doi: 10.1111/obr.12603

Etiology and Pathophysiology

Does stress influence sleep patterns, food intake, weight gain, abdominal obesity and weight loss interventions and vice versa?

N. R. W. Geiker[1], A. Astrup[2], M. F. Hjorth[2], A. Sjödin[2], L. Pijls[3] and C. Rob Markus[4,*]

[1]Clinical Nutrition Research Unit, Copenhagen University Hospital Herlev and Gentofte, Hellerup, Denmark, [2]Department of Nutrition, Exercise and Sports, Faculty of Science, University of Copenhagen, Copenhagen, Denmark, [3]Loekintofood-gcv/scs, Brussels, Belgium, and [4]Neuropsychology and Psychopharmacology, Faculty of Psychology and Neuroscience, Maastricht University, Maastricht, The Netherlands

Received 6 June 2017; revised 6 July 2017; accepted 22 July 2017

Address for correspondence: NRW Geiker, Clinical Nutrition Research Unit, Copenhagen University Hospital Herlev and Gentofte, Kildegårdsvej 28, DK-2900 Hellerup, Denmark. E-mail: nina.rica.wium.geiker@regionh.dk

*Correction added on 04 December 2017, after first online publication: this author's name was corrected to "C. Rob Markus"

Summary

Decades of research have reported only weak associations between the intakes of specific foods or drinks and weight gain and obesity. Randomized controlled dietary intervention trials have only shown very modest effects of changes in nutrient intake and diet composition on body weight in obese subjects. This review summarizes the scientific evidence on the role mental stress (either in or not in association with impaired sleep) may play in poor sleep, enhanced appetite, cravings and decreased motivation for physical activity. All these factors contribute to weight gain and obesity, possibly via decreasing the efficacy of weight loss interventions. We also review evidence for the role that lifestyle and stress management may play in achieving weight loss in stress-vulnerable individuals with overweight.

Keywords: Cortisol, cravings, sleep, visceral obesity.

Abbreviations: BDI, Beck Depression Inventory; GHQ, General Health Questionnaire; HADS, Hospital Anxiety and Depression Scale; HPA, hypothalamic–pituitary–adrenal axis; POMS, profile of mood states; PSS, Perceived Stress Scale.

Introduction

Stress can be defined as a negative emotional experience accompanied by biochemical, physiological and behavioural changes (1). Stress affects the majority of the population, and there is suggestive evidence that stress induces some types of depression (2,3). Stress can be induced not only by many factors, including insecurity in personal, social and professional life, but also by factors such as impaired or insufficient sleep (Table 1). Whether or not people might experience stress primarily depends on the meaning and importance assigned to such potential 'stressors'. In fact, an event becomes especially stressful when it is seen as highly relevant and threatening as well as inescapable. Thus, exposure to a 'stressor' does not inevitably cause stress. Deficiency in vitamin D, niacin, folate, vitamin B6, Vitamin B12 and omega-3 fatty increase the susceptibility to stress and depression (4,5). Furthermore, polymorphisms of the glucocorticoid receptor gene are associated with basal cortisol secretion and increase the vulnerability to stress (6,7). Experience or initiation of stress may be at a specific situation/life time event, i.e. gestational stress, referring to the stress that specifically occurs during pregnancy affecting mother and child, and social stress, referring to stress initiated from relationships or social environment; stress may also become chronic. In chronic stress, responses may

Table 1 List of stressful events and perceived chronic stressors

Major life event (lifetime)	Recent life event (within the last 12 months)	Life trauma	Perceived chronic stress
Parents' conflicts/separation/ divorce	Relationship difficulties	Parental neglect as child	Relationship problems
Loss of parent or other relative	Significant increase in arguments	Childhood/school social and learning problems	Divorce/separation
Loss of child by death or removed	Unfaithful significant other	Physical abuse/assault	Child-related stress
Separation from one or both parents	Divorce or separation	Threatened or witnessed assault	Chronic health/medical problems
Family or significant other with substance abuse issues	Ending of close relationship	Exposure to war or combat	Job/employment-related stress
Educational setback – failure	Death of a loved one	Kidnapping	Financial difficulties
Isolation and abandonment	Child or significant other	Death	Unemployment
Loss of home to natural disaster	Close family friend	Immediate family member or significant other (including suicide)	Legal problems
Victim of violence	Job-related stress	Emotional abuse	School-related stress
Observing violent victimization	Demotion/worse job	Serious accident	Living-situation stress
	Mobbing	Self/witnessed one	
	Transfer	Sexual abuse/assault	
	Pay loss	Trauma related to natural disaster	
	Job loss		
	Financial crisis		
	Bankruptcy		
	Going on welfare		
	Legal difficulties		
	Poor school performance		
	Failure or dropout		
	Living-situation stress		
	Moved to worse neighbourhood		
	Kicked out of house		
	Kicked child out of house		
	Natural disaster – loss of home		
	Physical assault		
	Serious accident or injury		
	House/car broken into		
	Unwanted pregnancy		
	Abortion/miscarriage		

The table is the authors' modification of the 'Cumulative Adversity Interview' by Sinha A & Jastreboff AM (11).

become accustomed while the stressor occurs or may persist long after the occurrence or even in absence of the stressor.

The high risk for weight gain, and particular accumulation of visceral adipose tissue, during chronic stress is thought to be related to activation of the hypothalamic–pituitary–adrenal axis (HPA-axis), sympathoadrenal activity as well as a negative affective consequence (negative mood causing 'emotional eating') (8–15). In obese individuals, chronic mental stress increases the risk of co-morbidities (16,17). It is a very old observation that abdominal obesity and the metabolic syndrome are tightly correlated to both mental stress and an unhealthy lifestyle composing of a poor diet, smoking, alcohol and lack of physical activity (18). Yet even though this stress/body weight relationship is gaining scientific approval, its potential causal role in clinical obesity seems to be grossly overlooked, in so far as there are only a few intervention studies that have examined the efficacy of stress management for success of obesity management.

The complex interaction among environmental, psychological, nutritional, societal and biological factors is absent in the scientific focus on stress as a potential cause of obesity. Many health professionals have identified certain foods and drinks as causal factors promoting obesity in stressed individuals and populations, thus potentially overlooking the real aetiology of obesity – and its real solution. We posit that an increased craving for, and consumption of, certain palatable foods and drinks high in carbohydrates and fat may only be mediators, not causes, and excessive focus on food and drink intake diverts attention from societal solutions that will prove effective.

The purpose of this review is to summarize the scientific evidence on the role mental stress (either or not in association with impaired sleep) may play in poor sleep, enhanced appetite, cravings and decreased motivation for physical activity. The concept may also contribute to explain the findings from both observational studies and randomized controlled trials that the effect on body weight of, e.g. high-fat sweet and/or savoury foods, is minor because effects are diluted by a large (unstressed) part of the population who do not gain weight or become obese even though they consume palatable foods. Also this review identifies major research gaps by indicating that studies are needed to determine whether or not interventions that reduce stress experience also facilitate body weight control in individuals with high stress vulnerability and to determine whether or not programmes combining stress management with dietary-related weight loss strategies might be more successful in stress-vulnerable individuals with overweight.

Stress in the society increases in parallel with abdominal obesity

Stress has been increasing in most industrial countries over the last 60 years, and it now affects the majority of the population. Lissner *et al*. studied the secular trends in lifestyle indicators in Sweden in 1968/1969 among 770 women and in 2004/2005 among 500 women. They found quite dramatic increases in the incidence of stress experiences ('individuals who have experienced stress occasionally or more during the last five years') in the population: the proportion of women who sometimes felt stressed doubled from 29 to 59%, and the proportion that felt they were continuously stressed increased from 6.0 to 15.6%, both among 38 and 50-year-olds (19). In the 2013 *Work Stress Survey* conducted by 'Harris Interactive', more than 80% of employed Americans reported that they were *stressed out* on the job, amid heavier workloads and low pay (20). This is quite an increase since a similar survey from 2001 where 35% responded their job to be harming their physical or emotional well-being (21). As these figures mostly rely on self-reported data, they thus may be confounded by more awareness and focus on stress as a societal and individual problem. However, there are many indications that a major increase in prevalence of stress is real, and the self-reported data are less likely to be confounded by secular attitudes and views. Anyway, the increased experience of stress seems to run in parallel with the increase in obesity rates, and particularly with the increase in abdominal fatness (Fig. 1). These observations are seen in both Europe and USA (19,22).

It is important to note that the increase in abdominal obesity occurs simultaneously with a constant, or even decrease of, average BMI and obesity prevalence in many countries, including USA and the Nordic countries (19,23–27). So while the good news is that obesity rates may have started to decline, the bad news is that the health hazard may continue to increase because abdominal obesity is more important than BMI. BMI does not reflect body *composition*. The increasing prevalence of sarcopenic obesity, i.e. the existence of a normal BMI but increased fatness at the expense of a subnormal lean body mass, could easily have excess negative affect stress experiences and the resulting increased cortisol secretion as contributing factors.

While stress may increase abdominal obesity at all ages, it is likely that stress during gestation in particular exerts a programming and/or differentiation effect on the offspring's brain stress-responsiveness to environmental adversity (28) and therefore predisposes the offspring to a higher BMI, obesity and abdominal fatness throughout the life span. Among putative causal factors are suggested prenatal adverse events like decreased sleep duration and experience of stress during gestation by the mother, as well as her eating behaviour and nutrition selection; these factors might exert a programming effect on the foetus's brain that increases the risk of weight gain and obesity (22). This subject has been reviewed in detail by Komlos and Brabec (29) and by Robinson and colleagues (22).

