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# Binge eating among young adults: association with sociodemographic factors, nutritional intake, dietary *n*-6:*n*-3 ratio and impulsivity

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## Abstract

Binge eating behaviour (BE) is the major symptom of binge eating disorder (BED). This study aimed to compare the nutritional intake in the presence or absence of BE, with a particular focus on dietary *n*-6:*n*-3 ratio, to assess the association between BE and impulsivity and the mediating effect of BMI on this association. A total of 450 university students (age 18–28 years) participated. The self-administered questionnaires were a semi-quantitative FFQ and the UPPS-P Impulsive Behavior Scale and the binge eating scale. The average BE score was 11·6 (SE 7·388), and 20 % of the total participants scored above the cut-off of 17, thus presenting BE with 95 % CI of 16·3, 23·7 %. Our study revealed that greater BMI, higher total energy intake, greater negative urgency and positive urgency scores were significantly associated with BE. Participants with high value of dietary *n*-6:*n*-3 ratio were 1·335 more at risk to present a BE compared with those with a lower value of this ratio ($P = 0{\cdot}017$). The relationship between BE score and UPPS domains score was not mediated by the BMI. This is the first study reporting a link between high dietary *n*-6:*n*-3 ratio and BE as well as the fact that BE was linked to both, negative and positive urgencies, and that the association between BE and impulsivity was not mediated by BMI. These findings can help to deal more efficiently with people suffering from BE, a symptom that can precede the development of BED.

**Key words:** **Binge eating: Impulsivity: Dietary *n*-6:*n*-3 ratio: BMI: Nutritional intake: Young adults**

Binge eating disorder (BED) is the most common eating disorder (ED) and an important public health problem worldwide. It continues to be an under-recognised and undertreated condition. Patients rarely spontaneously disclose binge eating symptoms because of embarrassment or shame. Binge eating behaviour (BE) is known as a behavioural symptom of BED, and the difference between BE and BED is that the latter is recognised as a psychiatric disorder by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR and DSM-5)[1].

BE is characterised by episodes of eating food more than a person would typically eat in a discrete period. In BED, these episodes occur at least once a week over 3 months[2,3] and are accompanied by a sense of loss of control and marked distress, in the absence of regular compensatory behaviours for weight loss[2,4].

BE is a public concern with serious physical and mental health consequences[5]. It was found to be associated with depression, anxiety and substance abuse[6], as well as compulsive behaviours, such as gambling[7] and binge drinking[8].

The lifetime prevalence of BE is estimated to be 4·9 % in women and 4·0 % in men in community samples[9]. While some reports state that BED is more common in women than men[9,10], BE is reported to be comparable with similar rates of frequency and functional impairment among women and men[11–13].

**Abbreviations:** BE, binge eating behaviour; BED, binge eating disorder; ED, eating disorder; FA, fatty acid.

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The differences between BED and BE are related to the fact that many individuals who engage in BE may not meet the full criteria of BED. Obese individuals are at 2–3 times increased risk of having disordered eating, compared with normal-weight individuals[14]. However, BE can also occur in healthy non-obese individuals[2,15].

Impulsivity is a broad term referring to a disposition towards behaviours that are unduly hasty, risky, and that lead to undesirous outcomes[16]. Individuals with high impulsivity and reward sensitivity experience an addictive response to certain foods, particularly to high-sugar and high-fat foods[17]. One compelling premise places impulsivity at the roots of loss of control during eating episodes[18]. Higher trait impulsivity and poor motor inhibitory mechanisms have been highlighted in individuals suffering from emotional eating independently from their weight[19].

Cross-sectional studies have found significant associations between impulsivity and ED that involve purging behaviour[20,21]. Impulsivity has not, however, been examined extensively in association with binge eating without purging behaviour. One study reported a significant association between impulsivity and binge eating in a nationally representative sample of US adolescents[22]. Others have shown that BE occurs in response to experiencing negative emotions[23,24] and research suggests individuals with elevated impulsivity are more likely to binge eat because of their tendency to engage in reckless actions under distress[25,26]. Furthermore, negative urgency was significantly linked to binge eating in preadolescents[27,28].

However, according to some studies, obesity itself may be the underlying cause. Obese individuals show elevated impulsivity both on the Stop-Signal task and Barratt impulsiveness questionnaire, compared with normal-weight controls with similar levels of impulse control disorders[29]. Some studies suggest that cognitive deficits associated with BE are really primarily associated with obesity and that obese individuals with or without BE exhibit the same types and severity of cognitive deficits[30–32].

