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PhD Dissertation

Effects of sleep deprivation on eating behaviour in a sample of individual reporting binge eating symptoms: a quasi-experimental study

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Part 2: An empirical contribution to the study of the relationship between sleep deprivation and eating behaviour in a sample of individual reporting binge eating symptoms

Introduction

Epidemiological studies reported an association between poor sleep and increased food intake and obesity at different ages. Emerging evidence (both cross-sectional and longitudinal) have shown that sleep quantity/quality and eating behaviour are strictly linked, and different underlying mechanisms may mediate or moderate this relationship (e.g. cognitive and emotional mechanisms). Moreover, experimental studies found that partial and acute sleep deprivation may increase food intake and may impair executive and emotional functioning.

The quasi-experimental study presented in this PhD dissertation aimed to evaluate the effect of a night of partial sleep deprivation (5 hours of sleep allowed) on food intake, executive functions and food craving compared to a night of habitual sleep. The potential mediating effect of executive functions (backward inhibition) and food craving (lack of control) and the potential moderating effect of emotional eating and depression were also taken into account.

The sample consisted of participants reporting binge eating symptoms (Binge Eating Group) and self-defined healthy control participants (Control Group). After an initial screening, both groups were invited to the laboratory after a night of habitual sleep and after a night of partial sleep deprivation, in a counterbalanced order. At their arrival in the lab, after both nights, they completed different computer tasks to assess executive functions and craving, valence and arousal for high palatable food images. They were then invited to have breakfast in the lab, in which food intake was unobtrusively measured. Participants were also asked to monitor their sleep through an electronic portable device and sleep diaries. Food intake over the course of the day was also assessed using food diaries. This complex study provides novel empirical evidence on the relationship between poor sleep and eating behaviour and the underlying mechanisms that could mediate or moderate this relationship. Indeed, it took into account many aspects of eating related behaviours, emotions and cognitions.

The present dissertation includes two parts. The first part presents the theoretical background on which is based the empirical contribution presented in the second part. The literature addressed in the first part aims to support the choice of the variables (independent, dependent, mediators and moderators) that were considered in the study and the hypotheses advanced. First, it presents the state of art on the link between poor sleep and obesity at different ages; then, the link between poor sleep and diet pattern and the results of experimental studies on the effect of sleep deprivation on food intake are discussed. Furthermore, it discusses the role of potential factors influencing this relationship such as biological (e.g. appetitive hormone disruption), cognitive (e.g. impaired executive functions), and emotional processes (e.g. emotional eating, negative affectivity). The first part also addresses the relation between poor sleep and eating disorders in order to explain why a sample of people reporting binge eating was selected for studying the link between poor sleep and eating behavior.

The second part presents a study conducted for testing different hypotheses related to the relationship between sleep and eating behavior. Although the study is one, for sake of clarity it is split into different chapters according to the specific hypotheses tested and the specific outcomes addressed. The first chapter presents the design and the general aims of the study, explains the methodology applied (self-report instruments and objective measures employed in the selection, screening phase and sleep assessment) and describes the procedure of the study and the statistical analyses plan.. It also includes the descriptive results on groups characteristics and results of the sleep manipulation check, as well as a brief discussion of these results. All the following chapters have a similar organization: they briefly summarize the relevant theoretical background on which the specific hypotheses tested were based, then they describe the specific methodology and instruments employed for measuring the variables and the relationships tested, the statistical analyses, the results and the discussion. Thus, the second chapter focuses on food intake taking into account the potential moderating role of emotional eating; the third discusses the results on

executive functions and its potential mediating role in affecting food intake. The fourth focuses on food craving and its potential role as mediator in increasing food intake. Finally, taking together the results presented in the empirical chapters, a general discussion and a conclusion are formulated, highlighting the clinical implication and the possible future directions.

Part 1: Theoretical background

1 Poor sleep and eating behavior: state of art

1.1 Poor sleep and obesity: epidemiological evidence

In recent years, obesity has become a global public health challenge (Swinburn et al., 2011). The worldwide prevalence of overweight and obesity combined has risen by 27.5% for adults and 47.1% for children between 1980 and 2013, with 2.1 billion with a BMI $\geq$ 25 Kg/h^2 (Ng et al., 2014). Obesity has been associated with higher cancer risk (Bhaskaran et al., 2014), cardiovascular diseases and mortality risk (World Health Organization, WHO, 2015).

The research interest in the sleep/obesity pathway has begun since the early 2000s and it continues to increase rapidly. Looking for modifiable factors associated with obesity that may be targeted in preventive interventions, sleep has been considered as a potential risk factor (Lombardo, 2016) since a parallel decline of sleep duration over the past century has been evidenced at least in children and adolescents (Matricciani et al. 2012). Currently, the National Sleep Foundation guidelines recommend 7–9 h of sleep per night for adults aged 18–64 years, and states that 6 h may have a negative impact on health and well-being. Poor sleep quality and quantity (e.g. persistent insomnia) has been associated with higher mortality risk (Parthasarathy et al., 2014) and cardiovascular disease (Sofi et al. 2014). Moreover, both obesity and poor sleep are often associated with increased risk of mental disorders, especially depression (Baglioni et al., 2011: de Wit et al., 2010).

Several reviews of the literature showed that poor sleep quantity/quality has been associated with obesity in children and adolescents (Liu et al. 2012; Magee et al., 2012), in adults (Patel et al., 2008) and in older adults (Norton et al., 2017). However, most of the epidemiological evidence are cross-sectional and suggest that this relationship is stronger in children and younger adults, while

longitudinal evidence are still lacking and controversial, particularly those including adults (Theorell-Haglöw & Lindberg, 2016).

In the next section, cross-sectional and longitudinal studies, as well as systematic reviews and meta-analyses that support (or not) the association between poor sleep and obesity will be discussed. Although cross-sectional associations do not provide causal relationship of sleep problems increasing risk for obesity, the majority of the empirical evidence on this relationship derived from cross-sectional designs. Few longitudinal studies that provide somewhat stronger causal evidence will be also discussed. Furthermore, most of the studies used subjective measures of sleep and nutritional status (e.g. self-report sleep duration and self-report weight and height), therefore the few studies using objective measures of sleep and obesity will receive particular attention.

1.1.1 Findings in children and adolescents

A systematic literature review by Patel et al. (2008) reported a strong and consistent association of short sleep duration with current and future obesity in children. Results of most of the examined studies (31 cross-sectional and 5 prospective) supported an independent association between short sleep duration and increased weight and this relationship appeared to wane with age. Consistently, another systematic literature review by Liu et al. (2012) of 25 cross-sectional and longitudinal studies including children and adolescents indicated a significant associations between short sleep duration and childhood overweight/obesity. Authors stated that children sleep duration might be a modifiable risk factor in preventing obesity. However, other systematic reviews (e.g. Guidolin & Gradisar, 2012) suggested to use caution in drawing direct link between shortened sleep duration and overweight/obesity in adolescence. In this literature examination, only cross-sectional studies found this association while prospective studies did not. According to the authors, the heterogeneity in study findings did not permit to establish temporal association and it remains inconclusive as to whether shortened sleep is a risk factor for overweight or obesity during adolescence.

Most of the cross-sectional pediatric studies usually find a negative linear association between habitual sleep duration and obesity or body weight (Marshall et al., 2008). A study by Garaulet et al. (2011) that examined 3311 adolescents, aged 12.5-17.49 years from 10 European cities, found that shorter sleepers showed higher values of BMI, body fat, waist and hip circumferences and fat mass index ($p<0.05$), particularly females. Similarly, another study by Lee and Park (2014) on 1187 adolescents aged 12-18 years showed that the probability of being overweight was increased in subjects who slept ≤ 5 h (OR: 2.04; 95% CI: 1.17- 3.5). Additionally, authors found that short sleep duration was associated with elevated blood pressure and long sleep duration was associated with hypertriglyceridemia, both linked with obesity risk.

Moreover, as noted by Marshall et al. (2008) also longitudinal studies showed a consistent negative linear association between baseline habitual sleep duration and later obesity. It should be noticed, however, the paucity of the number of observational studies examined (n=4). To date, longitudinal evidence showing an association between short sleep duration and subsequent weight gain are stronger in children compared to adults, as demonstrated by a systematic review of 20 longitudinal studies by Magee et al. (2012). This finding is also supported by a recent systematic review and meta-analysis by Fatima et al. (2015) examining 11 longitudinal studies (24,821 participants), that revealed that sleep duration was inversely associated with later BMI in children and adolescents. Subjects sleeping for short duration had twice the risk of being overweight/obese, compared with subjects sleeping for long duration. The results of this meta-analysis are consistent with those of previous findings and showed even stronger associations (OR: 2.15; 95% CI: 1.64–2.81) compared to those found by Chen et al. (OR: 1.58, 95% CI: 1.26–1.98), and Cappuccio et al. (OR: 1.89, 95% CI: 1.46–2.43) in 2008. Despite most of the studies reported a negative linear association between sleep duration and risk of obesity, other authors (Wang et al., 2016) found a U-shaped curvilinear association in both cross-sectional and longitudinal analyses on 48,922 3-year old children followed-up for 5 years. This finding indicated that both short and long sleep duration may be associated with higher risk of overweight/obesity in preschool-aged children.

In summary, all these findings support the need to consider sleep as a relevant issue in preventing obesity and overweight during childhood and adolescence although there is a need of increasing knowledge about the effects of sleep durations longer then recommended.

Studies using objective measures of sleep and/or obesity indicators.

A study by Liu et al. (2008) objectively measured sleep duration on 335 children and adolescents using 3 consecutive nights of polysomnographic measurements. Authors found that even after adjusting for potential confounding variables 1 hour less of total sleep was associated with approximately 2-fold increased odds of overweight (OR = 1.85), 1 hour less of REM sleep was associated with about 3-fold increased odds (OR= 2.91), and REM density and activity below the median increased the odds of overweight by 2-fold (OR = 2.18) and 3-fold (OR = 3.32) respectively.

Another thorough prospective study published in 2011 on Sleep by Silva and colleagues, objectively and longitudinally investigated the association between short sleep and BMI on 304 children aged 6-12 years, who underwent an unattended home polysomnograms (PSGs) and anthropometric measurements. Their results evidenced that children sleeping ≤ 7.5 hours at baseline had higher odds of being obese at follow-up (OR = 3.3, $p < 0.05$) than children who slept ≥ 9 hours. Moreover, they found a mean increase in BMI of 1.7 kg/m2 ($p = 0.01$) over the 5 years of follow-up for children who slept ≤ 7.5 hours compared to those who slept ≥ 9 hours. Another study by Martoni et al. (2016) using objectively measures of BMI and sleep (actigraphy) investigated the sleep patterns and food intake of 115 Italian primary school children. Authors found that groups based on BMI differed for sleep quantity (e.g. midpoint of sleep and amplitude), while they were similar for sleep quality and food intake patterns. Moreover, this study found that BMI was negatively predicted by sleep duration on schooldays.

Finally, another interesting study by Jarrin and colleagues (2013) aimed to assess whether anthropometrically measured obesity (waist and hip circumference, body mass index Z-score, and

percent body fat) were influenced by self/parent-reported sleep disturbances and sleep patterns, beyond sleep duration. This type of study is helpful to extend the understanding of the relationship between sleep and obesity due to the distinct physiological underlying mechanisms. After adjusting for many potential confounding variables and sleep duration, authors found that sleep disturbances were significantly associated with adiposity and body composition indices as well as sleep patterns of later bedtimes and wake-times during the weekdays and later weekend bedtimes were significantly associated with adiposity and body composition measures.

These findings provides strong evidence in support of the association of poor sleep (quantity and quality) with increased risk of obesity, since the greater internal validity and given the validity of objective measures employed.

1.1.2 Findings in adults

While epidemiological findings supports the effect of short sleep duration in increasing weight gain and risk of obesity in children and adolescents, evidence on adults are more controversial and this relationship is still not clear. Some studies reported a cross-sectional inverse relationship between short sleep duration and BMI (Patel et al., 2008). Prospective studies reported a U-shaped association between short sleep duration and obesity risk at baseline and found that short sleep duration was longitudinally negatively associated with increased obesity and weight gain risk (Hasler et al., 2004; Patel et al., 2006). Other longitudinal findings showed a significant negative association (e.g. Gunderson et al., 2008; Nishiura et al., 2010), while some others reported U-shaped association, indicating that both short and long sleep duration are linked to obesity risk in adults (Chaput et al., 2008). Gender differences were evidenced by some authors, showing a significant U-shaped association in women but not in men (Lopez-Garcia et al., 2008), and other reporting the opposite pattern of results with a significant U-shaped association in men and not in women (Watanabe et al., 2010). Hairston et al. (2010) evidenced also age difference: the U-shaped association was present in younger adults (≤40 y) but not in those older than 40 years.

Moreover,several other studies failed in evidencing an association between short sleep and obesity (e.g. Gangwisch et al., 2005; Lauderdale et al., 2009; Marshall et al., 2010)

Finally, two systematic reviews examining cross-sectional and longitudinal studies (Magee et al., 2012; Patel et al., 2008) found that results on adults are more mixed and inconsistent than those on children. Many of the included studies showed different pattern of results: U-shaped cross-sectional association, no association, positive cross-sectional but no longitudinal association or gender difference in this association (Patel et al., 2008). This may suggest either that methodological pitfalls explain these inconsistencies or that different potential mechanism may mediate or moderate this relationship. As suggested by different authors (e.g. Lombardo, 2016; Magee et al., 2012) the inclusion of adequate measurements of sleep (e.g. objective measures as polysomnography, actigraphy and subjective measures as validated self-reported questionnaires or sleep diaries etc.) taking into account the role of confounding, mediating, and moderating variables in these studies, would permit to extend our knowledge on this link and allow to draw more exhaustive conclusions.

1.1.3 Findings in older adults

Epidemiological evidence show that sleep changes across the lifespan, the main changes being a decrease of slow wave sleep and REM sleep and an increase of sleep latency (time needed to fall asleep) and light sleep (Ohayon et al., 2004) with age. Moreover, sleep is more fragmented and the number of awakenings as well the time spent awake at night also increase with age (Gooneratne et al., 2014; Ohayon et al., 2004). Notwithstanding the objective changes in sleep, there is evidence showing that older adults complain less sleep disturbances than younger adults (Grandner et al., 2012) and that sleep complaints are often secondary to their comorbidities and not to aging per se (Foley et al., 2004).

Recently, Norton et al. (2017) reviewed the literature evaluating the association of poor sleep with obesity in older adults. Authors suggested that, in general, the cross-sectional studies using subjective measures of sleep reported an association between poor sleep and obesity. Many of the

examined studies reported a U-shaped association, highlighting that in this population both short and long duration are associated with increased risk of obesity (e.g. Liu et al., 2013; Magee et al., 2010; Ohayon & Vecchierini, 2005; Tuomilehto et al., 2008). However, some of them did not report any association (e.g. Chaput et al., 2007; Chien et al., 2015; Gradner et al. 2015,). Although longitudinal findings are poor, they provide stronger evidence for a causal linkage between poor sleep and obesity. For example, a prospective study by Xiao et al. (2013) exploring the association between self-reported sleep duration and weight change over an average of 7.5 years of follow-up on 83,377 participants (aged 51–72), found that short sleep duration (<5 and 5-6 h) was associated with 0.66 kg (95% CI: .19, 1.13) and 0.43 kg (95% CI: .19, 1.13) more weight gain compared with normal sleep duration (7-8 hours). Short sleepers (<5 h) had an approximately 40% higher risk of developing obesity than normal sleepers (7-8).

Findings by Nagai et al (2013) on 13,629 participants (aged 40-79) showed that sleep duration did not significantly predict obesity, but among participants who were obese at baseline, those sleeping for 9+ hours per night were at increased risk for ≥ 5kg weight gain, with OR=1.36 (1.09-1.70). Additionally, a prospective investigation by Lopez-Garcia et al. (2008) reported that a weight gain of 5 kg was more frequent among women reporting ≤5 hours (OR: 3.41; 95% CI: 1.34, 8.69), 8 hours (OR: 3.03; 95% CI: 1.29, 7.12), and 9 hours (OR: 3.77; 95% CI: 1.55, 9.17) of sleep.

Moreover, studies employing objective measures of sleep quantity and quality, are generally consistent with those from studies using self-report measures, despite all of them have a cross-sectional design (e.g. Anderson et al., 2014; Cross et al., 2015; Kim et al., 2015; Patel et al., 2008, 2014; Rao et al., 2009; van den Berg et al., 2008).

