Chapter 5

Stress and Adiposity in Children

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Recent Advances in Obesity in Children

Abstract

Excessive caloric intake, insufficient physical activity and sleep deprivation are major lifestyle factors involved in the development of childhood overweight. Another recently identified potential predictor of overweight is chronic stress. Although children are not always recognized as being susceptible to stress, chronic exposure to stressful situations is not uncommon and may adversely affect their physiological and psychological health. After all, childhood is a period of incessant physical and brain development creating the foundation of adult psychopathology, lifestyle and adiposity.

This chapter aims to give a short literature overview on the stress-adiposity associations, its underlying pathways, some future research perspectives and finally prevention and intervention strategies to tackle the stress-adiposity association, particularly in children.

Some evidence on the stress-adiposity relation exists but the directionality is still unclear. Therefore, longitudinal studies are warranted. Recent reviews also highlighted the paucity of studies in the young age groups such as primary school children. Major underlying pathways are the stress hormone cortisol and lifestyle changes like diet, physical activity and sleep. More innovative suggested mechanisms are chronic inflammation and the composition and activity of the gut bacteria. Importantly, sex, age, cortisol reactivity and lifestyle can modify the strength or direction of the stress-adiposity association. From a pediatric perspective, the role of parents should of course also

be considered. Strategies to prevent or intervene in this stress-adiposity relationship should not only target children but also parents and schools. Relevant strategies are stress management like emotional regulation and lifestyle intervention.

Definition and Importance of Stress

Several definitions for stress exist. The most common definition is based on the transactional theory (interaction person/environment): "Stress involves a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" [1]. Consequently, stress arises when the demands of a situation exceed an individual's ability to cope and resolve the problematic situation, resulting in emotional, behavioural and cognitive disturbances [2]. Consequently, stress is an adaptive, dynamic state that starts with a stimulus, the stressor, which initiates stress appraisal. When homeostasis is threatened i.e. when there is a discrepancy between what is expected or the 'normal' situation (set point) and what is happening in reality (actual value), a physiological and psychological coping response will be initiated that induces arousal. The physiological stress response aims to regain homeostasis by giving priority to essential body functions. As shown in Figure 1, two endocrine systems are regarded as primary components of the stress response: the hypothalamic-pituitary-adrenal (HPA) axis with cortisol as end-product and the autonomic nervous

system (ANS) with the catecholamine adrenaline and noradrenaline as end-product.

Nevertheless, not all stressors will trigger emotional and/or behavioural disturbances and illness. After all, the stress response is a positive alarm by mobilizing physiological resources to initiate and improve performance. As a result, the body will try to regain the balance or homeostasis. The likelihood for a negative outcome is highest when the demands are high and the coping or control is low. Chronic, unpredictable and uncontrollable stressors are the most difficult to cope with. Unpredictable stressors will be accompanied by an absence of an anticipatory hormonal response, while uncontrollable and chronic stressors will be accompanied by high stress hormones with a reduced recovery afterwards. Since the arousal will sustain until the reason for the arousal is eliminated, these difficult situations will not lead to phasic arousal but to sustained, chronic arousal. It is this chronic arousal or chronic stress that can initiate pathophysiological processes of illness [4, 5] since the stress system will give priority to certain physical functions over other functions such as growth, digestion, immune function and reproduction. Cortisol as a glucocorticoid modulates the expression of approximately 10% of the human genes [6] amongst others those involved in inflammation and metabolism. And also the catecholamines exert regulatory effects on several systems such as the cardiovascular, pulmonary, hepatic, skeletal muscle and immune system. Accordingly, cancer, inflam-

matory, pulmonary, gastrointestinal and cardiovascular/metabolic diseases have been associated with stress [7-9]. Also adverse childhood experiences have been shown to induce allostatic load with changes in the nervous, endocrine and immune systems during childhood and future adulthood [10].

Also in children, stressors are highly prevalent. In the European IDEFICS sample of 4-11y old children, 53.4% lived in familial/social adversities (divorce, bad family climate, low socio-economic status, etc.) and 40.3% experienced at least one major negative life event such as a parental divorce [11]. More specifically, between 6-58% (average 23%) of the 11y olds in the international HBSC study reported to be pressured by schoolwork and between 2-23% (average 12%) reported to be bullied at school [12]. Mental health problems or emotional/behavioural problems affect 10-20% of children and adolescents worldwide [13,14]. In the international 2009/2010 HBSC study, between 70 and 96% (average 88%) of 11y olds reported high life satisfaction. Low life satisfaction might be explained by the high prevalence of psychosomatic complaints (physical complaints without a medical explanation) and psychological problems in childhood. Between 12 and 65% (average 28%) of the 11y olds reported these problems more than once a week [12]. Also in the European IDEFICS 4-11y old sample, 45.5% of children experienced at least one psychosomatic or emotional symptom,

with low emotional well-being during the last week being most frequently reported (38.2%) [15]. This childhood stress can be measured using questionnaires/interviews and also more objective biomarkers [16].

Short Literature Overview Regarding Stress-Adiposity Associations

In examining literature, the presence of a publication bias (i.e. mostly only studies with significant results are published) should be considered. Consequently, the discussed information is probably an overestimation of the reality. Moreover, stress will be considered in a broad definition including correlates such as depression.

Overall the literature underlines the importance of the bidirectional associations between mental health and adiposity [17]. Stress can increase adiposity through cortisol and lifestyle, while adiposity can increase stress mainly through lower self-esteem and stigma but also biological pathways like increased inflammation and cortisol dysregulation have been mentioned [18, 19]. The vicious cycle hypothesis can explain why people with stress or mental health problems will gain weight and people with a certain level of adiposity will show enhanced emotional eating and encounter more and more problems to control their weight. The evidence is relatively strong in teenagers and adults, but the strength and amount of evidence

in young children is much weaker [17]. Nevertheless, it should be considered that stress might also lead to weight decreases in some people. In paragraph 3.6, we go further into this topic by emphasizing the behavioural reactions to stress that might differ e.g. the level of physical activity.

In adults, a recent meta-analyses of longitudinal studies showed that depressed people had 58% more risk to become obese and obese people also had 55% more risk to become depressed [20]. In another meta-analysis, work and life stress also significantly increased BMI and/or waist longitudinally [21].

