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# Stress, Stress Reduction and Obesity in Childhood and Adolescence

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**Keywords**
Stress · Childhood and adolescent obesity · Stress biology · Cortisol · COVID-19 · Mindfulness

**Abstract**
***Background:*** Obesity in childhood and adolescence remains a great global health challenge. Stress exposure during childhood and adolescence is associated with a higher risk for obesity, yet the linkage between stress and obesity is multidimensional, and its biological and behavioral mechanisms are still not fully understood. ***Summary:*** In this literature review, we identified different types of stress exposure in children and adolescents, including first studied effects of the COVID-19 pandemic as a prolonged stress exposure and their association with obesity risk. We investigated studies on the connection of altered stress biology and behavioral pathways as well as intervention programs on stress reduction in children and adolescents with obesity. ***Key Messages:*** There is evidence that stress exposure in childhood and adolescence promotes biological and behavioral alterations that contribute to the multifactorial pathogenesis of obesity. COVID-19 related-stress presents the most current example of a negative influence on weight development in children and adolescents. However, longitudinal studies on the linkage between environmental, behavioral, and biological factors across development are few, and results are partly equivocal. Intervention programs to reduce stress in children through mindfulness might be a promising adjunctive tool in the prevention and treatment of childhood and adolescent obesity that could further offer proof of concept of theoretically elaborated cause-and-effect relationships.



## Introduction

Over the course of the past decades, obesity in children and adolescents has turned into a global health problem. In Western societies, 1 out of 5 children is obese [1, 2], and with obesity onset in early childhood, the risk to stay obese increases through adolescence into adulthood [3, 4]. Long-term consequences of obesity include high blood pressure, type 2 diabetes, metabolic syndrome, and cardiovascular disease [5, 6]. It is well established that the emergence of obesity is mediated through a variety of influential factors. Children and adolescents from low-income families with a low educational status or ethnic minorities are at a higher risk for overweight and obesity [7]. In addition to lifestyle factors, genetic predisposition, epi-

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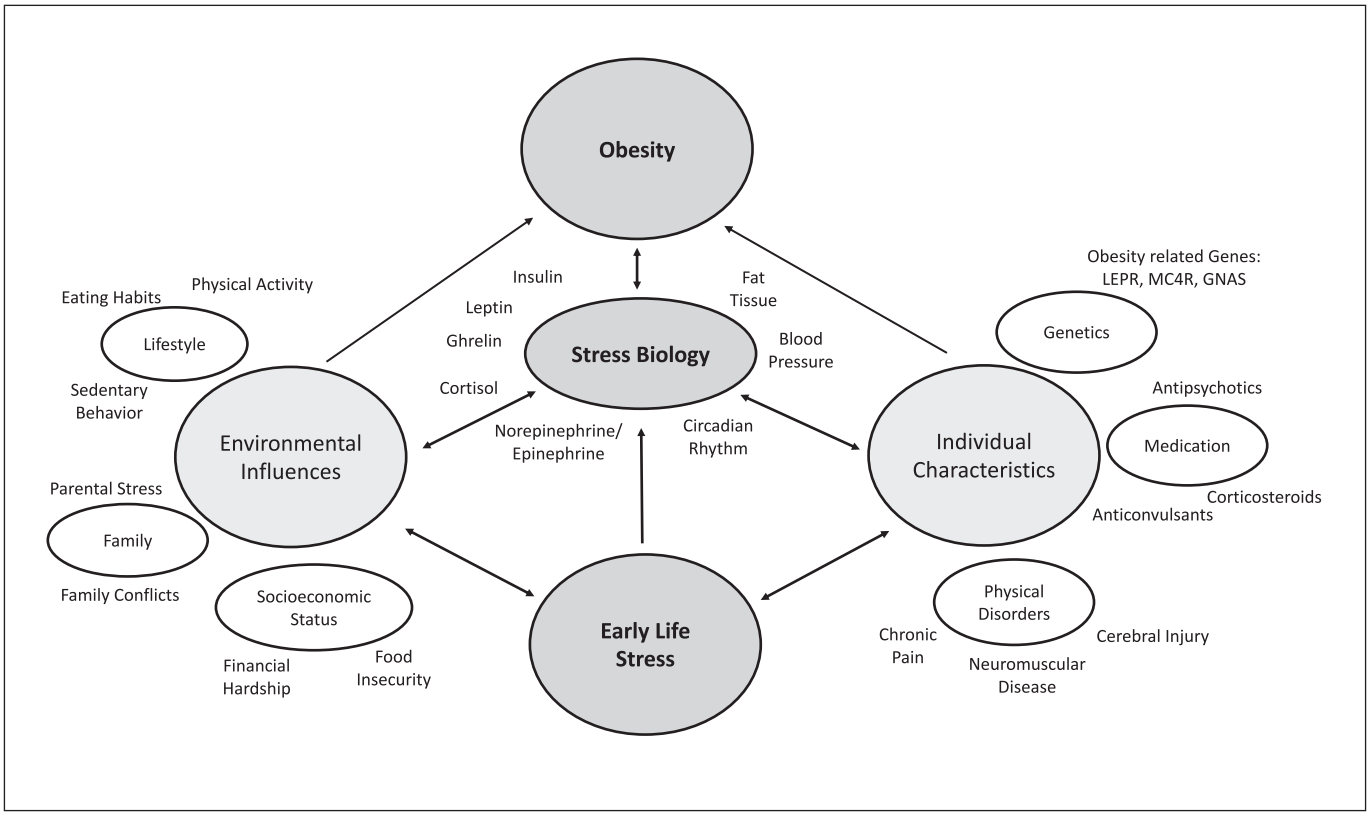