In summary, Norton et al. (2017) concluded the review stating that findings generally support the positive association between poor sleep (quality and quantity) and obesity. However, authors stated that this association seems to be not strong as in children or younger adults, probably due to higher comorbidity with medical conditions in older adults. They also evidenced the paucity of studies using objective measures of sleep and obesity, especially longitudinal and experimental studies,

which would help to establish the direction of the causal relation, and suggested to take into account possible mediators and moderators of this relationship such as psychological factors (e.g. emotion regulation, disordered eating etc.)

1.2 Poor sleep and food intake

After the examination of epidemiological findings about the relationship between poor sleep and BMI/obesity at different ages, an overview on the relationship of poor sleep with food intake will be presented in this section. First, the cross-sectional and longitudinal studies linking poor sleep and diet pattern/diet quality will be presented. Secondly, the experimental evidence linking poor sleep with food and caloric intake will be discussed, separately for children and adults.

1.2.1 Poor sleep and diet pattern/diet quality

In childhood, longitudinal evidence by Caspedes et al. (2016) in a cohort of 1,046 children followed from infancy to mid-childhood showed that chronic insufficient sleep predicted less favorable diet quality in mid-childhood.

Another study by Kjeldsen et al. (2014) tested whether objectively measured sleep duration and day-to-day variability in sleep duration (actigraphically measured) were independently associated with dietary risk factors for overweight and obesity in 676 children aged 8–11 years. A web-based food record for 7 consecutive days was used to assess dietary patterns and fasting blood samples were obtained for measurements of plasma leptin and ghrelin levels. The results of the study suggested that short sleep duration, high sleep duration variability and experiencing sleep problems are associated with a poor, obesity-promoting diet.

In adulthood, the influence of poor sleep (disrupted sleep pattern and/or short sleep duration) on diet pattern/diet quality have been cross-sectionally documented and also the opposite direction (namely the influence of eating pattern on sleep quality) has been investigated. For example, a recent large cross-sectional study by Yu et al. (2017) aimed to investigate the influence of dietary patterns on

sleep problems in a Chinese population. Authors found that two traditional northern dietary patterns (one characterized by high intakes of wheat and other staple food and the second by high intakes of meat, poultry, fish, eggs, fresh fruit, and dairy product) were associated to decreased prevalence of difficulties in initiating or maintaining sleep and with early morning awakenings.

Consistently with these findings, Mossavar-Rahmani and collegues (2017) found that both longer sleep duration and higher sleep efficiency (measured via actigraphy) were significantly associated with better diet quality (particularly with an increase in whole fruit intake and decrease in sodium intake) among US Hispanic/Latino adults. Moreover, recent cross-sectional evidence by Jahrami et al. (2017) revealed a negative significant ($r = -0.10$, $P < 0.05$) relationship between the consumption of energy drinks and sleep quality and patterns among college students.

In elderly, some studies have documented this relationship. For example, a study by Campanini et al. (2017) on 1596 older adults aged ≥60 years found that the adherence to a Mediterranean diet pattern was associated with lower risk of changes in sleep duration and with better sleep quality.

In the next section, findings from experimental studies that tested the effect of sleep manipulation on food intake and eating behavior will be discussed.

1.2.2 Sleep deprivation and food intake in children: experimental studies

As previously discussed, epidemiological evidence demonstrated a strong association between poor sleep (short sleep duration) and BMI and/or obesity in childhood and adolescence, stronger than that one reported in adulthood and elderly. Very few studies experimentally tested the presence of this association and how it can be explained.

A recent study by Mullins et al. (2017) experimentally investigated the immediate and recovery effects of acute sleep restriction on dietary intake in healthy preschool children. Sleep was objectively measured through actigraphy and dietary intake via parents' records. Results showed that acute sleep restriction (i.e. missed nap, delayed bedtime of ~2.3 h, habitual wake time) increased dietary intake on both the day of and the day after sleep restriction. Participants consumed

more kilocalories, sugar, carbohydrate and fat intake compared with a baseline day of habitual sleep duration.

More, a within-subjects, counterbalanced, crossover design by Hart et al. (2013) investigated the effect of experimental sleep manipulation (increase vs decrease sleep duration condition) on self-reported food intake, appetite-regulating hormones (leptine and ghrelin), and measured weight in children aged 8-11 years. Sleep was objectively measured through actigraphy and food was assessed using 24-hour dietary recalls. During the increased sleep duration condition compared to the decreased sleep condition, authors found that children reported to consume less energy, fasting morning leptin was significantly lower and children weighed less. However, no differences were found in reported macronutrient consumption nor in the fasting morning ghrelin.

Finally, a study by Beebe et al. (2013) investigated the effect of several nights of sleep restriction on adolescents' dietary intake, compared to several nights of nearly 9 h of sleep. Sleep was monitored via actigraphy and dietary intake was assessed using 24-h dietary recall interviews. Authors found that adolescents' diets after sleep restriction were characterized by higher glycemic index and glycemic load, as well as a trend toward greater consumption of calories and carbohydrates. An increase in the consumption of desserts and sweets during sleep restriction was also observed.

Despite the paucity of experimental studies including children and the small sample sizes, these finding provide empirical support to epidemiological evidence about the association of poor sleep with increased food intake, thus increasing the risk of overweight and obesity.

1.2.3 Sleep deprivation and food intake in adults: experimental studies

In 2012, a meta-analysis by Chapman and colleagues that aimed to investigate the relation between lifestyle factors (television watching, alcohol intake, and sleep deprivation) and increases in acute food intake, found that the short-term effect of these three factors on food intake was significant.

For sleep deprivation (5 experimental studies included), the Cohen's d was 0.49 while for alcohol intake and television watching Cohen's ds were 1.03 and 0.2 respectively.

Recently, an article by Al Khatib et al. (2016) including seventeen studies (n = 496) for a systematic review and eleven studies (n = 172) for a meta-analyses, assessed the effects of partial sleep deprivation on energy intake and energy expenditure. Authors found a significant increase of energy intake of 385 kcal per day following partial sleep deprivation compared to the control condition, whereas no effect was found on energy expenditure. Moreover, the observed increase in energy intake was characterized by significantly higher fat and lower protein intakes. However, the majority of the studies used laboratory settings. Therefore, as suggested by these authors, there is the need of studies investigating the effect of partial sleep deprivation on energy balance in an ecologically translatable setting, particularly as free-living conditions.

These findings are consistent with those of a previous systematic review exploring the impact of sleep duration on adiposity and energy intake, which had also suggested that sleep deprivation might increase food intake (Capers et al., 2015).

An experimental study by Hogenkamp et al. (2013) investigated the effect of a night of total sleep deprivation on portion size choice, comprising meal and snack items (measured using a computer-based task, in both fasted and sated state), and whether this depended on both hunger state and the type of food (snack or meal item) offered to young adults participants. Findings suggested that, when sleep deprived, participants chose larger portions at breakfast, irrespective of the type of food, and reported more hunger as compared to habitual sleep. This suggested the presence of a homeostatic mechanism inducing increased food intake. However, when participants were sated after breakfast, the added effect of sleep loss on portion size was observed only for snack items, indicating that a hedonic mechanism may promote food consumption after sleep deprivation.

Another study by Markwald and collegues (2013) showed that during sleep loss, participants consumed more energy than needed, particularly when food was easily accessible. Participants ate a smaller breakfast but consumed more calories as after dinner snacks, which contained more

carbohydrate, protein, and fibre content. Moreover, during sleep loss, they consumed more calories at night after dinner than calories consumed for any individual meal.

Consistently with these findings, Brondel et al. (2010) demonstrated that acute partial sleep deprivation (4 h of sleep) increased food intake in healthy men. Participants consumed more energy and reported more hunger before breakfast and dinner after sleep deprivation condition than after 8 h of sleep condition.

Previously, in 2011, St-Onge and colleagues demonstrated that experimentally induced short sleep duration (4 h of sleep) increased food intake of normal-weight men and women. Participants consumed more calories during the short sleep condition compared to the habitual sleep condition. Authors found that this effect was mostly due to increased consumption of fat.

Finally, further evidence by Nedeltcheva et al. (2009) showed that several days of sleep restriction (< 5.5 h) increased the consumption of calories from snacks with higher carbohydrate content especially from 19:00 to 07:00, but not meals intake.

Taken together, these results suggest that food intake is significantly increased following sleep deprivation as compared to intact sleep in healthy individuals. As can be derived from this section, the number of experimental studies including adults are certainly more numerous than those involving children and adolescents, while the opposite pattern has been observed regarding epidemiological evidence. This can be due to ethical problems concerning the inclusion of children and adolescents in experimental studies employing this methodology (e.g. absence from school, parental approvals, exposing children at risk of potential impairments).

1.2.4 Underlying mechanisms between poor sleep and food intake

The individual and environmental mechanisms underlying the relationship between poor sleep and increased food intake are still poorly understood. As suggested by Lundahl & Nelson (2015), factors influencing this relationship may exist at multiple levels as biological (e.g. appetitive

hormone disruption), cognitive (e.g. impaired executive functions, increased reward sensitivity), emotional (e.g. increased negative affect/emotional stress, lack of emotion regulation), and behavioural (e.g. increased impulsivity, impaired decision making).

1.2.4.1 Biological mechanism

In 2007, Knutson published an article entitled "The metabolic consequences of sleep deprivation", in which it was hypothesized that at least three pathways may be involved in the relationship of poor sleep with weight gain and increased risk of diabetes: alterations in glucose metabolism, upregulation of appetite, and decreased energy expenditure. In this article, Kristen Knutson presented a model that might explain the mechanism of positive energy balance in which energy intake is greater than energy expenditure, which would result in weight gain. In her literature review, she suggested that numerous endogenous and exogenous factors can influence both sides of this balance, and sleep has been previously associated with all these factors. Endogenous factors might be disturbances in glucose metabolism and alterations in orexigenic and anorexigenic hormones like ghrelin and leptin. Ghrelin sends signals from the stomach to the brain that trigger an increase in appetite (Van Der Lely et al., 2004), whereas leptin sends satiating signals to the appetite control centres in the hypothalamic region of the brain (Ahima et al., 2000). In addition to the biological mechanism, Knutson (2007) suggested that also the other factors as the increase in the time to eat when sleep deprived and a decrease in energy expenditure may affect food intake and weight gain, leading to obesity (figure 1).

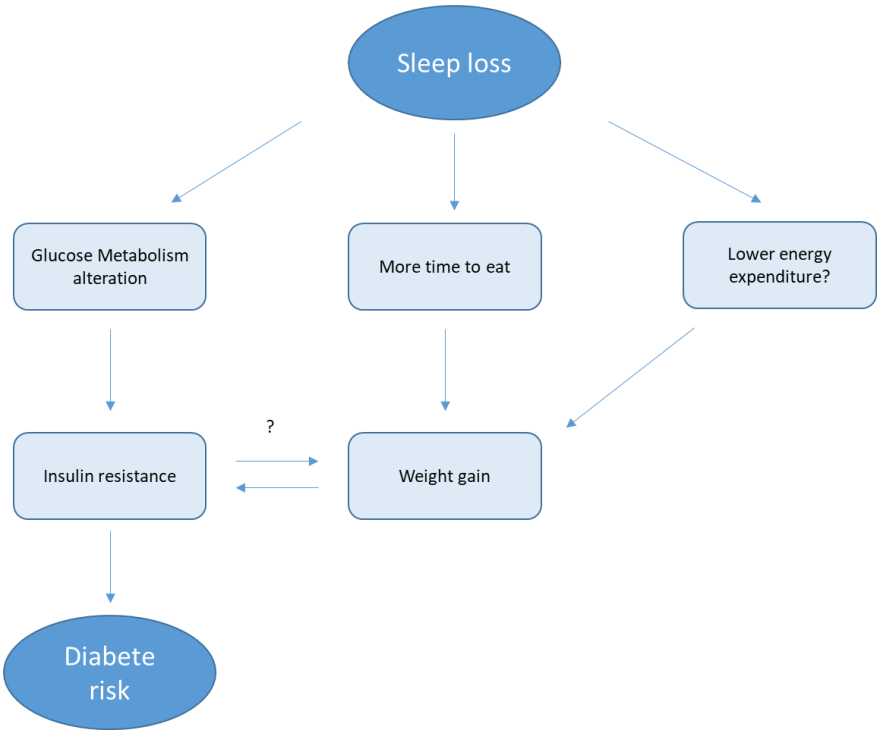


Figure 1 . Re-adapted schematic of the potential pathways leading from sleep loss to diabetes risk by Knutson (2007)

During the years, several studies experimentally investigated the effect of sleep deprivation on appetite hormones regulation. For example, one of the first authors investigating this pathway in healthy young man were Spiegel and colleagues (2004) in a randomized 2-period, 2-condition crossover clinical study. The study included two days of sleep restriction (4 hours in bed per night) and two days of sleep extension (10 hours in bed per night) under controlled conditions of caloric intake and physical activity. Compared to the sleep extension condition, the sleep restriction was associated with a reduction of 18% in leptin levels, and an increase of 28% in ghrelin levels. Moreover, authors observed an increase of 24% and 23% in self-reported hunger and appetite respectively. Particularly, appetite for calorie-dense nutrients with high carbohydrate content, including sweets, salty snacks, and starchy foods, increased.

A decrease of leptin level after sleep restriction has been observed also on children in the already mentioned study by Hart et al. (2013). Poor sleep (short sleep duration) was found to be associated with lower levels of leptin (Chaput et al., 2007) and high level of ghrelin (Taheri et al., 2004).

Also Schmid et al. (2008) found that participants reported stronger feelings of hunger and plasma ghrelin levels were higher after a night of total sleep deprivation compared to a night of 7 h of sleep or a night of 4.5 h of sleep (all *p*s < .05). However, their results did not show any difference in plasma leptin concentration. In fact, results on leptin alteration after sleep deprivation seem to be more mixed than ghrelin alteration. Some studies did not find the relationship between disrupted sleep patterns and leptin levels (e.g. Bosy-Westphal et al., 2008; Nedeltcheva et al., 2009).

Furthermore, a study by Omisade et al. (2010) showed that a single night of sleep restriction affected cortisol rhythms and increased morning leptin levels in a sample of young women. Indeed, they found that sleep restriction (3 hours of sleep allowed, from 05:00 to 08:00) significantly reduced morning salivary cortisol levels and increased leptin levels, elevated afternoon/evening cortisol area under the curve values, and slowed the decline in cortisol concentration during the day, compared to a night of 10 h sleep opportunity. Compared to other studies, this one reported the opposite pattern of results on leptin levels after sleep deprivation.

These findings suggested that sleep modulates hormone release and plays an important role in the control of blood glucose levels, and recurrent partial sleep deprivation may have a detrimental effect on appetite regulation, carbohydrate metabolism and endocrine function (Van Cautier et al., 2008). However, further investigations are needed to examine the short-term and long-term effects of poor sleep on metabolic processes in both children and adults.

1.2.4.2 Cognitive and neural mechanism

Executive functions (EFs) are commonly described as a set of cognitive processes involved in different psychological processes such as attention, memory, and emotion (Miyake et al., 2010). Results of experimental studies showed that sleep deprivation markedly impairs EFs (Cuyoumdjian et al., 2010; Gorgoni et al., 2014). Moreover, recent findings (Ballesio, 2017) evidenced that chronic sleep deprivation associated to insomnia symptoms are also related to impairment in executive functions (EFs). In addition, some studies showed that EFs impairment has been linked to

increased food intake in both children and adults (Lundhal & Nelson, 2015) even when the hunger is absent (Pieper & Laugero, 2013). Results on adults demonstrated that poor executive control was associated with higher caloric consumption, especially when the reinforcing value of the food was high (Rollins et al., 2010) and was associated with greater consumption of snacks and high-fat foods (Hall, 2012). Consistently with these findings, results on children showed that poor EFs proficiency was related to greater consumption of high calorie snacks (Riggs et al., 2010, 2012), whereas higher EFs proficiency was associated with the consumption of fruits and vegetables (Riggs et al., 2012). Moreover, a study by Cedernaes et al. (2014) found that total sleep deprivation impaired cognitive control in response to food stimuli in healthy young men, increasing the impulsivity in response to such food cues. Lundhal and Nelson (2015) highlighted the empirical evidence supporting the link between the tendency to behave impulsively (i.e. think, control, and plan insufficiently) with disrupted sleep patterns in both adult and paediatric samples. For example, sleep deprivation has been associated with greater difficulty in withholding inappropriate responses and reacting more quickly to behavioral tasks in adults (Anderson & Platten, 2011; Drummond et al., 2006). As suggested by Lundhal and Nelson, similar pattern of results have been found in children: both short sleep duration and sleep difficulties concurrently predict impulsive behaviors (Paavonen et al., 2009) and longitudinal evidence showed an association between sleep problems in childhood and problems with response inhibition in adolescence (Wong et al., 2010).