All ED are characterised by alterations in food choice, influencing directly the quantity and quality of nutrients intake[33]. The bulk of published data relies on food choices from laboratory test meals, in which the selection is restricted to foods decided on by the researchers and not by the patients[34]. Furthermore, nutritional intake studies focused more on anorexia nervosa or bulimia nervosa and very few on BE. It was reported that BE was associated with snacking and eating sweets[35] and with unbalanced diets[36], but researches are definitely needed on this subject. Among macro- and micronutrients, the role of PUFA in human health acquired growing interest in the last decades. The most important classes of PUFA are the *n*-3 fatty acids (FA), including $\alpha$-linolenic acid, EPA and DHA, and *n*-6 FA, including linoleic and arachidonic acids. The beneficial properties of *n*-3 FA on inflammatory, cardiovascular and the nervous system are recognised by several investigations[37–40]. In the brain, these agents modulate brain cell signalling, including dopaminergic and serotonergic pathways[41–43]. A well-balanced dietary *n*-6:*n*-3 ratio is fundamental for the development and functioning of the central nervous system[44]. In recent years, the effects of PUFA, with an emphasis on EPA and DHA, were investigated in several diseases such as psychosis, major depression, bipolar disorder, anxiety disorders, obsessive–compulsive disorder, posttraumatic disorder, ED, attention deficit hyperactivity disorder, autism spectrum disorders, substance abuse and borderline personality disorder[43,45–50]. Abnormal levels of *n*-3 and *n*-6 FA were observed in patients with anorexia nervosa[51,52], whereas in both BED and BE, there are no studies examining the level or intake of *n*-3 or *n*-6 FA from diet.

Hence, the objectives of this study were firstly to examine the sociodemographic variables associated with BE among young adults, secondly to compare the nutritional intake between BE individuals and those without BE, with a particular focus on dietary *n*-3 and *n*-6:*n*-3 ratio, and finally to assess the association between BE and impulsivity using the UPPS scale, as well as the mediating effect of BMI on this association.

## Materials and methods

### Ethical considerations

The protocol of the study was approved by the ethics committee of Saint-Joseph University of Beirut (reference USJ-2019-180). Informed written consent was acquired from all individuals prior to participating in the study.

### Survey procedure, sampling and data collection

This cross-sectional study was based on a survey conducted by four trained research assistants among the students of Saint-Joseph University, one of the largest universities in Lebanon, with students from all districts and regions. The questionnaires were presented to the participants in the same non-randomised order. Data collection started on January and was carried on until June 2019 (6 months). Inclusion criteria were: students above 18 years old and not suffering from any cognitive or chronic illnesses. From the 550 students randomly selected from each faculty of Beirut campus (the largest campus of the university), 450 agreed to participate. The sample size was calculated according to the formula that takes into consideration the number of independent variables to be included in the model: $n\ 50 + 8m$ ($m$ is the number of explanatory variables: sociodemographic characteristics, nutritional variables and UPPS score); given that $m = 16$, a minimum of 178 subjects has to be included in the study[53].

The face-to-face interview was divided into three steps: first, a research assistant explained the study and asked for a written informed consent. Then, the assistant collected sociodemographic information by asking direct questions to the participant. Finally, and since the last part of the questionnaire was self-administered, it was handed over to the participant to be filled without assistance. The self-administered questionnaires are internationally validated and reliable, namely the FFQ[54–56], the UPPS scale for impulsivity and the BE questionnaire. The time required for completion of the questionnaires was about 20-25 min.

### Participants and data collection

Sociodemographic variables of interest collected were age, sex, faculty, university, weight, height, living alone or not, tobacco

smoking, alcohol and caffeine consumption. BMI was calculated using the formula: weight (kg)/height$^2$ (m$^2$). It was then analysed in two different ways: as a continuous variable and also categorised according to the WHO cut-off points (underweight <18·5, normal 18·5–24·9, overweight 25–29·9 and obese >30 kg/m$^2$)[57]. The crowding index representing the number of people living in the same house divided by the number of rooms in the house (excluding the kitchen and bathrooms) was also calculated since it could affect sleep[58] and it reflects the socio-economic status of the participants.

### Study material

*Impulsivity.* The UPPS-P Impulsive Behavior Scale in its short version was used. It is a self-rated inventory with twenty items to measure five distinct personality pathways to impulsive behaviour[59,60]: negative urgency (four items), (lack of) perseverance (four items), (lack of) premeditation (four items), sensation seeking (four items) and positive urgency (four items). Items were rated on a four-point scale from Strongly Agree to Strongly Disagree. Average scores were calculated for each dimension. The UPPS has a good internal consistency as well as divergent and external validity. In this study, it showed a good Cronbach's $\alpha$ of 0·776.