Associations among mental stress, anxiety and well-being, and abdominal obesity and the underlying mechanisms

Observational studies [recent published studies are presented in Table 2, and older studies are presented in the meta-analyses by Wardle and colleagues (30)] have applied several of the existing psychometric screening tools (Table 3) to examine the association among mental stress, anxiety and depression and weight gain and abdominal obesity (31–36). The observational studies have demonstrated

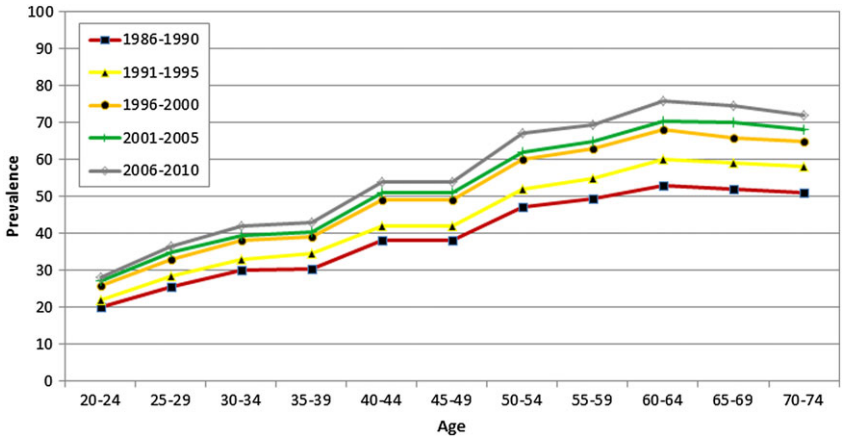

Figure 1 Abdominal obesity is increasing independently of BMI: US data. Reproduced with permission from Robinson *et al.* 2013 (22). [Colour figure can be viewed at wileyonlinelibrary.com]

Table 2 Long-term observational studies on stress and weight gain

Reference (country)	Design and duration	*N*	Objective	Variables	Results	Conclusion
Fowler-Brown *et al.* 2009 (31) (USA)	Prospective cohort, 13-year follow-up	1,172 (416 men and 756 women)	Examine relationship between perceived psychosocial stress and percent change in BMI	Anthropometry Perceived stress (PSS)	Men: No associations found Women: ↑perceived stress predicted ↑BMI	Emotional stress seems have a negative impact on weight change among women
Jackson *et al.* 2017 (54) (UK)	Longitudinal, 4-year follow-up	2,527 (1,061 men and 1,466 women)	Examine the association between adiposity and cortisol from hair as marker of stress	Anthropometry Hair cortisol	All subjects: ↑Hair cortisol: ↑body weight + −status, ↑waist circumference + −status, Obese subjects: ↑cortisol concentration Persistently obese subjects: ↑↑cortisol	Long-term exposure to elevated cortisol is associated with higher levels of adiposity
Manenschijn *et al.* 2011 (57) (the Netherlands)	Cohort, single measurement	122 (all men) 89 dayworkers and 33 shift workers	Examine the association between stress and BMI	Anthropometry hair cortisol	Shift workers had increased cortisol ↑Hair cortisol: ↑BMI	Increased cortisol was associated with BMI
Manenschijn *et al.* 2013 (56) (the Netherlands)	Cohort, single measurement	283 community-dwelling older people	Examine the association between stress and BMI	Anthropometry hair cortisol Medical history (self-reported)	↑Hair cortisol: ↑diabetes, ↑cardiovascular disease No association between cortisol and BMI	No association between cortisol and BMI
Michaud *et al.* 2015 (35) (Canada)	Cross-sectional, 3-month retrospective	511 (children)	Examine homework duration and the presence/absences of stress related to the homework and the association with childhood adiposity	Questionnaires on homework Physical activity	Stressed boys + long duration of homework: ↑total and trunk body fat, ↓physical activity and ↑screen time Girls: No associations	Boys with high workload and schoolwork-related stress have unfavourable adiposity indicators
Noppe *et al.* 2016 (58) (the Netherlands)	Cross-sectional, 3 months	3,019 (6-year-old children)	Examine the association between stress and childhood adiposity	Anthropometry Hair cortisol Hair glucocorticoid Hair cortisone	Cortisol and cortisone positively associated with BMI, fat mass and android/gynoid fat mass ratio. Glucocorticoid was not associated with adiposity any parameter	Long-term cortisol concentrations are associated with increased childhood adiposity and adverse body fat distribution
Olstad *et al.* 2016 (36) (Australia)	Cohort, single measurement	70 (all women, including 30 maternal–child pairs)	Examine the association between stress and adiposity	Weight and height Hair cortisol Perception of stress (PSS)	Women: ↑perception of stress ➔ ↑BMI No association between hair cortisol and BMI Children: no association between hair cortisol and *z*-BMI score	Perceived physiological measures are predictors of adiposity
Rivenes *et al.* 2009 (33) (Norway)	Observational, community based, single sample	60,704 (29,939 men and 30,765 women)	Examine the associations among obesity, anxiety and depression	Anthropometry Anxiety and depression (HADS) Medical history Lifestyle parameters	All subjects: Dose response relationship between anxiety and depression and hip–waist ratio Higher rates of depression among obese	Abdominal obesity was independently associated with depression and anxiety

(*Continues*)

Table 2 (Continued)

Reference (country)	Design and duration	*N*	Objective	Variables	Results	Conclusion
Rutters *et al*. 2015 (55) (the Netherlands)	Prospective, stressful life events 5 retrospective Metabolic syndrome 6.5-year follow-up	1,099 (517 men and 582 women)	Assessment of general health information via interview and following assessment of symptoms of metabolic disease and stressful life events.	Anthropometry background data Plasma glucose, cholesterol Blood pressure Stressful life events	Each stressful life event ➔ +0.86 cm in waist circumference and +13% in odds of developing metabolic syndrome Financial difficulties was the most stressful incident	Experience of stressful life events was associated with ncreased abdominal adiposity
Sims *et al*. 2008 (32) (USA)	Observational, single sample	159 (76 men and 83 women)	Examine the association between perceived stress and high fat eating	Anthropometry Demography Perceived stress (PSS) Eating behaviour	All subjects: ↑perceived stress: Haphazard planning and emotional eating Overweight and obese subjects with perceived stress: ↑snack eating	Greater perceived stress was associated with unhealthy eating
Tucker *et al*. 2010 (34) (USA)	Prospective cohort, 2 years	256 (all women)	Examine the relationship between emotional health and risk of weight gain	Anthropometry Physical activity Emotional health (GWBS)	Women with 'less than positive emotional health' had greater risk of weight gain Among depressed there was no greater risk than nondepressed	Women with less than positive emotional health are at greater risk of weight gain over time
Vanaelst *et al*. 2014 (53) (Belgium)	Cross-sectional, 2-year follow-up	355 (children, 186 boys and 169 girls)	Examine the relationship between stress and body composition	Anthropometry, stress parameters, salivary cortisol and lifestyle parameters	Both sexes: Negative life events, happiness: ↑BMI *z*-score Girls: Peer problems: ↑BMI *z*-score	Childhood stress positively related to overall and central adiposity

BMI, body mass index; GWBS, general well-being schedule; HADS, Hospital Anxiety and Depression Scale; PSS, Perceived Stress Scale.

Table 3 Psychometric screening tools

Abbreviation	Full name	Applied in study types	Identification	Administration	Length (items)	Target population
BDI	Beck Depression Inventory	Randomized controlled trial (59)	Symptoms of depression	Self-administrated	21	Adults and adolescents in the general population
GHQ	General Health Questionnaire	Randomized controlled trial (59)	Mood state	Self-administrated	28	Adults and adolescents in the general population
GWBS	General well-being schedule	Prospective (34)	Psychological well-being and distress	Self-administrated	18	Adults and adolescents in the general population
HADS	Hospital Anxiety and Depression Scale	Cross-sectional (33)	Anxiety and depression, two subscales	Self-administrated	14	Non-psychiatric patients at general hospital
POMS-standard*	Profile of mood states	Cross-over (42) Experimental (40,41)	Transient, distinct mood states and psychological distress	Self-administrated	65	Patients
PSS	Perceived Stress Scale	Retrospective (31) Cross-sectional (32) Randomized controlled trial (60,62,63) intervention (61)	Perception of stress	Self-administrated	10	Individuals with at least a junior high school education
STAI-Trait	State–Trait Anxiety Inventory	Experimental (40,41)	Experience of anxiety in state and trait situations	Self-administrated	40	Adults in the clinical setting and in research

*POMS also exist in a short form focusing on identifying levels of transient, distinct mood states; the questionnaire is reduced to 37 items.

clear associations between perceived stress and weight gain and abdominal fatness (measured by waist-to-hip ratio); specifically, the association between anxiety and waist-to-hip ratio followed a dose–response pattern (31,33,34). An association between stress and emotional eating behaviours is also established, specifically the increased snacking of sweets among obese subjects (32). While there are a quite high number of subjects, the results of these observational studies rely on self-reporting that is prone to error.