Taken together, these findings may suggest that one mechanism by which disrupted sleep patterns may increase food intake is via impairment of executive functions.

However, other cognitive and neural mechanisms may be implicated in this relationship, such as reward processing (Lundhal & Nelson, 2015). From a neural perspective, some regions of the brain involved in reward and attention undergo pronounced changes in response to high-calorie food cues (Berridge et al., 2010; Small et al., 2001; Stice and Yokum, 2016a; Ziauddeen et al., 2015). Some findings suggested that disrupted sleep patterns impact how the brain processes rewards (e.g. Chaput, 2013; Gujar et al., 2011), for example amplifying brain reactivity to pleasurable stimuli. A

recent study by Demos et al. (2017) using functional magnetic resonance imaging (fMRI) found an increased activity to food stimuli in regions of reward processing (e.g. nucleus accumbens/putamen) and sensory/motor signaling (e.g. right paracentral lobule) after short sleep condition (4 nights of 6 hours in bed) compared to long sleep condition (4 nights of 9 hours in bed). Authors also found that the whole brain analyses showed greater food cue-responsivity after short sleep in an inhibitory control region as the right inferior frontal gyrus (IFG) and the ventral medial prefrontal cortex (vMPFC), which has been implicated in reward coding and decision-making. Also another recent study by Katsunuma et al. (2017) investigated food-related responses in everyday sleep habits, indicating that unrecognized sleep loss (e.g. sleep debt accumulated in daily life) can promote brain hyper-reactivity to food cues compared to optimal sleep conditions. Consistently with these results, a study by Benedict et al. (2012) using fMRI demonstrated that acute sleep deprivation enhances the brain's response to hedonic food stimuli in normal-weight men, independently of plasma glucose levels. Particularly, they found increased activation of the right anterior cingulate cortex in response to food images after a night of total sleep deprivation compared to a night of habitual sleep.

However, some studies reported the opposite pattern of results, finding that poor sleep in youth was associated with decreased brain reactivity to reward (Hasler et al., 2012; Holm et al., 2009). As suggested by Lundhal and Nelson (2015) this effect of disrupted sleep pattern on reward processing (increasing or decreasing reward sensitivity) may have a detrimental impact on food intake, resulting in greater reward-seeking behavior given the heightened reinforcing value of the reward or, on the other side, in a compensatory increase in reward-seeking behaviour. In fact, the positive association of reward sensitivity with food craving, overeating and BMI, have been previously demonstrated (Davis et al., 2004, 2007; Franken & Muris, 2005; Stice & Yokum, 2016b). Moreover, a study by Nederkoorn et al. (2006) on children, found that sensitivity to reward was greatest in children who engaged in binge eating compared to healthy controls. These outcomes may have implications for weight loss interventions and the prevention or promotion of healthy diets, habits, and lifestyles since neural hyper-responsivity to food might be a key factor in

vulnerability to future weight gain (Cerolini et al., 2017). Identifying the reasons for hyper-responsivity to food cues in people who are gaining weight, are obese, or are candidates for bariatric surgery may be valuable when developing personalized programs (Cerolini et al., 2017).

In conclusion, executive functions and reward processing may be implicated in the relationship between poor sleep and increased food intake. To date, studies examining their potential mediating effects in the sleep/food intake pathway are very poor.

1.2.4.3 Emotional mechanism

Empirical studies have shown that the experimental manipulation of sleep duration can negatively impact both emotional functioning and cognitive performances on computer-based tasks, resulting in impaired positive affective response, emotion regulation, short-term memory, working memory, and aspects of attention (Vriend et al., 2013). Others have also found that sleep deprivation and sleepiness may impair executive and cognitive functioning such as inhibitory control over food intake, motivation, and mood (Alhola & Polo-Kantola, 2007; Burke et al., 2015; Killgore et al., 2013; Thatcher, 2008). Therefore, another underlying mechanism behind the relationship between poor sleep and food intake seems to be linked with emotion regulation. Both partial and chronic sleep deprivation have been shown to be closely linked to increased emotional reactivity and poorer emotion regulation (i.e. the capacity to implement strategies to change the intensity or valence of an emotion; Gross, 2015) among healthy individuals, and a complex interplay has been suggested to exist between these two dimensions (Cerolini et al., 2015). Sleep deprivation is understood to decrease individuals' capacity to regulate psychophysiological arousal and reactivity, thus leading to increased emotional lability, and particularly decreases in the capacity for effortful control of emotions and the pursuit of longer-term goals (van der Helm & Walker, 2009). The detrimental impact of sleep deprivation on mood and affect, with increases in the frequency and intensity of negative emotions, has been well documented (e.g. Baglioni et al., 2010; Kahn et al., 2013). Emotion disregulation has been highlighted as playing an important role in food intake, and it has

been hypothesized that it results in individuals displaying a greater tendency to use food as a strategy for coping with negative emotions, and thus lead to overeating when food is available (Leheer et al., 2015). Consistent with this is the finding that the use of dysfunctional strategies for regulating emotions is associated with increased food intake and especially increased consumption of comfort foods (Evers et al., 2010). Furthermore, the experimental induction of negative affect has been shown to cause greater food intake in laboratory settings, particularly among individuals with high levels of self-reported emotional eating (Wallis & Hetherington, 2004).

Emerging literature has also supported the relationship between sleep deprivation and higher levels of external eating, with an association documented among children (Burt et al., 2014). Similarly, poor sleep quality was associated with increased emotional eating among women (Dweck et al., 2014). Furthermore, experimentally induced negative affect was found to be associated with increased food intake among women with high levels of emotional eating reporting sleep deprivation (Dweck et al., 2014). Consistently, Strein and Coenders (2014) investigated the interaction between emotional eating and sleep duration on BMI change among 553 women and 911 men over 1 year follow-up. Authors found that the two-way emotional eating–sleep duration interaction on BMI change was present only in women but not in men: women with higher emotional eating showed a positive and significant association between short sleep duration and increased BMI. At the same time, women with short sleep duration showed a positive and significant association between emotional eating and increased BMI. These associations were significant even after controlling for possible confounders such as age, level of education, physical activity, doing sports, smoking, external eating, restrained eating, and poor general health and were stronger in women than in men.

Taken together, the findings summarized above support the hypothesis that sleep deprivation may increase food intake and trigger overeating through an emotional pathway. One of the limitations of the extant literature is that the majority of the existing studies experimentally investigating the effect of partial sleep deprivation on food intake have been conducted among healthy individuals,

thus limiting our capacity to understand how sleep deprivation might affect individuals with greater difficulties in regulating their food intake, especially in the context of negative affect (Kukk & Akkermann, 2016).

1.3 Poor sleep and eating disorders: a focus on binge eating

1.3.1 Poor sleep, insomnia and eating disorders

Poor sleep quantity and quality (e.g. insomnia) is considered as a transdiagnostic factor that may co-occur with several other mental disorders or increase the probability of comorbidity between disorders (Harvey, 2011). Patients with ED frequently experience interrupted sleep and early morning awakening, though they rarely spontaneously complain of it (Crisp, 1967; Crisp et al., 1971).

A study by Kim et al. (2010) demonstrated that sleep disturbances were highly prevalent (50.3%) among 400 female patients diagnosed with anorexia nervosa, bulimia nervosa, or binge eating disorder (BED). These patients presented also a more severe general symptomatological condition compared to those without sleep disturbances. However, this relationship may be indirect and it may due to the relationship of both insomnia and eating disorders with depression, which is known to be comorbid with both (e.g. Baglioni et al., 2011Slane et al., 2010). Two studies by Lombardo and colleagues (2013, 2015) investigated the hypothesis of the mediation by depression of the relation between poor sleep and ED severity both cross-sectionally and longitudinally. In the first study (Lombardo et al., 2013) 1019 female students were evaluated cross-sectionally. Results showed that increased severity of insomnia was associated with higher severity of disordered eating and both insomnia and disordered eating symptoms were related to depression. The mediation analysis evidenced a direct and an indirect relationship between insomnia symptoms and eating disorder symptoms trough the mediation of depression. In the second study (Lombardo et al.,

2015), 562 ED patients completed three self-report questionnaires at treatment admission and after six month of standard treatment (N= 271 patients). Authors found that poor sleep predicted severity of eating disorders symptoms through the mediation of depression at admission time. Moreover, the persistence of poor sleep after 6 months directly predicted the severity of the ED symptoms both directly and through the mediation of depression. Finally, poor sleep persistence predicted worse clinical conditions after 6 months of treatment.

1.3.2 Poor sleep and binge eating

In the last years, some studies indicated the presence of sleep disturbance in people with binge eating disorders and binge eating symptoms. Recently, Quick et al. (2016) have reported cross-sectional association between short sleep duration and binge eating among 1,252 U.S college students. Specifically, individuals sleeping < 8 hr compared to those sleeping ≥ 8 hr had significantly more negative eating attitudes, poorer internal regulation of food, and greater binge eating, even after controlling for gender, race/ethnicity, work time pressures, negative affect, and sleep disturbances. Another study by Trace et al. (2012) cross-sectionally investigated the presence of current self-reported sleep problems and lifetime binge eating in 3,790 women aged 20–47 years. Their results indicated that women reporting binge eating were 6.4% of the total sample and this symptomatology was positively associated with sleep disturbances such as not getting enough sleep, poor sleep, problems falling asleep, sleepiness during the daytime, and disturbed sleep, even after controlling for obesity.

Results consistent with these findings were reported by Vardar et al. (2004). They explored the subjective sleep qualities and the psychopathological features of BED in treatment-seeking obese patients (BED and non-BED group) and control subjects (control group). Results evidenced that BED obese group differed significantly from the non-BED obese group and the control group in the total score of The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) and in the sleep onset latency, thus indicating poorer sleep quality and more difficulties in initiating sleep. Lastly, there

are also evidence supporting the association between obstructive sleep apnea and binge eating and binge eating disorder in 1,099 obese patients candidate for bariatric surgery (Sockalingam et al., (2017).

Empirical evidence on children were reported by Tzischinsky & Latzer (2000), who objectively assessed sleep-wake cycles in a group of obese children with (N=13) and without binge eating (N=23) and in a normal-weight control group (N=25). Sleep was monitored by mini-actigraphs and self-report sleep questionnaires. Findings revealed that the three group differed significant in the actigraphic sleep parameters of sleep efficiency, sleep latency, and wakefulness during sleep. The group of obese children with binge eating slept significantly worse than the other two groups. The self-reported sleep was not different between obese children with and without binge eating though, except for significant higher complaint of mid-sleep awakening, snoring and restless sleep by both groups as compared to normal-weight group.

In conclusion, poor sleep seems to be linked with eating disorders severity. Especially, binge eating symptomatology have been demonstrated to be related to sleep disturbances and overall poor sleep.

1.3.3 Binge eating: symptomatology and main features

Binge eating behaviour is defined as consuming a large amount of food (significantly more food than most people would eat under similar circumstances) in a short period of time while experiencing a loss of control over eating (American Psychiatric Association, APA, 2013). This behaviour could be a symptom of different eating disorders such as Bulimia Nervosa or Anorexia Nervosa but, surely is the core symptom of the Binge Eating Disorder (BED) characterized also by several symptoms as eating too quickly, even when the person is not hungry; feelings of guilt, embarrassment, or disgust; binge eating alone to hide the behaviour; marked distress. A diagnosis of BED can be formulated only if these symptoms occur, on average, at least once a week over three months (APA, 2013). Binge eating behaviours may also occur lifetime in the normal population

without ED, for example it may happen to eat quickly and losing control over eating when one is very hungry, or when a large buffet or non-stop menu in restaurant/parties are presented. This disordered eating behaviour is frequently reported by college students and it is of particular concern because of its link to the development of eating disorders and obesity (Phillips et al., 2016). Generally, females students are more likely to report binge eating symptoms, and findings by Phillips et al. (2016) revealed that they reported also emotional concerns such as stress and negative affect prior to BE and poor body image and negative affect following episodes of BE, compared to males. Females also reported to prefer eating alone at home, whereas males were more likely to report BE socially on meal type foods. In addition, males reported more substance use, exercise, and hunger before a BE episode, with feeling satisfied or full after BE. Napolitano & Himes (2011) indicated that in college women, the prevalence of severe binge eating symptoms and BED has been estimated at 44% and 8.4%, respectively. Individuals who report binge eating symptoms are more prone to experience fluctuations in negative emotions and overeating (Kukk & Akkermann, 2016), lack of emotion regulation and consequent increased food intake (Ruscitti et al., 2016; Svaldi et al., 2014), negative affect, external and emotional eating (Schulz et al., 2010), difficulties in cognitive and emotional functioning (Kittel et al., 2012), more sleep disturbances (Trace et al., 2012), greater levels of eating, weight, and shape concerns and lower levels of appearance satisfaction and self-esteem (Herbozo et al., 2015), and more attentional bias toward food stimuli (Schmidt et al., 2016) compared to controls. In addition, recent studies and a systematic review have identified negative affect as one the main triggers precipitating binge eating (Berg et al., 2014; Leheer et al., 2014). Furthermore, consistent with the pathways described above, chronic sleep deprivation (e.g. insomnia) has been associated with binge eating (Trace et al., 2012), and depression (Baglioni et al., 2011), which are tightly related to increased food intake and obesity (de Zwann, 2001; Luppino et al., 2010).

1.4 Summary

The findings presented in this literature review provide strong evidence in support of the association of poor sleep (quantity and quality) with increased risk of obesity. Epidemiological evidence, cross-sectional and longitudinal studies overall support this link in children and adolescents, in adults and in older adults. However, the strongest evidence derived from studies in childhood, while results on adults and older adults are more controversial.

Experimental studies provide a strong contribution in demonstrating the causality pattern rather than the merely connection. In fact, it has been demonstrating that sleep deprivation may increase food intake, thus potentially enhancing the risk of obesity. However, despite some studies have investigated the biological underlying mechanisms implicated in the sleep/eating behavior pathway, very few authors have studied the other potential mechanisms involved, such as cognitive (e.g. the role of executive functions) and emotional mechanisms (e.g. emotion regulation, emotional eating, hedonic value of the food, food craving). Moreover, no other studies included individual reporting binge eating symptoms, while the literature reported a strong link between poor sleep and binge eating, and more in general, disordered eating.

Part 2: An empirical contribution to the study of the relationship between sleep deprivation and eating behaviour in a sample of individual reporting binge eating symptoms

1 General framework of the study

The scientific literature summarized in Part 1 of the present dissertation evidenced that poor sleep and obesity, as well as sleep deprivation and increased food intake, are strictly connected. However, underlying mechanisms are still poorly understood. In order to shed light on these potential mechanisms, a study was conducted assessing these variables in individual reporting binge eating symptoms. The following chapters of the present dissertation will give:

1) a general overview of the study describing aims, method and procedure, presenting the descriptive statistics of the sample and results of sleep manipulation check.
2) Evidence of the effect of sleep deprivation on food intake considering the moderation of emotional eating
3) Evidence of the effect of sleep deprivation on executive functions and the mediating role of EFs on food intake
4) Evidence of the effect of sleep deprivation on food craving and the mediating role of food craving on food intake.

1.1 Overview of the aims of the study

This study aims to provide novel contribution to the understanding of the relationship between poor sleep and eating behavior, examining different potential underlying mechanisms. It aims to evaluate the effect of a night of partial sleep deprivation on different outcomes related to eating behavior such as food intake, executive functions and craving for high palatable food images. The potential

mediation effect of executive functions and food craving in increasing food intake has been considered, as well as the effect of potential moderator such as self-reported emotional eating and depression. The sample included a group of individuals reporting binge eating symptoms (Binge Eating Group) and a group of healthy controls (Control Group). The lack of experimental studies investigating the role of acute partial sleep deprivation on food intake in individuals with binge eating limits our capacity to understand the mechanisms at play in the maintenance of binge eating symptomatology and therefore inform effective interventions. To evaluate this effect, a night of partial sleep deprivation (DN, 5h of sleep allowed) was compared to a night of habitual sleep (HN, the usual sleep length was allowed). After both nights, the performance in different computer tasks in the lab, the food intake during breakfast and over the course of the day were assessed.

We aimed to evaluate if food intake, food craving after the DN were higher compared to HN, and if this effect was stronger in individuals reporting binge eating. The potential mediating role of food craving in increasing food intake was also tested. Moreover, we wanted to investigate if executive functions were impaired after the DN, eventually considering their potential mediating role in increasing food intake. Furthermore, the moderating effect of self-report emotional eating and depression were also examined. To our knowledge, this is the first study evaluating the effect of sleep deprivation in participants reporting binge eating symptoms.