*Binge eating behaviour.* The binge eating scale is a sixteen-item self-report questionnaire designed to capture the behavioural (eight items, e.g. large amount of food consumed), as well as the cognitive and emotional (eight items, e.g. feeling out of control while eating, preoccupation with food and eating), features of objective binge eating in overweight and obese adults[61]. For each item, respondents are asked to select one of three or four response options, coded zero to two or three, respectively. Individuals' scores are summed and range from 0 to 46, with higher scores indicating more severe binge eating problems. Based on the binge eating scale total score, which ranges from 0 to 46, participants were categorised into three groups according to established severity cut-offs, which are: none (score < 17), mild-moderate (score of 18–26) and severe (score > 27)[48]. The binge eating scale was used as a screening measure to classify those with scores ≥17 as 'binge-eaters'. Importantly, the binge eating scale was created before BED was officially recognised as a psychiatric diagnosis[1] and thus is not intended to detect the presence of this disorder. Rather, it has been suggested that this measure be used as a brief screening tool to identify the severity of BE in overweight and obese adults, to tailor obesity interventions and to track treatment outcomes[61,62]. In this study, it showed a very good Cronbach's $\alpha$ of 0·828.

*Nutrient intake calculation.* A semi-quantitative 150-item FFQ containing Middle Eastern foods and local meals, validated by our research team in a previous study, was administered to participants[54,55].

To help them quantify the exact amount and portion of foods consumed, standard measuring cups and spoons, plastic food models and local food photos in frequently consumed sizes were used during the interview. The FFQ was subdivided into twelve food groups with open-frequency categories, used in decreasing order: daily, weekly, monthly, all over the year and never. The weight in grams of each food was multiplied by its frequency of consumption and divided, for example, by 7, if it was consumed just once a week. Participants' responses were then converted into average daily intake, in grams. The Nutrilog software (version 2.30) was used to analyse the food records of the FFQ, using databases from US Department of Agriculture, and *n*-6 and *n*-3 FA content of specific Lebanese food was derived from the American University of Beirut database.

### Statistical analysis

The statistical analyses were carried out using SPSS software for Windows (version 24.0). The significance level was set at 0·05. The characteristics of the sample were described using the mean and standard deviation for continuous variables and percentage for categorical variables. The prevalence of BE was calculated with a 95 % CI. Kolmogorov–Smirnov tests were performed to assess the normality distribution of continuous variables. In the initial stages, the univariate analyses were carried out using the Student's *t* test. Pearson was also used to evaluate the association between continuous variables. $\chi^2$ independence tests and Fisher exact tests were performed to assess the relationship between categorical variables. Logistic regression analysis was used with categorised BE as the dependent variable. Independent variables that showed associations with a *P* value <0·200 in univariate analyses were candidates for the multivariate model, according to the Enter method. Collinearity among independent variables was also tested, and variables highly correlated were excluded from the model; it has already been suggested not to include two independent variables where there is a correlation of 0·7 or more[53]. The explanatory variables included in the model were smoking, BMI, energy, MUFA as percentage of total fat, *n*-3, *n*-6:*n*-3 ratio, negative urgency, lack of premeditation and positive urgency.

The *n*-6:*n*-3 ratio was categorised into P66·6 (low, moderate and high) in order to further examine the association with BE. The cut-off values for *n*-6:*n*-3 ratio were chosen using the 66th percentiles; thus, the values >66·6th indicate a greater ratio. Logistic regression analysis was performed, and adjusted OR were obtained; these OR quantify better the strength of the associations between *n*-6:*n*-3 ratio and BE.

Mediation with regression analysis of BMI (as mediators M) on the relationship between impulsivity domain as the independent variable and BE as the dependent variable was conducted using a four-step approach, in which regression analyses and significance of the coefficients were examined at each step.

## Results

Out of 550 students approached, 450 (81·8 %) agreed to participate. Sociodemographic characteristics of the participants are summarised in Table 1. Of the 450 participants included in the study, 54·9 % were women. Participants' age varied between 18 and 28 years old. Mean BMI was 22·4 (SD 3·4) kg/m$^2$, and 74·4 % of participants had a normal body weight.

