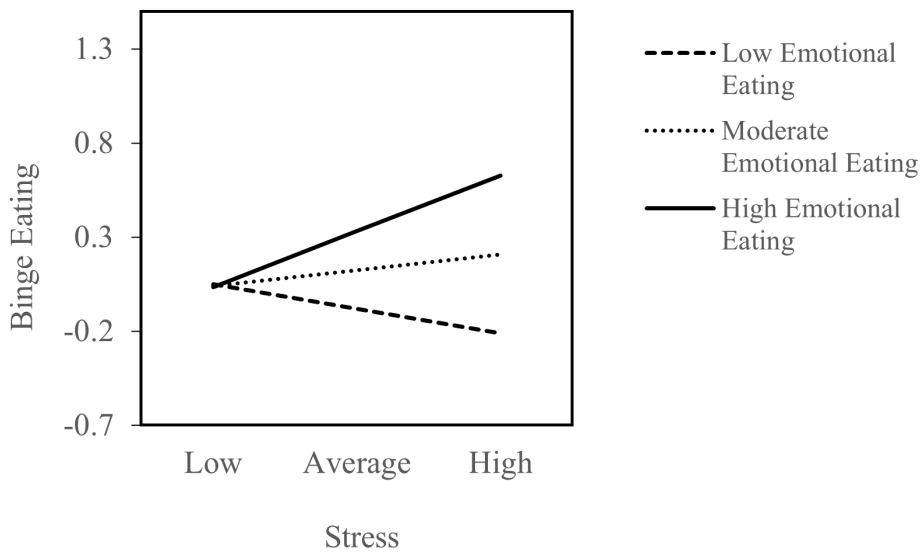
**Table 4***Relationship of stress on binge Eating moderated by eating styles*

	Effect	SE	t	p	95% Confidence Interval	
					Lower	Upper
<b>Emotional eating</b>						
Constant	.12	.07	1.92	.0594	-.01	.25
Stress	.13	.11	1.26	.2103	-.08	.34
<b>Emotional eating x Stress</b>						
Emotional eating	.02	.01	2.92	.0048	.01	.03
Emotional eating x Stress	.03	.01	3.54	.0007	.01	.05
<b>External eating</b>						
Constant	.17	.07	2.36	.0214	.03	.31
Stress	.29	.11	2.59	.0116	.07	.52
<b>External eating x Stress</b>						
External eating	.02	.01	2.26	.0272	.00	.05
External eating x Stress	.04	.02	2.46	.0165	.01	.08
<b>Restrictive eating</b>						
Constant	.18	.07	2.52	.014	.04	.33
Stress	.30	.12	2.57	.0123	.07	.53
<b>Restrictive eating x Stress</b>						
Restrictive eating	.02	.01	1.89	.0636	-.00	.04
Restrictive eating x Stress	.04	.02	2.01	.0416	.00	.08

**Note.** Emotional eating = DEBQ- Emotional eating; External eating = DEBQ External eating; Restrictive eating = DEBQ Restrictive eating; Stress = Mean of the Perceived Stress Scale-4 during the week; Binge eating = Total of binge eating episodes during the week.  $R^2$  = Coefficient of determination; SE = Standard Error;  $p = 0.05$ .

First, the emotional eating model adequately predicted BE [ $F(3,67) = 13.64$ ,  $R^2 = .38$ ;  $p < .0001$ ] (Table 4), specifically, the interaction of stress and emotional eating contributed 11.6% to explain the variance to the model [ $F(1,67) = 12.52$ ;  $R^2 = .12$ ;  $p = .0007$ ]. The results of this model indicate that at low to medium levels of emotional eating, the relationship between stress and BE is not accentuated. However, if the levels of emotional eating are high ( $t = 3.34$ ;  $p = .0014$ ), more BE episodes are observed in people who score high in stress.

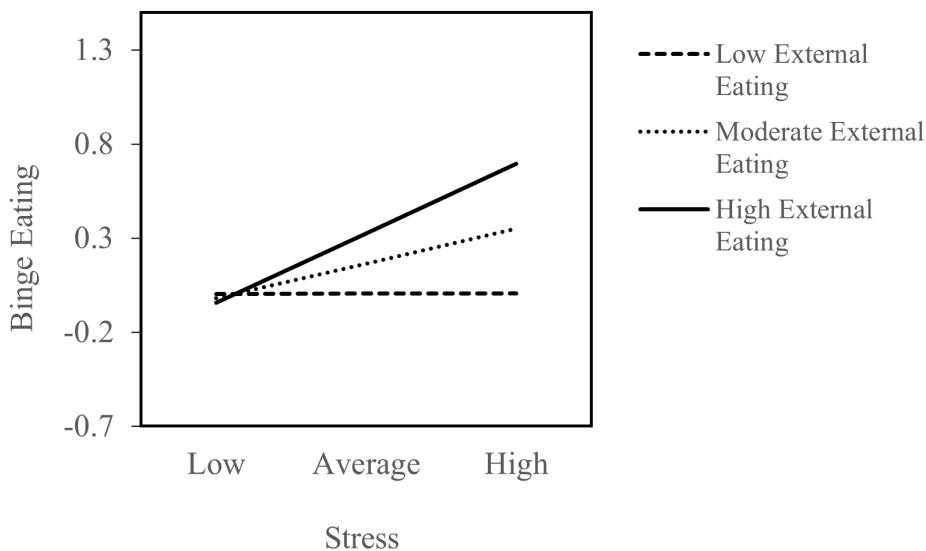
**Figure 2.** Simple effects of the interaction between emotional eating and stress on the prediction of binge eating.