During the past three decades, work and leisure time is favouring ever more technological features (and stressful situations) in expense of the more physical demanding tasks, and the effect on energy intake has been studied by Tremblay and colleagues (Table 4) (37–39). Although the stress tasks had no effect on appetite sensation markers, the energy intake was higher among female students, both in subsequent mealtime and over 24 h; this was not seen in male students. During the stress task, the students responded with an acute increase in cortisol, glucose and insulin. Westerterp-Plantenga and colleagues have investigated the effect of stress on 'eating in the absence of hunger' (Table 4) (40,41). They too did not find stress to influence appetite sensation but found that an increased energy intake satiated more following a stress test. There was a preference for highly palatable foods (high-calorie/high-sugar/high-fat foods, also called 'comfort foods'), especially among overweight and obese subjects. Personality traits influence the energy intake in reaction to stress differently. Individuals with a trait for being a 'restraint eater' (identified by score-12 in the Three-Factor Eating Questionnaire) have been excluded participation in several interventions of acute stress, even so, subjects with increasing restraint score reduced their energy intake in response to stress (37–39). Opposite to this, individuals prone to disinhibition during acute stress and also during stressful events increase their energy intake, also in the absence of hunger, and especially the intake of highly palatable foods (37,40).

The effect of acute stress on postprandial satiety and taste preferences is proposed to be influenced by brain activation of the reward areas, amygdala, hippocampus, reduction of cingulate cortex and increased cortisol (10,12,40–44). The mechanism involves close interaction between the HPA-axis and the sympathoadrenal system as a response to stress (2,10–12,43,45,46). Activation of the HPA-axis results in release of cortisol from the adrenal cortex, while the sympathoadrenal system acts through the adrenal medulla by releasing adrenalin and noradrenalin. Cortisol offers negative feedback to the HPA-axis, while the stress hormones from the sympathoadrenal system offer positive feedback and simultaneously increases the HPA-axis. From animal and human studies, it appears that the HPA-axis can quickly habituate in response to repeatedly and/or prolonged experiences of the same stressor, whereas the sympathoadrenal system often showed constant and stable activation patterns (47), hence continuing to activate the HPA-axis through, e.g. adrenalin. Evidence suggests that

Table 4 Experimental acute studies of stress on energy balance and food preference

Reference (country)	Design	*N*	Method	Variables	Results	Conclusion
Born *et al.* 2010 (44) (the Netherlands)	Crossover	9 (all women) normal weight	Stress *or* rest condition (control)	Wanting and liking Perception of food characteristics Plasma cortisol Hunger and satiety	Stress ➔ ↑energy, fat and protein intake, ↑preference for crispiness and fullness of food, ↑cortisol, ↓brain activation of reward areas	Stress reduced reward signalling and sensitivity and increased energy intake and preference for crispiness and fullness of food.
Chaput *et al.* 2008 (38) (Canada)	Crossover	14 (all female) normal weight	Simple mental work *or* demanding mental work *or* resting sitting (control) all followed by *ad libitum* lunch buffet	Energy intake (buffet only) Appetite sensation Plasma glucose, insulin and cortisol	Mental work ➔ ↑energy intake, ↑glucose, ↑insulin, ↑cortisol No difference in appetite sensation	Mental work induces positive energy balance.
Chaput *et al.* 2007 (37) (Canada)	Crossover	15 (all female) normal weight	Simple mental work *or* resting sitting (control) all followed by *ad libitum* lunch buffet	Energy intake Food preference Energy expenditure Appetite sensation Heart rate Blood pressure	Mental work ➔ ↑energy intake, no difference in appetite sensation	24-h net calorie surplus after session with mental work
Lemmens *et al.* 2011 (41) (the Netherlands)	Crossover	42 (16 men and 26 women) 27 normal weight, 15 visceral overweight	'Eating in the absence of hunger' subjects were offered two *ad libitum* meals with exposure to acute stress *or* rest test between the meals and after the last.	Anthropometry Appetite sensation Eating behaviour Assessment of mood and anxiety (POMS and STAI-trait) Heart rate	All subjects: Stress test ➔ ↑anxiety score, ↓mood score, ↑heart rate No difference in appetite sensation Overweight: Stress test ➔ ↑wanting for sweet and savoury, ↑energy intake, ↑intake of carbohydrate and fat	Visceral overweight subjects showed stress-induced food intake in the absence of hunger, resulting in an increased energy intake.
Pérusse-Lachance *et al.* 2013 (39) (Canada)	Crossover	35 (22 men, 13 women) normal weight	Mental work *or* exercise *or* resting sitting (control) all followed by *ad libitum* lunch buffet	Energy intake (lunch and all day) Energy expenditure Appetite sensation	Men: Mental work and exercise ➔ ↓energy intake Women: Mental work ➔ ↑energy intake All: No difference in appetite sensation	Men and women have specific food intake patterns when submitted to cognitive and physical stimuli.
Rutters *et al.* 2009 (40) (the Netherlands)	Crossover	129 (65 men, 64 women) normal and overweight	'Eating in the absence of hunger' after a fixed lunch subjects were exposure to acute stress *or* rest test and after 30 min presented an *ad libitum* selection of sweet and savoury snack foods.	Anthropometry Appetite sensation Eating behaviour Assessment of mood and anxiety (POMS and STAI-trait) Blood pressure	Stress test ➔ ↑anxiety score, ↓mood score, ↑energy intake, ↑sweet snack No difference in appetite sensation Correlation between anxiety score and energy intake	Exposure to stress is associated with eating in the absence of hunger.

POMS, profile of mood states; STAI: State–Trait Anxiety Inventory.

the intake of highly palatable foods as a response to stress often activates the brain reward system involving opioid, dopamine and endocannabinoid; this is suggested to promote the limbic system to induce dominant behavioural reinforcement in order to acquire palatable foods to blunt the activation of stress/sympathoadrenal system on the HPA-axis [for review see Cota *et al.* (48)] (8,43,46).

In addition to increasing the consumption of energy rich foods, a prolonged activation of the HPA-axis may promote abdominal, visceral fat accumulation as well as increase body weight via the increased cortisol secretion (46,49). For detailed review of the process, see the papers by Björntorp and Markus [e.g. Marcus and Björntorp (9,10)]. The excess production of cortisol does not only influence the accumulation of abdominal fat but also exerts adverse effects on medical health such as type 2 diabetes, hypertension, dyslipidaemia and cardiovascular disease (13,14,16,17,50–52). Some of these stress-related bodily consequences are commonly used as additional more objective physiological markers for stress, i.e. cortisol in hair and saliva, heart rate variability, resting heart rate and blood pressure. Evidence from observational studies using hair and saliva cortisol samples and/or blood pressure measures reveal that stress is positively related to increased BMI and specifically abdominal adiposity, both among children and adults (Table 2) (36,53–58). These results have also been repeated in human experimental studies, where negative mood and cortisol levels increased significantly as response to acute stress sessions; high cortisol compared with low cortisol responders were significantly more negatively affected/stressed (Table 4) (40–42,44).

Results from observational and experimental studies show that low well-being and high stress scores correlate with future weight gain by mechanisms that cannot be detected by usual self-reporting methods to measure energy intake and physical activity. Furthermore, the results indicate that chronic stress will cause excess weight with a high risk of centralized adiposity.

Stress and the effect of weight loss programmes

If mental stress actually is a psychological (negative affectivity) and physiological (sympathetic HPA-axis arousal, cortisol) situation that may promote energy intake, positive energy balance and preferential abdominal fat deposition, it is to be expected that particularly stressed individuals starting a weight loss diet will have all the odds against them for a successful outcome. Observations from a number of randomized weight loss trials indeed confirm that a high stress level and low state of mood (measured by psychometric screening tools; Table 3) hinder a successful weight loss outcome and increase the risk of dropout (31–34,59–61). Hence, stress poses an important barrier for weight loss efforts and improved health.

Despite ample research showing a strong link between stress and weight gain and that higher perceived mental stress reduces the success of weight loss, there are essentially no studies examining the efficacy of stress management as a tool for achieving weight loss and for weight loss maintenance. Only two small studies have been conducted to assess the effectiveness of stress coping for weight loss (62,63). Even though only one study revealed significant effects (62), weight loss was higher by adding stress management to lifestyle/weight loss programme among women with high prevalence of stress (measured by Perceived Stress Scale) compared with the effect of diet only. These two rather small randomized studies support the concept that stress management either alone or in combination with diet, exercise and behaviour modification can improve weight loss success. However, the trials were very small with limited statistical power and of short duration. Larger studies of longer duration with consecutive measures of stress, eating behaviour, appetite and cravings, as well as cardiometabolic biomarkers and detailed assessment of adiposity, are warranted.

Nutritional interventions for stress or weight management?

Observational studies have linked deficiency of certain B vitamins, and of minerals such as calcium and magnesium, and omega 3-fatty acids to increased risk of mental stress (4,5,64,65). Most of the evidence is derived from cross-sectional observational studies. However, Mishra *et al.* examined associations between levels of B vitamins with subsequent psychological distress in a cohort of British women and found that low dietary levels of vitamin B_{12} were associated with higher stress levels (66). A number of studies have examined the role of vitamin status for the development of stress during various disease conditions leading to chronic stress, and they suggest that current Recommended Dietary Allowance for vitamins and minerals may not take into account that there may be increased brain requirements of essential nutrients for optimal brain metabolism and psychic and mental function during stress (64).

Interestingly, some studies have revealed beneficial effects of dietary supplementation on stress resilience (67–71). Most of these studies have focused on either multivitamin and/or mineral supplementation or the influence of specific amino acids. Such foods are thought to interfere with brain biochemical processes involved in stress resilience and thus affect emotionality. A meta-analysis including eight studies found that combinations of B vitamins and minerals (calcium and magnesium) had beneficial effects on perceived stress, anxiety, mild psychiatric symptoms and certain aspects of everyday mood (Figs 2 and 3) (69).