**Table 1.** Participants' sociodemographic factors
(Mean values and standard deviations; numbers and percentages)

| | Total sample (*n* 450) | | | |
|---|---|---|---|---|
| | Mean | SD | *n* | % |
| Age (years) | 21·3 | 1·9 | | |
| Crowding index | 0·97 | 0·4 | | |
| BMI (kg/m$^2$) | 22·4 | 3·4 | | |
| BMI | | | | |
| Underweight | | | 39 | 8·7 |
| Normal weight | | | 335 | 74·4 |
| Overweight | | | 63 | 14·0 |
| Obese | | | 13 | 2·9 |
| Smoking status | | | | |
| Non-smoker | | | 365 | 81·1 |
| Smoker | | | 85 | 18·9 |
| Alcohol intake | | | | |
| No | | | 121 | 26·9 |
| Occasional | | | 241 | 53·6 |
| Up to 1 drink/week | | | 52 | 11·6 |
| More than 1 drink/week | | | 36 | 8·0 |

**Table 2.** Participants' nutritional intake
(Mean values and standard deviations; numbers and percentages, *n* 450)

| | Mean | SD |
|---|---|---|
| Total energy (kJ) | 10 417 | 4263 |
| Proteins (%) | 17·1 | 57·8 |
| Fat (%) | 38·6 | 14·5 |
| Carbohydrates (%) | 49·0 | 26·3 |
| Sugar (g) | 91·7 | 47·4 |
| SFA (% of total fat) | 20·5 | 14·6 |
| MUFA (% of total fat) | 26·5 | 14·5 |
| PUFA (% of total fat) | 11·8 | 6·8 |
| Fibre (g) | 19·1 | 8·7 |
| Cholesterol (mg) | 240·3 | 182·8 |
| *n*-6 (g) | 1·5 | 1·7 |
| *n*-6 (g/1000 kcal (4184 kJ)) | 5·4 | 5·9 |
| *n*-3 (g) | 0·2 | 0·3 |
| *n*-3 (g/1000 kcal (4184 kJ)) | 4·4 | 3·1 |
| *n*-6:*n*-3 ratio | 9·8 | 7·1 |

Table 2 presents the nutritional intake of the participants as minimum, maximum, mean and standard deviation. Total energy was presented in kJ, macronutrients in percentage of total energy intake while sugar, *n*-3, *n*-6 in g and cholesterol in mg. In addition, *n*-3 and *n*-6 were presented as intake in g per 1000 total kcal (4184 total kJ). The ratio of *n*-6:*n*-3 was also provided as well as the distribution of participants above or below the tertile value for this ratio.

UPPS-P and BE scores are presented in Table 3. The average BE score was 11·6 (SE 7·4), and 20 % of the total participants scored above the cut-off of 17 on the BE questionnaire, thus presenting BE with a 95 % CI of 16·3, 23·7.

### *Univariate analysis*

Table 4 presents the associations between categorised BE score and quantitative sociodemographic and nutritional variables, while Table 5 shows the associations between BE and UPPS-P domains.

**Table 3.** UPPS-P and binge eating behaviour (BE) scores of the participants
(Mean values and standard deviations; numbers and percentages, *n* 450)

| | Mean | SD | *n* | % |
|---|---|---|---|---|
| UPPS-P total | 42·6 | 7·3 | | |
| Negative urgency | 9·2 | 2·6 | | |
| Lack of perseverance | 7·2 | 2·5 | | |
| Lack of premeditation | 7·2 | 2·2 | | |
| Sensation seeking | 10·9 | 2·8 | | |
| Positive urgency | 8·3 | 2·9 | | |
| BE score | 11·6 | 7·4 | | |
| BE score categorised: | | | | |
| None to minimal BE 0–17 | | | 360 | 80·0 |
| BE ≥ 18 | | | 90 | 20·0 |
| Of which: | | | | |
| Moderate 18–26 | | | 72 | 16·0 |
| Severe >26 | | | 18 | 4·0 |

### *Multivariate analysis*

Independent variables highly correlated were not included in the same multivariate model (total UPPS and its domains), (Energy and Sugar), (*n*-6:*n*-3 and *n*-6 per 1000 kcal (4184 kJ)) (Table 6).

Participants with greater BMI and higher total energy intake were more prone at risk to develop BE compared with those with lower BMI ($P = 0·002$ and 0·049, respectively).

Participants with greater negative ($P = 0·011$) and positive urgency score ($P = 0·028$) were more at risk to have BE compared with those with a lower score.