**Note.** Moderating effects of emotional eating at low (Mean - 1SD); moderate (Mean) and high (Mean + 1SD) levels on the relationship between stress and BE. The independent variable is stress, the moderating variable is emotional eating, and the dependent variable is binge eating.

Secondly, the external eating model adequately predicted BE [ $F(3,67) = 6.62, R^2 = .23; p = .0006$ ] (Table 4), specifically, the interaction of stress and external eating contributed 7% to explained the variance of the model [ $F(1,67) = 6.05; R^2 = .70; p = .0165$ ]. The results of this model show that at low levels of external eating, the relationship between stress and BE is not accentuated. Nevertheless, if the levels of external eating are moderate ( $t = 2.59; p = .0116$ ) or high ( $t = 3.31; p = .0015$ ), more BE episodes are observed in people who score high on stress.

**Figure 3.** Simple effects of the interaction between external eating and stress on the prediction of binge eating



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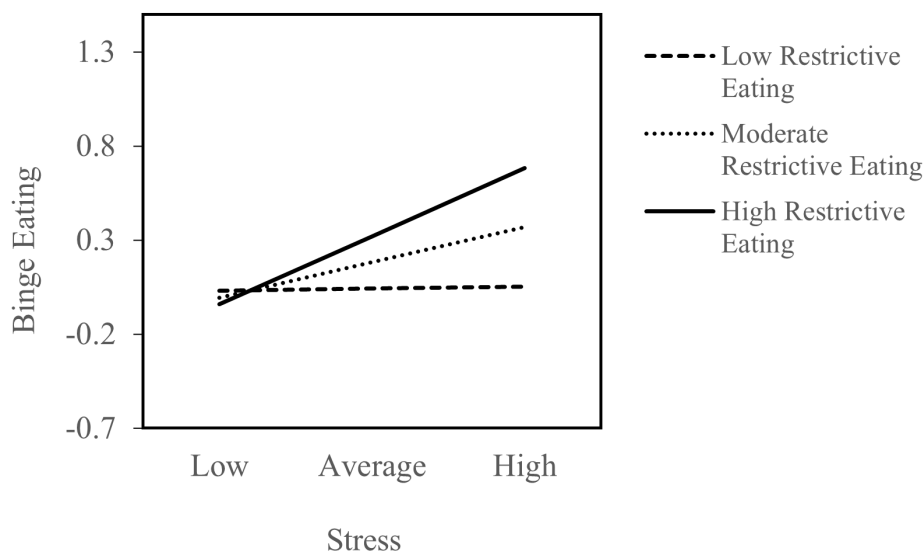
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**Note.** Moderating effects of external eating at low (Mean - 1SD); moderate (Mean) and high (Mean + 1SD) levels on the relationship between stress and BE. The independent variable is stress, the moderating variable is external eating, and the dependent variable is binge eating.

Thirdly, the restrictive eating model also predicted adequately BE [ $F(3,67) = 4.47$ ,  $R^2 = .17$ ;  $p = .0064$ ] (Table 4), specifically, the interaction of stress and restrictive eating contributed 5% to explain the variance of the model [ $F(1,67) = 4.32$ ;  $R^2 = .05$ ;  $p = .0416$ ]. The results of this model indicate that, at low levels of restrictive eating, the relationship between stress and BE is not accentuated. However, if the levels of restrictive eating are moderate ( $t = 2.57$ ;  $p = .0123$ ) or high ( $t = 3.16$ ;  $p = .0023$ ), more BE episodes are observed in people who score high on stress.

**Figure 4.** Simple effects of the interaction between restrictive eating and stress on the prediction of binge eating



**Note.** Moderating effects of restrictive eating at low (Mean - 1SD); moderate (Mean) and high (Mean + 1SD) levels on the relationship between stress and BE. The independent variable is stress, the moderating variable is restrictive eating, and the dependent variable is binge eating.

#### 4. Discussion

To the best of our knowledge, this paper is the first to examine the moderating role of eating styles in the relationship between stress and BE. In addition, this study has used EMA methodology to collect the data and avoid recall bias. Moreover, measures were taken during the 2020 spring lockdown in Spain, one of the countries most affected by COVID-19 at that time. The main finding of this study is that the three maladaptive eating styles moderate this relationship between stress and BE, as it was hypothesized.

Regarding, emotional eating, the proposed model explained 38% of the variance of the dependent variable. In this regard, our data show that individuals with high emotional eating are prone to experience more BE episodes when they feel stress, rather than people with medium or low levels of emotional eating. This finding might be understood under the light of the "escape theory" of BE [34], which postulates that BE can momentarily

relieve the negative emotions produced by stress. In addition, when emotional eaters binge, they may show a decrease in negative thoughts about themselves [35], thus, they may be able to improve their emotional state by bingeing. This finding is in line with other evidence pointing to a positive association between emotional eating, unhealthy food consumption, and stress in children and adolescents [36,37]. Also, Dos Santos Quarema et al. [38] found that emotional eating was associated with BE episodes during the lockdown. BE could be used to relieve the negative emotions present in a stressful situation [39], since emotional eaters seem to show difficulties in coping with emotions in a negative situation, resorting to food consumption to mitigate them [38].