In children and adolescents, less research has been performed but certainly not negligible since it is booming the last years [22]. A systematic review including 12 longitudinal studies [23] on clinical depression, perceived stress, anger/anxiety and behaviour concluded that the evidence is low for this relation in children/adolescents. Another review focused on the effect of household and individual stressors on overweight parameters [24]. Concerning the household stressors, self-esteem, financial strain, maternal depression, maternal distress and neglect showed significant associations with adiposity. For the individual stressors, depression was often examined and significant, although also more broad parameters such as future life goals, substance use and overall stress perceptions were significantly related to BMI. The used adiposity parameter was almost exclusively BMI (sometimes parentally reported) and not all studies were longitudinal. In table 1, an overview can be found of recent longitudinal

paediatric studies on stress-related predictors of adiposity.

Table 1: Overview of prospective studies about child and adolescent stress and negative affect as predictors of weight gain/overweight/adiposity: Prospective studies.

First author, year	Country	Follow-up duration	Sample (n) /gender (% girls)	Age or grades at baseline	Psychological predictor: Measurement tool	Adiposity measures	Results
Goodman (2002) [4]	USA	1 year	N=9374 (48.6%)	Grades 7-12 (12-18 years)	Depressive symptoms: CES-D	Measured and self-reported: BMI	Depressive symptoms increased risk of obesity in obese and non-obese subjects
Anderson (2010) [5]	USA	2 years	N=918 (100%)	Grade 6 (11 years)	Depressive symptoms: CES-D	Measured: BMI	Depressive symptoms associated with greater likelihood of obesity in white females
Bradley (2008) [6]	USA	11 years	N=1254 (48%)	2 years to grade 6	Externalizing and internalizing problems: CBCL	Measured: BMI	Positive association between externalizing or internalizing problems and BMI
van Jaarsveld (2009) [7]	UK	5 years	N=4065 (42%)	11-12 years	Perceived stress at a single time point and mean perceived stress over 5 years: PSS short form	Measured: waist, BMI	Perceived stress associated with Δ BMI z-scores, Δ waist High Mean of stress associated with 5-year BMI z-score trajectory and 5-year waist z-score trajectory
Midei (2009) [8]	USA	3 years	N=160 (48.7%)	14 years	Trait anxiety symptoms: STAIC. Anger and anxiety: Cook-Medley Hostility Scale	Measured: waist hip ratio	Higher anger associated with increased waist-hip ratio
Tanofsky-Kraff (2006) [9]	USA	4.2 years	N=146 (52%)	6-12 years	Depressive symptoms: CDI Child Symptom Inventory	Measured: body fat mass	No association
Rofey (2009) [10]	USA	3 years	N=285 (49%)	8-18 years	Depression and anxiety: K-SADS, K-SAFD-P (DSM-III and DMS-IV criteria)	Measured: BMI	Females: depression and anxiety associated with high BMI Males: anxiety associated with high BMI No association with depression
Aparicio (2013) [11]	Spain	3 years	N=229 (62%)	10 years	Depression and anxiety: SCARED, CDI MINI-Kid (DSM-IV criteria)	Measured: BMI, waist, body fat mass	Females: anxiety symptoms associated with Δ BMI and body fat mass; depression disorder associated with Δ waist Males: anxiety and depression associated with Δ BMI and waist Inverse relationship observed between major depression disorder and BMI
Larsen (2014) [12]	Netherlands	3 years	N=1465 (49.4%)	11.4-16.9 years	Depressive symptoms: CES-D	Measured: BMI	Females: depressive symptoms associated with higher zBMI Males: no association
Rhew (2008) [13]	USA	1 year	N=466 (46.2%) Sub-sample BMI measures: N=165	12 years mean	Depressive symptoms: MFQ	Measured and self-reported: BMI	BMI self-reported: Males: depressive symptoms associated with lower BMI than non-depressive symptoms Females: depressive symptoms associated with higher BMI than non-depressive symptoms BMI measures: no association
Michels (2014) [14]	Belgium	2 years	N=316	5-12 years	Negative events: Coddington Life Events Scale for Children Negative emotions: anger, anxiety, sadness Behavioural Problems: SDQ	Measured: BMI, waist-to-height ratio, fat percentage	Stress positively or negatively associated with adiposity depending on cortisol and life-style, which had a moderating effect
Stice (2005) [15]	USA	4 years	N=496 (100%)	11-15 years	Depressive symptoms: SADS for School-Age Children	Measured: BMI	No association
Jansen (2008) [16]	Netherlands	3 years	N=787 (49%)	9-10 years	Depressive symptoms and social anxiety : Rotterdam Youth Health Monitor RYM questionnaire, Short Depression Inventory for Children, Dutch social anxiety scale for children	Measured: BMI	No association
Chen (2010) [17]	USA	4 years	N=543 (100%)	10 years	Depressive symptoms: CSI (reported by parents) (DSM-IV criteria)	Measured: BMI	No association
Hammerton (2014) [18]	UK	1-2 years	N=289	9-17 years	Depressive disorder: CAPA (DSM-IV criteria)	Measured: BMI	No association

Child or adolescent emotional problems and adult overweight/adiposity							
Pine (1997) [19]	USA	8-10 years	N=644	9-18 years	Depressive symptoms: CES-D (DSM-III criteria)	Self-reported: BMI	Depressive symptoms associated with higher BMI
Pine (2001) [20]	USA	10 years	N=177	6-17 years	Depressive symptoms: SADS Lifetime Disorders version	Self-reported: BMI	Depressive symptoms associated with high BMI in adulthood
Franko (2005) [21]	USA	2-5 years	N=1554 (100%)	16 years	Depressive symptoms: CES-D	Measured and self-reported: BMI	Depressive symptoms associated with high BMI and with obesity
Anderson (2006) [27]	USA	22 years	N=661	15 years mean (9-18 years)	Depression and anxiety: DISC (DSM-IV criteria)	Self-reported: BMI	Females: positive relation Males: no association
Richardson (2003) [22]	USA	10 years	N=881	11-15 years	Depression disorder: DISC (DSM-III criteria)	Measured: BMI	Females: depression in later adolescence increases risk of obesity in adulthood; no association in early adolescence Males: no association
Ternouth (2009) [23]	UK	20 years	N=3359	10 years	Emotional problems: Rutter B scale	Self-reported: BMI	Females: childhood emotional problems predicted weight gain in women Males: no association
Hasler (2005) [24]	Germany	21 year	N=591 (50.5%)	19 years	Child depressive symptoms: SPIKE	Self-reported: BMI	Females and males: depressive symptoms before age 17 associated with increased weight gain
Duarte (2010) [25]	Finland	18-23 years	N=2209 (0%)	8 years	Depressive symptoms: Depression, CDI Emotion problems (Rutter questionnaire)	Measured: BMI	No association
Wickrama (2009) [26]	USA	6 years	N=11,404	12-19 years	Depressive symptoms: CES-D	Self-reported: BMI	No association

CDC: Centers for Disease Control and Prevention; CES-D: Center for Epidemiologic Studies Depression Scale; CBCL: Child Behaviour Checklist; PSS: Perceived Stress Scales; STAIC: State-Trait Anxiety Inventory for Children; CDI: Children's Depression Inventory; SCARED: Screen for Child Anxiety Related Emotional Disorders; MINI-KID: MINI-International Neuropsychiatric Interview for Children and Adolescents; MFQ: Mood and Feeling Questionnaire; SDQ: Strengths and Difficulties Questionnaire; RYM: Rotterdam Youth Monitor; SADS: Schedule for Affective Disorders and Schizophrenia; CSI: Child Symptom Inventory; CAPA: Child and Adolescent Psychiatric Assessment; DISC: Diagnostic Interview Schedule for Children; SPIKE: Structured Psychopathological Interview and Rating of the Social Consequences for Epidemiology.