Participants with high value of dietary *n*-6:*n*-3 ratio were 1·335 more at risk to present BE compared with those with lower value of this ratio ($P = 0·017$).

The mediating effect of BMI (Mediator M) on the relationship between BE (dependent variable) and total UPPS score (independent variable) was tested with a four-step analysis (Table 7). The results showed that the relationship between BE score and UPPS domains score was not mediated by the BMI.

## Discussion

Epidemiological BED research remains limited particularly across the Arab world, where overweight and obesity are primary public health issues[63,64].

Our results showed that BE occurrence among participants was 20 % and 4 % of those presented a severe form. BE reported frequency in the literature for the Western population is 1 % to 4·6 % with different tools and questionnaires[9]. Those frequencies seem higher in the Arab world, with 14·4 % previously reported in Lebanon[65], moderate to severe binge eating reported by one-third of a sample of youths in the United Arab Emirates[66] and up to 68·8 % in Saudi Arabia[67].

The majority of research conducted on ED has focused on the fact that body image concerns underlie the disorder. Furthermore, the consideration of internalised body image ideals is of particular relevance to populations from non-Western nations[68,69]. Sociocultural model of disordered eating suggests that the pressure to achieve Western ideals of thinness may engender body image concerns and disordered eating. This

**Table 4.** Associations between categorised binge eating behaviour (BE) score and quantitative sociodemographic and nutritional variables (Mean values and standard deviations; numbers and percentages, *n* 450)

| | BE* | *n* | Mean | SD |
|---|---|---|---|---|
| Age (years) | 0–17 | 360 | 21·2 | 1·9 |
| | ≥18 | 90 | 21·5 | 2·0 |
| | *P* | | 0·270 | |
| Crowding index | 0–17 | 360 | 0·98 | 0·4 |
| | ≥18 | 90 | 0·92 | 0·4 |
| | *P* | | 0·203 | |
| BMI (kg/m$^2$) | 0–17 | 360 | 22·0 | 3·1 |
| | ≥18 | 90 | 23·7 | 4·1 |
| | *P* | | 0·000† | |
| Energy (kJ) | 0–17 | 360 | 10 136·2 | 3952·6 |
| | ≥18 | 90 | 11 342·8 | 5174·8 |
| | *P* | | 0·016† | |
| Proteins (%) | 0–17 | 360 | 18·0 | 64·6 |
| | ≥18 | 90 | 13·7 | 2·6 |
| | *P* | | 0·534 | |
| Fat (%) | 0–17 | 360 | 38·9 | 16·0 |
| | ≥18 | 90 | 37·5 | 5·1 |
| | *P* | | 0·414 | |
| Carbohydrates (%) | 0–17 | 360 | 49·2 | 29·3 |
| | ≥18 | 90 | 48·430 | 5·9 |
| | *P* | | 0·807 | |
| Sugar (g) | 0–17 | 360 | 89·5 | 45·1 |
| | ≥18 | 90 | 100·4 | 55·3 |
| | *P* | | 0·051 | |
| SFA (% of total fat) | 0–17 | 359 | 20·9 | 15·5 |
| | ≥18 | 90 | 18·9 | 10·3 |
| | *P* | | 0·251 | |
| MUFA (% of total fat) | 0–17 | 360 | 26·9 | 14·9 |
| | ≥18 | 90 | 24·5 | 12·4 |
| | *P* | | 0·150 | |
| PUFA (% of total fat) | 0–17 | 360 | 12·0 | 6·8 |
| | ≥18 | 90 | 11·2 | 7·1 |
| | *P* | | 0·325 | |
| Fibre (g) | 0–17 | 360 | 18·944 | 8·7 |
| | ≥18 | 90 | 19·619 | 8·4 |
| | *P* | | 0·509 | |
| Cholesterol (mg) | 0–17 | 360 | 235·9 | 170·4 |
| | ≥18 | 90 | 257·8 | 226·1 |
| | *P* | | 0·310 | |
| *n*-6 (g) | 0–17 | 360 | 1·5 | 1·8 |
| | ≥18 | 90 | 1·4 | 1·4 |
| | *P* | | 0·606 | |
| *n*-6 (g/1000 kcal (4184 kJ)) | 0–17 | 360 | 5·6 | 6·2 |
| | ≥18 | 90 | 4·7 | 4·1 |
| | *P* | | 0·172 | |
| *n*-3 (g) | 0–17 | 360 | 0·2 | 0·3 |
| | ≥18 | 90 | 0·2 | 0·4 |
| | *P* | | 0·968 | |
| *n*-3 (g/1000 kcal (4184 kJ)) | 0–17 | 360 | 4·5 | 3·4 |
| | ≥18 | 90 | 3·9 | 1·5 |
| | *P* | | 0·020† | |