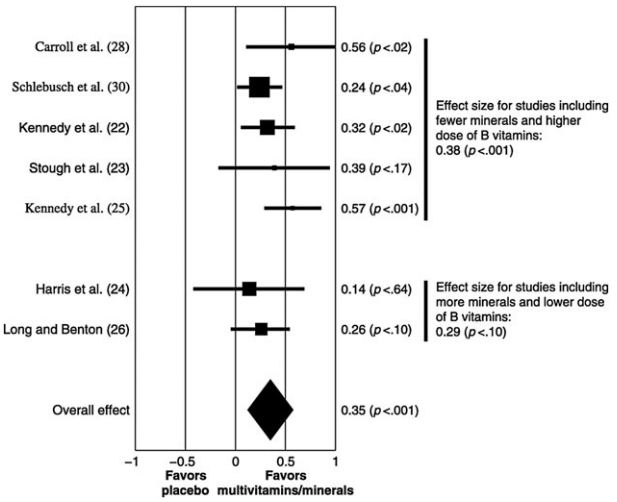

Figure 2 Forest plot of multivitamin/mineral supplementation on perceived stress. The size of the squares reflects the sample size, and the lines represent the upper and lower limits. Reproduced with permission from Long SJ & Benton D 2013 (69).

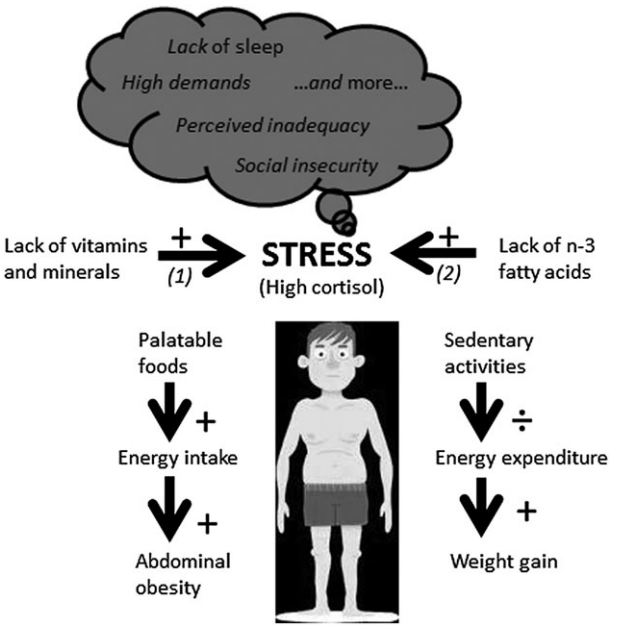

Figure 3 Stress may be a barrier to making diet and exercise changes needed to achieve long term control of body weight. Individuals with low intake of vitamins (especially B-vitamins), minerals and long chain n-3 fatty acids might have an increased susceptibility to stress.

Meta-analyses including clinical trials assessing the effect of omega-3 polyunsaturated fatty acids in the treatment of depressive disorders showed a clinically relevant improvement in patients with major depressive disorder and patients with depression (nonmajor); only 2 out of 19 studies included overweight subjects (70). Pure eicosapentaenoic acid was more effective than docosahexaenoic acid or if they were combined [for review see Grosso *et al.* (70)]. Omega-3 fatty acids also prove effective as adjunctive treatment for people with otherwise treatment-resistant depression, as monotherapy for childhood depression and in patients with recurring self-harm (72). Among healthy adults, 6 weeks of supplementation with fish oil showed a tendency to reduce cortisol concentration (71).

A broad range of well-controlled experimental studies also reveal possible beneficial effects of dietary amino acids and carbohydrates (including sugar) on stress resilience, particularly in stress-vulnerable subjects (73–75). For instance, in a human laboratory stress-exposure study, only healthy subjects with a trait to vulnerability to frequently experiencing stress (neuroticism) exhibited significant reductions in negative mood and cortisol responsiveness under acute stress by exposures to specific nutrients (73,76). More recent findings reveal that the administration of dietary tryptophan sources reduces acute stress-induced attention for negative mood and/or HPA-cortisol responses in healthy subjects with a genetic vulnerability for stress (75,77,78). These results are consistent with both the food amino acid tryptophan and carbohydrates/sugar found to promote activation of the brain serotonergic system, commonly known to control stress adaptation and mood (45).

It is interesting to note that these trials, both on the effect of vitamin/mineral and fatty acids supplementation, were not conducted in malnourished populations, but rather in population's representative for USA, Europe and South Africa. The optimal combination of vitamins, minerals and essential fatty acids has yet to be established, and it may vary depending on targeted effects and population. However, it is promising that a number of different mixtures of vitamins and minerals all have proved effective for reduction of mental stress within 30 d in normal individuals with perceived high levels of stress. This suggests that the therapeutic window for the intakes is quite broad and that they may be effective in a large proportion of the population today.

To summarize, studies confirm a relationship between nutrient supplementation and reduction in mental disorders, including stress. The supplements may serve as monoadjunctive or adjunctive treatment for already stressed individuals and also as prevention for healthy people, particularly among those susceptible to stress. To confirm a beneficial effect across the weight span, high-quality studies including overweight and obese subjects must be executed.

The influence of stress on sleep patterns and quality: links to obesity

Secular trends in sleep duration

Data from 10 to 15 different countries secular trends in adult sleep duration from 1960s to 2000s showed no consistency in change of sleep duration (19,79–82). They did

not report, however, the proportion of very short and very long sleepers, which might be of relevance to health. Studies specifically assessing poor and short sleep and very long sleep found both associated with increased levels of mental stress and obesity (83–86). Among children and adolescents, results are more consistent and show a decline in sleep duration of more than 1 h per night (87), most prevalent among children and adolescents of racial/ethnic minorities and those with low socioeconomic status (88). There is a large difference between countries in sleep duration specifically influenced by bedtime (Fig. 4); young adults have a greater variation in sleep schedules than do older adults (89). The apparent decrease in sleep duration among most populations has been suggested to be a part of the modern lifestyle with increased time spent in artificial light, watching television, using computers and working (81,90).

Sleep and weight gain/loss

Robust epidemiological evidence supports the potential role of inadequate sleep as contributing to the current high prevalence of obesity, more specifically abdominal adiposity, in both children and adults (Table 5) (91–97). There are longitudinal observations suggesting that sleep and body weight develop at least partly in parallel (98,99), so causality cannot be extrapolated from these cross-sectional observations. However, several experimental studies confirm the link between sleep and obesity (Table 6). All studies but one, including male adolescents (100), find increased energy intake and weight gain after acute sleep deprivation (101–105). The one study not finding an effect did, however, only measured energy intake at a subsequent breakfast and not like the others during several subsequent meals or days (Table 6). A crossover study found higher energy intake and increased abdominal adiposity among children sleeping less than average (106).

The increased energy intake in response to sleep deprivation may only to some extent be explained by the changes in appetite regulating hormones leptin, ghrelin and GLP-1 (103,105,107,108), and a following lower satiating effect of foods (109) and higher consumption of energy dense foods and sugar sweetened beverages (105,110). It is most likely that the reward-driven motivation also plays an important role (111). Sleep restriction is previously shown to increase the neuronal response (blood oxygen level dependent contrast imaging-response using visual food stimuli and functional magnetic resonance) to energy dense foods in normal-weight subjects (112,113) similar to what has been found during higher activity in the HPA-axis after an experimental stress condition (114). These studies are in support of a similarly enhanced reward-driven control of food intake with a preference for energy dense foods after these two different stressors, a preference that is more expressed among individuals with a high disinhibition eating behaviour trait (37,40,95).

In recent years, several studies have focused on sleep duration during weight loss periods. During 17 weeks of moderate caloric restriction among 123 overweight and obese adults, sleep duration and sleep quality at baseline predicted fat loss (115). Also among 150 obese (BMI 33.3 ± 3.5 kg/m^2) men and women (age 38.8 ± 8.6 years), sleep quality (assessed by Pittsburgh Sleep Quality Index) was associated with loss of body weight, fat mass and waist circumference (116). Furthermore, better subjective sleep quality among 245 women enrolled in a 6-month weight-loss programme increased the likelihood of successful weight loss by 33% (117). Supportive of this, an observational study found that an increase of sleep duration from (from <6 h/d to 7–8 h/d) over 6 years was associated with an attenuation in abdominal adiposity compared with those who maintaining their short sleep duration who gained 2.4 kg fat mass (96). Long-term randomized trials treating obesity with extension of sleep

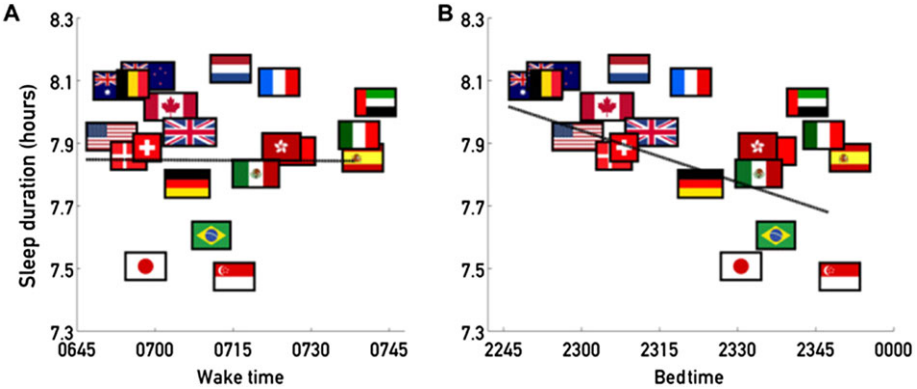

Figure 4 Sleep duration in 2016 measured by free mobile phone App. Reproduced with permission from Walch *et al.* 2016 (89). [Colour figure can be viewed at wileyonlinelibrary.com]