**Fig. 1.** Interactions of behavioral and biological factors on the development of obesity.

genetic mechanisms, and medical conditions (endocrine, cerebral, neuromuscular or medication-induced disorders) can contribute to the development of obesity [8]. In this multifactorial genesis, stress has been identified as a driver for obesity [6, 7]. Stress has generally been defined as a threatened state of homeostasis or a person's well-being by external or internal stimuli [9, 10] that prompt a complex neuroendocrine response. Alterations of the stress system through acute or chronic stress, specifically the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) and their mediators, have been subjects of past research and were implicated to contribute to the pathophysiology of overweight and obesity [11, 12]. Furthermore, recent research suggests interactions between environmental hits and gene expression through epigenetic modulation, which also applies to the interplay of stress and obesity [13]. Early life stress comprises traumatic events, like violence or abuse, as well as financial hardship and difficult home and family environment, yet the multiple mechanisms through which stress exposure in childhood elevates the risk for obesity are not well studied. This article aimed to elaborate how early life stress promotes obesity risk in childhood and adolescence. What are specific stressors in childhood and how do they affect the interacting biological and behavioral pathways toward obesity risk? Figure 1 demonstrates hypothesized factors and interactions of stress biology, behavior, and obesity during childhood that will be discussed in the following.

## Concepts of Stress Exposure in Childhood and Adolescence

Childhood and adolescence are crucial periods in life in which growth and mental and physical development are susceptible to many disruptive factors. Stress exposure, as one adverse influence on a child's development, has been linked with psychiatric disorders (i.e., depression and anxiety) [14, 15] and with a higher risk for obesity and metabolic syndrome [16–18]. Unraveling different types of stress exposure in childhood is one important step toward understanding the association of stress and an increased risk for childhood obesity.

*Family and Parental Influences*

Parents influence a child's risk for obesity on different levels. Prenatal stress exposure predicts a higher risk for metabolic disorders and higher body mass index (BMI) through elevated placental corticotropin-releasing hormone (CRH) concentrations [19]. Postnatal parental factors on weight development include parenting and feeding styles, as well as modeling healthy eating, physical activity, or preferences for sedentary behavior, and this modeling is also mediated through parental stress levels. Higher levels of parental stress were associated with using food as a reward and with more overeating among young adolescents [20]. Parental stress may lead to less energy resources to, for example, prepare family meals or limit screen/television time [21], and parent-perceived stress was also associated with fast food consumption and lower physical activity in children [22, 23]. Adolescents develop a more self-determined eating behavior, yet family influences on eating habits and lifestyle remain, and stress through inner familial conflicts can negatively influence behavioral patterns. While parent-adolescent conflicts favor restrained eating patterns, rather than emotional eating [24], verbal pressuring toward eating behavior or physical activity has significantly less effect than parental modeling. In a longitudinal, self-reporting study, overweight or obese adolescents ($n$ = 100) whose parents engaged in healthier behavior, that is, healthier food and physical activity, tended to show healthier dietary choices and higher levels of physical activity, and thus lower BMI [25].