Apart from the stress effects on adiposity, adiposity effects on stress also have been investigated in children [25,26]. Moderate levels of body dissatisfaction and a significant reduction in global self-esteem, social functioning and quality of life in obese youth was seen, but only few were depressed.

Mechanisms of Stress Effects on Adiposity

Several mechanisms in the effect of stress on adiposity have been described in literature, mainly divided in physiological and behavioural pathways, as will be discussed in the next paragraphs.

Firstly, direct metabolic changes (such as increased visceral fat disposition and a stimulation of appetite) are mainly caused by a dysregulation of the stress system and the production of stress hormones like cortisol. This is in agreement with the concept of stress-induced allostatic load leading to detrimental physiological consequences due to overactive endocrine reactions [8]. Research still introduces more and more plausible physiological pathways like the role of inflammation and gut bacteria.

Secondly, stress may indirectly facilitate adiposity through behavioural pathways such as maladaptive coping behaviours leading to an adiposity stimulating lifestyle: emotional eating of 'comfort' food (rich in sugar and fat), a disordered sleep and a lack of exercise with an increase in screen time. After all, anatomical links exist in the regulation of stress, energy and sleep [27] by anatomical and functional inter-correlations between regions of the hypothalamus: stress regulation in the paraventricular nucleus, hypocretin cells that regulate sleep in the lateral area and metabolism regulation in the arcuate nucleus.

The Stress Hormone Cortisol as Stimulator of Adiposity

Until now, the HPA axis appears to be involved in food intake and fat deposition more directly and by more complex pathways than the ANS axis [28].

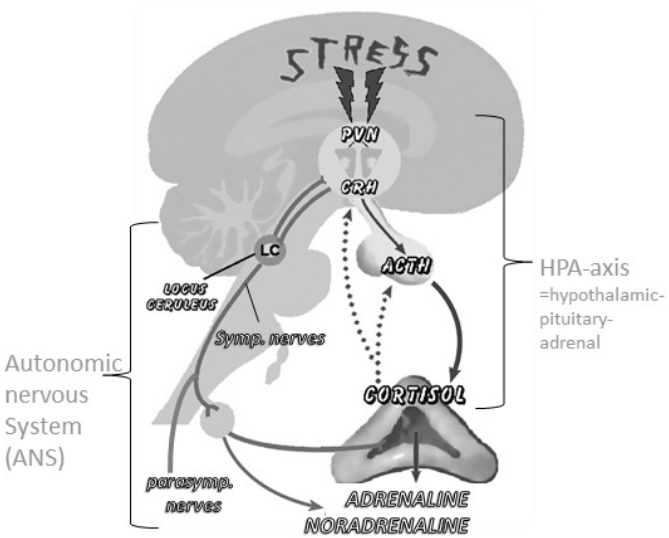

Figure 1: Physiology of the stress response [3]. (copyright obtained).
PVN= Paraventricular Nucleus; CRH= Corticotrophin Releasing Hormone; ACTH=Adrenocorticotropic Hormone; LC= Locus Coeruleus

In most cases, stress increases cortisol. This cortisol interacts with lipid metabolism in two ways as summarized in Figure 2 [29]. First, cortisol increases the amount of free circulating fatty acids by stimulating the lipoprotein lipase enzyme (LPL). These free fatty acids can then be used to accumulate fat in fat cells. Secondly, cortisol influences this fat storage. Increased adiposity is caused by hyperplasy (adipogenesis) and in the presence of insulin also by hypertrophy (lipogenesis). Cortisol may also influence lipolysis (degradation of adipose tissue triglycerides to fatty acids). Both prolipolytic and antilipolytic activity has been hypothesized, but the mechanisms are still unclear and may depend on duration, dose and location of cortisol exposure. Antilipolytic activity has mainly been observed in high cortisol concentrations and in the abdominal region. The fat storage chiefly occurs in the visceral fat cells since the cortisol receptors have a high density in this region [30].

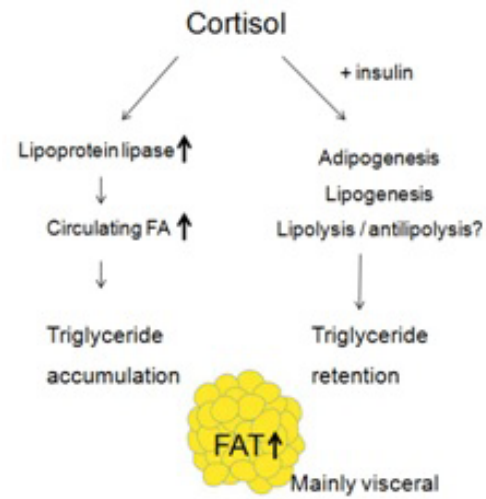

Figure 2: The effect of cortisol on adiposity.
FA= fatty acids

Mechanistic pathways have largely been investigated in animal studies, but some studies also tried to find this link in human observational studies. In identical twins (identical genetic information), a difference in visceral fat accumulation (rather than obesity in general) could be explained by higher psychosocial stress and cortisol and noradrenalin levels [31]. Nevertheless, it should be mentioned that some stress situations are not associated with increased but with no change or even a decrease in cortisol [32].

Dietary Changes in Relation to Stress

Stressed people may eat increased amounts of unhealthy food since eating functions as a way to cope with stress as it leads to distraction [33]. The underlying pathway is the increased cortisol during stress [34-37].