Table 5 Observational studies on sleep and weight gain

Reference (country)	Design and duration	*N*	Objective	Variables	Results	Conclusion
Chaput *et al.* 2008 (94) (Canada)	Prospective, 6 years	276 (117 men and 159 women)	Examine the relationship between sleep duration and subsequent body weight and fat gain	Anthropometry Covariates Sleep duration Dietary intake Energy expenditure	Short and long sleepers: ↑weight gain, ↑waist circumference, ↑ body fat	Short and long sleep duration predict a higher body weight and abdominal adiposity.
Chaput *et al.* 2011 (95) (Canada)	Prospective, 6 years	276 (117 men and 159 women)	Examine if the relationship between weight gain and short or long sleep duration is influenced by disinhibited eating behaviour	Anthropometry Covariates Sleep duration Dietary intake Energy expenditure Disinhibition eating behaviour	Subjects with high disinhibition eating behaviour + ↓sleep: ↑weight, ↑waist circumference, ↑energy intake	Subjects with high disinhibition eating behaviour trait were more sensitive to short and long sleep and had higher incidence of adiposity.
Chaput *et al.* 2012 (96) (Canada)	Prospective, 6 years	43 (22 men and 21 women)	Examine the effect of a favourable change in sleeping habits	Anthropometry Covariates Sleep duration	Short sleep ➔ short sleep: ↑weight, ↑waist circumference, ↑fat mass, short sleep ➔ average sleep: ↓weight, ↓waist circumference, ↓fat mass	Increasing sleep duration from short to average was associated with attenuation of abdominal adiposity.
Chaput *et al.* 2007 (97) (Canada)	Cross-sectional, single measurement	422 (211 boys and 211 girls)	Examine if short duration sleeping children are predisposed to abdominal adiposity	Anthropometry Covariates Sleep duration	Inverse association between daily sleeping hours and waist circumference	Short sleep duration favours abdominal adiposity in children.
Hjorth *et al.* 2014 (92) (Denmark)	Cross-sectional, longitudinal	785 (408 boys and 377 girls)	Examine the association between movement behaviours and fat mass index	Anthropometry Physical activity Sleep duration	↓Sleep duration: ↑fat mass index ↑Sleep disturbances: ↑fat mass index	Short sleep was independently associated with higher fat mass index.
Kjeldsen *et al.* 2014 (93) (Denmark)	Cross-sectional, 7 d	676 (children)	Examine the association between sleep duration and problems and proposed dietary factors for overweight and obesity	Anthropometry Covariates Sleep habits questionnaire Plasma leptin and ghrelin Dietary intake	Excess and depleted length of sleep: ↑energy density, ↑added sugar, ↑sugar sweetened beverages ↑Sleep problems: ↑energy density	Short and high sleep duration and experience of sleep problems are all associated with a poor, obesity promoting diet.

duration are ongoing, but results have not yet been published (118).

The link between stress and metabolic health is potentially mediated by sleep

There is extensive consensus that stress plays an important role in both the aetiology and persistence of sleep disturbances. For instance, sleep quality has been found to vary as a function of daily stressful life events (119–122), and hence, personality traits associated with stress vulnerability, like neuroticism, appear to be important predictors for sleep disturbances (123). In addition to such studies revealing relationships between mental stress experiences and sleep difficulties, there are also studies investigating the more direct interrelationships between sleep quality and activation of the HPA-axis, the major neuroendocrine system involved in stress responses and hence stress adaptation (45). In general, increased HPA-axis activation (as a function of metal stress) is found to impair sleep, most likely by increasing cortical arousal, resulting in lighter sleep and more nocturnal awakenings (124–126). These findings thus suggest that the negative effects of stress experience on sleep quality may, at least partly, be mediated by HPA-axis alterations.

Even more interesting, evidence for a simultaneous reverse causality is emerging in support of the notion that bad sleep could be seen as a stressor itself. Prior sleep studies have clearly shown that adequate (rapid eye movement sleep) sleep regulation is crucial for the overnight decrease in cognitive–emotional arousal caused by daily stressors (127). Conversely, a distortion of sleep regulation may then result in an inadequate inhibition (relief) of negative emotionality, causing a prolonged negative emotional state and reduced resilience to future adverse life events and hence further intensifying the impact of daily stress. In further support of this, recent evidence reveals that lack of sleep alters activation of the HPA-axis

Table 6 Experimental acute studies of sleep on energy balance

Reference (country)	Design	*N*	Method	Variables	Results	Conclusion
Bosy-Westphal *et al.* 2008 (103) (Germany)	Controlled intervention	14 (all women)	9 d; >8 h, >8 h, 7 h, 6 h, 6 h, 4 h, >8 h and >8 h of sleep per night	Anthropometry Oral glucose tolerance test Energy intake Energy expenditure Plasma insulin, ghrelin and glucose	↓sleep ➔ ↑energy intake, ↑leptin, ↑weight	Short-term sleep deprivation increased energy intake and led to a net weight gain
Brondel *et al.* 2010 (102) (France)	Crossover	12 (all men)	2 × 48 h of sessions with *either* ~8 h *or* ~4 h of sleep	Physical activity Perception of food Energy intake	~4 h sleep ➔ ↑energy intake, ↑hunger, ↑physical activity, ↑sleepiness	One night of sleep deprivation subsequently increased food intake
Broussard *et al.* 2016 (105) (USA)	Randomized crossover	19 (all men)	4 d of 8 h of bedtime versus 4 d of 4.5 h of bedtime all followed by 1 h of *ad libitum* lunch and dinner buffet	Anthropometry Energy intake Physical activity Plasma ghrelin, leptin	↓ sleep ➔ ↑ghrelin, ↑energy intake, ↑snack intake	Recurrent sleep deprivation facilitated increased energy intake and snacking
Calvin *et al.* 2013 (104) (USA)	Randomized, parallel	17 (11 men and 6 women) (thereof five men and three women randomized to sleep deprivation)	3 d run in followed by 8 d and nights of sleep deprivation versus normal sleep (control) followed by a recovery phase of 3 d	Anthropometry Energy intake Energy expenditure Plasma ghrelin, leptin	↓ sleep ➔ ↑energy intake, ↑weight	Recurrent sleep deprivation facilitated increased energy intake
Klingenberg *et al.* 2012 (100) (Denmark)	Randomized crossover	21 (all male adolescents)	2 × 3 nights with *either* 4 h *or* 9 h of sleep followed by *ad libitum* breakfast	Anthropometry Energy intake Energy expenditure Appetite sensation Plasma ghrelin, leptin	↓ sleep ➔ ↑energy expenditure, ↓energy intake	Short-term sleep deprivation is associated with small negative energy balance
Nedeltcheva *et al.* 2009 (110) (USA)	Randomized crossover	11 (6 men and 5 women)	2 × 14 d stay with *ad libitum* access to palatable food and *either* 5.5 h *or* 8.5 h of bedtimes	Anthropometry Energy intake Energy expenditure Preference of food Serum leptin, ghrelin	↓ sleep ➔ ↑carbohydrate intake, ↑snacking, particularly at evening and night times	Recurrent sleep deprivation facilitated excessive intake of snacks
NcNeil *et al.* 2013 (109) (Canada)		75 (all men)		Anthropometry Sleep quality Energy intake Appetite sensation	↓ sleep ➔ ↓sleep quality Short sleep ➔ ↓satiety	Short sleep reduced sleep quality and decreased satiety
Spiegel *et al.* 2008 (107) (USA)	Randomized crossover	12 (all men)	2 d of sleep restriction and 2 d of sleep extension	Anthropometry Physical activity Energy intake Leptin, ghrelin	↓ sleep ➔ ↑hunger, ↑appetite, ↑carbohydrate, ↑ghrelin, ↓leptin	Acute sleep deprivation is associated with increased hunger, appetite and energy intake
St-Onge *et al.* 2012 (113) (USA)	Crossover	30	4 h or 9 h of bedtime	Neuronal activity in response to food stimuli	↓ sleep ➔ ↑neuronal activity associated with reward activated in response to food stimuli	Restriction of sleep is associated with higher susceptibility to food stimuli

(128). They found that two nights of sleep restriction (4 h compared with 10 h in bed) in normal or moderately overweight men was associated with a 19% higher overall adrenocorticotropic hormone level and that the individual increases were strongly correlated ($-r$ = 0.63) with the individual amount of sleep loss. The overall levels of cortisol were elevated by 21% and, perhaps more importantly, they found that sleep restriction disrupted the normal circadian rhythms of these hormones.

Conclusions

The short conclusion is: Yes. Not only do the adverse effects of stress influence sleep patterns, food intake, weight gain, abdominal obesity and the effects of weight loss interventions but evidence also suggests that improving nutritional status and sleeping patterns may reduce the severity of stress and other mental disorders.

Mental stress and depression have increased dramatically over the last 50 years, now affecting a large proportion of the population similar in size to that affected by obesity. Several events in life may cause mental stress (Table 1), and impaired sleep and poor nutrition may increase the susceptibility of mental illness. Stress has been found associated with impaired sleep and to increase the emotional rewarding of palatable foods, thereby increasing obesity and in particular abdominal adiposity.

Not only does stress increase energy intake and a higher preference of palatable and energy dense foods but sleep deprivation also exerts a similar effect. Given the association between sleep deprivation and increased energy intake, it is worrying that the recommendations for sleep duration for children have declined (129). As stress can cause disruption and deprivation of sleep, and vice versa, a more holistic approach in treating obesity is desirable. There is emerging evidence from trials that stress management tools may facilitate weight loss (62,63), but it remains to be shown in well-powered randomized controlled trials whether stress reduction/coping programmes, improved sleep quality and dietary supplements of vitamins and minerals are feasible and efficient in diminishing food cravings and improve weight (loss) management.