*Socioeconomic Influences*

In addition to familial emotional stress factors, socioeconomic status (SES) is known to play a key role in the development of a healthy lifestyle in modern Western societies. A low SES can be considered a relevant social-contextual stressor for children as children from families with low-income experience food insecurity and worries over limited resources [26] which can significantly influence a family's diet and lifestyle [27]. Results from the German cohort study LIFE Child revealed an association of a higher SES with healthier lifestyle (i.e., healthier nutrition and less television time), more physical activity, and lower BMI among children and adolescents aged 3–18 years [28]. While the prevalence of childhood obesity in Western societies has generally plateaued during the past years, especially in groups with higher SES, there is evidence that in groups of low SES, obesity rates are still increasing [29]. A recent model on the causality between SES and obesity proposes different domains of stressors that, based on low SES, step-by-step promote obesogenic eating habits [30]. According to this model, low education and financial hardship as stress factors are linked to a lack of emotional bonding or neglect in the family which in turn result in low self-esteem, anxiety, and heightened vulnerability to stress in the child. A subsequent Danish longitudinal cohort study tested this model and confirmed that the mother's low educational status was clearly associated with a risk for obesity in adulthood for both genders [31], while family dysfunction and parental and offspring distress were associated with higher BMI only in girls.

*Eating Habits, Sedentary Behavior, and Physical Activity*

Both unhealthy emotional eating behavior and dietary patterns have been linked with stress in children and adolescents [32, 33]. Perceived, self-reported stress in adolescents and young adults was associated with an intake of more fatty, high-sugar, and energy-dense food and less fruits and vegetables [34, 35], a finding that was later confirmed also in children with an age range of 5–12 years [33]. Generally, in response to stress, around 50% of humans decrease their food intake, whereas the other 50% tend to increase it [34]. Among those individuals who increase their intake, chances are higher that they will engage in emotional eating. Emotional eating, defined as eating in response to negative emotions, but also external eating, food craving, and eating in the absence of hunger are related to overweight and obesity [33, 36]. Higher waking cortisol was also associated with higher consumption of sweet food in schoolchildren [37], thus supporting an association of stress biology and obesogenic eating. Furthermore, physical activity and sedentary behavior are independently linked to obesity. It seems likely that some individuals respond to stress by decreasing their physical activities, while others overindulge in sports to relieve stress. However, there is only little evidence of this association in children and adolescents. Balantekin et al. [38] implicated that children with a high amount of daily TV time increased their TV time after a stressful event, while in a recent Californian study with 143 children, participants engaged more in moderate to vigorous physical activity after elevated self-reported stress levels [39]. Having highlighted various aspects of stress in childhood and adolescence, we would like to review the current COVID-19 pandemic as a prolonged stressful event, particularly for children and adolescents, and its impact on weight development.

### Effects of the COVID-19 Pandemic on Children's Weight Development

There is evidence that natural catastrophes such as the earthquake in Fukushima as stressful events can also aggravate weight gain in children [40]. A current situation comparable to this is the COVID-19 outbreak in February 2020. Acute and chronic stress that can be explained by the pandemic-related life changes and possible sequelae for children and adolescents are worth investigating. The ongoing COVID-19 pandemic has severely disrupted children's and adolescents' daily routines mostly through the imposed measures to curb infection rates. School and kindergarten closures, lacking possibilities to keep up physical activities in clubs or at school, home confinements, and very limited opportunities for social interactions with peers can be considered severe stressors for children and adolescents. First data indicate negative psychological effects on anxiety, loneliness, or depression in children during lockdown [41–43] as well as higher parental stress levels and lower resilience through, for example, financial insecurity or the challenging balance of job and parental duties [44]. Children showed significantly lower scores in physical and psychological well-being during lockdown than pre-lockdown, an effect that was significantly stronger in children with medium or low SES [45]. Concerns are rising that the taken measures favor an increase in overweight and obesity caused by a decrease in physical activity, an increase in sedentary behavior, and higher intake of snacks and high-caloric (fast) food [46–48]. An observational study from Italy including 41 children implicated significant increases of high-caloric food and screen time combined with a significant decrease in physical activity [49]. A large German cohort study examined changes in BMI-standard deviation score (BMI-SDS) of 150,152 children from 2005 to 2019 with the respective changes from 2019 (pre-pandemic) to 2020 (after the onset of anti-pandemic measures). This study revealed a substantial weight gain across all weight and age-groups, reflected by an increase in mean BMI-SDS, an increase in the proportion of children gaining weight, and a decrease in the proportion of children losing weight. The highest effects on BMI gain were seen in younger children than adolescents and within the groups of children with overweight and obesity [Vogel, Geserick, Körner, Kiess, Pfäffle, et al., Int J Obes, accepted for publication, 2021]. This study is the first to highlight on a large scale the severe aggravation of obesity risk in children during the pandemic measures that still continue. One study linked higher COVID-19-specific stress with more non-nutritive use of food and snacks (e.g., emotional and instrumental feeding) and with greater child intake frequency of sweet and savory snacks [50]. On the other hand, they also observed more structure and positive interactions (e.g., eating with or engaging with child around mealtimes). These results are limited to the first months of COVID-19-related restrictions. As these restrictions still continue, more data over a longer period including valid stress measurements are desirable. However, the available data suggest a strong association of stress through profound changes in daily life and insecurity over ongoing restrictions, and negative effects on eating habits, physical activity, well-being, and consequently weight gain.