1.2 Method and procedure

1.2.1 Participants selection

Twenty-eight participants (age M= 23.75 ± 4.03, 21% male) were recruited from Sapienza University of Rome, through flyers, word of mouth, and electronic advertisements. After an initial online screening, during which participants were asked to complete self-report questionnaires and demographic information, eligible participants were contacted to take part in the study. Based on scores on the Binge Eating Scale (BES, Gormally et al., 1982) and the Disordered Eating Questionnaire (DEQ, Lombardo et al., 2004), we recruited 14 young adults reporting symptoms of

binge eating and 14 without any eating symptomatology (either of binge or restrictive eating). Inclusion criteria for the Binge Eating Group (BG) were scores above the cut-off of 17 on the Binge Eating Scale (BES ≥ 17), while for the Control Group inclusion criteria were scores equal or below both the cut-off of 17 on the BES (BES < 17) and the cut-off of 30 on the Disordered Eating Questionnaire (DEQ ≤ 30). Exclusion criteria for both groups were: presence of food allergies or intolerances, cardio-metabolic illness, particular diets such as being vegetarian or vegan, being underweight (BMI < 18.5), and current use of psychiatric medications. All participants voluntarily agreed to participate in the study and provided informed consent. They received a 20 Euro gift card (control group) or the opportunity to participate in a four-session emotion regulation group (binge eating group), in compensation for their time. The study was conducted in agreement with the Helsinki Declaration (World Medical Association, 2013) and employed the same methodology as another study (Lombardo et al., 2018) approved by the Ethical Committee of the Department of Psychology at Sapienza University of Rome on March 25th 2015.

1.2.2 Screening phase: materials

To screen for eligibility, we used the Binge Eating Scale (BES, Gormally et al., 1982) in the Italian version by Di Bernardo et al. (1998). The BES is 16-item questionnaire assessing the presence of certain binge eating behaviors such as controlling impulses towards food, feeling guilty after having eaten too much, having intrusive thoughts about eating, overeating in secret, etc. According to Imperatori et al. (2015) the BES has good internal consistency ($\alpha = 0.89$) and the total scale score is a reliable measure of binge eating severity. Based on the BES total raw scores, Marcus et al. (1988) identified a score of 17 as the clinical cut-off for the presence of binge eating. In this study Cronbach's $\alpha = 0.93$. Control participants were selected on the basis of the absence of any eating symptomatology using the BES and the Disordered Eating Questionnaire (DEQ, Lombardo et al., 2004). The DEQ is a one-factor scale that consists of 24 items assessing dysfunctional eating-related behaviour patterns, such as restrictive eating. This scale includes items regarding reducing

food intake to lose or maintain weight, ruminating and worrying about weight and body shape, and engaging in intense physical exercise to lose weight etc. It is based on DSM IV-TR criteria (APA, 2004) and the clinical cut-off score is 30. Cronbach's α in the validation study was 0.90 whereas in this study it was 0.94.

Emotional eating was assessed during screening using the Emotional Eating Scale (EES; Arnow, et al., 1995) in the Italian version by Lombardo and San Martini (2005). This 25-item scale is a valid and reliable instrument to assess eating behavior resulting from negative emotions. It includes three subscales: emotional eating after depression (EES-D), emotional eating after anxiety/confusion (EES-A), and emotional eating after anger (EES-C). In this study we used only the EES-D scale as a covariate for our analyses, since the existing link of depression with binge eating (Mussel et al., 1995) and obesity (de Zwann, 2001; Luppino et al., 2010) and the strong relationship between poor sleep and depression (Staner, 2010; Baglioni et al., 2011). Participants were asked to indicate the extent to which certain feelings lead them to feel an urge to eat (e.g. when they felt depressed, bored, etc.) on a five-point Likert scale ranging from "no desire to eat" to "an overwhelming urge to eat." In the original study of Arnow et al. (1995), the Cronbach's α was 0.72 for the Depression subscale. In this study, the Cronbach's α = 0.88.

Additional psychological components were also assessed, including depression and insomnia.

Depression was assessed through the Beck Depression Inventory-II (BDI-II, Beck et al., 1996), in the Italian version by Ghisi et al. (2006). The BDI-II is a 21-item self-report inventory assessing the presence and severity of depressive symptoms based on DMS-IV criteria (1994). Participants rated their endorsement of each symptom on a four-point scale ranging from 0 to 3, except for two items (16 and 18) that have seven options to indicate either an increase or decrease of appetite and sleep. Total scores of 0-13 are considered indicative of minimal-range depression, 14-19 as mild, 20-28 as moderate, and 29-63 as severe. The cut-off of 16 has been often considered as a good index for the

presence of clinical relevant depression. Cronbach's α in the English validation study was 0.91, whereas in the current study, Cronbach's α = 0.95.

Insomnia was assessed through the Insomnia Severity Index (ISI, Bastien et al., 2001), in the Italian version of Battagliese and Lombardo (2012). The ISI was employed to provide a measure of insomnia severity during the preceding two weeks. The brief scale includes items regarding difficulty falling asleep, difficulty staying asleep, problems waking up too early, and worries and satisfaction regarding sleep pattern and daily functioning. A total score is obtained by summing the responses to seven items, scored on a 5-point Likert scale, with total scores ranging from 0 (insomnia absent) to 28 (very severe insomnia). A score ≤ 7 is considered the cut-off for the absence of clinically significant insomnia. Cronbach's α in the validation study was 0.76 and in this study it was 0.77.

Menstrual phase was also recorded among female participants during the first morning in the laboratory asking the date of the first day of the last cycle.

1.2.3 Sleep assessment

During the duration of the days that followed both their normal and sleep-deprived nights, participants were asked to complete sleep diaries (Morin, 1993) that are considered the gold standard among subjective measure of sleep (Buysse et al., 2006). On both mornings, participants estimated a number of sleep parameters including what time they went to sleep, duration of nocturnal awakenings, what time they woke up, and the minutes it took to fall asleep. Consistent with previous work (e.g. Bastien et al., 2001; Cerolini et al. 2016, Devoto et al., 2003), using the data extracted from sleep diaries, we calculated the following sleep indices:

- Total time in bed (TIB), as the time a participant reported to lay in bed either sleeping or not;

- Total Sleep Time (TST) as TIB minus the minutes required to fall asleep and minus the minutes spent awaken after sleep onset;

-Sleep efficiency index (SEI) dividing TST by TIB and multiplying by 100.

	First day	*Second day*	*Third day*	*Fourth day*
Complete in morning				
Bedtime (date/time)	10:45 p.m. (4/10)			
Rise time (date/time)	7:00 a.m. (4/11)			
Estimated time to fall asleep	30 minutes			
Estimated number of awakenings and total time awake	5 times 2 hours			
Estimated amount of sleep obtained	4 hours			
Complete at bedtime				
Naps (number, time, and duration)	1 at 3:30 p.m. 45 minutes			
Alcoholic drinks (number and time)	1 drink at 8:00 p.m. 2 drinks at 9:00 p.m.			
List stresses of the day	Flat tire Argued with son			
Rate how you felt today 1 = Very tired/sleepy 2 = Somewhat tired/sleepy 3 = Fairly alert 4 = Wide awake	2			
Irritability level 1 = None 2 = Some 3 = Moderate 4 = Fairly high 5 = High	5			
Medications				

Figure 1. Sleep diary used to subjectively monitor sleep.

Moreover, an electronic portable device called Zeo (Inc., Newton, MA; Cellini et al. 2013, 2015) was used to objectively control participants' compliance to the sleep deprivation instruction, thus validating the sleep manipulation. Specifically, during both nights participants were instructed to wear the headband at 10 pm, specifically this was done to control whether they were actually awake until 1 am and woke up at 6 am during the deprivation night. They were also asked to avoid napping during the day following the habitual and the deprivation night. In fact, all participants reported to have followed the given instructions. Given the large amount of data, Zeo's data have not been analyzed for this dissertation.

Figure 2. The electronic tool used to objectively control the compliance.

1.2.4 General Procedure

Upon recruitment, participants were invited to an initial lab session during which they provided informed consent and received all the instructions and the materials (sleep and food diaries, and an electronic portable device called Zeo). After the researcher confirmed that participants understood how to record their sleep and food intake, participants scheduled two appointments to return to the lab during the following week. They were specifically asked for one of their appointments to follow a night of habitual sleep (HN) while the other should follow a night of partial sleep deprivation (DN) during which only 5 hours of sleep were allowed. The allowed time in bed was adapted based on subjects' habitual bedtime, in order to remove part of their initial and part of their final habitual sleep-time. The order of the sleep manipulations was counterbalanced among participants and between groups (HN-DN or DN-HN). When the DN occurred first, a night of restoration was required before the HN, since a restoration bias might occur after a night of poor sleep (Perlis et al., 2014). Upon their arrival in the lab, on both days, participants were asked to complete 2 computer tasks to measure executive functions and craving for high palatable food images. After that, a large and varied breakfast was provided (salty and sweet foods). Food intake was unobtrusively measured by the researchers. Participants were informed of this only in the final debriefing upon study completion, when they were asked to provide informed consent for the inclusion of these data. Two additional questions were also included: 1) Were you aware that the

researcher would measure your food intake? 2) If yes, did it affect your choices? In which ways? Figure 1 graphically illustrates the general procedure.

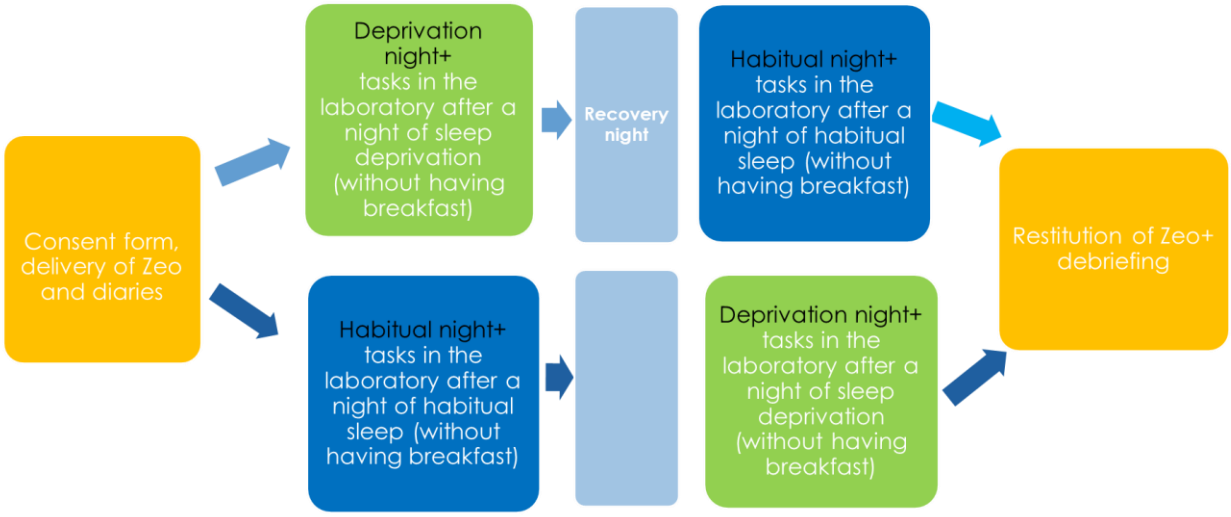


Figure 3. A schematic summary of the general procedure of the study. The order of the night was counterbalanced among participants.

1.3 Statistical analyses

All data analyses were conducted using the Statistical Package for Social Sciences (SPSS, IBM Corp., 2011) version 20.0 for Windows. Data were screened for errors and missing values. First, frequencies, descriptive statistics and internal consistencies for each scale of the self-report questionnaires were calculated. Group differences on demographic and self-reported measures were tested using Independent sample t-tests. A sleep manipulation check was performed using a 2 x 2 mixed design factorial ANOVA, Night (HN vs DN) x Group (Binge Eating Group vs Control), on the Total Sleep Time and the Sleep Efficiency Index.

1.4 Results

1.4.1 Group characteristics

Each group consisted of fourteen participants (11 females and 3 males). Five participants (Binge Eating Group = 2, Control Group = 3) were classified as being in menstrual phase (from day

1 to 6), four (Binge Eating Group =3, Control Group =1) were classified as being in the follicular phase (from day 7 to 13), one participant (Binge Eating Group) was in the ovulation phase, and twelve (Binge Eating Group = 5, Control Group = 7) were in the luteal phase. A chi-square test indicated that there were no significant differences in menstrual phase between the two groups (χ^2 = 2.533, p = .469).

Table 1 displays the means and standard deviations of the two groups for age, Body Mass Index (BMI) and the relevant self-report measures. Groups were compared though the t-tests for Independent Samples and results are also displayed in that table.

	Control Group n=14	Binge Eating Group n=14	t	*p*-value
Age	24.29 ± 4.250	23.21 ± 3.886	.696	.493
BMI	20.737 ± 1.277	24.577 ± 5.29	-2.64	.014
BES	2.071 ± 1.940	20.357 ± 3.201	-18.279	<.001
DEQ	7.143 ± 5.531	39.5 ± 13.637	-8.227	<.001
EES-D	7.429 ± 5.515	19.357 ±5.213	- 5.881	<.001
BDI-II	4.594 ± 3.926	15.909 ± 11.262	-3.55	.001
ISI	3.692 ± 2.869	7.462 ± 3.711	-2.897	.008

BMI= Body Max Index; BES= Binge Eating Scale; DEQ= Disordered Eating Questionnaire; EES-D= Emotional Eating Scale after Depression; BDI-II = Beck Depression Inventory-II; ISI = Insomnia Severity Index

Table 1. Groups' characteristics

As shown in Table 1, the groups differed on all the self-report measures as well as on BMI: the Binge Eating Group reported higher levels of binge eating symptoms, disordered eating, emotional eating, depression, and insomnia and presented a higher BMI compared to the Control Group. Particularly, in the Control Group any participant reported clinical depression (BDI-II> 16, n= 0) whereas in the Binge Eating Group were four (BDI-II >16, n= 4). Sub-threshold Insomnia were

reported by only one participant in the Control Group (ISI > 7, n = 1) and by 8 participants in the Binge Eating Group (ISI > 7, n = 8).

1.4.2 Sleep Manipulation check

The 2 x 2 mixed design factorial ANOVA Group (Binge Eating Group vs Control Group) x Night (HN vs DN) on the Total Sleep Time (TST) revealed a significant main effect of the Night, $F(1,26) = 141.54$, $p < .001$. During the Sleep Deprivation Night both groups slept less ($M = 274.21 \pm 31.94$ minutes) compared to the Habitual Night ($M = 427.43 \pm 56.751$ minutes). No main Group effect or interaction was found for the TST. No Group ($F_{(1,26)}= 1.80$, $p= .191$), Night ($F_{(1,26)}=0.94$, $p=.762$), or interaction effect ($F_{(1,26)}= 0.82$, $p= .373$) were found on the Sleep Efficiency Index (SEI).

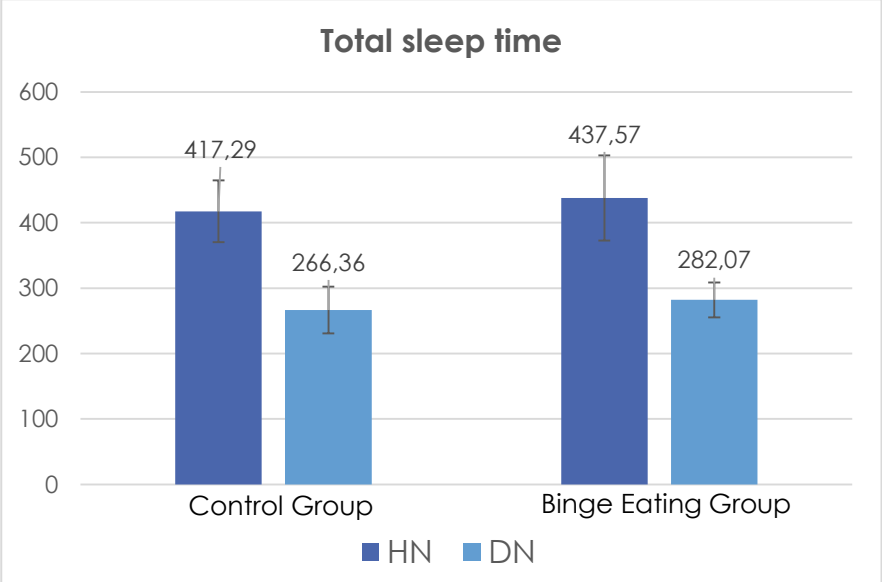


Figure 1. Total sleep time in minutes of both groups during Habitual Night (HN) and Deprivation Night (DN)

1.5 Discussion

Both groups (the group reporting binge eating symptoms and the healthy control group) differed significantly in terms of BMI, disordered eating, depression, emotional eating and insomnia severity. These findings are in line with previous studies suggesting that individuals reporting binge

eating symptoms have higher BMI and depressive symptoms (Mason et al., 2014), present disordered and restrictive eating, are more prone to emotional eating (Schulz et al., 2010) and report more sleep disturbances (Trace et al., 2012). The sleep manipulation check showed that both groups were effectively partially sleep deprived as they were supposed to be. Moreover, since they differed only in sleep duration while the efficiency of their sleep was comparable across nights and groups we are confident that the design was appropriate and sensitive and that the manipulation affected only sleep duration.