| | | *n*-6:*n*-3 ratio P66·6 | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | <12·4 | | 12·4+ | | Total | | |
| | BE categories* | *n* | % | *n* | % | *n* | % | *P* |
| Total sample | 0–17 | 247 | 69·8 | 107 | 30·2 | 354 | 100·0 | 0·015† |
| | ≥18 | 50 | 56·2 | 39 | 43·8 | 89 | 100·0 | |
| | Total | 297 | 67·0 | 146 | 33·0 | 443 | 100·0 | |

* BE score dichotomised: None to minimal (0–17), presence of BE (≥18).
† *P* values are significant.

could be one explanation of the high prevalence of BED observed in Lebanon.

Furthermore, even though most of the studies show that BE is more frequent in women[10], some recent studies have focused on an increase in ED amongst men, maybe due to social pressures and media influences regarding masculine and strong males' ideal body and serve binging as a method for achieving this goal[70]. In a recent study among Iranian college students, BE


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**Table 5.** Association between categorised binge eating behaviour (BE) scores and UPPS-P domains (Mean values and standard deviations; numbers and percentages)

| | Total sample (*n* 450) | | | |
|---|---|---|---|---|
| | BE* | *n* | Mean | SD |
| UPPS total | 0–17 | 360 | 42·0 | 7·1 |
| | ≥18 | 90 | 45·0 | 7·8 |
| | *P* | | 0·001† | |
| Negative urgency | 0–17 | 360 | 8·9 | 2·5 |
| | ≥18 | 90 | 10·1 | 2·7 |
| | *P* | | 0·000† | |
| Lack of perseverance | 0–17 | 360 | 7·1 | 2·4 |
| | ≥18 | 90 | 7·4 | 2·9 |
| | *P* | | 0·277 | |
| Lack of premeditation | 0–17 | 360 | 7·1 | 2·2 |
| | ≥18 | 90 | 7·5 | 2·3 |
| | *P* | | 0·075 | |
| Sensation seeking | 0–17 | 360 | 10·8 | 2·9 |
| | ≥18 | 90 | 10·9 | 2·7 |
| | *P* | | 0·862 | |
| Positive urgency | 0–17 | 360 | 8·1 | 2·9 |
| | ≥18 | 90 | 9·1 | 2·7 |
| | *P* | | 0·004† | |

* BE score dichotomised: None to minimal (0–17), presence of BE (≥18).
† *P* values are significant.

**Table 6.** Logistic regression of explanatory variables associated with categorised binge eating (*B*-coefficients with their standard errors; odds ratios and 95 % confidence intervals)

| | *B* | SE | df | Significance | OR | 95 % CI |
|---|---|---|---|---|---|---|
| Smoking | 0·281 | 0·311 | 1 | 0·366 | 1·324 | 0·720, 2·434 |
| BMI (kg/m$^2$)† | 0·114 | 0·036 | 1 | 0·002 | 1·120 | 1·044, 1·203 |
| Energy (kJ) | 0·000 | 0·000 | 1 | 0·466 | 1·000 | 1·000, 1·000 |
| MUFA (% of total fat) | −0·017 | 0·010 | 1 | 0·106 | 0·983 | 0·964, 1·004 |
| *n*-3 (g/1000 kcal (4184 kJ)) | −0·128 | 0·111 | 1 | 0·248 | 0·880 | 0·708, 1·093 |
| *n*-6:*n*-3 ratio P66·6*† | 0·289 | 0·130 | 1 | 0·027 | 1·335 | 1·034, 1·724 |
| Negative urgency† | 0·153 | 0·050 | 1 | 0·002 | 1·166 | 1·056, 1·287 |
| Lack of premeditation | 0·055 | 0·058 | 1 | 0·338 | 1·057 | 0·944, 1·183 |
| Positive urgency† | 0·090 | 0·045 | 1 | 0·046 | 1·095 | 1·001, 1·197 |

* P66.6: tertile value for this ratio, with distribution of participants above or below this tertile value.
† Significant.