In relation to external eating, a moderation effect was also found. The model explained 23% of the variance of the BE. Individuals with moderate and high levels of external eating (eating in response to visual or olfactory stimuli) increased food consumption in the face of this prolonged negative situation. In the specific case of the confinement situation, it must be highlighted that staying at home, with very restricted daily activities, could create a situation in which food may be more readily available. In fact, some studies have reported changes in dietary and cooking behaviors during the lockdown [40,41]. It could be speculated that external eaters in this prolonged negative situation show difficulties to distance themselves from appetitive stimuli (e.g., the refrigerator, the food, among others), thus increasing the likelihood of BE [42]. Our data is in line with previous studies that showed a positive relationship between external eating, BE, and food cravings [24,43,44], and also with stress [37,45].

Finally, results showed a significant interaction between stress and restrictive eating, predicting 17% of the variance. According to disinhibition theory, restrictive eaters have an all-or-nothing cognition about dieting. If they skip dieting, they are prone to think they have blown all progress and may end up overeating [46], possibly in the form of BE. In addition, it may be possible that these individuals, when faced with high perceived stress, decrease their self-regulation, precipitating food consumption [47]. Previous studies have found that restrictive eating is associated with increased food intake in response to emotions [25], especially negative ones [21,22,48].

In summary, the current study showed the effect eating styles have over binge eating under stressful situations. In this sense, this is a step forward toward identifying variables that can constitute a significant risk factor to BE. Considering these results, prevention and intervention strategies for BE should target the management of stress but also detect potential vulnerabilities concerning the eating style patterns and encourage the adoption of adaptive eating styles to prevent BE.

The current study presents some limitations that are necessary to highlight. First, the mobile app was only available for Android devices; this meant that some participants were excluded (e.g., iPhone users, users without storage capacity on the device or people without smartphones), possible skewing the results. Second, the sample consisted only of young adults, reducing the inferential power of the study, and making it difficult to generalize the current results to other demographic groups.

Future studies are needed to explore these results and extend them. In this regard, longitudinal studies with a more extensive and higher variability in the sample could be helpful to understand the relationship between stress and BE and how eating styles are linked to this relationship (e.g., including non-Caucasian sample or hidden populations). In addition, future studies may analyze these relationships in clinical populations (e.g., BE disorder) and examine whether these results are replicated, or the relationships have a different direction or power. Besides, although this study is pioneering in analyzing the influence of eating styles on the relationship between stress and BE, these are not the only variables that play a crucial role. In this regard, future studies to analyze the role of other variables such as self-esteem and mindfulness traits can help to better understand the relationship between stress and BE.

## 5. Conclusions

This study shows the influence of maladaptive eating styles such as emotional, external, and restrictive eating, on the relationship between perceived stress and BE, in the COVID-19 pandemic. The COVID-19 pandemic has involved prolonged exposure to a stressor by many people. Despite the tragedies experienced, research has used this particular time to observe the reactions of humans to this type of situation. Concretely, these results add evidence to those pointing out that dysfunctional eating styles can be considered important risk factors, which can trigger abnormal and maladaptive eating behaviors in stressful situations. Alternatively, more research is needed to consolidate evidence-based approaches to management dysfunctional intake styles and stress during stressful periods, in order to reduce binge eating.

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**Data Availability Statement:** The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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**Conflicts of Interest:** The authors declare no conflict of interest, financial or otherwise.