The high cortisol during stress increases reward sensitivity and appetite (see Figure 3). Cortisol stimulates the reward pathways (opioid and dopamine system) especially when people are on cognitive restraint. More importantly, cortisol also influences appetite hormones: it upregulates neuropeptide Y (NPY) and dysregulates insulin and leptin. The NPY increases appetite and reward. On the other hand, insulin and leptin decrease appetite and reward but due to the dysregulation the body becomes resistant and appetite and reward will be increased. Consequently, mainly rewarding food items will be consumed. These foods rich in sugar and fat have been called 'comfort food'. In pediatric literature, perceived stress has been cross-sectionally associated with more snacking [38] and longitudinally with sweet food intake in preadolescents [39] and with less fruit/vegetables and more snacks [40] or an overall lower diet quality [41] in adolescents. Also in children, cortisol has been associated with unhealthier intake, mainly sweet food consumption [42]. In laboratory experiments, preadolescents showed more snacking but especially those that were high in restraint, even when other stress coping behaviours were freely available [43,44].

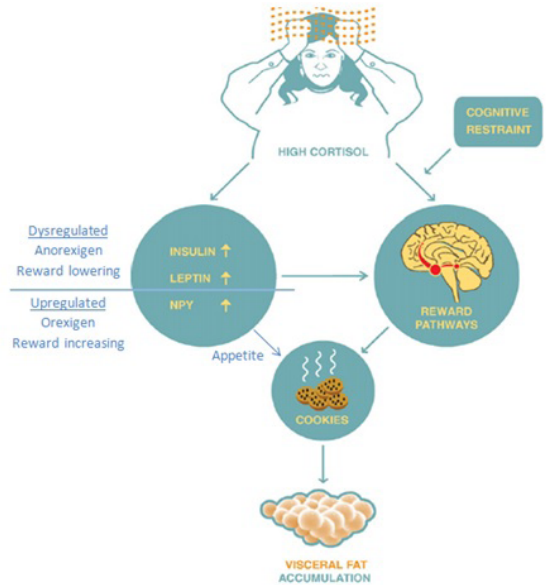

Figure 3: The effects of cortisol on food consumption [34] (slightly adapted, copyright obtained).

In this regard, the association between stress and food consumption must be visible as well in people's reports on their eating behaviour. Schlundt demonstrated that different eating behaviours can be observed during a two-week period, such as external eating, restrained eating, emotional eating or binge eating [45]. In relation to stress, emotional eating is the most important. People with an emotional eating behaviour have learned to label the negative feelings of stress as 'hunger' [46] and will think about food as an escape from stress [34,35]. According to Van Strien, external eating refers to a personal trait reflected in a tendency to overeat in reaction to food cues whereas restrained eating means eating less than wanted when exposed to food, which is not necessarily associated with overeating [47]. Both are however also linked with stress but the direction of the effect is less clear [33]. In the scarce literature on children and adolescents, negative emotions and problems have been associated mainly with emotional eating [48-50], although relations with the 3 eating behaviours have been found depending on sex and age [39].

In sum, the stress-diet relation is very complex involving multiple pathways, molecules and receptors in different brain regions. Even parallelisms of these stress-induced food cravings with addiction mechanisms have been found [51,52]. Apart from stress-induced increases of eating also stress-induced decreases of eating have been reported in the absence of palatable food, in people that

are less sensitive to emotional eating or in the case of very intense emotions (acute stress) [33,53]. Moreover, reversed causation might also be present by dietary patterns that influence stress and cortisol [54].

Physical Activity Changes in Response to Stress

In the natural stress reaction, energy is mobilized as preparation for activity in the fight/flight response. Consequently, physical activity would be an ideal stress reaction since it burns the mobilized energy and prevents energy storage. Nevertheless, stress is seldom leading to physical activity in our modern life and the energy is stored again [55].

The hypotheses on the stress-activity relation are shown in Figure 4. Stressed people probably might not have the motivation or the time to be active resulting in decreased activity. Nevertheless, physical activity can be used as a way of coping by some adults and children, especially those with a high usual level of physical activity [44,56]. Interestingly, the effect in children might also depend on age since positive longitudinal associations have been found in 6-8y old children, while negative longitudinal associations have been found in 9-11y old children [39]. Remarkably, most research has focused on the effect of physical activity on stress since it distracts from stress, it generates euphoric feeling, it increases social support and even decreases the stress response [55]. Nevertheless,

physical activity could also increase the stress level if there is high performance and time pressure. Recent reviews in children/adolescents showed a limited effect on stress although this is mostly based on cross-sectional findings [57,58].

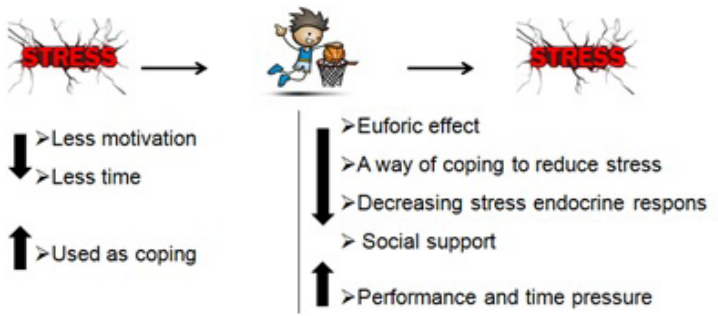

Figure 4: Associations between stress and physical activity.

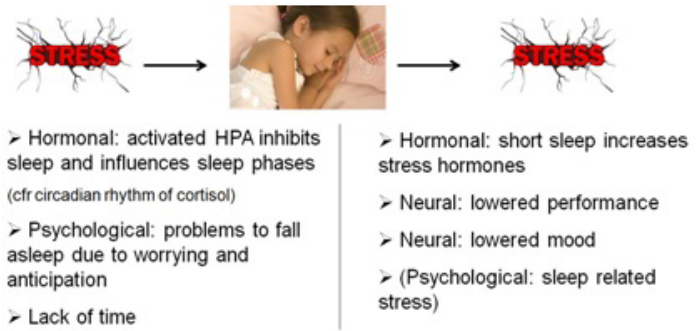

Figure 5: Associations between stress and sleep. HPA= hypothalamic-pituitary-adrenal axis

Apart from a decreased physical activity level, a simultaneous increase of screen time is hypothesized. Indeed, evidence in reviews on child/adolescent populations was even more consistent for an association between high sedentary screen time and poor mental health than for physical activity [57,59].

Sleep Changes in Response to Stress

Sleep is the metabolic antagonist of stress because of their opposite effects on heart rate, blood flow and hormones [60,61].

Not only sleep duration may be influenced by stress but also sleep quality such as long sleep latency, many awakenings or short time in important sleep phases such as the rapid eye movement (REM) and slow wave sleep phase [62].