We hypothesize that lifestyle changes in overweight and obese individuals with stress, depression, anxiety and negative mood should be combined with treatment targeted stress to achieve weight loss and maintaining this.

Perspectives and future research needs

Given the poor outcomes of current weight-control interventions to prevent and manage obesity, there is an urgent need to explore beneficial effects of possible new innovative weight-control treatment programmes. New strategies should investigate the effect of dietary alterations (low-energy diets followed by lifestyle changes) simultaneously with cognitive behavioural treatment, i.e. stress management. Such research may provide a new understanding, not only on the causes of obesity but also on the success of treatment.

Different forms of stress need to be clearly defined, and focus should be on the common, prevalent stress that is most often associated with weight gain and susceptibility to overweight and obesity (Table 1). We also need to elucidate the influence of these stressors on sleep quality and duration and then take into account the possible adverse additive or cumulative effect of stress, sleep, diet and obesity with emphasis on abdominal adiposity.

The findings of certain nutritional manipulations posit the intriguing possibility to be used to help achieve successful weight management particularly in stress-vulnerable overweight individuals. This possibility has not been investigated but is recently clearly supported by findings that glucose administration reduces snacking for high-fat sweet foods after stress exposure only in subjects with a genetic stress vulnerability (130). In light of the poor prognosis of weight loss programmes, these findings make it clear that there is a need to further examine the efficacy of dietary supplementation strategies on stress resilience, with an aim to improve the outcome of weight-management programmes particularly in stress-vulnerable overweight and obese individuals. Such trials should also assess a potential synergy between behavioural stress management programmes and nutrient supplementation and particularly examine if cravings for sweet and fat foods and drinks are diminished or normalized.

Conflict of interest statement

AA acts as consultant or member of advisory boards for The Global Dairy Platform, USA; Pathway Genomics, USA; Dutch Beer Knowledge Institute, the Netherlands; McCain, Canada; McDonalds, USA. As speaker at international congresses, travel expenses are often covered by the meetings, which are frequently supported by one or more corporate sponsors. He receives royalties as co-author/editor of a number of textbooks and diet/cookery books. Research at his department is funded by grants from both public and private sources. LP works for several public organizations as well as for several food and ingredient companies. NRWG, MFH, AS, LP and CRM declare no conflicts of interest.

Acknowledgements

We are grateful to Tina Cuthbertson for invaluable secretarial assistance and language editing.

References

1. Baum A. Stress, intrusive imagery, and chronic distress. *Health Psychol* 1990; **9**: 653.
2. Van Praag H. Can stress cause depression? *Prog Neuropsychopharmacol Biol Psychiatry* 2004; **28**: 891–907.
3. Wiegner L, Hange D, Björkelund C, Ahlborg G. Prevalence of perceived stress and associations to symptoms of exhaustion, depression and anxiety in a working age population seeking primary care—an observational study. *BMC Fam Pract* 2015; **16**: 1.
4. Lim SY, Kim EJ, Kim A, Lee HJ, Choi HJ, Yang SJ. Nutritional factors affecting mental health. *Clin Nutr Res* 2016; **5**: 143–152.
5. Tepper S, Dabush Y, Shahar DR, Endevelt R, Geva D, Ish-Shalom S. Vitamin D status and quality of life in healthy male high-tech employees. *Forum Nutr* 2016; **8**: 366.
6. van Rossum EF, Binder EB, Majer M *et al*. Polymorphisms of the glucocorticoid receptor gene and major depression. *Biol Psychiatry* 2006; **59**: 681–688.
7. Rosmond R, Chagnon YC, Chagnon M, Pérusse L, Bouchard C, Björntorp P. A polymorphism of the 5′-flanking region of the glucocorticoid receptor gene locus is associated with basal cortisol secretion in men. *Metabolism* 2000; **49**: 1,197–1,199.
8. Gibson EL. Emotional influences on food choice: sensory, physiological and psychological pathways. *Physiol Behav* 2006; **89**: 53–61.
9. Markus CR. Interaction between the 5-HTTLPR genotype, impact of stressful life events, and trait neuroticism on depressive symptoms in healthy volunteers. *Psychiatr Genet* 2013; **23**: 108–116.
10. Björntorp P. Do stress reactions cause abdominal obesity and comorbidities? *Obes Rev* 2001; **2**: 73–86.
11. Sinha R, Jastreboff AM. Stress as a common risk factor for obesity and addiction. *Biol Psychiatry* 2013; **73**: 827–835.
12. Oliver G, Wardle J, Gibson EL. Stress and food choice: a laboratory study. *Psychosom Med* 2000; **62**: 853–865.
13. Aschbacher K, Kornfeld S, Picard M *et al*. Chronic stress increases vulnerability to diet-related abdominal fat, oxidative stress, and metabolic risk. *Psychoneuroendocrinology* 2014; **46**: 14–22.
14. Block JP, He Y, Zaslavsky AM, Ding L, Ayanian JZ. Psychosocial stress and change in weight among US adults. *Am J Epidemiol* 2009; **170**: 181–192.
15. Björntorp P. The regulation of adipose tissue distribution in humans. *Int J Obes Relat Metab Disord* 1996; **20**: 291–302.
16. Harding JL, Backholer K, Williams ED *et al*. Psychosocial stress is positively associated with body mass index gain over 5 years: evidence from the longitudinal AusDiab study. *Obesity* 2014; **22**: 277–286.
17. Himmelstein MS, Incollingo Belsky AC, Tomiyama AJ. The weight of stigma: cortisol reactivity to manipulated weight stigma. *Obesity* 2015; **23**: 368–374.
18. Godfrey G. Observations on obesity. *JR Coll Gen Pract* 1971; **21**: 276–281.
19. Lissner L, Sjöberg A, Schütze M, Lapidus L, Hulthén L, Björkelund C. Diet, obesity and obesogenic trends in two generations of Swedish women. *Eur J Nutr* 2008; **47**: 424–431.
20. Workplace Stress on the Rise With 83% of Americans Frazzled by Something at Work. Corinthian Colleges Inc.: California, USA, 2013.
21. Harris I. Attitudes in the American workplace VII. 2001.
22. Robinson WR, Utz RL, Keyes KM, Martin CL, Yang Y. Birth cohort effects on abdominal obesity in the United States: the Silent Generation, Baby Boomers and Generation X. *Int J Obes (Lond)* 2013; **37**: 1,129–1,134.
23. Ng M, Fleming T, Robinson M *et al*. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. *The Lancet* 2014; **384**: 766–781.
24. Morgen CS, Rokholm B, Brixval CS *et al*. Trends in prevalence of overweight and obesity in Danish infants, children and adolescents—are we still on a plateau? *PLoS One* 2013; **8**: e69860.
25. Matthiessen J, Stockmarr A, Biltoft-Jensen A, Fagt S, Zhang H, Groth MV. Trends in overweight and obesity in Danish children and adolescents: 2000–2008—exploring changes according to parental education. *Scand J Public Health* 2014; **42**: 385–392.
26. Freedman DS, Ford ES. Are the recent secular increases in the waist circumference of adults independent of changes in BMI? *Am J Clin Nutr* 2015; **101**: 425–431.
27. Ford ES, Maynard LM, Li C. Trends in mean waist circumference and abdominal obesity among US adults, 1999–2012. *JAMA* 2014; **312**: 1,151–1,153.
28. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009; **10**: 434–445.
29. Komlos J, Brabec M. The trend of mean BMI values of US adults, birth cohorts 1882–1986 indicates that the obesity epidemic began earlier than hitherto thought. *Am J Hum Biol* 2010; **22**: 631–638.
30. Wardle J, Chida Y, Gibson EL, Whitaker KL, Steptoe A. Stress and adiposity: a meta-analysis of longitudinal studies. *Obesity* 2011; **19**: 771–778.
31. Fowler-Brown AG, Bennett GG, Goodman MS, Wee CC, Corbie-Smith GM, James SA. Psychosocial stress and 13-year BMI change among Blacks: the Pitt County Study. *Obesity* 2009; **17**: 2,106–2,109.
32. Sims R, Gordon S, Garcia W *et al*. Perceived stress and eating behaviors in a community-based sample of African Americans. *Eat Behav* 2008; **9**: 137–142.
33. Rivenes AC, Harvey SB, Mykletun A. The relationship between abdominal fat, obesity, and common mental disorders: results from the HUNT study. *J Psychosom Res* 2009; **66**: 269–275.
34. Tucker LA, Earl AB. Emotional health and weight gain: a prospective study of midlife women. *Am J Health Promot* 2010; **25**: 30–35.
35. Michaud I, Chaput JP, O'Loughlin J, Tremblay A, Mathieu ME. Long duration of stressful homework as a potential obesogenic factor in children: a QUALITY study. *Obesity* 2015; **23**: 815–822.
36. Olstad DL, Ball K, Wright C, Abbott G, Brown E, Turner AI. Hair cortisol levels, perceived stress and body mass index in women and children living in socioeconomically disadvantaged neighborhoods: the READI study. *Stress* 2016; **19**: 158–167.
37. Chaput J-P, Tremblay A. Acute effects of knowledge-based work on feeding behavior and energy intake. *Physiol Behav* 2007; **90**: 66–72.
38. Chaput J-P, Drapeau V, Poirier P, Teasdale N, Tremblay A. Glycemic instability and spontaneous energy intake: association with knowledge-based work. *Psychosom Med* 2008; **70**: 797–804.
39. Pérusse-Lachance E, Brassard P, Chaput J-P *et al*. Sex differences in the effects of mental work and moderate-intensity physical activity on energy intake in young adults. *ISRN nutrition* 2013; **2013**.
40. Rutters F, Nieuwenhuizen AG, Lemmens SG, Born JM, Westerterp-Plantenga MS. Acute stress-related changes in eating in the absence of hunger. *Obesity* 2009; **17**: 72–77.
41. Lemmens SG, Rutters F, Born JM, Westerterp-Plantenga MS. Stress augments food 'wanting' and energy intake in visceral overweight subjects in the absence of hunger. *Physiol Behav* 2011; **103**: 157–163.

42. Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology* 2001; **26**: 37–49.
43. Ulrich-Lai YM, Fulton S, Wilson M, Petrovich G, Rinaman L. Stress exposure, food intake and emotional state. *Stress* 2015; **18**: 381–399.
44. Born J, Lemmens S, Rutters F *et al*. Acute stress and food-related reward activation in the brain during food choice during eating in the absence of hunger. *Int J Obes (Lond)* 2010; **34**: 172–181.
45. Markus CR. Dietary amino acids and brain serotonin function; implications for stress-related affective changes. *Neuromolecular Med* 2008; **10**: 247–258.
46. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav* 2007; **91**: 449–458.
47. Schommer NC, Hellhammer DH, Kirschbaum C. Dissociation between reactivity of the hypothalamus–pituitary–adrenal axis and the sympathetic–adrenal–medullary system to repeated psychosocial stress. *Psychosom Med* 2003; **65**: 450–460.
48. Cota D, Tschöp MH, Horvath TL, Levine AS. Cannabinoids, opioids and eating behavior: the molecular face of hedonism? *Brain Res Rev* 2006; **51**: 85–107.
49. Kyrou I, Tsigos C. Stress hormones: physiological stress and regulation of metabolism. *Curr Opin Pharmacol* 2009; **9**: 787–793.
50. Chrousos GP. Stress and disorders of the stress system. *Nat Rev Endocrinol* 2009; **5**: 374–381.
51. Djurhuus C, Gravholt CH, Nielsen S, Pedersen SB, Møller N, Schmitz O. Additive effects of cortisol and growth hormone on regional and systemic lipolysis in humans. *Am J Physiol Endocrinol Metab* 2004; **286**: E488–EE94.
52. Sandeep TC, Andrew R, Homer NZ, Andrews RC, Smith K, Walker BR. Increased in vivo regeneration of cortisol in adipose tissue in human obesity and effects of the 11β-hydroxysteroid dehydrogenase type 1 inhibitor carbenoxolone. *Diabetes* 2005; **54**: 872–879.
53. Vanaelst B, Michels N, Clays E *et al*. The association between childhood stress and body composition, and the role of stress-related lifestyle factors—cross-sectional findings from the baseline ChiBS survey. *Int J Behav Med* 2014; **21**: 292–301.
54. Jackson SE, Kirschbaum C, Steptoe A. Hair cortisol and adiposity in a population-based sample of 2,527 men and women aged 54 to 87 years. *Obesity* 2017; **25**: 539–544.
55. Rutters F, Pilz S, Koopman AD *et al*. Stressful life events and incident metabolic syndrome: the Hoorn study. *Stress* 2015; **18**: 507–513.
56. Manenschijn L, Schaap L, Van Schoor N *et al*. High long-term cortisol levels, measured in scalp hair, are associated with a history of cardiovascular disease. *J Clin Endocrinol Metabol* 2013; **98**: 2,078–2,083.
57. Manenschijn L, van Kruysbergen RG, de Jong FH, Koper JW, van Rossum EF. Shift work at young age is associated with elevated long-term cortisol levels and body mass index. *J Clin Endocrinol Metabol* 2011; **96**: E1862–E1E65.
58. Noppe G, van den Akker E, de Rijke Y, Koper J, Jaddoe V, van Rossum E. Long-term glucocorticoid concentrations as a risk factor for childhood obesity and adverse body-fat distribution. *Int J Obes (Lond)* 2016.
59. Anton S, Martin C, Redman L *et al*. Psychosocial and behavioral pre-treatment predictors of weight loss outcomes. *Eat Weight Disord-St* 2008; **13**: 30–37.
60. Elder CR, Gullion CM, Funk KL, DeBar LL, Lindberg NM, Stevens VJ. Impact of sleep, screen time, depression and stress on weight change in the intensive weight loss phase of the LIFE study. *Int J Obes (Lond)* 2012; **36**: 86–92.
61. KH-C K, Bursac Z, DiLillo V, White DB, West DS. Stress, race, and body weight. *Health Psychol* 2009; **28**: 131.
62. Christaki E, Kokkinos A, Costarelli V, Alexopoulos E, Chrousos G, Darviri C. Stress management can facilitate weight loss in Greek overweight and obese women: a pilot study. *J Hum Nutr Diet* 2013; **26**: 132–139.
63. Cox TL, Krukowski R, Love SJ *et al*. Stress management–augmented behavioral weight loss intervention for African American women a pilot, randomized controlled trial. *Health Educ Behav* 2013; **40**: 78–87.
64. Folstein M, Liu T, Peter I *et al*. The homocysteine hypothesis of depression. *Am J Psychiatry* 2007; **164**: 861–867.
65. Bodnar LM, Wisner KL. Nutrition and depression: implications for improving mental health among childbearing-aged women. *Biol Psychiatry* 2005; **58**: 679–685.
66. Mishra GD, McNaughton SA, O'Connell MA, Prynne CJ, Kuh D. Intake of B vitamins in childhood and adult life in relation to psychological distress among women in a British birth cohort. *Public Health Nutr* 2009; **12**: 166–174.
67. Kennedy DO, Veasey R, Watson A *et al*. Effects of high-dose B vitamin complex with vitamin C and minerals on subjective mood and performance in healthy males. *Psychopharmacology (Berl)* 2010; **211**: 55–68.
68. Stough C, Scholey A, Lloyd J, Spong J, Myers S, Downey LA. The effect of 90 day administration of a high dose vitamin B-complex on work stress. *Hum Psychopharma Clin Exp* 2011; **26**: 470–476.
69. Long S-J, Benton D. Effects of vitamin and mineral supplementation on stress, mild psychiatric symptoms, and mood in nonclinical samples: a meta-analysis. *Psychosom Med* 2013; **75**: 144–153.
70. Grosso G, Pajak A, Marventano S *et al*. Role of omega-3 fatty acids in the treatment of depressive disorders: a comprehensive meta-analysis of randomized clinical trials. *PLoS One* 2014; **9**: e96905.
71. Noreen EE, Sass MJ, Crowe ML, Pabon VA, Brandauer J, Averill LK. Effects of supplemental fish oil on resting metabolic rate, body composition, and salivary cortisol in healthy adults. *J Int Soc Sports Nutr* 2010; **7**: 1.
72. Owen C, Rees A-M, Parker G. The role of fatty acids in the development and treatment of mood disorders. *Curr Opin Psychiatry* 2008; **21**: 19–24.
73. Markus C, Panhuysen G, Tuiten A, Koppeschaar H, Fekkes D, Peters M. Does carbohydrate-rich, protein-poor food prevent a deterioration of mood and cognitive performance of stress-prone subjects when subjected to a stressful task? *Appetite* 1998; **31**: 49–65.
74. Markus CR, Verschoor E, Smeets T. Differential effect of the 5-HTT gene-linked polymorphic region on emotional eating during stress exposure following tryptophan challenge. *J Nutr Biochem* 2012; **23**: 410–416.
75. Cerit H, Jans LA, Van der Does W. The effect of tryptophan on the cortisol response to social stress is modulated by the 5-HTTLPR genotype. *Psychoneuroendocrinology* 2013; **38**: 201–208.
76. Markus C, Panhuysen G, Jonkman L, Bachman M. Carbohydrate intake improves cognitive performance of stress-prone individuals under controllable laboratory stress. *Br J Nutr* 1999; **82**: 457–467.
77. Markus CR, De Raedt R. Differential effects of 5-HTTLPR genotypes on inhibition of negative emotional information following acute stress exposure and tryptophan challenge. *Neuropsychopharmacology* 2011; **36**: 819–826.
78. Capello AE, Markus CR. Effect of sub chronic tryptophan supplementation on stress-induced cortisol and appetite in subjects