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## References

1. American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders*; American Psychiatric Association, 2013; ISBN 0-89042-555-8. 378
2. Hudson, J.I.; Hiripi, E.; Pope, H.G.; Kessler, R.C. The Prevalence and Correlates of Eating Disorders in the National Comorbidity Survey Replication. *Biol. Psychiatry* **2007**, *61*, 348–358, doi:10.1016/j.biopsych.2006.03.040. 381
3. Kessler, R.C.; Berglund, P.A.; Chiu, W.T.; Deitz, A.C.; Hudson, J.I.; Shahly, V.; Aguilar-Gaxiola, S.; Alonso, J.; Angermeyer, M.C.; Benjet, C.; et al. The Prevalence and Correlates of Binge Eating Disorder in the World Health Organization World Mental Health Surveys. *Biol. Psychiatry* **2013**, *73*, 904–914, doi:10.1016/J.BIOPSYCH.2012.11.020. 383
4. Swanson, S.A.; Crow, S.J.; Le Grange, D.; Swendsen, J.; Merikangas, K.R. Prevalence and Correlates of Eating Disorders in Adolescents: Results From the National Comorbidity Survey Replication Adolescent Supplement. *Arch. Gen. Psychiatry* **2011**, *68*, 714–723, doi:10.1001/archgenpsychiatry.2011.22. 384
5. Preti, A.; De Girolamo, G.; Vilagut, G.; Alonso, J.; De Graaf, R.; Bruffaerts, R.; Demyttenaere, K.; Pinto-Meza, A.; Haro, J.M.; Morosini, P.; et al. The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *J. Psychiatr. Res.* **2009**, *43*, 1125–1132, doi:10.1016/J.JPSYCHIRES.2009.04.003. 385
6. Mitchison, D.; Touyz, S.; González-Chica, D.A.; Stocks, N.; Hay, P. How abnormal is binge eating? 18-Year time trends in population prevalence and burden. *Acta Psychiatr. Scand.* **2017**, *136*, 147–155, doi:10.1111/ACPS.12735. 386
7. Smink, F.R.E.; Van Hoeken, D.; Oldehinkel, A.J.; Hoek, H.W. Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *Int. J. Eat. Disord.* **2014**, *47*, 610–619, doi:10.1002/EAT.22316. 387
8. Hanson, J.A.; Phillips, L.N.; Hughes, S.M.; Corson, K. Attention-deficit hyperactivity disorder symptomatology, binge eating disorder symptomatology, and body mass index among college students. *J. Am. Coll. Heal.* **2019**, *68*, 543–549, doi:10.1080/07448481.2019.1583651. 388
9. Crowther, J.H.; Sanftner, J.; Bonifazi, D.Z.; Shepherd, K.L. The role of daily hassles in binge eating. *Int. J. Eat. Disord.* **2001**, *29*, 449–454, doi:10.1002/EAT.1041. 389
10. Dingemans, A.E.; Spinhoven, P.; van Furth, E.F. Predictors and mediators of treatment outcome in patients with binge eating disorder. *Behav. Res. Ther.* **2007**, *45*, 2551–2562, doi:10.1016/J.BRAT.2007.06.003. 390
11. Kim, Y.R.; Hwang, B.I.; Lee, G.Y.; Kim, K.H.; Kim, M.; Kim, K.K.; Treasure, J. Determinants of binge eating disorder among normal weight and overweight female college students in Korea. *Eat. Weight Disord. - Stud. Anorexia, Bulim. Obes.* **2018**, *23*, 849–860, doi:https://doi.org/10.1007/s40519-018-0574-2. 391
12. Zunker, C.; Peterson, C.B.; Crosby, R.D.; Cao, L.; Engel, S.G.; Mitchell, J.E.; Wonderlich, S.A. Ecological momentary assessment of bulimia nervosa: Does dietary restriction predict binge eating? *Behav. Res. Ther.* **2011**, *49*, 714–717, doi:10.1016/J.BRAT.2011.06.006. 392
13. Tuschen-Caffier, B.; Vögele, C. Psychological and Physiological Reactivity to Stress: An Experimental Study on Bulimic Patients, Restrained Eaters and Controls. *Psychother. Psychosom.* **1999**, *68*, 333–340, doi:10.1159/000012352. 393
14. Weissman, R.S.; Bauer, S.; Thomas, J.J. Access to evidence-based care for eating disorders during the COVID-19 crisis. *Int. J. Eat. Disord.* **2020**, *53*, 639–646, doi:10.1002/EAT.23279. 394
15. Aldhuwayhi, S.; Shaikh, S.A.; Mallineni, S.K.; Kumari, V.V.; Thakare, A.A.; Khan, A.R.A.; Mustafa, M.Z.; Manva, M.Z. Occupational Stress and Stress Busters Used Among Saudi Dental Practitioners During the COVID-19 Pandemic Outbreak. *Disaster Med. Public Health Prep.* **2021**, 1–7, doi:10.1017/DMP.2021.215. 395
16. Buckland, N.J.; Swinnerton, L.F.; Ng, K.; Price, M.; Wilkinson, L.L.; Myers, A.; Dalton, M. Susceptibility to increased high energy dense sweet and savoury food intake in response to the COVID-19 lockdown: The role of craving control and acceptance coping strategies. *Appetite* **2021**, *158*, 105017, doi:10.1016/J.APPET.2020.105017. 396
17. Agüera, Z.; Núria, M.L.; José, M.S.J.; Fernández-aranda, F. A review of binge eating disorder and obesity. *Neuropsychiatric* 397

