

# Comprehensive analysis of the relationship between ultra-processed food consumption and food addiction at one-year follow-up in older adults with metabolic syndrome

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## FULL-LENGTH REPORT



In the originally published version affiliation 15 was erroneously linked to María Gomis-González. The error was corrected on 27 March 2026.

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## ABSTRACT

**Background:** Ultra-processed foods (UPFs) are theorised to exhibit addictive properties within the framework of the Food Addiction (FA) model, attributable to their high palatability, sugars, salt, saturated/trans fats, and caloric density. **Aims:** To evaluate the association between UPFs consumption

and FA presence, and to examine whether reducing UPFs intake after one year of intervention is associated to decreased FA scores. *Methods:* The sample included 429 Mediterranean older adults with overweight/obesity and metabolic syndrome from the PREDIMED-Plus-Cognition sub-study. FA presence was evaluated with the Yale Food Addiction Scale 2.0, and the nutritional information through validated food frequency questionnaires. UPFs was categorised according to the NOVA system and divided into consumption tertiles. Data were analysed using repeated measures ANOVA, structural equation modelling, and logistic regression to compare FA changes across tertiles of UPF reduction after one-year follow-up. *Results:* Baseline YFAS categorisation into three levels (positive-probable-negative) showed significant differences between the second and third tertile of UPFs consumption, with the highest tertile of consumption having greater likelihood of worse FA status. After one year, the likelihood of change in the FA levels was higher only for individuals within the highest decreases in the UPFs consumption ( $OR = 1.67, p = 0.040$ ). *Discussion and Conclusions:* These findings indicate that a reduction in the consumption of UPFs may contribute to the improvement of FA symptoms, providing novel insights into the association between UPFs and the presence of FA. Future research should focus on populations with higher UPFs consumption and investigating the long-term effects of dietary quality on FA symptoms.

#### KEYWORDS

food addiction, mediterranean diet, obesity, ultra-processed foods

## INTRODUCTION

Obesity is a complex, chronic disease with a multifactorial aetiology. A wide range of aspects are involved in its determination, including genetic, behavioural, physiological, environmental, and socio-cultural factors that interplay in an intricate manner (González-Muniesa et al., 2017). In the last decades, there has been a significant increase in the prevalence of obesity on a global scale. A recently conducted pooled analysis of population-representative studies estimated that by the year 2022, 43% of adults had overweight (Phelps et al., 2024). Several environmental changes in recent decades have been identified as significant contributors to weight gain. In this context, the transition to dietary patterns associated with overconsumption of foods of low nutritional quality has been identified as one of the most significant (Lingvay, Cohen, Roux, & Sumithran, 2024). Therefore, recent research has focused on the relationship between the obesity pandemic and the increase in the intake of ultra-processed foods (UPFs) (Askari, Heshmati, Shahinfar, Tripathi, & Daneshzad, 2020; González-Palacios et al., 2023; Lane et al., 2024; Moradi et al., 2023). The consumption of such industrially produced foods, which contain high levels of refined carbohydrates and/or added fats, as well as flavour additives and texturisers that enhance palatability, has not only been associated with a higher risk of adverse health outcomes (Lane et al., 2024), but has also been proposed as strong candidates for having

an addictive potential (Gearhardt, Bueno, DiFeliceantonio, et al., 2023a).

In view of similarities between the overconsumption of highly palatable foods and addictive behaviours, and as the scientific understanding of addiction evolves, an increasing number of clinicians and scientists have suggested that some types of this excess weight may be a form of ‘food addiction’ (FA) (Davis et al., 2011; Gearhardt, Grilo, DiLeone, Brownell, & Potenza, 2011; Volkow, Wise, & Baler, 2017; Wenzel, Weinstock, & McGrath, 2020). In this regard, two separate strands of thought about the FA concept have been proposed. The first perspective focuses on the similarities between the consumption of specific food types, specially UPFs, and addictive mechanisms associated with substance abuse disorders (SUDs) (Gearhardt, Davis, Kushner, & Brownell, 2011). Evidence suggests that FA and SUDs share neurocognitive mechanisms involving hyperactivation of reward circuits (mesolimbic dopamine pathways), heightened cue reactivity, and deficits in inhibitory control, paralleling addictive processes observed in substance use (Gearhardt et al., 2023b; Volkow, Koob, & McLellan, 2016). Conversely, other authors emphasise in the parallelism presented between FA and other behavioural addictions, whereby the central aspect of the addiction process is the act of eating as the addictive process, rather than considering a particular type of food as an addictive substance in itself (Hebebrand et al., 2014). Current evidence suggests that both mechanisms are valid and may coexist across different subgroups of patients (Jiménez-Murcia et al., 2019). This heterogeneity model is supported by previous findings, which indicate that FA is not a unitary phenomenon but rather encompasses distinct profiles (Jiménez-Murcia et al., 2019). Some of these are driven by reward sensitivity to food cues, while others by compulsive eating behaviours linked to emotional dysregulation. Thus, FA may integrate features of both substance and behavioural addiction models. The present study acknowledges both frameworks, recognising that, while substance-related mechanisms, such as those associated with UPFs, play a role, eating behaviours themselves also involve behavioural vulnerabilities.

The FA Model, proposed by Gearhardt et al., suggests that not all types of foods have an addictive potential (Gearhardt et al., 2023a). The consumption of UPFs (highly palatable, sugary, salty, rich in saturated and trans fats), with high caloric content appear to have an addictive effect (Gearhardt et al., 2023a). The Food and Agriculture Organization of the United Nations (FAO) defined UPFs as formulations of ingredients predominantly used exclusively by industry, and typically created through industrial processes and techniques (Monteiro, Cannon, Lawrence, Laura Da Costa Louzada, & Machado, 2019). These industrial processes and ingredients are designed with the objective of creating longer lasting, ready-to-consume, and hyperpalatable foods, with higher economic profit for the industry (Monteiro et al., 2019). The potential addictive effect produced by UPFs would include the search for, and compulsive consumption of, certain foods, despite the adverse physical and psychological consequences;

the activation of the same brain regions associated with the reward pathways, as well as the presence of tolerance, abstinence, persistent desire, or an inability to reduce this pattern of consumption (Gearhardt, Davis, et al., 2011). In addition to individual risk factors, there are also environmental aspects that play a significant role in the addiction process (Nutt, King, Saulsbury, & Blakemore, 2007). It has been evidenced that changes in the surrounding environment, like when a substance becomes inexpensive, easily accessible, heavily marketed, and socially acceptable to use, the responses to that substance will increase. In the case of FA, there is a social justice implication that needs to be remarked, as addictive drugs are not necessary for surviving, but food is (Gearhardt et al., 2023b). Due to their accessibility and affordability, UPFs have become a significant proportion of the total caloric intake for an increasing number of individuals worldwide, particularly those experiencing food insecurity (Gearhardt et al., 2023a), but also in those with other individual vulnerabilities, such as the presence of an eating disorder (ED) (Munguía, Camacho-Barcia, et al., 2022; Munguía, Gaspar-Pérez, et al., 2022). In this context, although evidence for its status as a distinct nosological entity is limited, FA shows strong transdiagnostic relevance. Rather than a separate diagnostic category, FA is increasingly viewed as a construct that spans disorders, including not only ED and other mental health conditions, such as behavioural addictions, but also obesity and disordered eating conditions (Fernandez-Aranda, Karwautz, & Treasure, 2018; Horsager, Færk, Gearhardt, Lauritsen, & Østergaard, 2022). FA core symptoms, including craving, loss of control, and persistent consumption despite adverse consequences, mirror criteria for SUDs and overlap with features of ED, particularly binge eating disorder and bulimia nervosa. Ongoing debates within the two main classification systems in psychiatry, Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD), question whether FA warrants independent classification or should be conceptualised as a dimensional phenomenon crossing diagnostic boundaries. Framing FA as transdiagnostic highlights shared neurobiological and behavioural mechanisms, such as dysregulated reward processing and compulsive eating, which manifest differently across clinical presentations (Fernandez-Aranda et al., 2018). This perspective supports research and treatment approaches targeting common underlying processes rather than rigid diagnostic categories.