2 The effect of sleep deprivation on food intake

2.1 Introduction

Emerging evidence suggests that poor sleep quantity and quality are linked to increased food intake both in children (Beebe et al., 2013; Chaput, 2016; Tatone-Tokuda et al., 2012), and adults (Hogenkamp et al., 2013; St-Onge, 2016) and that this may constitute a risk-factor for obesity (Fatima et al., 2014; Lombardo, 2016). A recent systematic review and meta-analysis by Al Khatib et al. (2016) found that energy intake was significantly increased following partial sleep deprivation as compared to intact sleep in healthy individuals. These findings are consistent with those of a previous systematic review exploring the impact of sleep duration on adiposity and energy intake, that had also suggested that sleep deprivation might increase food intake (Capers et al., 2015). However, to date, the individual and environmental mechanisms underlying this relationship are still poorly understood. Factors influencing this relationship may exist at multiple levels (Lundahl and Nelson, 2015), including biological (e.g. appetitive hormone disruption), cognitive (e.g. impaired executive functions, increased reward sensitivity), and behavioral processes (e.g. increased impulsivity, impaired decision making). However, one of the mechanisms that has so far received less attention involves emotional pathways, specifically the role of increased negative affect and emotional stress on increased food intake.

Both partial and chronic sleep deprivation have been shown to be closely linked to increased emotional reactivity and poorer emotion regulation (i.e. the capacity to implement strategies to change the intensity or valence of an emotion; Gross, 2015) among healthy individuals, and a complex interplay has been suggested to exist between these two dimensions (Cerolini et al., 2015). Sleep deprivation is understood to decrease individuals' capacity to regulate psychophysiological arousal and reactivity, thus leading to increased emotional lability, and particularly decreases in the capacity for effortful control of emotions and the pursuit of longer-term goals (van der Helm & Walker, 2009). The detrimental impact of sleep deprivation on mood and affect, with increases in the frequency and intensity of negative emotions, has been well documented (e.g. Baglioni et al., 2010; Kahn et al., 2013). Consistent with this, empirical studies have shown that the experimental manipulation of sleep duration can negatively impact both emotional functioning and cognitive performances on computer-based tasks, resulting in impaired positive affective response, emotion regulation, short-term memory, working memory, and aspects of attention (Vriend et al., 2013) and several studies have also found that sleep deprivation and sleepiness may impair executive and cognitive functioning such as inhibitory control over food intake, motivation, and mood (Alhola& Polo-Kantola, 2007; Burke et al., 2015; Killgore et al., 2013; Thatcher, 2008).

Emotion disregulation has been highlighted as playing an important role in food intake, and it has been hypothesized that it results in individuals displaying a greater tendency to use food as a strategy for coping with negative emotions, and thus lead to overeating when food is available (Leheer et al., 2015). In particular, Escape Theory (Heatherton & Baumeister, 1991) posits that among individuals with high levels of emotional eating, overeating may occur in the context of narrowed attention as an attempt to regulate negative emotions and avoid threatening stimuli, leading to greater focus on the immediately available food, and thus greater food intake. Consistent with this is the finding that the use of dysfunctional strategies for regulating emotions is associated with increased food intake and especially increased consumption of comfort foods (Evers et al., 2010). Furthermore, experimental inductions of negative affect have been showed to cause greater

food intake in laboratory settings, particularly among individuals with high levels of self-reported emotional eating (Wallis & Hetherington, 2004).

Emerging literature has also supported the relationship between sleep deprivation and higher levels of external eating, with an association documented among children (Burt et al., 2014). Similarly, poor sleep quality was associated with increased emotional eating among women (Dweck et al., 2014). Furthermore, experimentally induced negative affect was found to be associated with increased food intake among women with high levels of emotional eating reporting sleep deprivation (Dweck et al., 2014).

Taken together, the findings summarized above support the hypothesis that sleep deprivation may increase food intake and trigger overeating through an emotional pathway. One of the limitations of the extant literature is that the majority of the existing studies experimentally investigating the effect of partial sleep deprivation on food intake have been conducted among healthy individuals, thus limiting our capacity to understand how sleep deprivation might affect individuals with greater difficulties in regulating their food intake, especially in the context of negative affect (Kukk&Akkermann, 2016), as occurs in Binge Eating. Binge eating is defined as consuming a large amount of food (significantly more food than most people would eat under similar circumstances) in a short period of time while experiencing a loss of control over eating (APA, 2013). Individuals who report binge eating symptoms are more prone to experience fluctuations in negative emotions and overeating (Kukk & Akkermann, 2016), lack of emotion regulation and consequent increased food intake (Ruscitti et al., 2016; Svaldi et al., 2014), negative affect, external and emotional eating (Schulz et al. 2010), difficulties in cognitive and emotional functioning (Kittel et al., 2012), and more sleep disturbances (Trace et al., 2012) compared to controls. In addition, recent studies and a systematic review have identified negative affect as one the main triggers precipitating binge eating (Berg et al., 2014; Leheer et al., 2014). Furthermore, consistent with the pathways described above, chronic sleep deprivation (e.g. insomnia) has been associated with binge eating (Trace et al., 2012),

and depression (Baglioni et al., 2011), which are tightly related to increased food intake and obesity (de Zwann, 2001; Luppino et al., 2010). In addition, habitual short sleep duration (< 8 h) has been also associated with lower flexibility and competence in terms of eating, poorer internal regulation of food, and higher rates of binge eating compared to longer sleep duration (≥ 8 h) (Quick et al. 2016). To date, however, the role of acute sleep deprivation on food intake among individuals with binge eating symptomatology is poorly understood. Thus, the current study aimed to investigate the effect of partial sleep deprivation on food intake in a group of individuals reporting binge-eating symptoms (Binge Eating Group) and in a group of healthy controls (Control Group). The literature summarized above led to the hypothesis that sleep deprivation might increase caloric intake in participants reporting binge eating. In addition, given the previously documented role of emotional eating as a mechanism for increased food intake, we also sought to examine the contribution of emotional eating, supposing that it may increase the effect of sleep deprivation in people reporting binge eating.

2.2 Food intake assessment

Participants were provided with and asked to complete food diaries that included serving size references, in which they reported their food and beverage intake over the course of the whole day. Participants were asked to specify the nature, quantity, and preparation of each food consumed all over the day. During the laboratory sessions after both the habitual and deprivation nights, participants were invited to help themselves with foods from a large and varied breakfast, and select whatever they wanted. The meal options included sweet and salty foods such as biscuits, croissants, Nutella (a chocolate spread), and savory crackers, as well typical Italian breakfast drinks such as fruit juice, coffee or cappuccino etc. Food intake was assessed counting pieces (e.g. for biscuits) or weighing (e.g. for Nutella) the food before and after consumption using a calibrated electronic scale (maximum capacity 3 Kg, accurate to 1 g).

Using the website http://sapermangiare.mobi/, sponsored by the Research Centre for Eating and Nutrition (CRA-NUT) and The Ministry of Agricultural, Food and Forestry Policies, each food and beverage consumed at breakfasts in the lab and reported in the food diaries were converted into total caloric (Kcal) and macronutrient (carbohydrate, fat, protein, fibre and alcohol) content. Measures of caloric and macronutrients intake at breakfast and at each main meal and snack were computed by summing up the calories and macronutrients contents of each food/beverage consumed after the Habitual and the Deprivation nights.

2.3 Statistical analyses

A mixed design factorial ANOVA, Night (HN vs DN) x Group (Binge Eating Group vs Control Group), was used to analyze the Food Intake at breakfast in the lab. Based on the literature, a 2 x 2 mixed design factorial ANCOVA was performed controlling for Emotional Eating. When significant interactions between the covariate and the factors were found, following Pedhazur and Schmelkin (1991) suggestions, two groups were formed based on a median split: High EES-D and Low EES-D. Then, a series of 2 x 2 mixed design factorial ANOVAs were performed considering Group (High EES-D vs Low EES-D) and Night (HN vs DN) as factors and the total energy and the macronutrients contents eaten at breakfast as outcome variables. Similar analyses (mixed design factorial ANOVAs and ANCOVAs) were conducted to examine the effects of the factors Group (High EES-D vs Low EES-D) and Night (HN vs DN) on the Daily Food Intake data. Given the small sample size, and thus the reduced power of the statistical test, we decided to perform Group (High EES-D vs Low EES-D) x Night (HN vs DN) ANOVAs regardless of the significance of the interaction between the Night and the covariate.

2.4 Results

2.4.1 Food intake at breakfast

The 2 x 2 mixed design factorial ANOVA Group (Binge Eating Group vs Control Group) x Night (HN vs DN) on the Total Energy consumed at breakfast in the lab showed no main effect either of the night, $F_{(1,26)} = 2.03$, $p = .166$, or of the group ($F_{(1,26)} = 1.93$, $p = .177$), or any significant Group x Night interaction ($F_{(1,26)} = 0.96$, $p = .340$). The analysis using fibre content as the dependent variable revealed a main effect of the Night ($F_{(1,\ 26)} = 7.71$, $p = .010$). Thus, after the Sleep Deprivation Night both groups consumed less fibre ($M = 2.69 \pm 1.762$) than after the Habitual Night ($M = 3.43 \pm 2.35$). The main effect of the Group and the interaction were both non significant. No significant effects were found for carbohydrate, fat, or protein intake during breakfast in the lab.

2.4.2 Food intake at breakfast in the lab controlling for Emotional Eating

Based on the existing literature on the putative role of Emotional Eating (Dweck et al., 2014), we performed a 2 x 2 mixed design factorial ANCOVA, using the EES-D scale as a covariate. Results revealed a significant Night x EES-D interaction ($F_{(1,25)} = 6.52$, $p = .017$) and a significant main effect of the Night ($F_{(1,25)} = 8.69$, $p = .007$). Given the significant interaction between the covariate and the within subjects factor, we further divided the sample based on EES-D scores. Using a median split, a Low EES-D (EES-D $\leq$ 15) and a High EES-D (EES-D > 15) group were created. The two groups almost perfectly overlapped with the original groups created on the basis of self-reported binge eating symptoms, except for two individuals: one classified into the Binge Eating Group reported low EES-D and one classified into the Control Group reported high EES-D. A new 2 x 2 mixed design factorial ANOVA examining Group (Low EES-D vs High EES-D) x Night (HN vs DN) effects on Total Energy consumed during breakfast in the lab was performed. Results revealed a significant Group x Night interaction ($F_{(1,26)} = 4.42$, $p = .045$) and a marginal main Group effect ($F_{(1,26)} = 3.257$, $p = .083$). Analyses of simple effects revealed that individuals reporting low EES-D scores ate less at breakfast when sleep-deprived ($M = 397.09 \pm 182.72$) than the habitual night of sleep ($M = 488.72 \pm 242$; $t = 2.66$, $p = .020$). Participants reporting high EES-

D ate less at breakfast after both nights as compared to the low EES-D group (M = 302.94 ± 205.63; and M = 442.91 ± 204.7 respectively). Results are displayed in Figure 1.

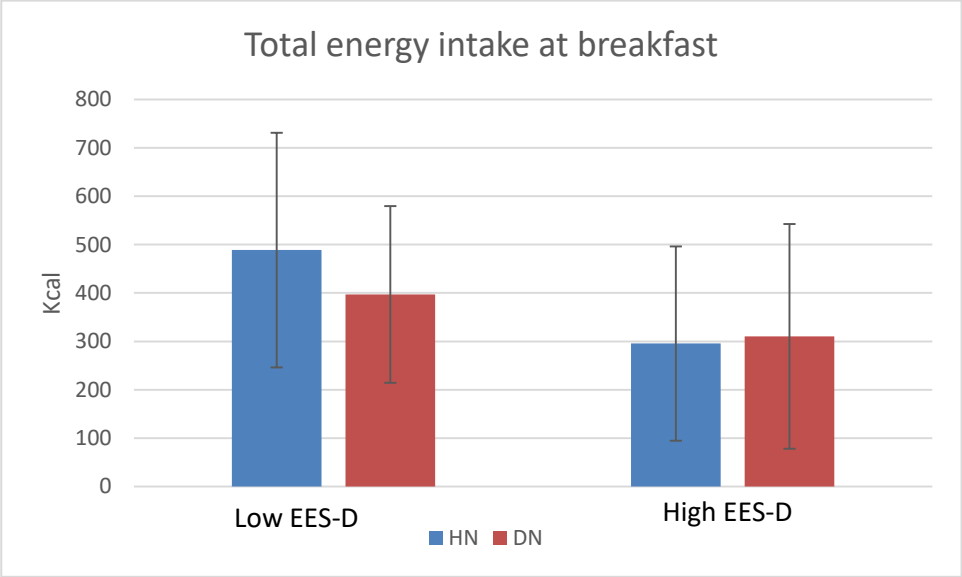


Figure 1. Total energy (kcal) consumed at breakfast by Low EES-D and High EES-D

Mixed 2 x 2 design factorial ANCOVAS were also performed on carbohydrates, fat, proteins and fibre intake during the breakfast in the lab. Results for carbohydrate intake revealed a significant Night x EES-D interaction ($F_{(1,25)}$ = 7.16, p = .013) and a significant main effect of the Night ($F_{(1,25)}$ = 9.53, p = .005). Given the significant interaction between the factor Night and the covariate, here too we performed a mixed design factorial 2 x 2 ANOVA considering the Groups defined on the basis of EES-D scores (Low EES-D vs High EES-D) x Night (HN vs DN) on consumed carbohydrate content. Results revealed a significant Night x Group interaction ($F_{(1,26)}$ = 5.96, p = .022) and a main Group effect ($F_{(1,26)}$ = 4.31, p = .048). Simple effects revealed that after the Sleep Deprivation Night, the Low EES-D group consumed significantly fewer carbohydrates (M = 70.96 ± 31.66) compared to the Habitual Night (M = 56.64 ± 20.95, t = 2.77, p = .016). Moreover, after the habitual night, the Low EES-D group consumed more carbohydrates (M = 79.96 ± 31.66) compared to High EES-D group (M = 41.03 ± 29.02, t = 2.70, p = .015). The main effect of group

indicated that the Low EES-D group consumed overall more carbohydrates (M = 63.80 ± 25.05) compared to the High EES-D group (M = 42.526 ± 29.048). Results are displayed in Figure 2.

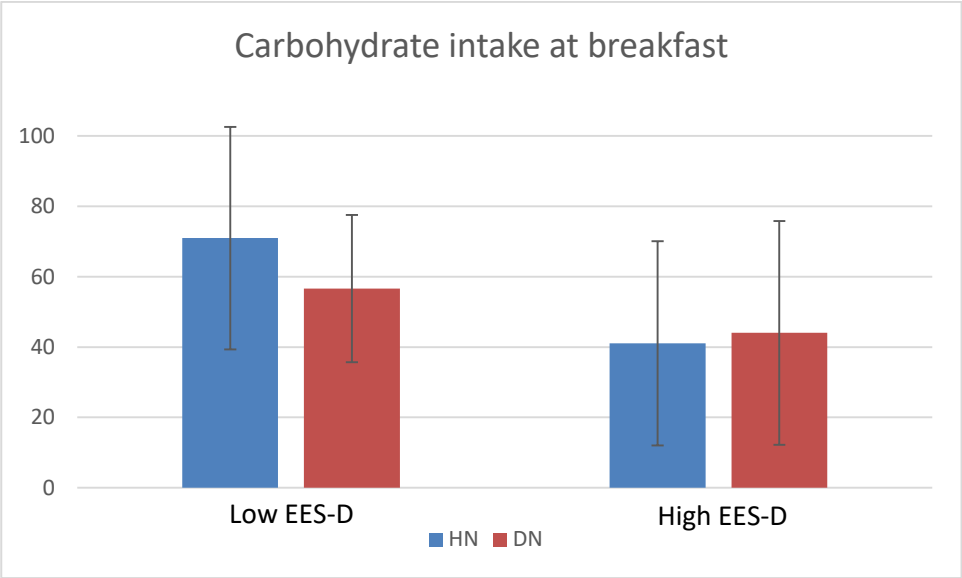


Figure 2. Carbohydrate content consumed at breakfast by Low EES-D and High EES-D.