Apart from the fact that stressed people might have a lack of time for long sleep, also genetic, hormonal, neural and psychological mechanisms exist (see Figure 5). On a hormonal level, short sleep increases stress hormones and these hormonal changes inhibit sleep and influences important sleep phases. The difficult combination of stress with sleep can be demonstrated by the circadian rhythm of the stress hormone cortisol: cortisol has a peak in the morning when awakening and very low levels in the night when preparing to sleep. On neural level, the regulation of sleep, behaviour and emotions are closely related: interac-

tions of sleep loss on amygdala and prefrontal cortex functions [63,64] and also anatomical interconnections between different centres of the hypothalamus that regulate sleep and stress [27] have been found. Consequently, sleep loss will be associated with deteriorated performance and mood. On psychological level, sleep is essential for proper emotion regulation while stress can result in problems to fall asleep due to rumination or next day anticipation. Rarely, sleep related stress is reported: being stressed because of worrying about the sleeping problems [65].

A recent review showed that the sleep-stress relation in children and adolescents is likely bidirectional: most evidence shows that sleep problems or insufficient sleep exacerbate emotional and behavioural difficulties, while mood disturbances and anxiety perhaps compromise sleep patterns. Nevertheless, the field is full of discrepancies recommending more longitudinal research in different age groups [63].

More Explorative Mechanisms Between Stress and Adiposity

Figure 6 shows the interaction of stress with inflammation and gut bacteria. Although less clinical and community research has been done on these possible pathways between stress and adiposity, existing research (mainly preclinical) shows they are promising.

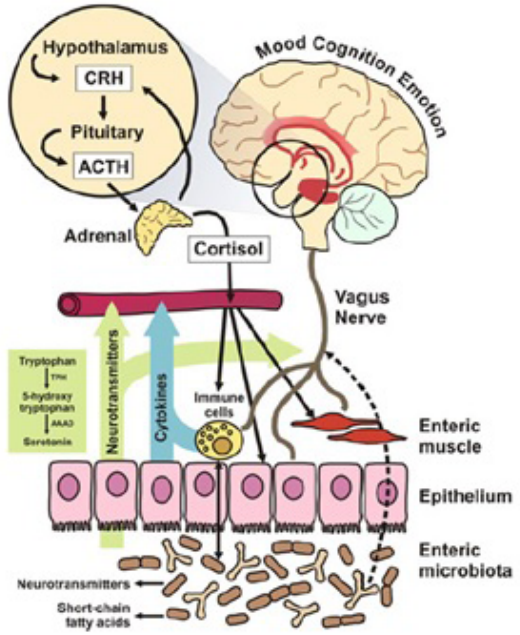

Figure 6: The interaction of stress with inflammation and gut bacteria [66] (copyright obtained).

Stress might initiate a low grade inflammatory process, a situation of chronic and systemic inflammation not triggered by external factors (e.g. infections), in two ways. First, indirectly, emotional distress can lead to an unhealthy lifestyle (e.g. sleep problems, imbalanced diet) that subsequently induces inflammation. Directly, stress-induced catecholamines stimulate the so-called NFκB

pathway to macrophages and pro-inflammatory cytokine production leading to low grade inflammation [67,68]. Interestingly, this pro-inflammatory NFκB pathway is a key modulator for brain inflammation. Indeed, peripheral inflammation can become central inflammation by inflammatory processes in the hypothalamus, the central energy metabolism regulator. In contrast to cachexia-inducing inflammation by pathogens, this metabolic inflammation (due to low-grade inflammation and unhealthy diet) may result in increased food intake and finally adiposity by resistance to the anorexigenic factors leptin and insulin [69,70]. Also the other direction is possible: adiposity might increase stress due to increased inflammation. After all, excessive fat will trigger inflammatory cytokine production that may lead to increased "stress sensitivity" by reaching the brain and affecting stress-related neurocircuitry, neuroendocrine activity and neurotransmitters [71,72].

A second novel pathway between stress and adiposity are the gut bacteria. The human gut contains 1011 bacteria per gram intestinal content that play a role in optimal body function but the composition can easily be disturbed e.g. by an unhealthy diet or by medication use. Apart from their intestinal functions such as food fermentation, gut microbiota have important immunological functions since they help protecting us against pathogens by preventing colonization, preventing invasion of the gut barrier and controlling the immune inflammatory responses [73].

The current evidence shows that obese people have a reduced gut microbiota diversity and a different composition of microbiota species [74]. A bidirectional relation has been suggested here. Disturbed gut microbiota can be involved in adiposity development as microbiota metabolites have been shown to interfere with satiety regulation and energy-expenditure. In addition, signals from the gut microbiota can increase inflammation and stimulate fat storage [75]. On the other hand, a changed gut microbiota spectrum might also be a consequence of adiposity due to the individual's unbalanced diet and/or other potential mechanisms.

Less evidence exists in humans on the stress-microbiota relation. Again, a theoretical bidirectional relationship between the gut microbiota and the stress-regulatory centre in the brain can be hypothesized [76]. This is sometimes called the 'microbiota-brain-gut axis' since microbiota play an active role in the gut-brain communication. In the stress-microbiota relation several pathways exist: neural (autonomic and enteric nervous system), neuro-endocrine (including the enterochromaffin cells in the gut epithelium and effects of short-chain fatty acid produced by the gut microbiota) and neuroimmune (cytokines) pathways. Preclinical data on the stress-microbiota relation is booming the last years [66]. Animal models of stress have revealed interesting gut microbiota alterations as a result of stress; and in the opposite direction germ-free animals and antibiotic and probiotic treatment in animals showed

changes in behaviour and related biochemistry such as cortisol activity [66]. This preclinical data emphasizes that the gut microbiota is an attractive target for therapeutic strategies. In humans, probiotic or prebiotic treatment is the most straight-forward method to start with experimental research and when its effect is proven whole-diet changes can be considered as a more natural alternative. In humans, a few small probiotic interventions in healthy adults have been performed to change emotional state [77,78]. Also in children, probiotics have been tested for several diseases like genitourinary, enteric, allergic and atopic disorders [79]. Nevertheless, no such probiotic interventions in humans with depression have been published up to now.

Factors That Might Moderate the Stress-Adiposity Relationship

The stress-adiposity relation might not be significant in all people. For example, it is well-known that age and sex might be important moderators [17], but also cortisol and lifestyle might be moderators.