### Stress Biology and Obesity

The biological stress system includes the HPA axis with corticotropin-releasing hormone and adrenocorticotropic hormone as central mediators and glucocorticoids as end-effectors in the periphery, and the SNS with the catecholamines norepinephrine and epinephrine [51]. Whereas in normal conditions, activation of the stress system to everyday stressors results in a response limited to the time of stress exposure before returning to the baseline state, chronic stress can cause a prolonged hyperactivation of the stress system, that is, the HPA axis [11, 52]. Chronically elevated cortisol levels increase appetite, lead to hypersecretion of insulin, and thus insulin resistance and (visceral) fat accumulation in the long term [51]. Moreover, chronic stress induces hypertrophy of adipocytes, promotes the conversion of preadipocytes to mature adipocytes, and activates stromal fat immune cells [53–55]. Chronic stress has also been linked to the development of nonalcoholic fatty liver disease through induction of hepatic oxidative stress and inflammation [56]. It has been implicated that an altered HPA axis regulation contributes to the risk of overweight and obesity; however, the directionality of this association is not conclusively resolved. Of note, one should be aware of different methodological approaches for the investigation of the HPA axis that may reflect different aspects of cortisol homeostasis and may lead to diverging results. Whereas cortisol is bound to its transporter protein transcortin in plasma, urinary and salivary cortisol samples better reflect free cortisol levels. Hereby, salivary samples represent the cortisol fraction which has been filtered from plasma and thereby account for its clearance. Scalp hair cortisol samples and 24 h urinary samples may reveal long-term systemic levels of cortisol [53]. Scalp hair sam-

**Table 1.** Descriptive characteristics from studies examining the association of stress, stress reactivity, and obesity in children and adolescents

| | Sample size, *N* | Age, years | Study design | Measures of stress response | Results |
|---|---|---|---|---|---|
| Dockray et al. [66] | 111 | 8–13 | Cross-sectional | Salivary cortisol (5 samples) | Higher cortisol reactivity associated with higher BMI in girls |
| Lumeng et al. [67] | 331 | 3–4 | Cross-sectional | Salivary cortisol, intercept, and diurnal slope | Direct association of hypocortisolism and overweight in girls |
| Ruttle et al. [68] | 346 | 11, 13, 15, 18 | Cross-sectional and longitudinal | Salivary cortisol and diurnal slope | Low cortisol levels and flattened cortisol slopes associated with higher BMI at age 18 years |
| Veldhorst et al. [70] | 40 | 8–12 | Cross-sectional | Scalp hair cortisol | Higher scalp hair cortisol concentration in children with obesity |
| Doom et al. [12] | T1: 380<br>T2: 330<br>T3:257 | 2.9–5.2<br>3.2–7.1<br>7.0–10.2 | Longitudinal | sAA<br>Salivary cortisol (intercept, diurnal slope, and reactivity) | Overweight/obesity in preschool predicted future lower morning levels of cortisol and sAA, blunted cortisol reactivity, and a lower sAA slope across the day in middle school |
| Genitsaridi et al. [78] | 300 | 4–18 | Cross-sectional | Scalp hair cortisol | No significant association between hair cortisol and obese, overweight, or normal BMI or waist-to-hip ratio<br>Higher hair cortisol in pre-pubertal girls than boys |

BMI, body mass index; sAA, salivary alpha amylase.