differing in 5-HTTLPR genotype and trait neuroticism. *Psychoneuroendocrinology* 2014; **45**: 96–107.
79. Bin YS, Marshall NS, Glozier N. Secular trends in adult sleep duration: a systematic review. *Sleep Med Rev* 2012; **16**: 223–230.
80. Bin YS, Marshall NS, Glozier N. Sleeping at the limits: the changing prevalence of short and long sleep durations in 10 countries. *Am J Epidemiol* 2013; **177**: 826–833 kws308.
81. Van Cauter E, Knutson K, Leproult R, Spiegel K. The impact of sleep deprivation on hormones and metabolism. *Medscape Neurol Neurosurg* 2005; **7**.
82. Bonke J. Trends in short and long sleep in Denmark from 1964 to 2009, and the associations with employment, SES (socioeconomic status) and BMI. *Sleep Med* 2015; **16**: 385–390.
83. Olson R, Thompson SV, Wipfli B *et al*. Sleep, dietary, and exercise behavioral clusters among truck drivers with obesity: implications for interventions. *J Occup Environ Med* 2016; **58**: 314–321.
84. Charles LE, Slaven JE, Mnatsakanova A *et al*. Association of perceived stress with sleep duration and sleep quality in police officers. *Int J Emerg Ment Health* 2011; **13**: 229.
85. Iftikhar IH, Donley MA, Mindel J, Pleister A, Soriano S, Magalang UJ. Sleep duration and metabolic syndrome. an updated dose–risk metaanalysis. *Ann Am Thorac Soc* 2015; **12**: 1364–1372.
86. Ryu SY, Kim KS, Han MA. Factors associated with sleep duration in Korean adults: results of a 2008 community health survey in Gwangju metropolitan city. *Korea J Korean Med Sci* 2011; **26**: 1,124–1,131.
87. Matricciani L, Olds T, Petkov J. In search of lost sleep: secular trends in the sleep time of school-aged children and adolescents. *Sleep Med Rev* 2012; **16**: 203–211.
88. Keyes KM, Maslowsky J, Hamilton A, Schulenberg J. The great sleep recession: changes in sleep duration among US adolescents, 1991–2012. *Pediatrics* 2015; **135**: 460–468.
89. Walch OJ, Cochran A, Forger DB. A global quantification of "normal" sleep schedules using smartphone data. *Sci Adv* 2016; **2**: e1501705.
90. Chahal H, Fung C, Kuhle S, Veugelers P. Availability and night-time use of electronic entertainment and communication devices are associated with short sleep duration and obesity among Canadian children. *Pediatr Obes* 2013; **8**: 42–51.
91. Cappuccio FP, Taggart FM, Kandala N *et al*. Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* 2008; **31**: 619.
92. Hjorth MF, Chaput J-P, Ritz C *et al*. Fatness predicts decreased physical activity and increased sedentary time, but not vice versa: support from a longitudinal study in 8- to 11-year-old children. *Int J Obes (Lond)* 2014; **38**: 959–965.
93. Kjeldsen JS, Hjorth MF, Andersen R *et al*. Short sleep duration and large variability in sleep duration are independently associated with dietary risk factors for obesity in Danish school children. *Int J Obes (Lond)* 2014; **38**: 32–39.
94. Chaput J-P, Després J-P, Bouchard C, Tremblay A. The association between sleep duration and weight gain in adults: a 6-year prospective study from the Quebec Family Study. *Sleep* 2008; **31**: 517.
95. Chaput J-P, Després J-P, Bouchard C, Tremblay A. The association between short sleep duration and weight gain is dependent on disinhibited eating behavior in adults. *Sleep Med* 2011; **12**: S13.
96. Chaput J, Despres J, Bouchard C, Tremblay A. Longer sleep duration associates with lower adiposity gain in adult short sleepers. *Int J Obes (Lond)* 2012; **36**: 752–756.
97. Chaput J-P, Tremblay A. Does short sleep duration favor abdominal adiposity in children? *Int J Pediatr Obes* 2007; **2**: 188–191.
98. Gonnissen HK, Adam TC, Hursel R, Rutters F, Verhoef SP, Westerterp-Plantenga MS. Sleep duration, sleep quality and body weight: parallel developments. *Physiol Behav* 2013; **121**: 112–116.
99. Westerterp-Plantenga MS. Sleep, circadian rhythm and body weight: parallel developments. *Proc Nutr Soc* 2016; **75**: 431–439.
100. Klingenberg L, Chaput J-P, Holmbäck U, Jennum P, Astrup A, Sjödin A. Sleep restriction is not associated with a positive energy balance in adolescent boys. *Am J Clin Nutr* 2012; **96**: 240–248.
101. Hart CN, Carskadon MA, Considine RV *et al*. Changes in children's sleep duration on food intake, weight, and leptin. *Pediatrics* 2013; **132**: e1473–e1e80.
102. Brondel L, Romer MA, Nougues PM, Touyarou P, Davenne D. Acute partial sleep deprivation increases food intake in healthy men. *Am J Clin Nutr* 2010; **91**: 1,550–1,559.
103. Bosy-Westphal A, Hinrichs S, Jauch-Chara K *et al*. Influence of partial sleep deprivation on energy balance and insulin sensitivity in healthy women. *Obes Facts* 2008; **1**: 266–273.
104. Calvin AD, Carter RE, Adachi T *et al*. Effects of experimental sleep restriction on caloric intake and activity energy expenditure. *Chest J* 2013; **144**: 79–86.
105. Broussard JL, Kilkus JM, Delebecque F *et al*. Elevated ghrelin predicts food intake during experimental sleep restriction. *Obesity* 2016; **24**: 132–138.
106. Hjorth MF, Sjödin A, Dalskov S-M *et al*. Sleep duration modifies effects of free ad libitum school meals on adiposity and blood pressure. *Appl Physiol Nutr Metab* 2015; **41**: 33–40.
107. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med* 2004; **141**: 846–850.
108. Gonnissen HK, Hursel R, Rutters F, Martens EA, Westerterp-Plantenga MS. Effects of sleep fragmentation on appetite and related hormone concentrations over 24 h in healthy men. *Br J Nutr* 2013; **109**: 748–756.
109. McNeil J, Drapeau V, Gallant A, Tremblay A, Doucet E, Chaput J. Short sleep duration is associated with a lower mean satiety quotient in overweight and obese men. *Eur J Clin Nutr* 2013; **67**: 1,328–1,330.
110. Nedeltcheva AV, Kilkus JM, Imperial J, Kasza K, Schoeller DA, Penev PD. Sleep curtailment is accompanied by increased intake of calories from snacks. *Am J Clin Nutr* 2009; **89**: 126–133.
111. Zheng H, Lenard N, Shin A, Berthoud H-R. Appetite control and energy balance regulation in the modern world: reward-driven brain overrides repletion signals. *Int J Obes (Lond)* 2009; **33**: S8–S13.
112. St-Onge M, Wolfe S, Sy M, Shechter A, Hirsch J. Sleep restriction increases the neuronal response to unhealthy food in normal-weight individuals. *Int J Obes (Lond)* 2014; **38**: 411.
113. St-Onge M-P, McReynolds A, Trivedi ZB, Roberts AL, Sy M, Hirsch J. Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. *Am J Clin Nutr* 2012; **95**: 818–824.
114. Born JM, Martens MJ, Rutters F, Lemmens SG, Goebel R, Westerterp-Plantenga MS. High HPA-axis activation disrupts the link between liking and wanting with liking and wanting related brain signaling. *Physiol Behav* 2012; **105**: 321–324.
115. Chaput J-P, Tremblay A. Sleeping habits predict the magnitude of fat loss in adults exposed to moderate caloric restriction. *Obes Facts* 2012; **5**: 561–566.
116. Filiatrault M, Chaput J, Drapeau V, Tremblay A. Eating behavior traits and sleep as determinants of weight loss in overweight and obese adults. *Nutr Diabetes* 2014; **4**: e140.
117. Thomson CA, Morrow KL, Flatt SW *et al*. Relationship between sleep quality and quantity and weight loss in women

participating in a weight-loss intervention trial. *Obesity* 2012; **20**: 1,419–1,425.
118. Cizza G, Marincola P, Mattingly M *et al*. Treatment of obesity with extension of sleep duration: a randomized, prospective, controlled trial. *Clin Trials* 2010; **7**: 274–285.
119. Barclay NL, Eley TC, Rijsdijk FV, Gregory AM. Dependent negative life events and sleep quality: an examination of gene-environment interplay. *Sleep Med* 2011; **12**: 403–409.
120. Mezick EJ, Matthews KA, Hall M *et al*. Intra-individual variability in sleep duration and fragmentation: associations with stress. *Psychoneuroendocrinology* 2009; **34**: 1,346–1,354.
121. Vahtera J, Kivimäki M, Hublin C *et al*. Liability to anxiety and severe life events as predictors of new-onset sleep disturbances. *Sleep* 2007; **30**: 1,537–1,546.
122. Åkerstedt T, Orsini N, Petersen H, Axelsson J, Lekander M, Kecklund G. Predicting sleep quality from stress and prior sleep—a study of day-to-day covariation across six weeks. *Sleep Med* 2012; **13**: 674–679.
123. Calkins AW, Hearon BA, Capozzoli MC, Otto MW. Psychosocial predictors of sleep dysfunction: the role of anxiety sensitivity, dysfunctional beliefs, and neuroticism. *Behav Sleep Med* 2012; **11**: 133–143.
124. Buckley TM, Schatzberg AF. On the interactions of the hypothalamic–pituitary–adrenal (HPA) axis and sleep: normal HPA axis activity and circadian rhythm, exemplary sleep disorders. *J Clin Endocrinol Metab* 2005; **90**: 3,106–3,114.
125. Rodenbeck A, Huether G, Rüther E, Hajak G. Interactions between evening and nocturnal cortisol secretion and sleep parameters in patients with severe chronic primary insomnia. *Neurosci Lett* 2002; **324**: 159–163.
126. Steiger A. Sleep and the hypothalamo–pituitary–adrenocortical system. *Sleep Med Rev* 2002; **6**: 125–138.
127. Jackson ML, Sztendur EM, Diamond NT, Byles JE, Bruck D. Sleep difficulties and the development of depression and anxiety: a longitudinal study of young Australian women. *Arch Womens Ment Health* 2014; **17**: 189–198.
128. Guyon A, Balbo M, Morselli L *et al*. Adverse effects of two nights of sleep restriction on the hypothalamic–pituitary–adrenal axis in healthy men. *J Clin Endocrinol Metabol* 2014; **99**: 2,861–2,868.
129. Matricciani LA, Olds TS, Blunden S, Rigney G, Williams MT. Never enough sleep: a brief history of sleep recommendations for children. *Pediatrics* 2012; **129**: 548–556.
130. Markus CR, Jonkman LM, Capello A, Leinders S, Hüsch F. Sucrose preload reduces snacking after mild mental stress in healthy participants as a function of 5-hydroxytryptamine transporter gene promoter polymorphism. *Stress* 2015; **18**: 149–159.