The relation between stress and adiposity can be sex-dependent since sex differences have been reported in all the underlying aspects. Sex differences have been shown in the prevalence, developmental pathways during childhood and manifestation of psychopathology [80] and in handling stressful situations in children [81]. Sex differences in children's peer context have also been reviewed

with girls being more sensitive to the friendship status, being more exposed to a wide variety of peer stressors and receiving higher levels of emotional provisions in their friendships [82]. Women generally show an increased responsiveness to stress due to effects of sex hormones on glucocorticoid receptors, the brain corticotrophin releasing hormone action, adrenal responsiveness to ACTH and cortisol feedback inhibition [83]. Although they have a lower overall dietary intake, women tend to prefer more the sweet/fatty food items [84]. Moreover, women experience enhanced rewarding and enhanced appetitive emotional learning resulting in higher levels of craving and eating disorders [83]. Women also tend to have more potent satiation reactions to leptin and serotonin while lower satiety reactions to insulin and cholecystokinin [83]. Finally, women are more likely to develop obesity [85], but in children and adolescents obesity tend to be higher in boys [86]. Anyhow, the literature is inconsistent: most studies report stronger stress-adiposity correlations in women and girls [17,87,88] especially when focusing on the effect of adiposity on stress, but sometimes higher correlations in men have been reported [21].

Especially in children, also age can be a moderator. Some discussion in literature exists about the age group that is most vulnerable to stress-induced adiposity. A study in 4065 adolescents reported consistent cross-sectional stress-adiposity relations during four measurement waves but no longitudinal relations [89]. This finding sug-

gests that the relations may already be established during childhood. Also in a sample of 2278 children, behavioural problems during childhood predicted adiposity better than those during adolescence [90]. In contrast, other research hypothesizes that adiposity development would be more clearly visible during adolescence and adulthood [86,91]. A first explanation might be the puberty-related increases in stress response and emotional reactivity [92] and of course also in fat deposition. Apart from biological reasons, the natural process of children's transition to adolescence is the decreasing parental control. From the age of 10 on, decreasing daily interactions with their family have been found [93]. Children's diet and emotional eating behaviour has previously been correlated with that of their parents [94-96]. Nevertheless, a recent meta-analysis/review has shown that the parent-child relation should not be overstated and that there is a trend of diminishing influence the last decade by changing society (i.e. increasing importance of other influencing players like school and peers) [97]. Moreover, the intake of sweet foods in 12-year olds was influenced by their emotional eating behaviour above the parental influence. Also decreased parental influence with age has been reported for sleep duration [98] and for physical and sedentary behaviour [99].

Importantly, the above mentioned mediating factors, cortisol and lifestyle might also be moderators, as shown in Figure 7. After all, not everybody seems to react in the same way to psychosocial problems since it is depending on behavioural preferences and personal reactivity [33,44,83,100, 101]. For example, emotional state has been associated with both increased and decreased food intake [33], with decreased or increased activity [39], and with increased, decreased or no change in cortisol [32]. As a result, the relation between psychosocial markers and adiposity might not be significant in all cases. Psychosocial-adiposity relations might only appear in those with an unhealthy lifestyle behaviour since these people are more prone to physiological changes towards adiposity. Indeed, high cortisol and sweet food intake were vulnerability factors and high physical activity a protective factor in stress-induced adiposity in a longitudinal study of 5-12y old children [102]. Similarly in 4-6y old children, low quality-of-life was associated with increased adiposity in those with high sedentary time, high soft drink intake, high fat or low fruit intake and low quality-of-life was also associated with decreased adiposity in those with high physical activity, low sedentary time and high fruit intake [103]. Also a study in 8-11 year old girls has shown cortisol as a moderator in the relation between stress events and abdominal fat: a higher number of school-related negative events was related to more abdominal fat for girls with a high cortisol morning output [104]. And in adolescents, physical activity was found to buffer (i.e. moderate) the effect of stress on adiposity and metabolic syndrome [105,106].

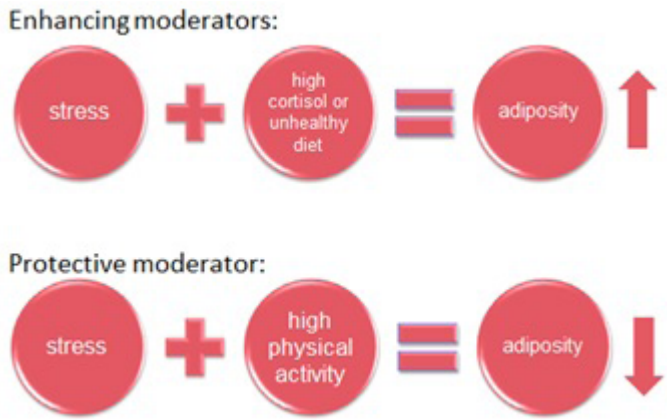

Figure 7: Cortisol and lifestyle as moderators in stress-adiposity.

A Paediatric Perspective: The Role of Parents

Parental psychopathology might predict child psychopathology [107,108]. It has also been related to child adiposity [109,110] as parental stress has been associated with lower parenting quality by increasing the children's fast food intake and decrease their vegetable consumption [111,112]. In addition, the parental influence is essential in the process of stress coping, where parents serve as role models and influence the family atmosphere [113]. Children learn emotion regulation skills early through interactions with their parents or caregivers, as well as through family factors like family expressivity or communication

[114]. A negative parental response to children's emotions teaches children to avoid rather than understand emotions [115].

Apart from psychopathology, parents play a pivotal role in children's lifestyle. Parents and family members influence children's food choices not only through food availability, eating patterns at home, and role modelling [116], but also through parenting. Parental feeding practices could lead to unhealthy eating behaviours and childhood obesity [117], since parents can pressure children to eat or restrict their diet or can use sweets as rewards or to change their temperament. Children also learn from their parents' behaviour, for example by using food to calm negative emotion. A laboratory study conducted on 25 preschool children and their mothers showed that children of mothers who use food for emotion regulation consume more sweet palatable foods in the absence of hunger [118]. Similarly, a cross-sectional study in 497 children and mothers found that maternal feeding behaviours affect emotional eating in children [119]. These practices may negatively impact development of self-regulation of eating. Finally, it is strongly supported that certain parenting styles, such as the authority and permissive parenting style, increase the risk of overweight [120], especially those children who fail to delay gratification or have poor self-regulation [121]. Indeed, it is suggested that emotion regulation mediates the relation of parental attachment with emotional eating or unhealthy food consumption

and sedentary activities in children [119,122].

In the perspective of adiposity-induced stress, parents' own body image and their perceptions of their child's overweight highly influence the well-being of obese children and the way in which they perceive themselves [120,123]. Criticism and over-encouragement to lose weight might decrease children's self-esteem and body image.