- 2021, 35, 57–67, doi:10.1007/s40211-020-00346-w. 420
18. Sulkowski, M.L.; Dempsey, J.; Dempsey, A.G. Effects of stress and coping on binge eating in female college students. *Eat. Behav.* **2011**, *12*, 188–191, doi:10.1016/J.EATBEH.2011.04.006. 421
19. Verstuyf, J.; Vansteenkiste, M.; Soenens, B.; Boone, L.; Mouratidis, A. Daily Ups and Downs in Women’s Binge Eating Symptoms: The Role of Basic Psychological Needs, General Self-Control, and Emotional Eating. *J. Soc. Clin. Psychol.* **2013**, *32*, 335–361, doi:10.1521/JSCP.2013.32.3.335. 422
20. Van Strien, T.; Herman, C.P.; Anschutz, D.J.; Engels, R.C.M.E.; de Weerth, C. Moderation of distress-induced eating by emotional eating scores. *Appetite* **2012**, *58*, 277–284, doi:10.1016/J.APPET.2011.10.005. 423
21. Hill, D.; Conner, M.; Clancy, F.; Moss, R.; Wilding, S.; Bristow, M.; O’Connor, D.B. Stress and eating behaviours in healthy adults: a systematic review and meta-analysis. *Health Psychol. Rev.* **2021**, doi:10.1080/17437199.2021.1923406. 424
22. Cardi, V.; Leppanen, J.; Treasure, J. The effects of negative and positive mood induction on eating behaviour: A meta-analysis of laboratory studies in the healthy population and eating and weight disorders. *Neurosci. Biobehav. Rev.* **2015**, *57*, 299–309, doi:10.1016/J.NEUBIOREV.2015.08.011. 425
23. van Strien, T.; Frijters, J.E.; Bergers, G.P.; Defares, P.B. The Dutch Eating Behavior Questionnaire (DEBQ) for Assessment of Restrained, Emotional, and External Eating Behavior. *Eat. Disord.* **1986**, *5*, 295–326. 426
24. Černelič-Bizjak, M.; Guiné, R.P.F. Predictors of binge eating: relevance of BMI, emotional eating and sensitivity to environmental food cues. *Nutr. Food Sci.*, doi:10.1108/NFS-02-2021-0062. 427
25. Macht, M. How emotions affect eating: A five-way model. *Appetite* **2008**, *50*, 1–11, doi:10.1016/J.APPET.2007.07.002. 428
26. *Obesity: preventing and managing the global epidemic. Report of a WHO consultation.*; Switzerland, 2000; Vol. 894;. 429
27. Cebolla, A.; Barrada, J.R.; van Strien, T.; Oliver, E.; Baños, R. Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite* **2014**, *73*, 58–64, doi:10.1016/J.APPET.2013.10.014. 430
28. Shiffman, S.; Stone, A.A.; Hufford, M.R. Ecological Momentary Assessment. *Annu. Rev. Clin. Psychol.* **2008**, *4*, 1–32, doi:10.1146/annurev.clinpsy.3.022806.091415. 431
29. Herrero, J.; Meneses, J. Short Web-based versions of the perceived stress (PSS) and Center for Epidemiological Studies-Depression (CESD) Scales: a comparison to pencil and paper responses among Internet users. *Comput. Human Behav.* **2006**, *22*, 830–846, doi:10.1016/J.CHB.2004.03.007. 432
30. Mason, T.B.; Do, B.; Chu, D.; Belcher, B.R.; Dunton, G.F.; Lopez, N. V. Associations among affect, diet, and activity and binge-eating severity using ecological momentary assessment in a non-clinical sample of middle-aged fathers. *Eat. Weight Disord. - Stud. Anorexia, Bulim. Obes.* **2021**, *1*, 1–9, doi:https://doi.org/10.1007/s40519-021-01191-8. 433
31. Smith, K.E.; Mason, T.B.; Schaefer, L.M.; Juarascio, A.; Dvorak, R.; Weinbach, N.; Crosby, R.D.; Wonderlich, S.A. Examining intra-individual variability in food-related inhibitory control and negative affect as predictors of binge eating using ecological momentary assessment. *J. Psychiatr. Res.* **2020**, *120*, 137–143, doi:https://doi.org/10.1016/j.jpsychires.2019.10.017. 434
32. Hayes, A.F. Partial, conditional, and moderated moderated mediation: Quantification, inference, and interpretation. *Commun. Monogr.* **2017**, *85*, 4–40, doi:10.1080/03637751.2017.1352100. 435
33. O’Brien, R.M. A Caution Regarding Rules of Thumb for Variance Inflation Factors. *Qual. Quant.* **2007**, *41*, 673–690, doi:10.1007/S11135-006-9018-6. 436
34. Paxton, S.J.; Diggins, J. Avoidance Coping, Binge Eating, and Depression: An Examination of the Escape Theory of Binge Eating. *Int. J. Eat. Disord.* **1997**, *22*, 83–87, doi:10.1002/(SICI)1098-108X(199707)22:1. 437
35. Heatherton, T.F.; Baumeister, R.F. Binge eating as escape from self-awareness. *Psychol. Bull.* **1991**, *110*, 86–108, doi:10.1037/0033-2909.110.1.86. 438
36. Michels, N.; Sioen, I.; Braet, C.; Eiben, G.; Hebestreit, A.; Huybrechts, I.; Vanaelst, B.; Vyncke, K.; De Henauw, S. Stress, emotional eating behaviour and dietary patterns in children. *Appetite* **2012**, *59*, 762–769, doi:10.1016/J.APPET.2012.08.010. 439