A 2 x 2 mixed design ANCOVA on fats intake evidenced a main effect of the Night ($F_{(1, 26)}$ = 5.09, p = .033) and a significant interaction Night x EES-D ($F_{(1,26)}$ = 4.28, p = .049). Following the same procedure, a mixed design ANOVA showed no significant results. Only a marginal interaction Night x Group ($F_{(1,26)}$ = 2.74, p = .110) was found. Simple effects showed that the Low EES-D group tended to consum less fats after the Deprivation Night (M = 15.51 ± 10.02) compared to the Habitual Night (M = 18.45 ± 11.44, t = 1.83, p =.090).

A 2 x 2 mixed design ANCOVA on fibre intake revealed a main effect of the Night ($F_{(1,26)}$ = 7.12, p = .013) and no interaction between Night and the covariate. Both Groups consumed less fibre after the Deprivation Night (M = 2.69 ± 0.325) compared to after the Habitual Night (M = 3.43 ± 0.41). No significant results were found on proteins intake during the Test Meal in the lab.

Since during the DN participants had more time before going to bed (at 1 AM) and therefore more time to eat, an additional analysis was performed on the number of snacks and caloric content

consumed during the evening (after dinner and before going to sleep) preceding the HN and the DN. No differences were found between the Nights and the Groups in both number or Kcal consumed.

2.4.3 Daily Food Intake

The 2 x 2 mixed design factorial ANOVA Group (Binge Eating Group vs Control Group) x Night (HN vs DN) performed on the Total Daily Energy revealed no significant result either for the Night ($F_{(1,26)}$ = .013, p = .910), for the group ($F_{(1,26)}$ = .31, p = .580), or for the interaction Group x Night ($F_{(1,26)}$ = 2.18, p = .151). The means of the two groups seemed to differ as shown in Figure 3. The Control Group ate more (not significantly though) after Sleep Deprivation compared to the Habitual Night.

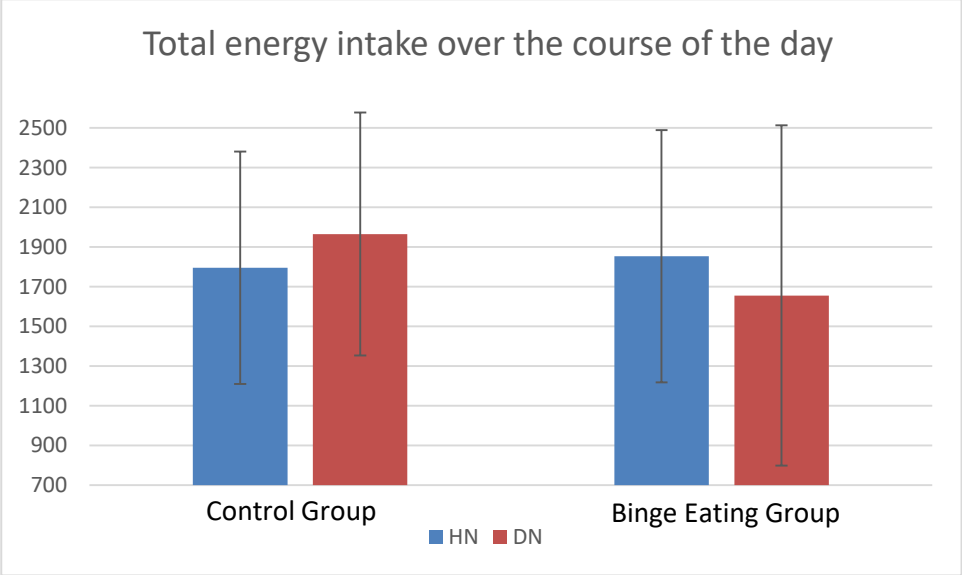


Figure 3. Total Daily Intake (Kcal) of Binge Eating Group and Control Group

A marginal significant main effect of the Group ($F_{(1,26)}$ = 4.05, p = .055) was found on daily fibres intake. The Binge Eating Group consumed overall less fibre (M = 11.60 ± 4.13) than the Control Group (M = 14.83 ± 4.37). No significant results were found on carbohydrates, fats and proteins content.

2.4.4 Daily Food Intake controlling for Emotional Eating

The 2 x 2 mixed design factorial ANCOVAs did not reveal any significant main effects or interactions on total energy and macronutrients consumed during the day. Nevertheless, a 2 x 2 mixed design factorial ANOVA with Group (Low EES-D vs High EES-D) x Night (HN vs DN) performed on Total Daily Energy intake highlighted a significant Night x Group interaction ($F_{(1,26)}$ = 4.28, p = .049), however, the main effect of the Night and the Group were not significant. Simple effects revealed that the Low EES-D group ate significantly more after the Deprivation Night (M = 1907.42 ± 611.17) compared to the Habitual Night (M = 1673.36 ± 507.565, t = -2.44, p = .030). Figure 4 shows the Means and Standard Deviations of the Total Daily Energy intake during the day in the two groups after both nights.

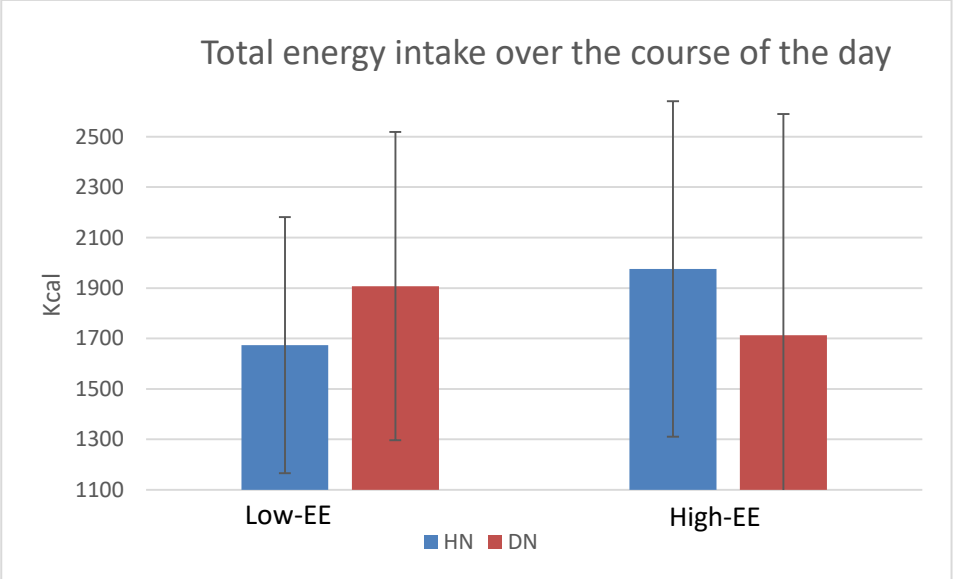


Figure 4. Total Daily Intake (Kcal) consumed by Low EES-D and High EES-D

Results on daily carbohydrate intake revealed a marginally significant Night x Group interaction ($F_{(1,26)}$ = 4.21, p = .051). Simple effects revealed no significant differences, although the means seemed to be in line with the results in terms of total energy intake: the Low EES-D group tended to consume more carbohydrates after sleep deprivation (M = 217.22 ± 91.45) than after a habitual night of sleep (M = 191.81 ± 77.34, t = -1.66, p = .120). The same marginally significant interaction

Night x Group was found on protein content ($F_{(1,26)}$ = 4.19, p = .051). The analyses of simple effect revealed that after the HN, High EES-D ate more proteins (M = 80.61 ± 36.07) than Low EES-D (M = 55.39 ± 27.31, t = -2.09, p = .047). A marginal difference (t = -2.01, p = .066) was also found between the proteins content consumed by Low EES-D after the DN, that was higher (M = 72.58 ± 27.72) compared to the HN (M= 55.39 ± 27.31). Analyses performed on fat and fibre intake did not reveal any differences, however the group means also consistently displayed a similar pattern with the low EES-D group consuming more after sleep deprivation than after the habitual night of sleep.

2.4.5 Daily Snacks

A 2 x 2 mixed design factorial ANOVA Group (Binge Eating group vs Control Group) x Night (HN vs DN) on the number of Snacks consumed in the subsequent day revealed a significant effect of the Night ($F_{(1,26)}$ = 7.12, p = .013). After the Sleep Deprivation Night, both groups consumed more Snacks (M = 2.32 ± 1.76) compared to after the Habitual Night (M = 1.68 ± 1.25). However, findings regarding the Total Energy derived from snack consumption throughout the day did not reveal any significant effects, except for a marginal effect of the Group ($F_{(1,26)}$ = 3.72, p = .065). The control Group ate overall more (M = 315.15 ± 206.87) compared to Binge Eating Group (M = 189.57 ± 128.42). A marginal main effect of the Group ($F_{(1,26)}$ = 3.77, p = .063) on carbohydrate intake from snacks was also found. Specifically, the Control Group consumed more carbohydrates (M = 39.13 ± 24.77) compared to the Binge Eating Group (M = 22.32 ± 20.88).

2.4.6 Daily Snacks controlling for Emotional Eating

The mixed design factorial ANCOVAs revealed no significant effects in terms of Total Energy intake from snacks, or their fat, carbohydrate, and protein content. Results in terms of fibre content controlling for EES-D, showed a significant main effect of the Night ($F_{(1,25)}$ = 4.44, p = .045). Thus, after the Sleep Deprivation Night both groups reported more fibre intake from daily snacks (M =

1.83 ± 2.96) than after the Habitual Night (*M* = 1.23 ± 1.72). No significant results emerged using groups based on EES-D.

2.5 Discussion

This study was the first to evaluate the effect of partial sleep deprivation on food intake in participants reporting symptoms of binge eating vs healthy controls, controlling for the role of emotional eating. We hypothesized that sleep deprivation would increase caloric intake both at breakfast in the lab and during the day especially in participants reporting binge eating symptoms. Overall our findings provided only partial support for this. We found that sleep deprivation may increase snack consumption regardless of binge eating symptoms. In addition, our findings revealed that daily food intake may increase after sleep deprivation in individuals who do not report emotional eating.

However, neither the group nor the sleep deprivation predicted differences in the caloric intake as hypothesized. The only exception was the effect of sleep manipulation on the amount of fibre consumed during breakfast in the lab and in the number of snacks consumed during the day after sleep deprivation and a marginally significant effect of the Group on the snacks consumed during the day. Our results are partially consistent with a study by Nedeltcheva et al. (2009), who found that sleep curtailment (5.5 h) was followed by increased intake of calories from snacks. However, the effect of partial sleep deprivation on Kcal intake in our study was not significant. Probably the participants ate more frequently light or diet food, thus resulting not increasing the total energy consumed.

These results, however, should be interpreted taking into account also emotional eating, as suggested for instance by Dewck et al. (2014). We found that sleep deprivation affected mainly the group with low emotional eating: individuals reporting low emotional eating in presence of depressive mood consumed significantly fewer Kcal and carbohydrates during breakfast and more Kcal and carbohydrates during the whole day after sleep deprivation compared to the day following

the habitual night. This finding is in contrast with our expectation that the sleep manipulation would be associated with increased food intake in both groups, but particularly in the group reporting binge eating symptoms and high depressive emotional eating. However, this pattern of results among healthy controls is in line with previous results by Markwald et al. (2013) who showed that after sleep loss, participants ate smaller breakfasts but ate more carbohydrates, proteins, and fibres over the course of the following day, especially at night after dinner. In general, these results are also consistent with the findings by Al Kathib et al. (2016) regarding the effect of partial sleep deprivation in increasing food intake in healthy participants.

In contrast with our expectations, however, we did not find the same pattern of results among individuals reporting binge eating and with high scores of emotional eating. Similarly, a previous study by Dingemans et al. (2009) that aimed to experimentally test the induction of negative and positive mood on caloric intake of snacks in a laboratory setting in individuals with and without Binge Eating Disorder, found no main effect of the mood induction and of the group. Moreover, participants with high scores of emotional eating overall consumed fewer Kcal and carbohydrates during breakfast and from snacks during the day, compared to individual with low scores of emotional eating. A number of factors may help to account for these unexpected findings. First, our study employed an ecological partial sleep deprivation induction (5 h) and this may not have been sufficient to provoke an increase in food intake among individuals with binge eating symptoms who are known to report higher rates of insomnia severity (Trace et al., 2012). It is possible that at least some of the participants in this group were used to be chronically sleep deprived and that a reduction of sleep to 5 hours was insufficient to result in increased negative affectivity and higher food intake. Second, these individuals may skip breakfast more often that controls and this may explain why they consumed fewer calories during breakfast in the lab. A previous study conducted by Harvey et al. (2011) revealed that individuals with binge eating may skip breakfast more often than any other meals and have irregular eating patterns. Third, as suggested by a recently published

experimental study (Kemps et al., 2016), restrained eaters who expected to eat high-calorie foods may be able to activate their dieting goal, thereby limiting their food intake. Given that our participants were asked to maintain their daily habits and behaviours, this may have triggered restrictive goals during the days of the study, resulting in eating overall fewer Kcal and carbohydrates during breakfast and during snacks. Indeed in our study, participants in the binge eating group reported also higher scores on the Disordered Eating Questionnaire that is sensitive not only to binges but also to eating restriction in the past month. In addition, since it is well know that individuals who report binge eating and emotional eating often eat and overeat when they are alone (APA, 2013), social desirability and shame related to eating in the lab as well as underreporting on food journals may also have contributed to these findings. However, when in the final debriefing participants were asked if they were aware of the food intake measurement during the breakfast in the lab and if it could have influenced their choices, part of them (N = 12) reported to be aware but anyone reported to be influenced by this knowledge in their food consumption (N = 0), at least they were not willingly to admit it openly.

3 The effect of sleep deprivation on executive functions

3.1 Introduction

Executive functions (EFs) are commonly described as a set of cognitive processes involved in different psychological processes such as attention, memory, and emotion (Miyake et al. 2010). Some studies showed that EFs impairment has been linked to increased food intake in both children and adults (Lundhal & Nelson, 2015) even when the hunger is absent (Pieper & Laugero, 2013). Results on adults demonstrated that poor executive control was associated with higher caloric consumption, especially when the reinforcing value of the food was high (Rollins et al., 2010) and was associated with greater consumption of snacks and high-fat foods (Hall, 2012). Consistently, results on children showed that poor EFs proficiency was related to a greater consumption of high calorie snacks (Riggs et al., 2010, 2012), whereas higher EFs proficiency was associated with the consumption of fruits and vegetables (Riggs et al., 2012). Although emerging evidence suggested that executive function underlie eating behaviour, the cognitive processes underlying binge eating and BED are currently poorly understood (Van den Eynde et al., 2011). For example, a recent study by Kittel et al. (2017) investigating EFs in adolescents with and without BED and obesity, found that adolescents with BED and obesity displayed significantly poorer inhibitory control compared to normal-weight adolescents.

Parallel, some experimental findings suggested that sleep deprivation markedly impairs EFs (Cuyoumdjian et al., 2010; Gorgoni et al., 2014). A study by Cedernaes et al. (2014) found that total sleep deprivation impaired cognitive control in response to food stimuli in healthy young men, increasing the impulsivity in response to such food cues.

Taken together, these findings may suggest that one mechanism by which disrupted sleep patterns may increase food intake is via impairment of executive functions (Lundhal & Nelson, 2015).

In the present study we aimed to evaluate if: 1) sleep deprivation may impair EFs; 2) there were difference in EFs between the two groups; 3)the potential executive dysfunction after sleep deprivation might mediate the hypothesized increase in food intake.

3.2 Executive function assessment

The study followed the same procedure of a previous study (Ballesio et al., 2017). Participants were asked to complete a Task Switching (Sdoia & Ferlazzo 2008), which was used to assess the executive processes of switching attention and backward response inhibition. Participants sat in comfortable seat in front of a computer screen. They were instructed to apply three different rules (indicated as A, B, C) in consecutive trials, presented in rapid succession according to random sequences of triplets of rules (A-A-A, A-B-A, C-B-A). For each trial, a geometric figure (a circle, a rhombus or a square) appeared in the screen. Then, a number (from 1 to 9) appeared inside the geometric figure. Based on the geometric figure (which indicated the rule to apply), participants were asked to judge whether the number was odd or even; bigger or smaller than five or central (3, 4, 6, 7) or extreme (1, 2, 8, 9). Number 5 was never shown. On one hand, the performance in repetitive trials, in which the rule to apply is the same as the previous one (e.g. A-A-A) is better than the performance in non-repetitive trials, in which participants are asked to switch from one rule to another (A-B-A, C-B-A). This switch cost is generally interpreted as the reconfiguration of the cognitive resources available by the control processes (e.g. Logan, 2003; Monsell, 2003). On the other hand, switching back to a recently executed task is harder than switching back to a less recently executed task.