Some Future Research Perspectives

A good design and methodology are important. The literature review stressed the importance of longitudinal studies since some relations may perhaps be bidirectional. These large-scale longitudinal studies ideally are interdisciplinary to cover both the biological and psychological aspects. They should also use multiple methods to examine all the relevant determinants and outcomes e.g. objective measurements of stress and lifestyle are necessary next to the self-reports. Moreover, several aspects of stress (perceived stress, stress reactivity, stress sensitization…) need to be considered. Nevertheless, also experimental studies are warranted to further prove the suggested causalities. In the context of paediatric studies, information should be collected of the environmental factors like parental mental health and lifestyle control. Besides, several other methodological improvements for future research can be targeted: in stress-biomarkers (new are the alpha-amylase measurements and the hair cortisol, but older techniques like salivary cortisol need to be optimized in

collection compliance), in lifestyle measures (quantification of food intake, objective measurement of sleep quality, advanced technology to combine different real-time measures) but also in the determination of fat percentage and fat distribution.

Although the research on stress-adiposity is in an upward trend, several aspects are still not fully clear. Apart from the need of more research in the above mentioned explorative mechanisms gut bacteria and inflammation, future research could also consider other aspects as shown in Figure 8. (1) The predictor stress can be considered in a life perspective: perinatal stress could already lay an important foundation for future health. (2) The outcome of stress could even be looked at broader by considering the whole metabolic syndrome. (3) Finally, some underlying aspects need further attention such as appetite hormones and reward sensitivity. Overall, the main challenge is to identify biological and behavioural correlates of adversities and of the person's resilience or vulnerability to this health deteriorating allostatic load.

Perinatal Stress as Exposure

Experiences of stress already start in the perinatal life. Examples of such experiences are maternal stress, nutrient restriction, growth restriction and intra-uterin toxin/pharmaceutical exposure, but also include parental neglection and infections postnately. Maternal stress can increase cortisol levels in the mother and subsequently en-

hance fetal cortisol exposure. This impacts cortisol stress reactivity by effects on the HPA and limbic development and function (e.g. receptor levels that finally determine the negative feedback) [124] but it also increases ANS reactivity [125] leading to overall excessive stress responsitivity. Moreover, prenatal stress affects hormonal regulation of hunger and satiety (leptin, ghrelin, insulin) and adipogenesis (partially due to epigenetic changes) [126]. In conclusion, perinatal stress may instigate increased susceptibility to stress-induced adiposity. Consequently, longitudinal birth cohorts starting at pregancy or even before conception are warranted. They can utilize a life-course perspective on stress-adiposity by examining the role of prenatal stress and its effects on later life stress reactions and adiposity.

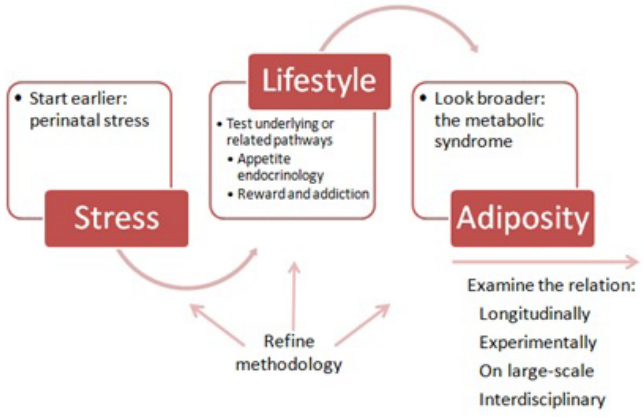

Figure 8: Aspects for future research in the stress-adiposity relation in children.

Metabolic Syndrome as Outcome

Continuous high cortisol does not only stimulate fat deposition. It could also account for inflammation, high blood pressure, insulin resistance, accelerated blood coagulation and disturbed blood lipid levels (e.g. cholesterol). All these complications fit together in the metabolic syndrome that finally can lead to cardiovascular diseases and diabetes [30]. Stress-induced metabolic changes could be one of the mechanisms that increase metabolic syndrome both (1) directly due to its interaction with the ANS, growth, appetite, metabolism and immune axes and (2) indirectly through stress-induced adiposity [127,128]. Importantly, the cortisol activating enzyme 11β-hydroxysteroid dehydrogenase-1 is present at several tissue levels that are associated with the metabolic syndrome: the liver, pancreas, muscles, vasculature and adipose tissue [129,130]. In summary, it would be of interest that studies on the effects of stress should consider the broad outcome of metabolic syndrome, after correction for other metabolic predictors.

Hormonal Pathways in Appetite

More research is needed on the detailed hormonal pathways in the stress-adiposity relation. This may provide potential targets for treatment of adiposity in people with an overactive HPA axis. It is hypothesised that cortisol influences reward and appetite by upregulating NPY (increased appetite and reward) and dysregulating

insulin and leptin levels (the body becoming resistant to their appetite and reward reduction) [34]. Also in adolescents, the link between stress with insulin and leptin has been reported [131]. Recent literature also suggest a stress induced change of other hormones like ghrelin (orexigen and mood-regulating) [132-134] and adiponectin (anti-inflammatory, insulin-sensitizing, energy-regulating and possibly anti-depressant) [135,136].

Brain Reward and Addiction Pathways

Psychological behavioural characteristics have been linked with the development, maintenance and treatment resistance of adiposity, in particular related to the high food intake. First of all, obese children are more cue-responsive: they are more vulnerable to food cues and consequently will react to them by eating the food [137]. Obese children have also been shown to be more impulsive than the lean children: they find it more difficult to resist food intake [138]. This is reflected in enhanced disinhibition (the lack to control inhibition) and enhanced reward sensitivity (a high motivation to strive for immediate reward despite punishment rather than waiting for a delayed but larger reward).

These findings encourage the study of these disinhibition and reward concepts in the stress-diet relation. Is there a co-existence with stress? Might they act as moderators in the stress-diet relation? Can they explain personal differences in stress-induced eating (more versus less intake)? Very recently, some preliminary evidence

has been published that stress-reactive individuals show diminished sensitivity to reward, but not punishment, under acute stress [139]. The question remains what the effects of chronic stress are on reward. A possible biological mechanism linking these concepts with stress is the physiological interaction of stress with the mesocorticolimbic dopaminergic reward system [128,140]. These interactions with the reward/motivation pathways and dopaminergic signalling squeak parallelisms with addiction pathways [52,141]. After all, the increased salience of comfort food induces a conditioning with greater wanting and seeking of these foods. In animal studies, the same brain regions were involved in food cravings as in drug cravings and food restriction induced cortisol increments that lead to this addictive conditioning of greater food seeking [142]. These findings initiated the terminology 'food addiction' [51,52]. In an obese sample of children and adolescents, 29% reported themselves as addicted to food [143]. Interestingly, cross-sensitization of food and addictive substances (drugs, alcohol, nicotine) might happen, resulting in a co-occurrence of addiction and stress/psychiatry [52,144].