37. Hou, F.; Xu, S.; Zhao, Y.; Lu, Q.; Zhang, S.; Zu, P.; Sun, Y.; Su, P.; Tao, F. Effects of emotional symptoms and life stress on eating behaviors among adolescents. *Appetite* **2013**, *68*, 63–68, doi:10.1016/J.APPET.2013.04.010. 462  
463
38. dos Santos Quaresma, M.V.; Marques, C.G.; Magalhães, A.C.O.; dos Santos, R.V.T. Emotional eating, binge eating, physical inactivity, and vespertine chronotype are negative predictors of dietary practices during COVID-19 social isolation: A cross-sectional study. *Nutrition* **2021**, *90*, 111223, doi:10.1016/J.NUT.2021.111223. 464  
465  
466
39. Ricca, V.; Castellini, G.; Lo Sauro, C.; Ravaldi, C.; Lapi, F.; Mannucci, E.; Rotella, C.M.; Faravelli, C. Correlations between binge eating and emotional eating in a sample of overweight subjects. *Appetite* **2009**, *53*, 418–421, doi:10.1016/j.appet.2009.07.008. 467  
468  
469
40. Rodríguez-Pérez, C.; Molina-Montes, E.; Verardo, V.; Artacho, R.; García-Villanova, B.; Guerra-Hernández, E.J.; Ruiz-López, M.D. Changes in Dietary Behaviours during the COVID-19 Outbreak Confinement in the Spanish COVIDiet Study. *Nutrients* **2020**, *12*, 1730, doi:10.3390/nu12061730. 470  
471  
472
41. Ruiz-Roso, M.B.; Padilha, P. de C.; Mantilla-Escalante, D.C.; Ulloa, N.; Brun, P.; Acevedo-Correa, D.; Peres, W.A.F.; Martorell, M.; Aires, M.T.; Cardoso, L. de O.; et al. Covid-19 Confinement and Changes of Adolescent's Dietary Trends in Italy, Spain, Chile, Colombia and Brazil. *Nutrients* **2020**, *12*, 1807, doi:10.3390/nu12061807. 473  
474  
475
42. Touyz, S.; Lacey, H.; Hay, P. Eating disorders in the time of COVID-19. *J. Eat. Disord.* **2020**, *8*, 1–3, doi:10.1186/s40337-020-00295-3. 476  
477
43. Burton, P.; J. Smit, H.; J. Lightowler, H. The influence of restrained and external eating patterns on overeating. *Appetite* **2007**, *49*, 191–197, doi:10.1016/J.APPET.2007.01.007. 478  
479
44. Mason, T.B.; Lewis, R.J. Profiles of Binge Eating: The Interaction of Depressive Symptoms, Eating Styles, and Body Mass Index. *Eat. Disord.* **2014**, *22*, 450–460, doi:10.1080/10640266.2014.931766. 480  
481
45. Kalkan Uğurlu Y.; Mataracı Değirmenci D.; Durgun H.; Gök Uğur H. The examination of the relationship between nursing students' depression, anxiety and stress levels and restrictive, emotional, and external eating behaviors in COVID-19 social isolation process. *Perspect. Psychiatr. Care* **2021**, *57*, 507–516, doi:10.1111/PPC.12703. 482  
483  
484
46. Ruderman, A.J. Dietary Restraint. A Theoretical and Empirical Review. *Psychol. Bull.* **1986**, *99*, 247–262, doi:10.1037/0033-2909.99.2.247. 485  
486
47. Baumeister, R.F.; Heatherton, T.F.; Tice, D.M. When Ego Threats Lead to Self-Regulation Failure: Negative Consequences of High Self-Esteem. *J. Pers. Soc. Psychol.* **1993**, *64*, 141–156. 487  
488
48. Evers, C.; Dingemans, A.; Junghans, A.F.; Boevé, A. Feeling bad or feeling good, does emotion affect your consumption of food? A meta-analysis of the experimental evidence. *Neurosci. Biobehav. Rev.* **2018**, *92*, 195–208, doi:10.1016/J.NEUBIOREV.2018.05.028. 489  
490  
491  
492



Annex 7: First certificate  
from the Comitè Ètic  
d'Investigació en Humans  
de la Comissió d'Ètica en  
Investigació Experimental  
of the Universitat de  
València.



**D. José María Montiel Company**, Profesor Contratado Doctor Interino del departamento de Estomatología, y Secretario del Comité Ético de Investigación en Humanos de la Comisión de Ética en Investigación Experimental de la Universitat de València,

CERTIFICA:

Que el Comité Ético de Investigación en Humanos, en la reunión celebrada el día 1 de marzo de 2018, una vez estudiado el proyecto de tesis doctoral titulado:  
*“Relación entre los patrones de ingesta y dieta y la vulnerabilidad al consumo de alcohol en adultos jóvenes”*, número de procedimiento H1513854038939, cuya responsable es Dña. Tamara Escrivá Martínez, dirigida por Dña. Rosa María Baños Rivera, ha acordado informar favorablemente el mismo dado que se respetan los principios fundamentales establecidos en la Declaración de Helsinki, en el Convenio del Consejo de Europa relativo a los derechos humanos y cumple los requisitos establecidos en la legislación española en el ámbito de la investigación biomédica, la protección de datos de carácter personal y la bioética.

Y para que conste, se firma el presente certificado en Valencia, a dos de marzo de dos mil dieciocho.





Annex 8: Second  
certificate from the Comitè  
Ètic d'Investigació en  
Humans de la Comissió  
d'Ètica en Investigació  
Experimental of the  
Universitat de València



El comité Ético de Investigación en Humanos de la Comisión de Ética en Investigación Experimental de la Universitat de València,

CERTIFICA:

Que el Comité d'Ètica d'Investigació en Humans , en la reunió celebrada el día 02 de Diciembre de 2021 , una vez estudiado el proyecto de tesis doctoral : "*Relación entre consumo de dieta grasa y consumo de alcohol en jóvenes: un estudio a través de Evaluación Ecológica Momentánea* ", con número de registro1821046 .

Cuyo/a responsable es D/Dña.

TAMARA ESCRIVA MARTINEZ , dirigida por D/Dña. ROSA MARIA

BAÑOS RIVERA

ha acordado informar favorablemente el mismo.

Y para que conste, se firma el presente certificado





Annex 9: Spanish version of  
the Binge Eating Scale.



## BES

Instrucciones: A continuación, se encuentran 16 grupos de tres o cuatro oraciones. Lee con cuidado cada una de las oraciones de cada grupo y marca la que mejor describe cómo te sientes con respecto a los problemas que has tenido para controlar tu forma de comer.