The Task Switching paradigm allowed to compute two indices of performance: the Switch Cost Index, which measures reflect increased mean reaction times on the third trial of switching triplets (A-B-A,C-B-A), vs repetition triplets (A-A-A), and the Backward Inhibition Index, which reflects slower mean reaction times on alternating trials (A-B-A) vs non-alternating trials (C-B-A) sequences (Ferlazzo et al., 2014).

3.3 Statistical analyses

First, mean reaction times were checked for errors. The Switch Cost Index and the Backward Inhibition Index were calculated. All the scores were re-calculated excluding the errors. Since data were normally distributed, we used parametric tests. Moreover, one subject reported only 11 correct responses after the habitual night and 6 correct responses after the deprivation night, while the mean of the correct responses in both groups was 52 for the HN and 51 for the DN. For this reason, this subject was excluded from the analyses. Two separated mixed design factorial ANOVAs Group (Binge Eating Group vs Control Group) x Night (HN vs DN) were performed on the number of correct responses, Switch Cost Index and on the Backward Inhibition Index. After that, two further ANCOVAs were performed on the same variables inserting BDI-II score as covariate. This was done since previous literature evidenced poor EFs in depression (Snyder et al., 2015).

Given the significant effect found on Backward Inhibition, and in order to test the potential mediating effect of this EF on food intake, we performed two different mediation models using the SPSS macro PROCESS (Hayes, 2012). We finally explore the indirect effect through one-tail bivariate correlations between Backward Inhibition and total energy at breakfast, dividing per group.

3.4 Results

3.4.1 Correct responses, Switch Cost and Backward Inhibition

First, no significant results were found on the number of correct responses and on Switch Cost Index, while a significant interaction Night x Group emerged on the Backward Inhibition Index (table 1).

	Number of correct responses	Switch Cost Index	Backward Inhibition Index

	$F_{(1,25)}$	p	$F_{(1,25)}$	p	$F_{(1,25)}$	p
Effect of the Night	2.87	.103	0.43	.518	0.03	.864
Interaction NightxGroup	0.03	0.87	0.75	.785	4.68	**.04**
Effect of the Group	0.25	0.62	0.53	.473	1.13	.297

Table 1.Results of mixed model ANOVAs on Switch Cost Index and Backward Inhibition Index.

The Fisher least significant difference (LSD) post hoc test revealed that Bing Eating Group differed significantly from Control Group in the HN ($F_{(1,25)}$= 5,93, p= .020). LSD post hoc test comparing the two nights did not evidence significant difference in the Control Group ($F_{(1,25)}$= 1.91, p= .170) and in the Binge Eating Group ($F_{(1,25)}$= 2.84, p=.105).

Furthermore, as can be derived from Figure 2, a 2X2 ANCOVA inserting the BDI-II as covariate, the same interaction Night x Group ($F_{(1,24)}$= 6.96, p= .014) was found. LSD post hoc tests showed that the significant difference between Groups in the HN remained ($F_{(1,24)}$= 6.32, p= .019). Moreover, LSD post hoc test comparing the two nights revealed a significant difference in the Backward Inhibition in the Binge Eating Group ($F_{(1,24)}$=4.69, p= .040), and a marginal difference in the Control Group in the opposite direction ($F_{(1,24)}$= 3.60, p= .070).

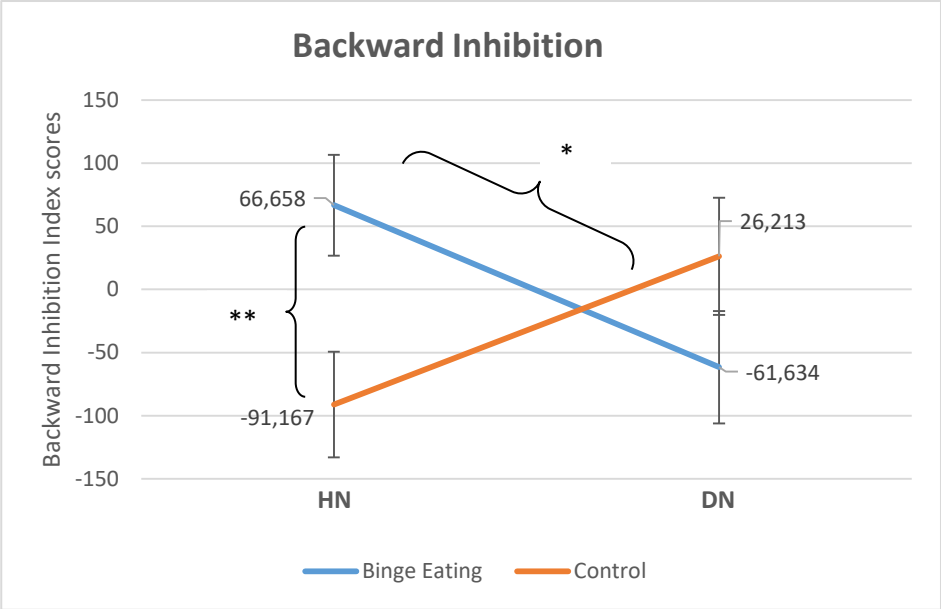


Figure 1. Interaction Night x Group in the Backward Inhibition scores, controlling for depression.

These results suggest that after the DN the Backward Inhibition is lower in the Binge Eating Group, whereas it is higher in the Control Group, though this difference is not significant.

No other results were found on the other variables controlling for BDI-II.

3.4.2 The mediation of EFs on food intake at breakfast

In order to evaluate the putative mediation role of EFs on food intake after both nights, we computed two mediation models inserting the score of Backward Inhibition as mediator.

The models included the Group (Binge Eating vs Control) as independent variable, the score of Backward Inhibition as mediator, and the Total Energy intake at breakfast, separately for each night, as dependent variable.

As can be derived by Figure 2, given the significant indirect effect found, the mediation model in the HN was significant. In fact, the Group predicted the total energy consumed at breakfast, through the mediation of the Backward Inhibition.

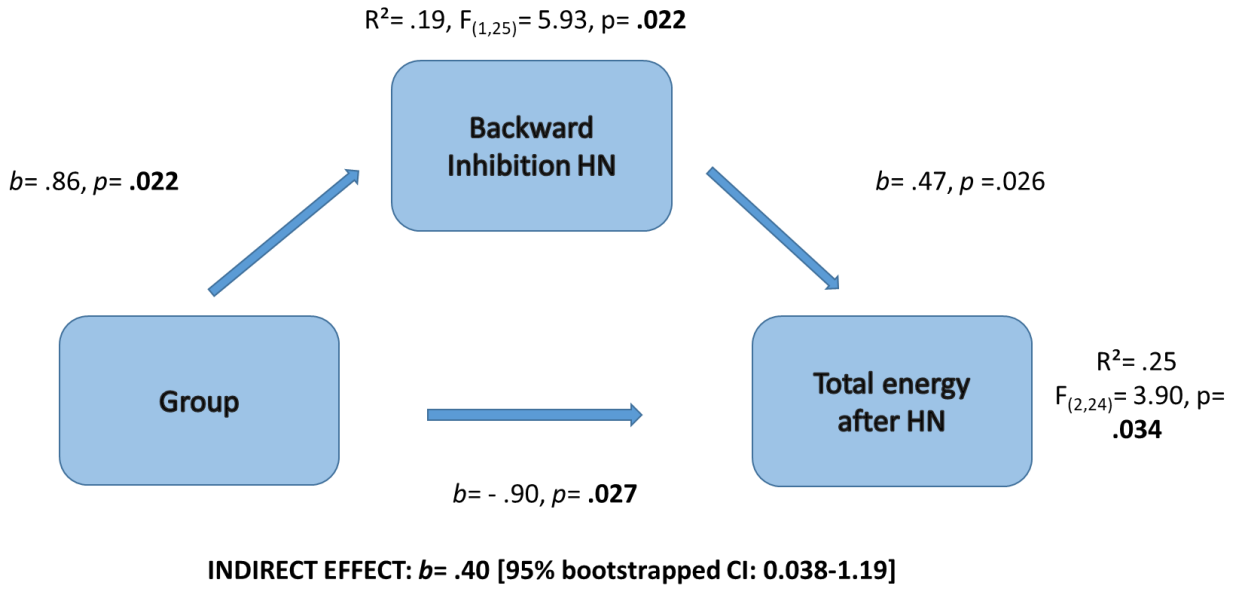


Figure 2. Mediation model in the Habitual Night.

In order to explore this indirect effect, one-tail bivariate correlations divided per group showed that Backward Inhibition after the HN was positively correlated with Total Energy after HN only in Binge Eating Group (r= .535, p= .024), thus evidencing more backward inhibition associated with more caloric intake.

On the contrary, the second model was not significant, showing no mediation of EFs on food intake after sleep deprivation.

3.5 Discussion

To our knowledge, this is the first study evaluating the effect of sleep deprivation on EFs in a sample of individuals reporting binge eating symptoms. Findings revealed that sleep deprivation had an impact on EFs, particularly on Backward Inhibitions as reported by previous studies (Cuyoumdjian et al., 2010; Gorgoni et al., 2014). Moreover, the Backward Inhibition was impaired in individuals reporting binge eating symptoms but not in the control group, which showed the inverse pattern of results, though this trend was not significant. In explanation of this result, we can speculate that the Control Group employed more effort after the deprivation night in order to compensate the deficits or to cope with the sleepiness. These efforts may have paradoxically

improved their performance in the Task Switching. Moreover, no main effect of the group was found as previous studies did, that observed a relative EFs weaknesses in inhibitory control in overweight and obese participants with BED (Manasse et al., 2014; Mobbs et al., 2011; Svaldi et al. 2014) and an association between increased BMI and poorer executive function test performance (Gunstad et al., 2007). Results on Switch Cost revealed no difference between groups and between nights as in other studies, that did not report any EFs impairments (e.g. Monica et al., 2010; Wu et al., 2013). Probably, in the present study the sleep manipulation (5 hours of sleep allowed) was not big enough to induce the expected impairment also on this EF.

The mediation analyses performed on food intake at breakfast revealed a positive influence of Backward Inhibition in increasing food intake after a night of habitual sleep, only in individuals reporting binge eating, while this mediation effect was not significant at all after sleep deprivation in both groups. This may be explained in different ways: 1) probably, after a night of habitual sleep, having more control on their cognitive processes, as switching from a task to another and inhibiting the previous task, may be connected with more cognitive flexibility, thus translating in more propensity to consume more food during a buffet ad libitum. On the contrary, when they felt disinhibited, they may activate their restrictive goal, especially in social context, when are not alone. 2) The assessment of backward inhibition did not reflect a direct measure of the behavioural or impulsive control (this is only an indirect measure), thus limiting our capacity to extend this result to the real life, outside the laboratory. 3) In addition, only one measure of EFs (the Task Switching) has been used in this study, thus limiting our capacity to extend the findings to other cognitive processes involved. The small sample size and the inter-variability may have strongly influenced our results.

Taken together these results evidence the need of further studies investigating the effect of sleep deprivation of EFs and their potential role in increasing food intake, especially in individuals reporting disordered eating symptoms.

4 The effect of sleep deprivation on craving

4.1 Introduction

Food craving is commonly defined as an intense desire to eat a particular food (Hill, 2007). It has been differentiated from hunger since food craving tends to be more intense and more specifically related to particular foods (Hill, 2007). For example, in Western societies, these foods usually are energy dense (high fat and/or sugar content) and have high palatability (Rodríguez-Martín and Meule, 2015). Moreover, food craving can be considered as a multidimensional experience that includes cognitive (e.g. thinking about food), emotional (e.g. desire to eat or changes in mood), behavioural (e.g. seeking and consuming food), and physiological (e.g. salivation, arousal) aspects (Cepeda-Benito et al., 2000; Nederkoorn et al., 2000).

State craving changes has been observed after visual or imaginative exposure to food stimuli (e.g. Bullins et al., 2013; Lombardo et al., 2016). Different levels of craving may also affect the physiological state, such as the cardiac reactivity (Frings et al., 2015) and they may be associated with neural activation related to reward and motivational salience (Ulrich et al., 2016).

Moreover, as largely discussed in the previous chapters, an increase of food intake and hunger related hormones alterations have been demonstrated after sleep deprivation (Al Khatib et al., 2016). However, findings on self-reported hunger and craving after sleep loss, independently of food intake, are very controversial (Markwald et al., 2008; Omisade et al., 2010; Schmid et al., 2008).

In parallel, several studies found higher levels of food craving in individuals with eating disorders such as bulimic disorder (Van den Eynde et al., 2012) and binge eating disorder (Ng & Davis, 2013) compared to control groups. Lombardo et al. (2016) found that eating disorders symptoms positively correlated with different aspects of food craving, especially with the fear of losing control

over food intake. In fact, the loss of control is a crucial symptom of binge eating behaviour (APA, 2013).

Given the literature summarized above, this study aimed to evaluate the effect of a night of partial sleep deprivation on food craving, evaluating if it differed between nights and between groups, before and after a Craving Induction Task. We further hypothesized that Binge Eating Group would be more sensitive to food craving and food craving changes after sleep deprivation. Finally, we aimed to investigate the potential mediating role of food craving, particularly related to the sense of lack of control, on food intake at breakfast after both nights.

4.2 Craving assessment

Both groups were assessed before breakfast during and after a Craving Induction Task. Participants were presented with 3 blocks of images: 12 neutral stimuli (non-food stimuli) selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2005), 12 images of high palatable sweet foods (e.g. cake, ice-cream, chocolate etc., see Figure 1), and 12 images of high palatable salty foods (e.g. pasta, sandwich, chips etc., see Figure 2). The first block presented was always the neutral one, while the second and the third blocks were counterbalanced across participants and between nights.

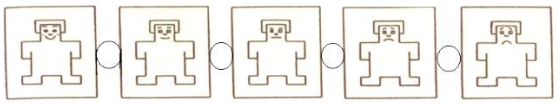
Figure 1. Example of high palatable sweet food

Figure 2. Example of high palatable salty food

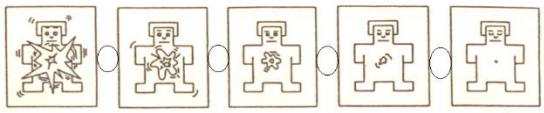
Participants were asked to evaluate the valence, arousal and the craving evoked by each block of images before and after being exposed to each block of images. Valence and arousal were evaluated using the Self assessment Manikin (SAM, Bradley & Lang, 1994; Image 1). Craving was assessed asking participants to indicate how much they desired to eat their favourite foods. Valence, arousal and craving were first assessed before starting the Craving Induction Task, They were then invited to observe each image for how long they wanted and to press the spacer button on the keyboard to proceed with the next image. As the pause screen appeared after each block, they were instructed to complete the questions and the SAM. Images were presented on a computer screen by Super Lab Pro, version 2.0 (by Cedrus, https://cedrus.com/) that also recorded time spent by participants viewing each image.

Valuta sulle seguenti scale quanto:

ti senti in uno stato **piacevole** o **spiacevole**



ti senti **attivato** o **calmo**



Valuta, tracciando un segno verticale sulla linea seguente, quanto è forte il desiderio di *consumare uno o più dei tuoi cibi preferiti*:

Figure 3: Analogic scales presented before and after each block of images.

During image inspection, skin conductance and heart rate were also recorded using a portable device by Satem (Psycholab_VD 13SV model and Pc-Soft, Satem software, version 5.0). Each participant sat in a comfortable seat in front of a 17 inches screen in which the images were presented. Before beginning the Craving induction task three electrodes were located on the participant's body, two on both wrists and one on the left handbreadth, for recording Heart Rate (HR). Two other electrodes were located on the last phalanx of the index and middle fingers of the participant's dominant hand for recording Skin Conductance (SC) Level. Before and after the Craving Induction Task, participants were also asked to complete a self-report questionnaire: the Food Craving Questionnaire-State (FCQ-S; Cepeda-Benito et al., 2000; Italian version by Lombardo, Iani & Barbaranelli, 2016). It measures the food craving state, which could be experienced when a temporary increase of the food craving occurs, especially following specific or stressful situations (e.g., food deprivation, stressful events; Cepeda-Benito et al., 2000). The FCQ-S consisted of 15 items belonging to 5 different subscales: intense desire to eat (1,2,3); anticipation of positive reinforcement that may result from eating (4,5,6); anticipation of relief from negative states and feelings as a result of eating (7,8,9); lack of control over eating (10,11,12) and craving as a physiological state of hunger (13,14,15). The validity of the Italian version of the FCQ-S and its sensitivity to state changes in food craving following food stimuli exposure have been demonstrated (Lombardo et al., 2016). Cronbach's αs of all the subscales pre and post exposure showed good internal consistency.