In this regard, older adults with metabolic syndrome represent a clinically relevant population for the study of maladaptive eating behaviours. This group is characterised by metabolic dysregulation, chronic low-grade inflammation, and neurobiological alterations, such as impaired insulin signalling and dopaminergic dysfunction, which may heighten vulnerability to compulsive eating patterns (Džidić-Krivić et al., 2025). Evidence suggests that age-related changes in dopamine receptor availability and executive functioning further compromise reward regulation and cognitive flexibility, increasing susceptibility to addictive eating behaviours (Berry et al., 2016; Leung et al., 2025).

Moreover, chronic inflammation, along with pro-inflammatory dietary patterns, may also exacerbate these mechanisms by influencing appetite regulation and accelerating cognitive decline and brain ageing (Dunk et al., 2025). A comprehensive understanding of these interconnected metabolic, neurobiological, and psychological vulnerabilities, examined through a longitudinal design that tracks changes and identifies the progression over time, is essential for the development of targeted interventions.

In view of the exposed evidence, the general objective of this study is to contribute to this growing area of research by exploring the association between the consumption of UPFs and FA in Mediterranean older adults with overweight or obesity and metabolic syndrome. Moreover, specifically, we aimed to examine the cross-sectional association between the consumption of UPFs and FA at baseline. Additionally, we sought to determine whether a reduction in UPFs intake after one year of follow-up was associated with improvements in the severity and presence of FA. Lastly, we aimed to explore the potential role of psychological factors, specifically impulsivity, depressive symptoms, and cognitive functioning, in shaping the relationship between UPFs consumption and FA.

Based on previous evidence, we hypothesise that higher UPFs consumption at baseline would be associated with greater FA severity and a higher likelihood of FA presence. We further theorised that participants who achieved greater reductions in UPF intake during the one-year follow-up would exhibit significant decreases in FA scores and prevalence. Finally, we hypothesised that higher impulsivity and depressive symptoms, as well as lower cognitive functioning, would be linked to greater FA severity and could amplify the impact of UPF consumption on FA.

## MATERIALS AND METHODS

### Study design, participants, and study population

This observational, prospective study was conducted in the PREDIMED-Plus-Cognition cohort, a sub-study conducted within the PREDIMED-Plus study. PREDIMED-Plus was a large ( $n = 6,874$ ), 6-year multicentre, parallel-group, randomised clinical trial conducted in 23 different centres across Spain (Martínez-González et al., 2019). Its primary aim was to evaluate the effect of a lifestyle intervention on the primary prevention of cardiovascular disease, and on weight loss. PREDIMED-Plus participants were recruited between September 2013 and December 2016, and were randomized into either a weight-loss lifestyle intervention or a control group. The intervention group received recommendations to increase their adherence to an energy-reduced Mediterranean diet (MedDiet), physical activity promotion, and psychosocial support. In contrast, participants in the control group only received general, usual care recommendations to follow an energy-unrestricted MedDiet. Participants enrolled in the intervention group engaged in individual interview sessions and motivational group sessions on a

monthly basis during the initial year of the intervention, subsequently reducing to bi-monthly sessions. In addition, they were provided with free extra virgin olive oil (1 L per month) and nuts (500 g per month). The control group, meanwhile, received the standard healthcare provided by primary care professionals for the management of metabolic syndrome, along with all the written material and instructions on adhering to the traditional MedDiet, and on lifestyle recommendations. Furthermore, every six months, they were also invited to participate in the group's sessions. In these sessions, and with the aim of promoting the MedDiet and encouraging compliance with the trial, the participants received a free supply of virgin olive oil (6 L every 6 months) and nuts (3 kg every 6 months). Inclusion criteria comprise an age range of 55–75 years for men, and 60–75 years for women, the presence of overweight or obesity ( $27 \text{ kg/m}^2 \geq$  body mass index [BMI]  $< 40 \text{ kg/m}^2$ ), and the met of at least three of the five criteria for metabolic syndrome (elevated waist circumference, elevated triglycerides, reduced high-density lipoprotein cholesterol, elevated blood pressure, and elevated fasting glucose) at baseline (Alberti et al., 2009). Conversely, exclusion criteria encompassed illiteracy or incapacity to give written informed consent, permanent or long-stay residence in a care home, documented history of previous cardiovascular disease, inability to follow the recommended diet (including religious reasons, swallowing disorders, food allergies to any component of MedDiet), or to carry out physical activity, history of obesity surgery or current use of weight loss medication, serious psychiatric disorders, alcohol consumption of  $>50\text{g}$  or drug use, among others. The current study included  $n = 429$  participants from the PRE-DIMED-Plus-Cognition sub-study, recruited in four Spanish centres: Hospital del Mar Medical Research Institute, Bellvitge University Hospital, Rovira i Virgili University, and Valencia University. The present sub-study aimed to deepen assessments of psychological and cognitive domains in a smaller sample of participants. The participants' flowchart and data availability are shown in Figure A1, the analytic sample consists exclusively of individuals with complete data for the measures used in the analyses. The study protocol with further information is available at: <http://www.predimedplus.com>. The trial was registered in the International Standard Randomized Controlled Trial Registry, and details can be found at <http://www.isrctn.com/ISRCTN89898870>.

## Assessment

**Ultra-processed foods consumption.** The participants' dietary intake was assessed by trained dietitians through a 143-item validated semi-quantitative food frequency questionnaire (FFQ) (Fernández-Ballart et al., 2010). The FFQ was completed at baseline and annually at each follow-up visit. Nutritional information (total energy, macro and micronutrients) was obtained from the FFQ and estimated using Spanish food composition tables (Mataix, 2003; Moreiras, Carbajal, Cabrera, & Cuadrado, 2005). For the UPFs determination, items in the FFQ were categorized

according to the NOVA food classification system (Monteiro et al., 2019). This classification system groups all foods according to the extent and purpose of food processing into four groups: Group 1: unprocessed or minimally processed foods, Group 2: processed culinary ingredients, Group 3: processed foods, and Group 4: ultra-processed foods. The consumption of UPFs in this study was estimated using 36 different items of the FFQ classified as Group NOVA 4, as a previous analysis from this cohort (González-Palacios et al., 2023). The UPFs intake was expressed in grams per day and later converted as percentage of consumption within the total daily dietary intake ( $[\text{intake of UPFs in grams}/\text{total dietary intake in grams}] \times 100$ ). For analytical purposes, participants' UPFs percentage of daily intake was later categorised into tertiles. The sample was ranked and divided into three equal sized groups: T1 (lowest consumption: values between 0.01 to 1.00%), T2 (intermediate consumption: values between 1.01 to 6.00%), and T3 (highest consumption: values between 6.01 to 37.00%). Cut-off points for tertiles were determined empirically from the sample distribution.

**Food addiction.** The presence of FA was assessed using the Spanish validated version of the Yale Food Addiction Scale, version 2.0 (Gearhardt, Corbin, & Brownell, 2016; Granero et al., 2018). This scale is a 35 self-report questionnaire that assesses the symptom criteria for substance use disorder in DSM-5 in the context of food intake. It is based on an eight-level Likert scale that ranges from 0 = never to 7 = every day. There are three different approaches to deriving the scale. Firstly, a continuous symptom count score ranging from 0 to 11 is used, with 11 reflecting the number of diagnostic criteria met. Secondly, a diagnosis of FA is based on the number of symptoms, and clinically significant impairment or distress. Thirdly, a group named as 'FA probable' was considered for those participants that met criteria for sub-clinical or subthreshold (only 1 symptom criteria, or more than 3 symptoms without the presence of related impairment). Prior studies have highlighted the clinical relevance of subthreshold or subclinical presentations, given the substantial proportion of individuals who do not meet full diagnostic criteria; but nonetheless experience significant distress (Okasha, 2011). Recent research has also examined subthreshold food addiction in adults using the YFAS-2, and has applied an operational definition consistent with that used in the present study (Constant, Som, Val-Laillet, Moirand, & Thibault, 2025). The Cronbach's alpha internal consistency in the present sample was  $\alpha = 0.911$ .