In summary, the links with brain reward and addiction pathways advocates the integration of neuroimaging (e.g. activity in the dopaminergic centres) and behavioural research (e.g. behavioural tasks) in stress-adiposity research. Finally, this could shed light on new scientific perspectives and as such lead to new treatment alternatives.

Strategies to Tackle the Stress-Adiposity Relation

Strategies to tackle the stress-adiposity relation can be generated on different levels and discovering diverse aspects as shown in Figure 9.

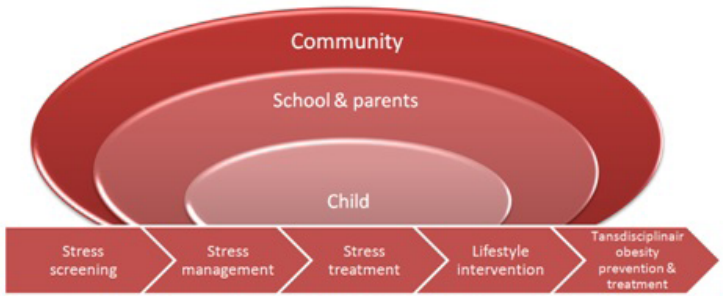

Figure 9: Aspects and levels in tackling the stress-adiposity relation in children.

A first aspect is to screen for the presence of psychosocial stressors (e.g. traumatic events; for a practical review see [145]) and symptoms. As such, high-risk children can be identified and monitored. A second step is training the management of stress to make children capable to cope with stress. A logical third step when stress levels are increased implies the treatment of stress [146]. Apart from pharmacological treatment, also cognitive behavioural therapy, relaxation techniques (mediation, respiratory control and neuromuscular relaxation), hypnosis, biofeedback and even physical activity have been applied. A fourth step is to intervene in stress-associated lifestyle factors such as emotional eating and decreased physical activity. When extreme adiposity has been developed yet, a final step is the transdisciplinary adiposity treatment with a special focus on psychological support in the obese [147].

Intervening on Stress Management

Since stress is not inevitable, focus should be laid on stress management skills. Prevention in children ought to create a firm basis and transferable skills for further life. As stated above, parents have a special role to play in the education of their kids by being a role-model and by their parenting style. Therefore, parenting support training in group can focus on interpersonal warmth, family activities, responsiveness and assertive parenting skills with strict rules that are age-appropriate [148]. Effective childhood stress interventions are tailored (to culture, age and sex), evidence-based, long term, have a holistic approach, involve the children themselves and use competent actors [149,150]. Tailoring to kids can be achieved by using face pictures for stress expression and cartoons or power hero cards for skills training [151].

In stress, resilience is important and may be thought of as the characteristic or ability to positively adapt to and/or rebound from significant adversity and distress. Therefore, some potential stress management methods compromise [148]:

- Self-awareness and self-esteem
- Pro-active thinking skills (in contrast to rigid thinking of 'must' and 'should')
- Social development of relationships (respect, positive feelings, conflict management)
- Emotional regulation and coping techniques

Indeed, adaptive emotional regulation is essential to healthy psychological functioning. In children, it is an important predictor of risk behaviours and mental problems [152,153]. Children who do not develop adequate emotion regulation strategies will be less able to survive in new environments where they face conflicts. The use of maladaptive emotion regulation can generate a vicious circle, since it may increase negative emotions which are involved in developing maladaptive strategies [154].

Several intervention studies and randomised control trials have recently been conducted in adults to test the effectiveness of emotion regulation strategies in regulating adiposity and food intake [155]. The most frequent strategies are based on the regulatory skills and correspond to new approaches within the cognitive behavioural psychological therapies. So, mindfulness therapy, acceptance and commitment therapy, self-compassion therapy (loving-kindness therapy), emotionally-focused therapy and dialectical behaviour therapy lead to less psychological distress, less binge eating episodes, less emotional eating,

less food cravings, healthier eating patterns, weight loss less personal barriers to physical activity, and improved self-efficacy to weight loss. Despite a few non-significant findings [156], these strategies are a promising approach for obesity treatment and prevention.

Although the evidence is modest in children [157-159], better emotion regulation skills were associated in observational research with a healthier diet (more fruit/vegetables and less snack food, although not all studies reported significant findings), higher physical activity and, in some cases, a healthier weight status. Interventions in children/adolescents are almost not existing. Nevertheless, they are promising since an emotion regulation intervention in adolescents indicated a positive impact on physical activity, BMI and psychological outcomes [160].

Intervening on Lifestyle

Lifestyle can be a moderator in the stress-adiposity relation. Consequently, stress-induced lifestyle changes have to be targeted. After all, lifestyle behaviour can track from childhood into adulthood [161-164]. The transition period of childhood is therefore a critical phase for these interventions. To prevent overweight, the environment (e.g. at home, at school etc.) needs to be an 'activity encouraging, healthy food zone' that minimizes opportunities for stress-induced eating but maximizes opportunities for physical activity.

Emotional eating is an easy choice in our food-abundant environment. This form of hedonic eating (eating for pleasure rather than for energy needs) is further stimulated by changes in the social norms on food since there is substantial encouragement to eat in all circumstances and few prohibitions against doing so [165]. Therefore, parents and children should be made aware of this stress-induced eating and problem-solving coping skills should be highlighted as an alternative for coping stress by food consumption [166,167]. Emphasis should be put on the contextual and especially familial environmental factors of children's dietary behaviour. Parents should make an example by their own eating style and by lowering the availability of unhealthy food. Also, parental stress should be treated as a risk factor since it can directly and indirectly enhance children's fast food consumption and lower their vegetable consumption [111,112].

Concerning physical activity, a review reported that young people have a consistent desire to be active but that they are often constrained by external factors such as school policy or curricula, parental rules in relation to safety and convenience, and physical environmental factors [168]. Since physical activity is a perfect reaction to stress [55], physical activity must be encouraged and long screen time must be discouraged.

A recent meta-analysis indicated that intervening in lifestyle such as diet and physical activity is less efficacious than intervening in stress management on itself and the

efficacy of interventions overall increased from childhood and adolescence through late middle age [169]. Consequently, intervening in inter alia emotional eating in children will be a though problem to deal with in the future.

Approaches on Multiple Levels

All these aspects can be tackled at several levels. Some aspects of intervention preferentially take place at the individual level e.g. personalized psychotherapy. Nevertheless, most interventions are school interventions since the school is an easy way to reach all children at once with adult role models and the social network of peers. The school based intervention can be comprehensive (adapting the school environment and involving the parents and community) or curriculum-based. Apart from school interventions, the parents or family remain important actors in the child's environment to target both stress and lifestyle. Also more universal community intervention programs (outside school or family) belong to the possibilities. The internet-based interventions constitute an important promising subgroup [170].

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