4.3 Statistical analyses

A 2x2x2 mixed design factorial ANOVAs Night (HN vs DN) x Group (Binge Eating Group vs Control) x Stimulus (sweet vs salty) was performed on inspection times of each block of images. In this case, we decided to exclude from the analyses the neutral stimuli exposure times since it was always presented as the first block of images and was voluntary not counterbalanced. Then, several 2x2x4 Night (HN vs DN) x Group (Binge Eating Group vs Control) x Stimulus (baseline vs neutral stimuli vs sweet foods vs salty foods) were performed on self-reported valence, arousal and desire to consume a food. Moreover, two separate mixed design factorial ANOVAs Night (HN vs DN) x Group (Binge Eating Group vs Control) x Stimulus (neutral stimuli vs sweet foods vs salty foods) were performed on physiological indices of skin conductance (SC) and heart rate (HR). In the analyses of the physiological indices, for SC one subject's record from the Binge Eating Group was not available due to technical problems and for HR, one subject's record from Control Group and two subjects' record from Binge Eating Group were not available due to technical problems. For these reasons, analyses conducted on physiological indices did not include data from these participants.

After that, five 2x2x2 mixed design factorial ANOVAs Night (HN vs DN) x Group (Binge Eating Group vs Control) x Moment (pre vs post exposure) were performed considering respectively each scale of the FCQ-S. Finally, two mediation analyses were performed using the macro PROCESS on SPSS (Hayes, 2012) separated for night, inserting the groups as independent variable, the score of Lack of Control after the Craving Induction Task as mediator, and the Total Energy consumed at breakfast as dependent variable.

4.4 Results

4.4.1 Times of inspection

Results on times of inspection of neutral stimuli, sweet and salty food-stimuli did not reveal any significant effect or interaction.

4.4.2 Self-reported valence, arousal and desire to consume food

Results on self-reported valence showed a main effect of the Stimulus ($F_{(3,24)}$= 11.87, p < .001). Post hoc tests revealed that both groups rated sweet foods as the most pleasant (M= 5.96 ± 0.23) compared to baseline (M= 5.45 ± 0.23, p < .001), neutral stimuli (M= 5.04 ±0.24, p < .001) and salty foods (M= 5.61 ± 0.23, p = .012). Salty foods were rated as more pleasant than neutral stimuli (p = .011).

The same main effect of the Stimulus was found on self-reported desire to consume food ($F_{(3,24)}$= 23.47, p <.001). Post hoc test revealed that sweet foods are rated as the most desire eliciting (M= 5.27 ± 0.37) compared to baseline (M= 4.16 ± 0.34, p <.001), neutral stimuli (M= 3.96 ± 0.36, p <.001) and salty foods (M= 4.72 ±0.38, p= .008). Participants reported more desire to consume food after the view of salty food images compared to baseline (p = .017) and to neutral stimuli images (p= .003).

Results on self-reported arousal evidenced a different pattern of results. A main effect of the Stimulus was significant ($F_{(3,24)}$= 8.08, p< .001), though baseline was rated as the most arousing (M= 5.05 ± 0.22) compared to neutral stimuli (M= 4.39 ± 0.21, p= .001), sweet stimuli (M= 4.54 ±0.26, p= .001) and salty stimuli (M= 4.71 ± 0.25, p= .033). The other differences were not significant. Only a marginal effect was found between salty and sweet stimuli (p= .056): participants rated as more arousing salty foods than sweet foods.

4.4.3 Craving state changes before and after exposure

Results of mixed design factorial ANOVA Group (Binge Eating Group vs Control Group) x Night (HN vs DN) x Moment (pre vs post exposure) on each scale of the FCQ-S, showed that after exposure, all participants reported higher Lack of Control (main effect of the Moment: $F_{(1,26)}$=6.23, p=.019). A main effect of the group was also found ($F_{(1,26)}$=5.81, p=.023): Binge Eating Group reported higher Lack of Control compared to Control Group after both nights. LSD post hoc tests

following a marginal interaction Night x Group ($F_{(1,26)}$= 3.06, p= .095) revealed that Binge Eating Group reported higher Lack of Control after DN (M= 5.07 ± 0.49) compared to after HN (M= 5.43 ±0.45, p= .005). All participants reported also an higher Intense Desire to Eat ($F_{(1,26)}$= 16.17, p< .001) after the exposure of the Craving Induction Task. No significant results were found on the three subscales related to physiological hunger, anticipation of positive reinforcement and anticipation of relief from negative states.

4.4.4 Physiological indices during the Craving Induction Task

Findings on the physiological index of SC revealed a significant main effect of the Stimulus ($F_{(2,25)}$= 4.79, p= .013) and a marginal effect of the Night ($F_{(1,25)}$= 3.50, p= .073). Regarding the main effect of the Stimulus, LSD post hoc tests revealed that during the exposure of sweet foods SC increased (M= 10.40 ±1.19) compared to neutral stimuli (M= 9.95 ±1.14, p= .024). SC was also higher during the exposure to sweet food compared to salty food (M= 9.98 ±1.14, p= .007). Regarding the marginal effect of the Night: after DN, the mean SC was lower (M= 9.36 ± 1.15) compared to after HN (M= 10.86 ± 1.28). Results on HR did not reveal any significant effect or interaction.

4.4.5 The mediation role of food craving in increasing food intake

In order to evaluate the putative mediation role of food craving on food intake after both nights, we computed two mediation models inserting the score of Lack of Control after the exposure to the Craving Induction Task as mediator, respectively for each night.

The first model included the Group (Binge Eating vs Control) as independent variable, the score of Lack of Control post exposure after the HN as mediator, and the Total Energy intake at breakfast after the HN. As illustrated in Figure 1, results showed that this model was not significant: the mediation hypothesis has not been confirmed. Only the direct effect of the Group on the Total Energy is significant and this is in line with the results presented in the chapter 3 “Food Intake”.

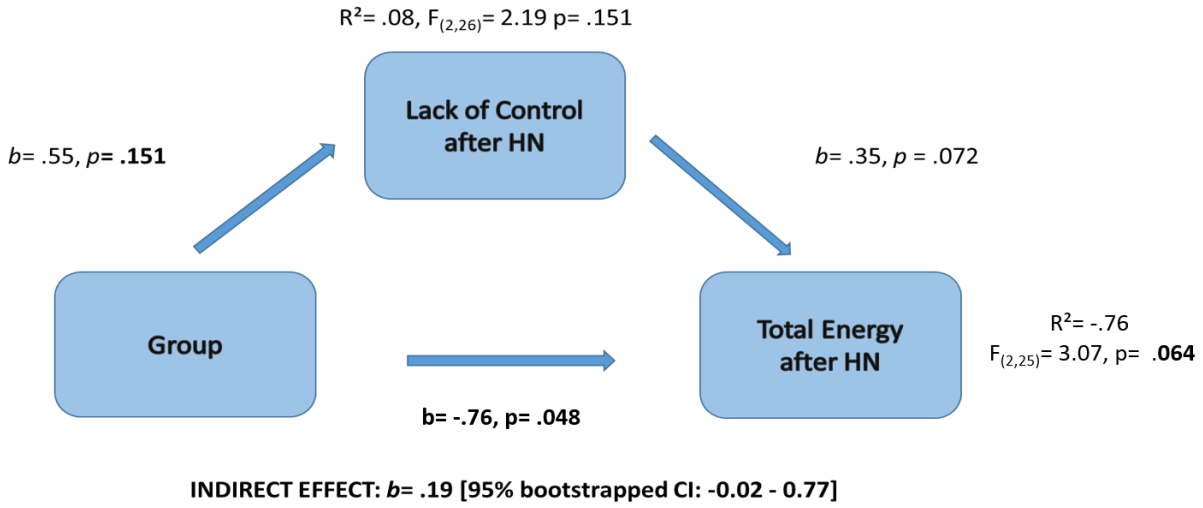


Figure 1. The mediation model for the Habitual Night.

The second model included the Group (Binge Eating vs Control) as independent variable, the score of Lack of Control post exposure after the DN as mediator, and the Total Energy intake at breakfast after the DN as dependent variable. As illustrated in Figure 2, the mediation model is significant.

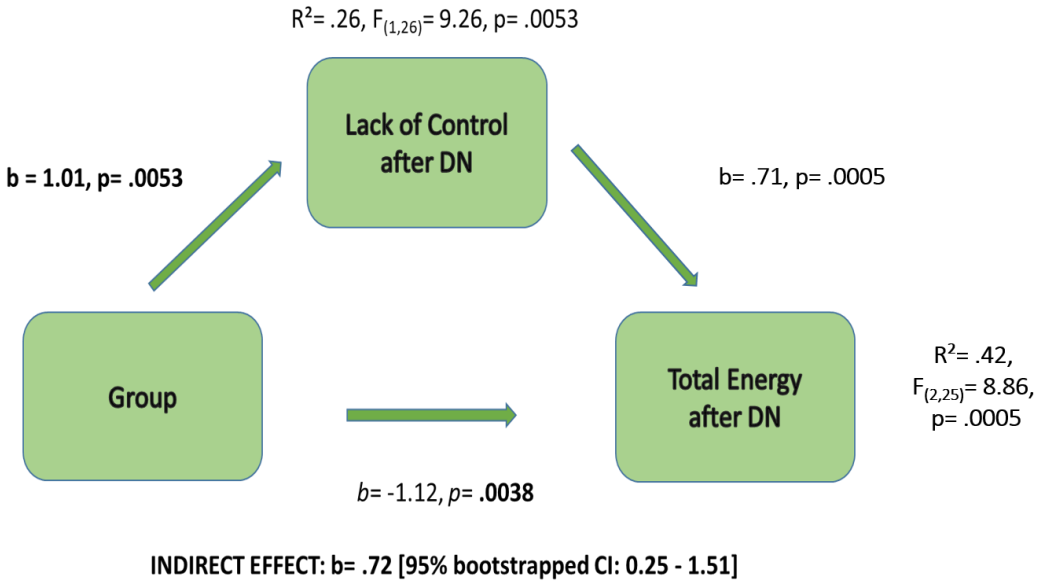


Figure 2. The mediation model for the Deprivation Night

In order to explore this indirect effect, one-tail bivariate correlations divided per group showed that Lack of Control post exposure after the DN was positively correlated with Total Energy after DN in

Binge Eating Group (r= .694, p= .006), while this correlation was only marginal in Control Group (r= .518, p= .058), thus highlighting the important mediating role of lack of control in increasing caloric intake after sleep deprivation in both group, particularly in Binge Eating Group.

4.5 Discussion and conclusion

This study is the first to examine the effect of partial sleep deprivation on craving in participants reporting or not binge eating symptoms. Very few studies previously investigated the physiological activation during food cues exposure, resulting in the alteration of autonomous nervous system functioning indices, such as heart rate and skin conductance (e.g. Yela Bernabé et al., 2013), but no one after sleep deprivation.

In this study, findings revealed an effect of the group on food intake at breakfast after sleep deprivation (Binge Eating Group ate more), mediated by the increase in the lack of control over eating. The analyses conducted on physiological indices showed a trend of lowered skin conductance after a night of partial sleep deprivation, as compared to habitual sleep, probably indicating less physiological activation during the Craving Induction Task.

Although no significant effect or interaction involving the night was found on the Craving Induction Task, this task enhanced the level of craving and valence and the physiological activation (skin conductance) for sweet foods stimuli compared to neutral and salty stimuli. This may be related to hedonic value of sweet food in enhancing craving levels and to the Italians' breakfast preference for sweet foods (such as biscuits, cakes, croissants etc.) compared to salty foods. However, no significant differences were found in the observation times between sweet and salty stimuli.

Finally, the induction of craving was effective since all participants reported higher desire to eat and higher lack of control after the exposure. These significant state craving changes were also reported by previous studies (e.g. Lombardo et al., 2016). Binge eating was more sensitive to the craving induction compared to the Control Group, especially in the sense of lack of control over eating as hypothesized. Moreover, the lack of control experienced after the sleep deprivation

mediated the effect on food intake at breakfast in this group. This is also in line with the results found on EFs, which evidenced a lack of inhibitory control after sleep deprivation in individuals reporting binge eating, compared to a night of habitual sleep.

Further studies investigating physiological activation for food stimuli and craving after sleep deprivation may be useful to understand how homeostatic and hedonic mechanisms may intervene in the relationship of poor sleep with increased food intake and obesity.

5 General discussion and conclusion

This novel experimental study aimed to evaluate the effect of partial sleep deprivation (5 h of sleep allowed) on food intake, taking into account some of the potential moderators and mediators of this effect in a sample of 14 individuals reporting binge eating symptoms and 14 healthy controls denying any eating pathology. Food intake, executive functions and food craving were assessed after a night of habitual sleep and after a night of partial sleep deprivation.

In summary, findings revealed that sleep deprivation partially affected food intake and results may vary between groups and taking into account emotional eating when depression occurs. Moreover, the effect of sleep deprivation was also evident on executive functions (backward inhibition) and food craving (lack of control). The exploratory analyses performed on their mediation role on subsequent food intake at breakfast revealed significant results, partially confirming our hypotheses. However, some of the results found are complex and may need further investigations.

This study offers a novel contribution in understanding the link and the underlying mechanism between partial sleep deprivation and food intake. However, it also has several limitations.

First, the sample size is quite small and included individuals reporting binge eating symptoms, not individuals with a diagnosis of Binge Eating Disorder. This may have affected our results and led to an underestimation of the effect that might have emerged in a larger clinical sample. Moreover, the sleep deprivation induced may not have been sufficient to produce the expected pattern of behaviours in this population, given that this population reports frequent sleep disturbance and poor sleep (Trace et al., 2012). Acute sleep deprivation of less than 4 hours might have been more effective, and would be interesting to investigate in the future. However, the presence of an ecological induction of sleep deprivation and the daily monitoring of sleep and food intake through diaries increased the ecological validity of the study. Another limitation is present in the selection and screening of the sample. We recruited and screened participants through self-report measures of

binge eating and disordered eating symptoms. Further studies should include measures such as clinical interviews, which are considered the gold standard for a valid selection of a clinical sample. More, the intra and inter-individual differences within each group (e.g. habitual eating patterns, chronotype, sleep disorders, etc.) may have contributed to increase the internal variability, thus negatively influencing our results. Future studies should take into account also these variables and use objective measures to monitor sleep as, for example, the actigraphy.

Finally, this study contributes to shedding light on the effect of partial sleep deprivation on food intake in individuals reporting or not binge eating symptoms and emotional eating. Our findings suggest that a single night of partial sleep deprivation may increase the daily food intake in people that habitually do not binge and do not eat in response to negative emotions. On the contrary, people who report high binge eating symptoms and high emotional eating are probably used to experience a lack of sleep, they may need much more sleep curtailment for exhibiting the detrimental effects of sleep loss on eating behaviour. However, we found that after a night of sleep deprivation the food craving, particularly the sense of lack of control experienced after the induction of craving, may act as mediator of the subsequent food intake, especially in individuals reporting binge eating symptoms. More, we found that after sleep deprivation the inhibitory control was lower in individuals reporting binge eating compared to healthy controls. However, the mediation role of inhibitory control in increasing food intake was found only after the habitual night of sleep, in the reverse way (higher cognitive control associated to increase food intake) in the binge eating group. In the future, it would be interesting to test additional sophisticated mediation models that include all the mediators at the same time or moderated mediation models that include mediators and moderators simultaneously. Indeed, these further analyses have not been performed yet due to the small sample size and the large amount of potential variables. Therefore, we have selected only three variables per model. Future studies with larger sample size should address this

limit in order to extend our knowledge on the underlying mechanisms involved in the sleep/increased food intake pathway in people with binge eating.

In conclusion, based on the literature summarized in this dissertation and based on the results of this preliminary study, we can support the idea that sleep is a fundamental factor in influencing eating behavior and eating related processes (e.g. cognitive and emotional) but also many other factors may intervene. Its relationship with obesity surely need further investigations (especially longitudinal ones) to deeply examine the mechanisms involved. However, promoting a good sleep hygiene may be fundamental in preventing obesity and overweight at different ages, particularly in children and young adults. Finally, extending this preliminary results to a broader context, especially considering clinical implications, as improving sleep quality and quantity (e.g. through cognitive behavioural therapy for insomnia, CBT-I, which is considered the gold standard in the treatment of chronic insomnia disorder) may have potential beneficial effect in the treatment of obesity and many mental disorders, such as eating disorders. Similarly, interventions that address emotion regulation may be useful in individuals reporting symptoms of eating disorders, such as binge eating, and sleep problems.

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