**Biochemical, anthropometric, and blood pressure measurements.** At baseline and at one-year follow-up, fasting blood samples were collected to determine circulating levels of triglycerides, total cholesterol, HDL-cholesterol and LDL-cholesterol, fasting glucose, glycated haemoglobin (HbA1c), and insulin. The homeostatic model assessment for insulin resistance (HOMA-IR) was estimated through the following formula: fasting glucose levels [mg/dL]  $\times$  fasting insulin levels [ $\mu\text{U/mL}$ ]/405,13 (Matthews et al., 1985).

At each visit, participants' height and weight were measured in light clothes and without shoes, in duplicate (the average value was used for analysis), by trained staff using a wall-mounted stadiometer and calibrated scales, respectively. The BMI was calculated as weight (kg)/height (m) squared. The waist circumference (cm) was determined midway between the lowest rib and the iliac crest with use of an anthropometric tape.

Systolic and diastolic blood pressure was measured using a validated semi-automatic oscillometer (Omron HEM-705CP).

**Psychological variables.** Different Spanish-validated questionnaires were administered at baseline and after one year of follow-up to measure different psychological variables. These were administered by trained psychologists.

Trait impulsivity was assessed using the UPPS-P Impulsivity Scale (Whiteside, Lynam, Miller, & Reynolds, 2005), which includes five different subscales (negative urgency, positive urgency, lack of premeditation, lack of perseverance, and sensation seeking) rated on a 4-point Likert scale. The validated Spanish version (Verdejo-García, Lozano, Moya, Alcázar, & Pérez-García, 2010) showed an overall good reliability across subscales, ranging from  $\alpha = 0.74$  for positive urgency to  $\alpha = 0.83$  for negative urgency. In our sample, internal consistency for the total score was  $\alpha = 0.907$ .

Depressive symptomatology was measured using the Spanish validated version of Beck Depression Inventory-II (BDI-II) (Sanz, Perdigón, & Vázquez, 2003), a 21-item self-report questionnaire scored from 0 to 63. Internal consistency in our sample was  $\alpha = 0.871$ .

Cognitive function was evaluated using the Spanish validated version of the Montreal Cognitive Assessment (MoCA) (Ojeda del Pozo, del Pino Sáez, Ibarretxe Bilbao, Schretlen, & Peña Lasa, 2016), a screening tool covering multiple domains (e.g., memory, attention, executive function) with a maximum score of 30. Internal consistency in our sample was  $\alpha = 0.814$ .

**Other clinical and sociocultural variables.** A general questionnaire was administered to all participants at the baseline and one-year follow-up visits, with the objective of collecting information on socio-demographics, lifestyle, previous medical conditions, and medication use. Leisure-time physical activity was assessed using a Spanish validated questionnaire (Molina et al., 2017), and this data was later used to determine total physical activity as metabolic equivalent of task minutes per week (METs/week). Civil status was dichotomized into married, or all others, including single, separated, widow(er) and divorced. Prevalence of type 2 diabetes, hypertension and hypercholesterolemia were also used as dichotomous variables (yes or no).

## Statistical analysis

The data analysis was done with Stata18 for Windows. First, the differences between the groups at baseline were tested with chi-square ( $\chi^2$ ) for categorical variables and analysis of variance (ANOVA) for quantitative variables. These

procedures included pairwise comparisons and the calculation of their effect size through the standardized Cramer's- $V$  and Cohen's- $d$  coefficients. Finner's method was also used for controlling the increase in the Type-I error due to the multiple significance tests.

Second, changes in UPF and FA levels over the follow-up period were examined using repeated measures ANCOVA. Separate analyses were conducted for each measure, defining as the intra-subject factor (dependent variable) the assessment time (baseline versus 1-year follow-up), while the intervention, treatment group, and baseline BMI were included as covariates to control for their potential confounding effects. Furthermore, logistic regression analyses were conducted to compare the prevalence of participants exhibiting a decrease in FA levels across the three groups generated from tertiles of UPF reduction. In these models, the outcome variable was coded as 0 for participants without a decrease, and 1 for participants with a decrease, and the analyses were similarly adjusted for the intervention treatment group and baseline BMI.

Path analysis, implemented through Structural Equation Models (SEM), assessed the underlying relationships between the main variables of the study at baseline. The rationale for defining the model specification was based on the theoretical background; provided by the cumulated evidence, all parameters were freely estimated, and the maximum-likelihood estimation method was employed for the estimations. With the aim to obtain parsimonious models and increasing statistical power, an initial model was defined with all the potential effects (direct and indirect links), and next, the parameters with no statistical significance were deleted and the model was re-specified and adjusted. Goodness of fit was tested as usual, and it was considered adequate for: non-significant result in the  $\chi^2$  test ( $p > 0.05$ ), Root Mean Square Error of Approximation, RMSEA < 0.08, Bentler's Comparative Fit Index, CFI > 0.90, Tucker-Lewis Index TLI > 0.90, and Standardized Root Mean Square Residual SRMR < 0.10.

## Ethics

All participants provided written informed consent. According to the ethical standards of the Declaration of Helsinki, by the Research Ethics Committees, all the participating institutions approved the study protocol and procedures (Clinical Research Ethics Committee (CEIC) from the Bellvitge University Hospital-IDIBELL: PR240/13; CEIC Parc de Salut Mar, and IDIAP Jordi Gol—IMIM: PI13/130; CEIC Corporativo de Atención Primaria de la Comunitat Valenciana—University of Valencia: CEIC del Hospital Universitari Sant Joan de Reus y IDIAB Jordi Gol—Universitat Rovira i Virgili: 2011-005398-22; 13-07-25/7proj2).

## RESULTS

Demographic characteristics of participants, following the distribution into tertiles defined by UPF consumption, are described in Table 1.

Table 1. Descriptive for the total sample at baseline, and according to tertiles of baseline ultra-processed food consumption

		Total sample (n = 429)		UPF T1 (n = 142)		UPF T2 (n = 143)		UPF T3 (n = 144)		$\chi^2$	df	p
		n	%	n	%	n	%	n	%			
Sex	Male	206	48.0%	50	35.2%	72	50.3%	84	58.3%	15.78	2	<.001*
	Female	223	52.0%	92	64.8%	71	49.7%	60	41.7%			
Civil status	Single	15	3.5%	5	3.5%	6	4.2%	4	2.8%	3.06	6	.801
	Married	335	78.1%	107	75.4%	112	78.3%	116	80.6%			
	Divorced-separated	30	7.0%	10	7.0%	12	8.4%	8	5.6%			
	Widowed	49	11.4%	20	14.1%	13	9.1%	16	11.1%			
School	University (high)	40	9.3%	9	6.3%	17	11.9%	14	9.7%	19.58	6	.003*
	University (grade)	34	7.9%	6	4.2%	10	7.0%	18	12.5%			
	Secondary	124	28.9%	40	28.2%	32	22.4%	52	36.1%			
	Primary	231	53.8%	87	61.3%	84	58.7%	60	41.7%			
Employment	Employed	78	18.2%	15	10.6%	31	21.7%	32	22.2%	27.21	8	.001*
	Work at home	45	10.5%	23	16.2%	15	10.5%	7	4.9%			
	Retired	275	64.1%	95	66.9%	87	60.8%	93	64.6%			
	Unemployed (incomes)	18	4.2%	2	1.4%	5	3.5%	11	7.6%			
Group weight	Unemployed (no-incomes)	13	3.0%	7	4.9%	5	3.5%	1	0.7%	4.01	4	.405
	Over-weight	118	27.5%	45	31.7%	40	28.0%	33	22.9%			
	Obesity I (BMI 30-35)	211	49.2%	68	47.9%	72	50.3%	71	49.3%			
Age (years-old)	Obesity II (BMI > 35)	100	23.3%	29	20.4%	31	21.7%	40	27.8%	9.455	2; 426	<.001*
	Mean	SD	Mean	SD	Mean	SD	Mean	SD				
		65.27	4.63	66.59	4.40	64.87	4.50	64.35	4.70			

Note. UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%. BMI: Body Mass Index; SD: standard deviation. df: degrees of freedom. \*Bold: significant comparison.