ples from the posterior vertex as a marker for long-term endogenous cortisol concentrations have been shown to also correlate with self-reported stress [57]. Cortisol follows a circadian pattern of secretion, characterized by peak levels in the early morning, followed by a gradual decline over the day [52]. Diurnal cortisol slope, as measured in repeated samples across the day, and cortisol reactivity to stress exposure (e.g., cortisol measurements in response to a social stressor like the Trier Social Stress Test) are further established measurements of the HPA axis function. Of note, there is a homeostasis between the biological active metabolite cortisol and its inactive derivate cortisone which lacks a hydroxy group. The enzyme 11β-hydroxysteroid dehydrogenase 2 (11β-HSD2) catalyzes the inactivation, whereas 11β-HSD1 converts cortisone to its active form cortisol. Those conversions occur in different tissues including kidney, liver, and adipose tissues [58]. Dysregulation of the SNS has been connected with obesity, hypertension, and the metabolic syndrome [59, 60]. Chronic sympathetic overdrive and reduced epinephrine-stimulated lipolysis through resistance to catecholamines are suspected to contribute to fat accumulation. However, SNS activity varies in different body tissues and is partly mediated through, for example, increased levels of the adipocyte-derived hormone leptin [61].

*Evidence for an Altered Stress Biology in Children and Adolescents with Obesity*

In adults, some study results implicate that patients with overweight or obesity show blunted diurnal HPA axis functioning with lower morning levels and less cortisol change in the diurnal slope, although overall results remain equivocal [62, 63]. Fewer studies have examined the association of diurnal cortisol patterns and obesity in childhood and adolescence, and some of them will be reviewed in this article. Most studies exploring the association of cortisol levels as a marker for HPA axis activity and obesity in children have been cross-sectional, and results were mixed (Table 1). Pediatric studies on SNS dysregulation in the context of obesity development are rare, and while some suggest chronic sympathetic overdrive in obesity [64], others implicated reduced parasympathetic and sympathetic activities in association with increasing BMI-SDS [65]. Dockray et al. [66] found higher cortisol reactivity associated with a higher BMI in pre-pubertal girls but not in boys, which is in contrast with results from a study by Lumeng et al. [67], where overweight was associated with hypocortisolism (low morning cortisol and flattened diurnal slope) in low-income girls from emotionally stressful homes. Two longitudinal studies with large cohorts examined different age-groups: one compared pre- and middle school-aged children and the sec-

**Table 2.** Descriptive characteristics of studies on mindfulness-based interventions in children and adolescents with overweight or obesity

| | Sample size, *N* | Age, years | Study design | Trial duration, weeks | Results |
|---|---|---|---|---|---|
| Stavrou et al. [85] | 23 IG<br>26 CG | 9–15 | Diet + PA + MBI versus diet + PA | 8 | Significant BMI reduction in IG and reduced depression and anxiety |
| Emmanouil et al. [82] | 16 IG<br>20 CG | 8–17 | Diet + PA + MBI versus diet + PA | 8 | Significant reduction of waist-to-hip ratio in IG and higher night cortisol in IG |
| Shomaker et al. [84] | 29 IG<br>25 CG | 12–17 | Mindfulness versus health education | 6 | No significant BMI changes between groups |
| López-Alarcón et al. [83] | 17 IG<br>12 CG | 10–14 | MND-CNI versus CNI | 16 | Significant decrease in BMI, anxiety scores, and ghrelin, and a light decrease in serum cortisol in IG |

IG, Intervention Group; CG, Control Group; PA, physical activity; MBI, mindfulness-based intervention; MND-CNI, mindfulness-based Intervention + Conventional Nutritional Intervention; CNI, conventional nutritional intervention; BMI, body mass index.



ond study focused on adolescents. Ruttle et al. [68] showed that adolescents at age 11 years with lower morning cortisol and flattened slope presented with higher BMI across adolescence and at age 18 years. However, a recent study by Doom et al. [12] considered salivary alpha-amylase (sAA) as a biomarker for the SNS [69] combined with cortisol measurements. Here, they revealed that overweight and obesity at preschool age predicted lower future morning levels of cortisol and sAA, blunted cortisol reactivity, and a lower sAA slope across the day at middle school age. These results suggest a downregulation of the HPA axis and the SNS with excess of adipose tissue [12, 68], and provide information on the directionality of an association of stress biology and obesity. Cross-sectional studies that applied scalp hair cortisol measurements report differing results: while 2 studies with smaller cohorts described higher scalp hair cortisol concentrations in obese children [70, 71], a large recent study by Genitsaridi et al. demonstrated that scalp hair cortisol did not differ significantly among obese, overweight, and normal-BMI children and adolescents. Of note, results from studies with adult participants suggest a direct association of obesity and cortisol levels within the adipose tissue through higher regeneration of cortisone through 11β-HSD1 in adipocytes [72, 73]. These diverging results might partly be due to differences between the examined cohorts regarding age, sample size, ethnicity, SES etc., and different methodological approaches (self-reported stress vs. stress tests and longitudinal vs. cross-sectional, different types of cortisol samples). Still, they implicate that a dysregulation of the HPA axis and the SNS are involved in the pathophysiology of obesity in children and that an early exposure to chronic stress in life is associated with a higher risk for later obesity. However, the directionality of disrupted stress biology in obesity remains to be solved, which highlights the need for extended longitudinal research in this field. Furthermore, it is likely that more than one biological system is involved in the pathogenesis of obesity. Leptin and insulin are induced by glucocorticoids and were suggested to contribute to central regulations of food reward; additionally, abnormal cortisol concentrations favor elevated neuropeptide Y secretion, a hypothalamic feeding signal [74, 75]. Next to endocrine disturbances, epigenetic changes have been proposed to promote obesity through, for example, endocrine-disrupting chemicals [76]. One study revealed that certain DNA methylation sites interact with adverse childhood experiences to predict BMI, and some of these methylation sites were in genes associated with obesity risk [77].