**Table 7.** Four-step analysis of the mediating effect of BMI (mediator M) on the relationship between binge eating behaviour (BE) (dependent variable) and UPPS score (independent variable)*
(*B*-coefficients with their standard errors; odds ratios and 95 % confidence intervals)

| | | Unstandardised coefficients | | Standardised coefficients | | | |
|---|---|---|---|---|---|---|---|
| Four-step analysis | | *B* | SE | *β* | *t* | Significance† | 95 % CI for *B* |
| Step 1‡ Dependent variable: BE | UPPS score | 0·208 | 0·047 | 0·206 | 4·461 | 0·000 | 0·116, 0·299 |
| Step 2§ Dependent variable: BMI | UPPS score | 0·049 | 0·022 | 0·106 | 2·259 | 0·024 | 0·006, 0·092 |
| Step 3‖ Dependent variable: BE | BMI | 0·646 | 0·098 | 0·298 | 6·608 | 0·000 | 0·454, 0·838 |
| Step 4¶ | BMI | 0·605 | 0·097 | 0·279 | 6·257 | 0·000 | 0·415, 0·795 |
| Dependent variable: BE | UPPS | 0·178 | 0·045 | 0·177 | 3·957 | 0·000 | 0·089, 0·266 |

* Since, there were significant relationships from steps 1 through 3, the mediation was possible, and we proceed to step 4. At step 4, UPPS score was significant when BMI is controlled and this finding did not support mediation.
† *P* values are significant.
‡ Step 1: Single regression analysis with UPPS score predicting BE.
§ Step 2: Single regression analysis with UPPS score predicting BMI.
‖ Step 3: Single regression analysis with BMI predicting BE.
¶ Step 4: Multiple regression analysis with BMI and UPPS score predicting BE.

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occurrence was not different between males and females[71]. Our results (multivariate analysis) showed no statistical difference in BE occurrence between males and females, and this seems to highlight the fact that neither BE prevalence nor sex differences are similar between Western and Middle-eastern population owing to probable socio-cultural disparities.

Greater BMI and higher total energy intake were associated with BE in our study. Weight fluctuation is a common phenomenon in all subjects suffering from ED; however, because binge eaters do not engage in inappropriate compensatory behaviours such as purging, they seldom have a low body weight and mostly they tend to be of normal or higher than average weight. Besides, their disrupted abnormal relationship with food creates specific food rituals, mostly directed towards high energetic preferences. In an experiment conducted by Dalton *et al.*[72], among normal/lean and overweight/obese binge type and normal individual, those having higher BMI had a tendency to consume more energy content, with a net preference towards sugary and fatty food choices.

Long-chain PUFA have important physiological functions and play important structural and functional roles in the human brain and affect monoaminergic neurotransmission, dendritic arborisation, synapse formation and ion channel function[73,74]. *n*-3 PUFA have been shown to possess anti-inflammatory and antioxidant[75] properties, while *n*-6 or *n*-6 PUFA are generally seen as pro-inflammatory, and a high *n*-6:*n*-3 ratio is thought to have adverse health effects[47,76].

As a result of the worldwide increased consumption of ready-made and processed foods during the last decades, the dietary *n*-6:*n*-3 ratio is constantly growing[77,78].

Our results showed a dietary ratio of *n*-6:*n*-3 ratio close to 10:1, which is lower than contemporary Western diets, characterised by a ratio of about 15:1, reflecting deficient intake of *n*-3 FA and excessive intake of *n*-6 FA[76]. Published national Lebanese data had shown a net decline in fish consumption due to its high cost[79]. In contrast, eating French fries, oil-fried chicken and meat is frequent because it is easier to cook and cheaper to prepare or buy than sophisticated meals, especially among our sample of university students. However, the ratio observed in our study is still lower than typical western diets, probably because Lebanese diet retains some characteristics of the Mediterranean diet, *n*-3 dietary sources, such as fish, walnuts and purslane.

While no differences were observed in the dietary intake of *n*-3 and *n*-6 between participants with or without BE, the ratio of dietary *n*-6:*n*-3 was significantly different and this is reported for the first time: participants with high value of dietary *n*-6:*n*-3 ratio were 1·335 more at risk to present BE compared with those with a lower value of this ratio.