## Comparison of the groups at baseline

Table 2 presents the results of the analyses conducted to examine the association between UPF consumption and FA at baseline (first objective of the study). The first comparison in this table corresponds to an ANOVA, assessing differences in the mean YFAS total score across the three UPF-level groups. The other two comparisons involve  $\chi^2$  tests, evaluating differences in the prevalence of YFAS risk categories across the UPF levels. No differences between groups were identified for the YFAS total score, nor for the YFAS binary screening group (positive versus negative). However, the classification of the YFAS in three levels (positive-probable-negative) achieved differences for the contrast

between the groups: UPF T2 versus UPF T3, being the highest tertile of consumption characterised by the higher likelihood of worse FA state (Fig. 1).

The comparisons between groups for the remaining study measures are presented in Table 3. Mean differences for quantitative variables were assessed using ANOVA, whereas differences in the prevalence of hypertension, type 2 diabetes, and hypercholesterolemia were examined using  $\chi^2$  tests. These results indicate that the T1 of UPFs consumption was characterised by achieving lower mean scores in alanine aminotransferase and aspartate aminotransferase as compared with UPFs T2, lower weight and waist circumference as compared with UPFs T3, lower intake of total kcal and total grams of carbohydrates as compared with the other

Table 2. Comparison of Food Addiction (FA) at baseline according to tertiles of consumption of Ultra-processed Food

	UPF T1 (n = 142)		UPF T2 (n = 143)		UPF T3 (n = 144)		UPF T1 vs UPF T2		UPF T1 vs UPF T3		UPF T2 vs UPF T3	
	Mean	SD	Mean	SD	Mean	SD	p	d	p	d	p	d
FA: YFAS total score	1.68	1.26	1.62	1.27	1.96	1.25	.937	0.04	.168	0.22	.079	0.27
YFAS: total score												
FA: YFAS screening score	n	%	n	%	n	%	p	C-V	p	C-V	p	C-V
Positive	7	4.9%	5	3.5%	12	8.3%	.547	0.036	.248	0.068	.083	0.102
Negative	135	95.1%	138	96.5%	132	91.7%						
Probable	7	4.9%	5	3.5%	12	8.3%	.832	0.036	.125	0.121	<b>.047*</b>	0.146
Negative	116	81.7%	119	83.2%	103	71.5%						

Note. UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%. FA: Food Addiction; SD: standard deviation; YFAS: Yale Food Addiction Scale. \*Bold: significant comparison.

†Bold: effect size into the range mild-moderate to high-large.

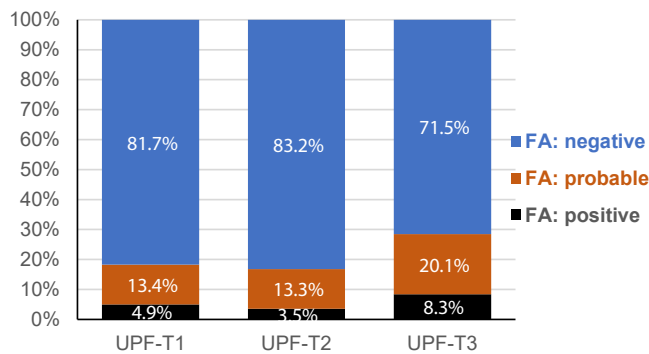


Fig. 1. Prevalence of Food Addiction (FA) screening score by tertiles of Ultra-processed Food consumption at baseline

Note. UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%.

two conditions, and higher percentages of total protein and total lipids in grams as compared with the other two tertiles. The highest tertile of UPFs consumption reported the highest mean for insulin and HOMA-IR as compared with the other tertiles, and higher monounsaturated fatty acids (MUFA) grams as compared with the first tertile of UPFs consumption. Other variables with differences between all the groups were saturated fatty acids (g and %), and trans fatty acids, with the lowest values among the UPF T1 and the highest among the UPF T3.

### Assessment of the changes during follow-up

Table 4 displays the results obtained in the assessment of the changes observed for the UPFs consumption and the FA levels (results obtained in repeated measures ANCOVA, adjusted for the covariates: intervention group and baseline BMI). Separate analyses were conducted for the total sample, as well as for the subsamples defined according to FA screening status and UPF consumption groups. For the complete sample, a significant decrease for both the UPFs consumption and the FA total scores was identified. The UPF level also decreased for the participants, independent of the FA level at baseline (see first graph in Fig. 2). However, the FA levels only achieved significant decreases for participants grouped at UPF T1 and UPF T3 at baseline (see second graph in Fig. 2).

### Assessment of the associations between the changes in UPF and FA during follow-up

Table 5 presents the results of the logistic regression analysis (adjusted for intervention group and baseline BMI), conducted to examine the association between the reduction in UPF consumption after one year of intervention and the presence of FA (second objective of the study). The upper section of the table reports the prevalence of individuals with and without a pre–post decrease in the YFAS raw score. The lower section presents the comparison across the three

groups defined by tertiles of pre–post changes in UPF levels (see also Fig. 3). The pairwise comparison (obtained in logistic regression adjusted by the intervention group and the BMI at baseline), achieved statistical differences comparing the prevalences registered within the tertiles 3 and 2 ( $OR = 1.67$ ,  $p = .040$ ). This evidenced that the likelihood of change in the FA levels is higher only for individuals within the highest decrease in the UPF levels.

### Path analysis to identify relationship patterns at baseline

Figure 4 presents the path diagram with the standardized coefficients of the SEM estimated using baseline measures, allowing visualization of the significant relationships among the main variables of the study. This SEM identifies the role of UPF consumption and YFAS scores in relation to BMI and BDI levels, while accounting for the additional contributions of sex, impulsivity, and cognitive functioning. Adequate fitting was obtained for the final model ( $\chi^2 = 7.71$  [ $p = .657$ ],  $RMSEA = 0.002$  [95%CI: 0.001 to 0.043],  $CFI = 0.999$ ,  $TLI = 0.999$ , and  $SRMR = 0.022$ ). Appendix Table A1 contains the complete results for this model.

Regarding the BMI, higher values were directly related with higher UPF and FA levels. Indirect mediational links were also identified: a) men increased UPF levels, and this profile was associated to higher likelihood for BMI; b) females and participants with higher impulsivity increased FA levels, which next contributed to higher BMI.

Concerning the depression levels, worse states were directly associated to women and participants with higher FA and impulsivity levels. The indirect links for this measure were: a) being women, which contributed to increased FA, and next to higher depression symptom levels; and b) higher impulsivity, which impacted in increased FA, resulting in a worse depression state. No significant associations were found for cognitive function.

### Path analysis to identify relationship patterns during the follow-up

Figure 5 presents the path diagram, including the standardized coefficients of the SEM, examining the longitudinal associations among the main study variables. This model, adjusted for treatment group, illustrates how changes in these variables over time are interrelated, and highlights the significant pathways that account for the temporal dynamics observed throughout the follow-up period. Adequate fitting was obtained for the final model ( $\chi^2 = 71.79$  [ $p = .163$ ],  $RMSEA = 0.020$  [95%CI: 0.001 to 0.037],  $CFI = 0.994$ ,  $TLI = 0.991$ , and  $SRMR = 0.041$ ). Appendix Table A2 contains the complete results for this model.