### GRUPO 1

1. No me siento preocupado(a) de mi peso o mi talla cuando estoy con otros.
2. Me siento preocupado(a) de cómo luzco para los demás, pero normalmente esto no me hace sentirme decepcionado(a) de mí mismo(a).
3. Me siento preocupado(a) acerca de mi apariencia y peso y esto me hace sentir decepcionado(a) de mí mismo(a).
4. Me siento muy preocupado acerca de mi peso y frecuentemente siento una pena intensa y disgusto por mí mismo(a). Trato de evitar contactos sociales debido a mi preocupación por mi apariencia.

### GRUPO 2

1. No tengo ninguna dificultad para comer lentamente y de manera adecuada.
2. Aunque parece que «devoro» la comida no termino sintiéndome «lleno» por haber comido mucho.
3. Algunas veces como muy rápido y después me siento incómodamente lleno.
4. Siempre o casi siempre me trago la comida sin masticala y cuando esto pasa me siento incómodamente «lleno(a)» porque he comido demasiado.

### GRUPO 3

1. Me siento capaz de controlar mis ganas de comer cuando yo quiero.
2. Siento que he fallado en controlar mi alimentación más que una persona promedio.
3. Me siento incapaz de controlar mis ganas de comer.
4. Me siento desesperado porque no soy capaz de controlar mi manera de comer.

### GRUPO 4

1. No acostumbro comer cuando estoy aburrido(a).
2. ALGUNAS VECES me pongo a comer cuando estoy aburrido(a), pero SIEMPRE O CASI SIEMPRE soy capaz de mantenerme ocupado(a) y dejar de pensar en la comida.

3. SIEMPRE O CASI SIEMPRE me pongo a comer cuando estoy aburrido(a), pero ALGUNAS VECES puedo hacer otra actividad para dejar de pensar en la comida.
4. SIEMPRE O CASI SIEMPRE me pongo a comer cuando estoy aburrido(a) y nada parece ayudar me a romper este hábito.

#### GRUPO 5

1. GENERALMENTE como cuando me siento físicamente con hambre.
2. ALGUNAS VECES como algo impulsivamente a pesar de que no tengo hambre.
3. MUCHAS VECES como cosas que realmente no disfruto para satisfacer mi sensación de hambre a pesar saber que físicamente no necesito comer en ese momento.
4. A pesar de que físicamente no tengo hambre, tengo una sensación de hambre en mi boca que sólo es satisfecha comiendo cosas que me llenan la boca, como un sándwich. Cuando hago esto, algunas veces escupo la comida para no engordar.

#### GRUPO 6

1. No siento culpa ni me odio después de comer de más.
2. A VECES siento culpa o me odio después de comer de más.
3. SIEMPRE O CASI SIEMPRE siento culpa o me odio después de comer de más.

#### GRUPO 7

1. Cuando hago dieta y la rompo porque como de más, puedo volver a controlar mi forma de comer.
2. Cuando hago dieta y la rompo comiendo algo «prohibido», ALGUNAS VECES siento que «me equivoqué» y como aún más.
3. Cuando hago dieta y la rompo porque como de más, MUCHAS VECES siento que «cometí un error» y como aún más.
4. SIEMPRE O CASI SIEMPRE hago dieta y la rompo porque tengo un atracón. Parece que mi vida transcurriera entre «atracones» y «tener hambre».

#### GRUPO 8

1. MUY POCAS VECES O NUNCA como tanto como para sentirme incómodo.
2. ALGUNAS VECES, aproximadamente 1 vez al mes, como tanto que al final termino sintiéndome muy «lleno».

3. MUCHAS VECES durante el mes tengo periodos donde como grandes cantidades de comida, ya sea a la hora de la comida o entre comidas.
4. SIEMPRE O CASI SIEMPRE como tanta comida que me siento incomodo(a) al acabar de comer, y a veces hasta con un poco de náuseas.

#### GRUPO 9

1. Mi ingesta de calorías no es muy baja ni muy alta.
2. ALGUNAS VECES después de comer demasiado, trato de disminuir mi ingesta de calorías casi a cero para compensar el exceso que había comido.
3. SIEMPRE O CASI SIEMPRE como demasiado durante la noche. Generalmente no tengo hambre durante el día pero como demasiado en la noche.
4. Durante mi vida adulta hay semanas en que tengo largos periodos donde prácticamente me mato de hambre y luego como de más. Parece ser que mi vida transcurre entre «atracones» y «hambre».

#### GRUPO 10

1. Normalmente soy capaz de parar de comer cuando yo quiero. Sé cuándo: «suficiente es suficiente».
2. ALGUNAS VECES me da compulsión de comer y parece que no puedo controlar mi manera de comer.
3. MUCHAS VECES me da una urgencia por comer y pareciera que no la puedo controlar, pero otras veces si la puedo controlar.
4. SIEMPRE O CASI SIEMPRE me siento incapaz de controlar mi urgencia por comer. Tengo miedo de no poder parar de comer cuando yo quiera.

#### GRUPO 11

1. No tengo ningún problema para parar de comer cuando me siento «lleno».
2. Puedo parar de comer cuando me siento lleno, pero A VECES como demasiado y me siento «muy lleno».
3. MUCHAS VECES tengo el problema para parar de comer y me siento incómodamente «lleno».
4. SIEMPRE O CASI SIEMPRE soy incapaz de parar de comer cuando yo quiero y algunas veces ha sido necesario inducirme el vómito, usar laxantes o diuréticos para aliviar mi sensación de estar «muy lleno».