It has been suggested that the ratio between *n*-6 and *n*-3 intakes might be a more important indicator of status than the absolute intake of either because it reflects the mutual competition of the two PUFA types[80]. For example, even when *n*-3 intake is high, this might be counteracted by an even higher intake of *n*-6[81]. Furthermore and even though it was speculated by some authors that *n*-6-docosapentaenoic acid is a buffer to prevent the possible catastrophic effects of DHA (*n*-3) depletion on brain and visual function[82], there is a consensus that an increased ratio of *n*-6:*n*-3 PUFA in the diet is an important risk factor of major chronic disorders[83]. In a cross-sectional study investigating the association between cognitive decline and dietary intake of PUFA[84], it was shown that the *n*-6:*n*-3 ratio was associated with cognitive decline. Another study also reported an association between dementia or cognitive decline and the ratio of *n*-6:*n*-3 FA[85]. In these studies, the dietary ratio of *n*-6:*n*-3 FA was not explicitly delineated. Vercambre *et al.* stated a mean ratio of *n*-6:*n*-3 FA of 9·4 and a positive association between increasing dietary levels *n*-6:*n*-3 and cognitive decline[86]. In animal models, it was found that mice fed with a diet presenting *n*-6:*n*-3 ratio of 2·5 performed better in spatial learning and memory than animals fed with a diet presenting a *n*-6:*n*-3 ratio of 7·5[87,88]. As to the relation between dietary intakes of PUFA and brain concentrations, transgenic Alzheimer disease-mouse models that were fed with a diet presenting a 2·8 ratio of *n*-6:*n*-3, showed a lower brain concentration of arachidonic acid and a lower DHA:arachidonic acid ratio than mice fed with a *n*-6:*n*-3 dietary ratio of 10·4[89] and diets containing a low *n*-6:*n*-3 ratio resulted in relatively low ratios in total brain homogenates[90]. These studies seem to indicate a direct relationship between dietary ratio of *n*-6:*n*-3 PUFA and brain fat composition.

Apart from memory and learning, an association between high dietary *n*-6:*n*-3 ratio and psychiatric illnesses was reported: the increased intake of *n*-6 essential FA and the reduced consumption of foods containing *n*-3 FA have been hypothesised to correlate with depression[76]. In anorexia nervosa, multiple studies have consistently demonstrated very profound distortions in serum and dietary PUFA profiles, compared with normal[91,92]. As for BE, this is the first report of a link between high dietary *n*-6:*n*-3 ratio and BE occurrence. This important finding needs to be consolidated in future larger studies.

Finally, total impulsivity score and urgency domains were significantly associated with high BE scores in our study, and this association between BE and impulsivity was not mediated by BMI, as previously mentioned in some articles. Research suggests individuals with elevated impulsivity are more likely to binge eat[25,26]. One study reported a significant association between impulsivity and binge eating in a nationally representative sample of US adolescents[22]. Others have shown that BE occurs in response to experiencing negative emotions[23,24]. Furthermore, negative urgency was significantly linked to binge eating in preadolescents[27,28]. Heightened negative urgency, or the tendency to behave impulsively when experiencing (and/or attempting to avoid) negative emotions, appears to characterise both problem eating and drinking behaviours[25,60,93–97]. In our study, both negative and positive urgencies were higher among participants with greatest BE scores. This is the first report on the impact of positive urgency on BE showing that BE was not only linked to negative but also to positive urgency (impulsive behaviour when experiencing positive emotions). This finding, if confirmed in future investigations, will help professionals understand the full panel of emotions, negative and positive, that provoke binge eating episodes and deal more efficiently with this behaviour that was classically considered until now as a way of coping or avoiding negative emotions.

Several limitations should be considered. This was a young sample, but BE is also present in older age groups. The study

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was neither designed nor powered to examine the influence of co-morbidities on BE, an issue that warrants further study in future. For example, attention deficit hyperactivity disorder is common in BE and has interesting overlap neuro-biologically (including in terms of pharmacotherapy)[98]. FFQ is a limitation in gathering data on foods consumed. An additional limitation is that the blood measures of *n*-6 and *n*-3 FA are the gold standard for estimating intakes compared with estimations based on FFQ's and the correlation between *n*-6 and 3 FA in foods shows weak relationships with blood levels, especially *n*-3 levels. This is another limitation which must be acknowledged. Finally, the current findings may not generalise to clinical settings, since the study recruited from the university. Notwithstanding these limitations, several findings in this study are of utmost importance: this is the first study reporting a link between high dietary *n*-6:*n*-3 ratio and BE occurrence as well as the fact that BE was not only linked to negative but also to positive urgency and that the association between BE and impulsivity was not mediated by BMI. These important findings, consolidated in future larger studies, can help professionals understand the importance of a balanced dietary *n*-6:*n*-3 ratio as well as the full panel of emotions, negative and positive, that provoke binge eating episodes and deal more efficiently with this problem.

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All data are made available by authors upon request.

All authors approved the manuscript and approved submission for publication.

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