At baseline, as compared to men, women obtained lower UPF levels, but higher FA and depression levels. As expected, significant associations were obtained between each measure at baseline and at 1 year of the follow-up. In addition, higher FA at baseline achieved significant longitudinal capacity for predicting higher BMI and depression levels 1-year later, higher BMI at baseline contributed to

Table 3. Comparison of metabolic and dietary measures according to tertiles of Ultra-processed Food consumption at baseline

	UPF T1 (n = 142)		UPF T2 (n = 143)		UPF T3 (n = 144)		UPF T1 vs UPF T2		UPF T1 vs UPF T3		UPF T2 vs UPF T3	
	Mean	SD	Mean	SD	Mean	SD	p	d	p	d	p	d
Glucose (mg/dL)	117.26	31.04	114.38	28.58	116.76	28.58	.712	0.10	.990	0.02	.791	0.08
Insulin (mIU/mL)	16.95	8.12	17.97	8.27	20.64	8.89	.594	0.12	<b>.001*</b>	0.43	<b>.028*</b>	0.31
HOMA-IR	5.00	3.00	5.10	2.70	6.07	3.34	.964	0.03	<b>.012*</b>	0.34	<b>.025*</b>	0.32
Triglycerides (mg/dL)	152.33	71.17	156.57	75.26	172.13	85.91	.900	0.06	.100	0.25	.239	0.19
Total cholesterol (mg/dL)	208.55	39.09	210.45	42.19	205.97	37.82	.922	0.05	.860	0.07	.634	0.11
LDL-cholesterol (mg/dL)	127.71	35.00	126.92	33.51	123.04	31.31	.980	0.02	.496	0.14	.616	0.12
HDL-cholesterol (mg/dL)	52.60	13.52	52.24	13.86	49.44	11.06	.973	0.03	.118	0.26	.184	0.22
Albumin (g/dL)	4.39	0.64	4.48	0.24	4.44	0.53	.254	0.20	.661	0.09	.754	0.11
HbA1c(%)	6.19	0.76	6.12	0.75	6.04	0.74	.728	0.09	.265	0.19	.706	0.10
Alanine aminotransferase (U/L)	24.43	11.10	28.72	13.45	26.71	12.78	<b>.016*</b>	0.35	.308	0.19	.393	0.15
Aspartate aminotransferase (U/L)	22.53	7.61	25.51	9.45	24.08	8.05	<b>.012*</b>	0.35	.299	0.20	.355	0.16
Systolic blood pressure (mm Hg)	141.02	16.42	141.74	13.23	139.47	15.38	.922	0.05	.685	0.10	.443	0.16
Diastolic blood pressure (mmHg)	78.37	10.95	80.62	7.65	80.87	9.30	.131	0.24	.082	0.25	.976	0.03
Total physical activity energy expenditure (MET min/week)	913.01	874.70	800.12	758.47	776.09	731.10	.484	0.14	.343	0.17	.967	0.03
Weight (kg)	82.39	13.05	85.59	12.72	88.83	14.29	.132	0.25	<b>&lt;.001*</b>	0.47	.123	0.24
BMI (kg)	32.18	3.45	32.35	3.28	32.95	3.50	.909	0.05	.160	0.22	.334	0.18
Waist circumference(cm)	106.31	9.76	106.99	10.86	109.63	10.07	.853	0.07	<b>.024*</b>	0.33	.095	0.25
Total energy intake (Kcal)	2,169	452.66	2,437	498.33	2,591	692.77	<b>&lt;.001*</b>	<b>0.56<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.72<sup>†</sup></b>	.065	0.26
Carbohydrates total (g)	214.60	58.19	249.09	75.48	260.88	94.37	<b>.001*</b>	<b>0.51<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.59<sup>†</sup></b>	.437	0.14
Carbohydrates total (%)	39.39	6.05	40.53	6.63	39.78	6.88	.341	0.18	.880	0.06	.626	0.11
Proteins total (g)	97.95	19.34	101.45	19.88	102.15	22.97	.366	0.18	.234	0.20	.960	0.03
Proteins total (%)	18.37	3.25	16.86	2.46	16.12	2.58	<b>&lt;.001*</b>	<b>0.52<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.76<sup>†</sup></b>	.083	0.29
Lipids total (g)	96.07	22.77	107.87	24.09	119.52	33.00	<b>.001*</b>	<b>0.50<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.83<sup>†</sup></b>	<b>.002*</b>	0.40
Lipids total (%)	39.92	6.11	40.14	6.15	41.71	5.90	.956	0.04	<b>.045*</b>	0.30	.090	0.26
MUFA (g)	50.20	13.71	55.83	13.35	60.40	16.36	<b>.005*</b>	0.42	<b>&lt;.001*</b>	<b>0.68<sup>†</sup></b>	<b>.030*</b>	0.31
MUFA (%)	20.89	4.39	20.84	4.11	21.23	4.02	.995	0.01	.798	0.08	.740	0.09
PUFA (g)	16.23	4.91	17.94	5.92	20.14	7.97	.080	0.31	<b>&lt;.001*</b>	<b>0.59<sup>†</sup></b>	<b>.015*</b>	0.31
PUFA (%)	6.76	1.57	6.61	1.61	7.00	2.15	.785	0.09	.514	0.13	.180	0.21
Saturated fatty acids (g)	23.53	7.09	27.94	7.08	31.93	11.09	<b>&lt;.001*</b>	<b>0.62<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.90<sup>†</sup></b>	<b>.001*</b>	0.43
Saturated fatty acids (%)	9.72	1.99	10.36	1.85	11.02	1.77	<b>.016*</b>	0.33	<b>&lt;.001*</b>	<b>0.69<sup>†</sup></b>	<b>.013*</b>	0.36
Trans fatty acids (%)	0.49	0.32	0.69	0.35	0.81	0.51	<b>&lt;.001*</b>	<b>0.61<sup>†</sup></b>	<b>&lt;.001*</b>	<b>0.77<sup>†</sup></b>	<b>.034*</b>	0.28
Prevalences	n	%	n	%	n	%	p	C-V	p	C-V	p	C-V
Hypertension	105	73.9%	110	76.9%	110	76.4%	.559	0.035	.632	0.028	.915	0.006
Type 2 Diabetes	43	30.3%	43	30.1%	35	24.3%	.969	0.002	.257	0.067	.272	0.065
Hypercholesterolemia	77	54.2%	70	49.0%	74	51.4%	.373	0.053	.631	0.028	.680	0.024

Note. UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%. BMI: Body Mass Index; HbA1c: glycated haemoglobin; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; MUFA: Monounsaturated fatty acids; PUFA: Polyunsaturated fatty acids; SD: standard deviation. \*Bold: significant comparison. <sup>†</sup>Bold: effect size into the range mild-moderate to high-large.

higher FA levels 1-year later, and higher impulsivity also contributed to higher FA levels at the end of the follow-up.

## DISCUSSION

The findings of the present study offer novel perspectives on the association between the consumption of UPFs and the presence of FA in an older adult population with overweight

or obesity. Our results indicated significant differences between baseline tertiles of UPFs consumption, as measured by percentage of total daily dietary intake, and the classification of FA levels (positive, probable, and negative). In comparison with the mid-tertile of consumption, the highest tertile of UPF consumption was characterised by a higher likelihood of a worse FA state. However, non-significant associations were identified for the total score of the YFAS, nor for the binary positive versus negative presence of FA. As stated

Table 4. Changes in the mean scores for the Ultra-Processed Foods and the Food Addiction during the follow-up

		Baseline		1 year follow-up		p	d
		Mean	SD	Mean	SD		
UPF	Total sample (n = 429)	7.86	6.09	4.74	4.15	<.001*	<b>0.60</b> <sup>†</sup>
UPF	FA- at baseline (n = 405)	7.79	6.03	4.73	4.19	<.001*	<b>0.59</b> <sup>†</sup>
	FA+ at baseline (n = 24)	9.15	7.33	4.86	3.61	<b>.010</b> *	<b>0.74</b> <sup>†</sup>
UPF	FA- at baseline (n = 338)	7.68	6.16	4.60	4.26	<.001*	<b>0.58</b> <sup>†</sup>
	FA probable at baseline (n = 67)	8.33	5.20	5.39	3.84	<.001*	<b>0.64</b> <sup>†</sup>
	FA+ at baseline (n = 24)	9.15	7.33	4.86	3.61	<b>.010</b> *	<b>0.74</b> <sup>†</sup>
FA: YFAS total	Total sample (n = 429)	1.75	1.23	1.45	1.05	<.001*	0.27
	UPF T1 at baseline (n = 142)	1.68	1.23	1.39	1.00	<b>.007</b> *	0.26
	UPF T2 at baseline (n = 143)	1.62	1.20	1.48	1.07	.160	0.13
	UPF T3 at baseline (n = 144)	1.96	1.24	1.48	1.08	<.001*	0.41

Note. UPF: ultra processed food; FA: food addiction; SD: standard deviation; YFAS: Yale Food Addiction Scale.

UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%. Results adjusted by the intervention group and BMI at baseline. \*Bold: significant comparison. †Bold: effect size into the range mild-moderate to high-large.