## GRUPO 12

1. Como lo mismo cuando estoy con otros (familia, reunión social) que cuando estoy solo.
2. ALGUNAS VECES cuando estoy con otras personas no como tanto como quisiera porque me siento preocupado acerca de mi forma de comer.
3. MUCHAS VECES como pequeñas cantidades de comida cuando hay gente a mí alrededor porque me siento avergonzado de mi forma de comer.
4. SIEMPRE O CASI SIEMPRE me siento muy avergonzado por comer de más y elijo tiempos para comer de más cuando sé que nadie podría verme.

## GRUPO 13

1. Hago tres comidas al día y SÓLO ALGUNAS VECES como bocadillos entre comidas.
2. Hago tres comidas al día, pero MUCHAS VECES como bocadillos entre éstas.
3. Cuando como muchos bocadillos me salto las comidas regulares.
4. Hay periodos que parece que estuviera comiendo todo el tiempo, sin ninguna comida planeada.

## GRUPO 14

1. No pienso mucho acerca de tratar de controlar comer cosas que en realidad no deseo.
2. ALGUNAS VECES pienso acerca de tratar de controlar mi urgencia por comer.
3. MUCHAS VECES paso mucho tiempo pensando acerca de cuanto comí o acerca de tratar de no comer más.
4. SIEMPRE O CASI SIEMPRE estoy pensando en «comer o no comer». Siento que vivo para comer.

## GRUPO 15

1. No pienso que la comida sea «lo más importante».
2. Tengo antojos fuertes de comida, pero sólo por periodos cortos de tiempo.
3. HAY DÍAS que parece que no puedo pensar en otra cosa que no sea la comida.
4. LA MAYORÍA DE LOS DÍAS estoy preocupado acerca de la comida. Siento como si viviera para comer.

## GRUPO 16

1. SIEMPRE O CASI SIEMPRE puedo distinguir si estoy físicamente hambriento o no. Como lo suficiente como para satisfacerme.
2. A VECES me siento inseguro de saber si estoy físicamente hambriento o no. Cuando pasa esto me es difícil saber qué tanto debo comer para satisfacerme.
3. Aunque pienso que debería saber cuántas calorías debo comer, no tengo ni la menor idea cual es la cantidad «normal» de comida para mí.





Annex 10: Spanish version  
of the modified Yale Food  
Addiction Scale 2.0.



## mYFAS 2.0

Esta encuesta pregunta sobre tus hábitos alimentarios del año pasado. Las personas a veces tienen dificultad para controlar la cantidad que comen de ciertos alimentos, como, por ejemplo:

- Dulces como helado, chocolate, donuts, galletas, pastel, caramelos
- Almidones como pan blanco, panecillos, pasta y arroz
- Aperitivos salados como papas fritas, rosquilletas y galletas saladas
- Alimentos ricos en grasa como bistec, tocino, hamburguesas, pizza y papas fritas
- Bebidas azucaradas como gaseosas, refrescos, bebidas para deportistas y bebidas energéticas

Cuando las siguientes preguntas se refieran a "DETERMINADOS ALIMENTOS", por favor piensa en CUALQUIER alimento o bebida similar a los enumerados en los grupos de alimentos o bebidas anteriores o CUALQUIER OTRO alimento con el que hayas tenido dificultad en el último año.

EN LOS ÚLTIMOS 12 MESES:	Nunca	Menos de una vez al mes	Una a vez al mes	2-3 veces al mes	Una vez a la semana	2-3 veces a la semana	4-6 veces a la semana	A diario o más
1. He comido hasta el punto de sentirme físicamente enfermo.	0	1	2	3	4	5	6	7
2. He pasado mucho tiempo sintiéndome lento o cansado por comer en exceso.	0	1	2	3	4	5	6	7

3. He evitado el trabajo, la escuela o las actividades sociales porque tenía miedo de comer en exceso allí.	0	1	2	3	4	5	6	7
4. Si tuviera problemas emocionales por no comer ciertos alimentos, los comería para sentirme mejor.	0	1	2	3	4	5	6	7
5. Mi comportamiento alimentario me ha causado mucho malestar.	0	1	2	3	4	5	6	7
6. Tuve problemas significativos en mi vida debido a la comida y al comer. Estos pueden haber sido problemas con mi rutina diaria, trabajo, escuela, amigos, familia, o salud.	0	1	2	3	4	5	6	7
7. Mi exceso de comida me impidió cuidar de mi familia o hacer tareas domésticas.	0	1	2	3	4	5	6	7
8. Seguí comiendo de la misma manera a pesar de que mi alimentación me ha causado problemas emocionales.	0	1	2	3	4	5	6	7
9. Comer la misma cantidad de comida no	0	1	2	3	4	5	6	7

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me ha dado tanto placer  
como solía hacerlo.

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10. Tenía tantas ganas  
de comer ciertos  
alimentos que no podía  
pensar en otra cosa

0	1	2	3	4	5	6	7
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11. Lo intenté y no pude  
reducir o dejar de comer  
ciertos alimentos.

0	1	2	3	4	5	6	7
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12. Estaba tan distraído  
al comer que podría  
haberme herido (por  
ejemplo, al conducir un  
coche, al cruzar la calle  
o al operar con  
maquinaria).

0	1	2	3	4	5	6	7
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13. Mis amigos y  
familiares estaban  
preocupados por lo  
mucho que yo comía.

0	1	2	3	4	5	6	7
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