## Interventions on Stress Reduction in Children and Adolescents

In consideration of the mounting evidence of a strong association of stress, altered stress biology, and obesity in children and adolescents, stress management could be a target in the prevention and treatment of obesity. Study results from investigations among adults on stress reduction, mindfulness, and obesity-related outcomes such as eating behavior, perceived stress, cortisol levels, and body weight have revealed controversial results [79–81]. To date, only few studies have explored mindfulness-based interventions in pediatric groups. Mindfulness is a psy-

chological technique that focuses on awareness, meditation, and attention and includes heterogeneous practices, such as breathing techniques, guided imagery, yoga, progressive muscle relaxation, or cognitive restructuring in order to improve coping with stress. A selection of recent pediatric studies on stress management interventions in youth with obesity is demonstrated in Table 2. All studies included children and adolescents with overweight or obesity and compared conventional interventions in diet and physical activity with mindfulness-based interventions in addition to the conventional program [82–85]. All but 1 study found a significant decrease in BMI or waist-to-hip ratio in the mindfulness-based intervention groups. Measurements of stress biology, like cortisol levels and appetite-regulating hormones such as insulin and ghrelin, were considered in 2 studies [82, 83]. Emmanouil et al. [82] found significantly higher night cortisol levels in the mindfulness intervention group after the intervention. The most recent study by López-Alarcón et al. [83] measured ghrelin, serum cortisol and insulin, saliva cortisol (awakening response and slope), BMI change, and anxiety scores among adolescents with obesity and elevated anxiety scores. Herein, they demonstrated significant declines in anxiety scores, ghrelin, and BMI as well as a marginal decrease in serum cortisol in the mindfulness-based intervention group compared to the control group [83]. The aforementioned studies have several limitations that underline the need for more research in the field. As mindfulness comprises very heterogeneous techniques, the programs applied in the studies varied in their intensity and methods and are therefore difficult to compare. As mentioned earlier, sample sizes in all studies were relatively small which is probably due to strict inclusion criteria and high time expenditure of interventions, and duration of intervention varied between the studies. Moreover, group assignments were not strictly randomized [83]. It could therefore be argued that participants and their parents had a higher intrinsic motivation in the mindfulness-based intervention group which might have influenced the positive outcome on other parameters. Nevertheless, results are encouraging that combining mindfulness as a tool for stress reduction with conventional intervention programs might have positive influence on perceived well-being, biological stress, appetite regulation, and weight reduction in children and adolescents with obesity.

## Conclusion

Obesity in childhood and adolescence is a multidimensional challenge that requires interdisciplinary solutions. We have underlined different behavioral and biological pathways in the context of early life stress that have an impact on the risk for obesity during childhood and adolescence. The interaction of biological and behavioral pathways is complex and possibly bidirectional. Early alterations of the biological stress system are likely to shape later health outcome. To further understand the directionality and impact of different types of stress exposure on weight development, longitudinal studies with objective measurements of biological and behavioral factors are needed. Implementing interventions to prevent or reduce early life stress is a promising approach for tackling obesity at an early stage.

## Conflict of Interest Statement

The authors have no conflict of interest to declare.


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## Author Contributions

C.K., A.M. and W.K. conceived the article concept and structure. C.K. reviewed the literature and wrote the first draft of the article, A.M., W.K., and R.S. discussed and finalized the first draft of the manuscript. All authors revised the work critically and approved the final version of the manuscript.

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