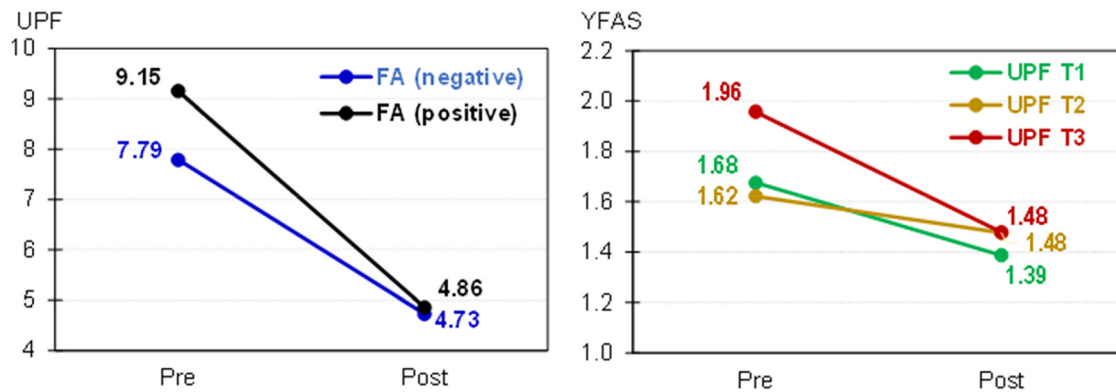


Fig. 2. Changes in Ultra Processed-food (UPF) consumption and Food Addiction (FA) during one year follow-up

Note. UPF: ultra processed food. FA: food addiction. UPF-T1: tertile 1 of % of ultra-processed food consumption/day, values between 0.01 to 1.00%. UPF-T2: tertile 2 of % of ultra-processed food consumption/day, values between 1.01 to 6.00%. UPF-T3: tertile 3 of % of ultra-processed food consumption/day, values between 6.01 to 37.00%. Results adjusted by intervention group and BMI at baseline.

Table 5. Prevalence of patients with and without decrease in the FA screening score by tertiles of change of consumption of Ultra-processed Foods

		Change UPF-T1 (n = 143)		Change UPF -T2 (n = 142)		Change UPF -T3 (n = 141)	
		n	%	n	%	n	%
Decrease in the YFAS	No	94	65.7%	98	69.0%	83	57.6%
	Yes	49	34.3%	44	31.0%	61	42.4%
<i>*Pairwise comparisons</i>		B	SE	p	OR	95% CI for OR	
UPF-T2 vs UPF-T1		-0.142	0.253	.576	0.868	0.528	1.426
UPF-T3 vs UPF-T1		0.369	0.247	.134	1.447	0.892	2.346
UPF-T3 vs UPF-T2		-0.511	0.249	.040	1.667	1.023	2.716

Note. YFAS: Yale Food Addiction Scale. Change UPF-T1: change in ultra processed food into tertile1 (decreases equal or lower than 17.0). Change UPF-T2: change in ultra processed food into tertile2 (decreases between 17.1 to 78.0). Change UPF-T3: change in ultra processed food into tertile3 (decreases higher than 78.1).

\*Results of the logistic regression adjusted for the intervention group and baseline BMI.

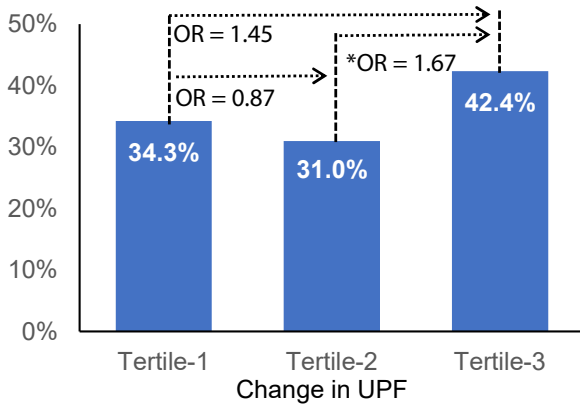


Fig. 3. Prevalence of patient with decrease in the FA screening score by tertiles of change of consumption of Ultra-processed Foods  
 Note. Change UPF: change in ultra processed food. OR: odds ratio (adjusted for the intervention group and baseline BMI).  
 \*Bold: significant parameter.

in recent systematic reviews and meta-analyses, there is limited evidence regarding the association of addictive eating behaviours and dietary profiles, patterns, and nutrient intake (Pursey, Skinner, Leary, & Burrows, 2021; Reche-García, Piernas, Martínez-Rodríguez, Sánchez-Guerrero, & Hernández-Morante, 2022). Most studies assessed in these evaluated total calorie intake and nutrient consumption. Following a systematic review of the extant evidence, Pursey et al. found that, compared to individuals without FA, adults with YFAS-diagnosed FA had significantly higher energy, total fat, saturated, mono and poly-unsaturated fatty acids, and carbohydrate intake (Pursey et al., 2021). Of the studies reviewed, one assessed the consumption of UPFs as defined by the NOVA classification system, and FA presence in children with overweight. Their findings indicated a tendency for higher consumption of UPFs in children with a diagnosis of FA (Filgueiras et al., 2019). In line with this,

a recent study aimed to compare the intake of UPFs and FA among young adults, showing a positive association between percentage energy from NOVA UPFs and FA status in young adults aged between 18 and 35 years (Whatnall, Clarke, Collins, Pursey, & Burrows, 2022). Yet, no results have been reported for this type of assessment in older adults. The findings of the meta-analysis performed by Reche-García et al. are of particular interest in this context. This work concluded that, while the presence of FA was not associated with higher energy intake in the general population, it was linked to higher intake of processed and energy-dense foods and higher total calorie intake among individuals with overweight or obesity (Reche-García et al., 2022). The higher likelihood of worse FA status in highest UPF tertile aligns with substance-related mechanisms, suggesting that hyper-palatable UPFs may activate reward pathways similar to addictive substances. However, the absence of differences in total YFAS scores suggests that FA may not be fully explained by UPF intake alone, pointing to additional behavioural factors.

Furthermore, results from this study showed that, after one year of follow-up, a reduction in UPFs intake was associated with a decrease in FA total scores and FA presence. For the complete sample, a significant decrease for both the UPFs and the FA total scores was identified. However, the FA levels only achieved significant decreases for participants grouped at the first and third baseline tertiles of UPFs consumption, interestingly, the ones with highest FA scores. In addition, when we assessed the changes in both UPF consumption and FA scoring after one year of follow-up, results suggested that the likelihood of change in the FA levels was higher only for those individuals within the highest decreases in UPF consumption. It has been previously reported that FA has a negative impact on the treatment outcomes of patients with obesity and ED (L. Camacho-Barcia, Munguía, Gaspar-Pérez, Jimenez-Murcia, & Fernández-Aranda, 2022; Halbeisen et al., 2025).

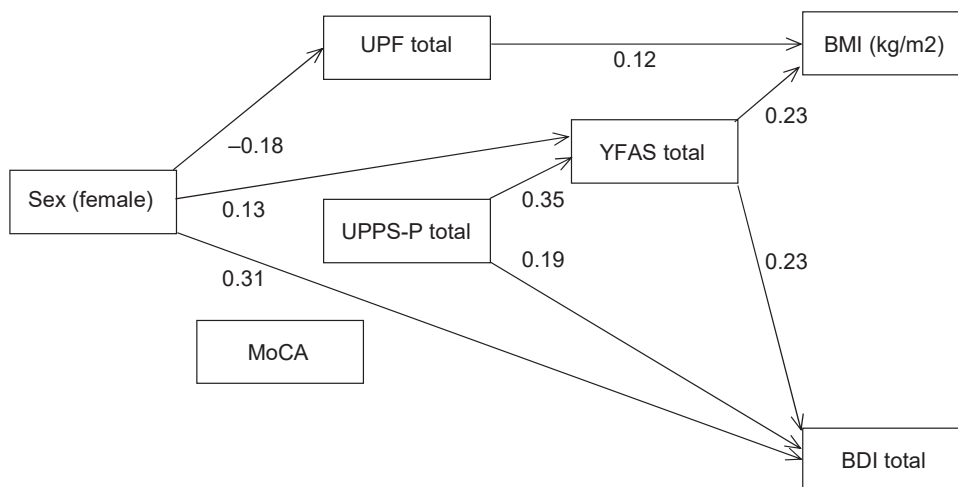


Fig. 4. Path diagram with the standardized coefficients (results at baseline)

Note. Only significant coefficients retained in the model. BDI: Beck Depression Inventory; BMI: Body Mass Index; MoCA: Montreal cognitive assessment; UPF: Ultra-processed food; UPPS-P: Impulsive Behaviour Scale; YFAS: Yale Food Addiction Scale.

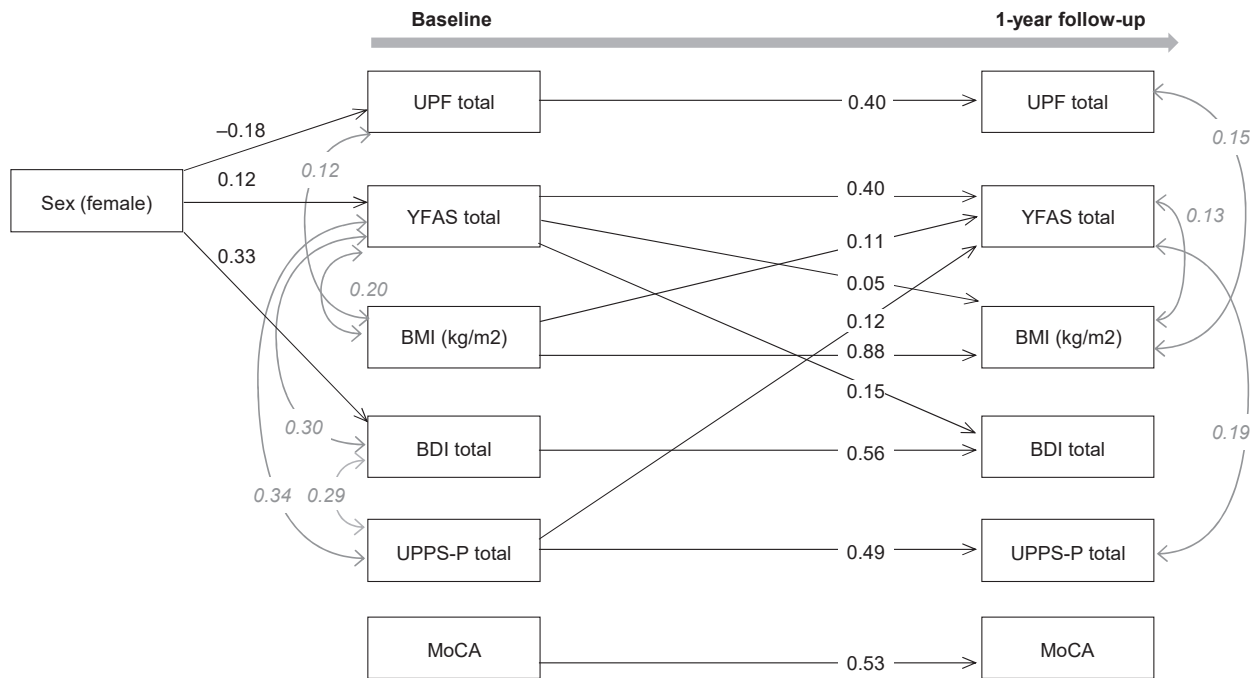


Fig. 5. Path diagram with the standardized coefficients

Note. Only significant coefficients retained in the model. BDI: Beck Depression Inventory; BMI: Body Mass Index; MoCA: Montreal cognitive assessment; UPF: Ultra-processed food; UPPS-P: Impulsive Behaviour Scale; YFAS: Yale Food Addiction Scale. Results adjusted by intervention group.

It can act as a predictor or mediator of adverse outcomes, including lower weight loss, higher levels of dropouts, and lower levels of remission (L. Camacho-Barcia et al., 2022). Though there is scarce evidence regarding specific interventions targeting FA, studies show that in patients with obesity and FA, weight reduction can decrease the FA symptoms, particularly following metabolic surgery (Reche-García et al., 2024). Nevertheless, little is known about how specific changes in dietary patterns can affect FA. Some authors have proposed that low-carbohydrate interventions may contribute to improve the FA symptoms (Carmen et al., 2020; Unwin et al., 2022); however, no long-term results have been reported. In the context of biological indicators of FA, it has been observed in animal models that foods rich in simple carbohydrates may be involved in addictive processes. Results suggest that the consumption of high sugar and fat foods in the form of binge eating could lead to elevated dopamine levels in the brain, specifically in the mesolimbic dopaminergic system, and trigger behaviours similar to substance addiction (Avena, Rada, & Hoebel, 2009). However, if the behavioural aspect of FA is also taken into consideration, such as the loss of control over consumption, increased cravings, and excessive and continued intake despite negative consequences, the foods suggested to be most implicated are those with a high level of palatability, high in refined carbohydrates, fats and salt (Munguía, Camacho-Barcia, et al., 2022; Schulte, Avena, & Gearhardt, 2015, 2017). Considering the industrial modifications of UPFs to enhance their palatability, and the adding of these nutrients in high concentrations, it is possible to suggest that

the decrease in UPFs contributed to the reduction in FA scores. Individuals in both intervention and control groups in this study were recommended to adhere to a Mediterranean diet pattern. Participants were instructed not only in incorporating fresh, seasonal and regional products naturally dense in high quality nutrients, but to also increase the home-made preparations. It is worth mentioning that, despite both FA and non-FA groups achieving weight loss after the intervention, as previously evidenced in other analyses of this sample (Camacho-Barcia et al., 2021), the progression of BMI during the follow-up differed in each FA condition. At baseline, individuals with FA exhibited significantly higher BMI. After one year of follow-up, both FA and non-FA groups achieved significant weight loss, however, non-significant differences in BMI were observed between groups. These findings may suggest that changes in BMI may not be the only factor influencing FA symptoms, but that the improvement in the nutritional quality may also play a role. The observed improvement in FA symptoms following a UPF reduction, independent of weight loss, may underline the behavioural component of FA, where modifying eating patterns reduces compulsive consumption. This supports the hypothesis that FA reflects both substance-related and behavioural vulnerabilities.

The results from the structural equation models revealed no significant direct associations between UPFs consumption and the presence of FA. Nevertheless, interestingly, both baseline, cross-sectional and longitudinal analyses found links with both FA and UPFs and BMI. In the longitudinal analysis, a higher FA presence at baseline achieved a

significant capacity for predicting a higher BMI after one year of follow-up, which significantly correlated with UPF consumption. Despite the literature evidencing a solid relationship between FA and BMI, it would have been interesting to analyse further the obesity dimension, including body composition and adipose tissue distribution, when including the consumption of UPFs in the analysis. This is particularly relevant in light of previous findings from the Predimed-Plus study, which showed that a 10% daily increment in consumption of UPF was associated with significantly greater accumulation of total fat, visceral fat, and android-to-gynoid fat ratio (Konieczna et al., 2021). One potential hypothesis that can be derived from this work is that, given body composition is affected by the consumption of UPFs, including these variables in the model could lead to the identification of other types of mediating effects or direct associations. It is unfortunate that the available measurements in this subsample of the study do not permit further investigation of this hypothesis. Nonetheless, these findings strengthen the interpretive framework by linking observed changes to proposed mechanisms: UPFs' high palatability and nutrient composition may drive reward sensitivity, while structured dietary interventions could reduce compulsive eating behaviours. Together, these mechanisms could explain why UPF reduction predicts FA improvement.

In accordance with the findings of earlier analyses in this sample (Mallorquí-Bagué et al., 2021), the results indicated that higher baseline impulsivity contributed to higher FA levels at the end of the one-year follow-up. A distinct pattern has been previously observed in relation to FA and an inability to regulate behaviour, that correlates with overconsumption of food and lack of inhibitory control (Maxwell, Gardiner, & Loxton, 2020). Surprisingly, in our model, cognitive function appears to be unrelated to the consumption of UPFs or the FA scoring. The independence of baseline cognition and changes over time with UPFs and FA is somewhat unexpected, given that prior results have shown that baseline cognition influences outcomes in terms of both cognition and BMI after a period of follow-up (Soldevila-Domenech et al., 2021). There is a possibility that the reduced consumption of UPFs in this cohort hides the identification of a potential association, or it may suggest the presence of alternative mechanisms contributing to the association.

To the best of our knowledge, this is the first study to assess the association of UPFs and FA in an older adult Spanish sample. However, our results must be considered with caution, as they are subject to certain limitations. As is the case with any observational study, the possibility of residual or unmeasured confounding could not be eliminated. The study cohort, comprising a Mediterranean older adult population with metabolic syndrome, does not reflect the general population; consequently, the extrapolation of results is not possible. Furthermore, despite adjustments made in the analyses to mitigate the intervention's effects, the possibility of a residual effect remains. It should be noted, however, that both groups were advised to improve the

quality of their dietary patterns, which resulted in a decrease in the consumption of UPFs. In addition, the present sample demonstrated minimal clinical variability regarding FA, showing a prevalence number (5.6%) underneath the one reported for the general population (8%) (Hauck, Weiß, Schulte, Meule, & Ellrott, 2017). Future studies should target populations with higher FA scores. Moreover, despite the usefulness of FFQs in epidemiological studies for dietary assessment, as a subjective method, it is necessary to acknowledge the potential limitations in accurately estimating dietary information. It is important to note that there is significant variability in UPFs consumption reported on a global scale, not only due to geographical location, but also in relation to age, sex, BMI, and the presence of overweight or obesity (Marino et al., 2021). The mean consumption of UPFs in our study population is substantially lower than what has been reported worldwide, which may be attributable to factors such as older age, Spanish origin, and adherence to a Mediterranean diet. A further key limitation is the age of the dataset, which was collected prior to recent food environments and public awareness regarding UPFs. Nevertheless, evidence indicates that UPF consumption patterns have remained consistently high over the past decade, with recent studies confirming similar intake levels and associated health risks (Wang et al., 2025; Williams, Couch, Emmerich, & Ogburn, 2025). Likewise, neurocognitive mechanisms that underpin eating behaviours are considered to be relatively stable over time, and not substantially influenced by changes in food environments (Stover et al., 2023). Moreover, the dataset is both large and well-characterised, and includes validated measures, thus providing a robust foundation for examining long-term associations (Landberg et al., 2024). Further studies should aim to extend these findings to a younger sample that mirrors the general populations' UPF consumption patterns.

In summary, this study provides novel insights into the association between the consumption of UPFs and the presence of FA in an older Mediterranean population with overweight or obesity and metabolic syndrome. Our findings suggest that a reduction in the consumption of UPFs may contribute to the improvement of FA symptoms, independent of weight loss. Future research should focus on populations with higher UPF consumption, incorporating body composition measurements and investigating the long term-effects of dietary quality on FA symptoms. Considering the limited evidence on effective interventions for FA in individuals with obesity without eating disorders, these findings highlight the importance of integrating nutritional strategies, particularly those targeting UPFs, into prevention and treatment efforts for eating-related addictive behaviours.

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## Appendix

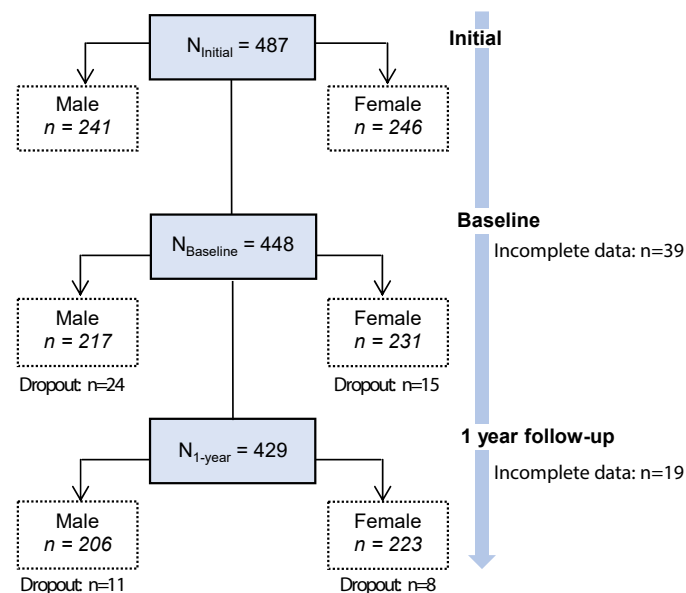


Fig. A1. Flowchart for the sampling

Table A1. Complete results obtained in the path analysis (results at baseline)

Structural		Coeff.	SE	T-stat	p	95%CI Coeff.	
UPF-baseline	Sex (female)	−0.1788	0.0464	−3.86	<.001	−0.2697	−0.0879
BMI-baseline	UPF-baseline	0.1213	0.0464	2.61	.009	0.0303	0.2123
	YFAS-baseline	0.2279	0.0455	5.00	<.001	0.1386	0.3171
YFAS-baseline	UPPS-P-baseline	0.3452	0.0409	8.43	<.001	0.2649	0.4254
	Sex (female)	0.1281	0.0444	2.89	.004	0.0411	0.2152
BDI-Baseline	YFAS-baseline	0.2349	0.0445	5.27	<.001	0.1476	0.3222
	UPPS-P-baseline	0.1898	0.0442	4.30	<.001	0.1033	0.2763
	Sex (female)	0.3069	0.0399	7.69	<.001	0.2286	0.3852

Note. Coeff: standardized coefficient. SE: standard error. T-stat: T-statistic. 95%CI: 95% confidence interval.

Table A2. Complete results obtained in the path analysis (SEM adjusted by treatment type)

Structural		Coeff.	SE	T-stat	p	95%CI Coeff.	
UPF-Baseline	Sex (female)	−0.1788	0.0464	−3.86	<.001	−0.2697	−0.0879
UPF-1year	UPF-baseline	0.4192	0.0387	10.83	<.001	0.3433	0.4950
YFAS-baseline	Sex (female)	0.1228	0.0474	2.59	.001	0.0299	0.2156
YFAS-1year	YFAS-baseline	0.3973	0.0423	9.39	<.001	0.3144	0.4802
	BMI-baseline	0.1076	0.0428	2.52	.012	0.0238	0.1914
	UPPS-P-baseline	0.1163	0.0448	2.60	.009	0.0285	0.2041
BMI-1year	YFAS-baseline	0.0522	0.0191	2.74	.006	0.0148	0.0895
	BMI-baseline	0.8751	0.0108	80.88	<.001	0.8539	0.8963
BDI-1year	YFAS-baseline	0.1542	0.0394	3.91	<.001	0.0769	0.2314
	BDI-baseline	0.5585	0.0337	16.56	<.001	0.4923	0.6246
BDI-Baseline	Sex (female)	0.3323	0.0417	7.96	<.001	0.2505	0.4141
UPPS-P-1year	UPPS-P-baseline	0.4932	0.0363	13.59	<.001	0.4220	0.5643
MoCA-1year	MoCA-baseline	0.5257	0.0324	16.21	<.001	0.4622	0.5892
<i>Covariances</i>		<i>Coeff.</i>	<i>SE</i>	<i>T-stat</i>	<i>p</i>	<i>95%CI Coeff.</i>	
UPF-baseline	BMI-baseline	0.1202	0.0467	2.57	.001	0.0287	0.2116
UPF-1year	BMI-1year	0.1487	0.0470	3.16	.002	0.0565	0.2409
YFAS-baseline	BMI-baseline	0.1981	0.0427	4.64	<.001	0.1145	0.2817
YFAS-baseline	BDI-baseline	0.3033	0.0432	7.01	<.001	0.2186	0.3881
YFAS-baseline	UPPS-P-baseline	0.3431	0.0418	8.20	<.001	0.2611	0.4250
YFAS-1year	BMI-1year	0.1278	0.0466	2.74	.006	0.0364	0.2191
YFAS-1year	UPPS-P-1year	0.1865	0.0463	4.02	<.001	0.0956	0.2773
BDI-baseline	UPPS-P-baseline	0.2872	0.0443	6.48	<.001	0.2003	0.3740

Note. Coeff: standardized coefficient. SE: standard error. T-stat: T-statistic. 95%CI: 95% confidence interval.

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