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Editorial

Early Prevention of Childhood Obesity: Another Promise or a Reliable Path for Battling Childhood Obesity?

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A recently published article by Gillman and Ludwig [1] strongly advocates for early obesity prevention. Indeed a child's obesity life course appears to be determined by the age of 6 years as shown in compiled data on the natural course of BMI distribution from birth to 14 years (fig. 1) in three German birth cohorts (Kiel Obesity Prevention Study (KOPS) [2], Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study [3], Multicenter Allergy Study (MAS) [4]) in a collaboration facilitated by the German Adiposity Network (EPI Germany; *www.kompetenznetz-adipositas.de/kompetenznetz/konsortien.html?L = 1*). The patterns in boys and girls were very similar. Among the obese children with a BMI above the 97th percentile at 6 years 50.0% (95% CI 33.3–66.7%) remained obese, and 40.0% (95% CI 26.7–53.3%) became overweight (BMI > 90th percentile). Among overweight children with a BMI between the 90th and 97th percentile at 6 years 24.2% (95% CI 13.6–34.9%) stayed overweight until age 14 years, and 27.3% (95% CI 16.7–37.9%) became obese, whereas only 2.1% (95% CI 1.6–3.1%) of the children who had a BMI below the median at the age of 6 years developed obesity at the age of 14 years. Overweight at age 5 was the strongest predictor of being overweight at age 10 years after adjustment for several co-factors, as also demonstrated in another German cohort [5]. Therefore, any effort to avoid an elevated BMI within the first 6 years of life appears to be strongly justified. But is there sufficient evidence for effective preventative action?

Gillman and Ludwig [1] based their argument on evidence from animal studies demonstrating irreversible derangements in offspring's adiposity and metabolism related to adverse pre- and perinatal conditions. Such proof of principle, however, is difficult to translate into effective prevention programs in humans, although the authors suggested that a large

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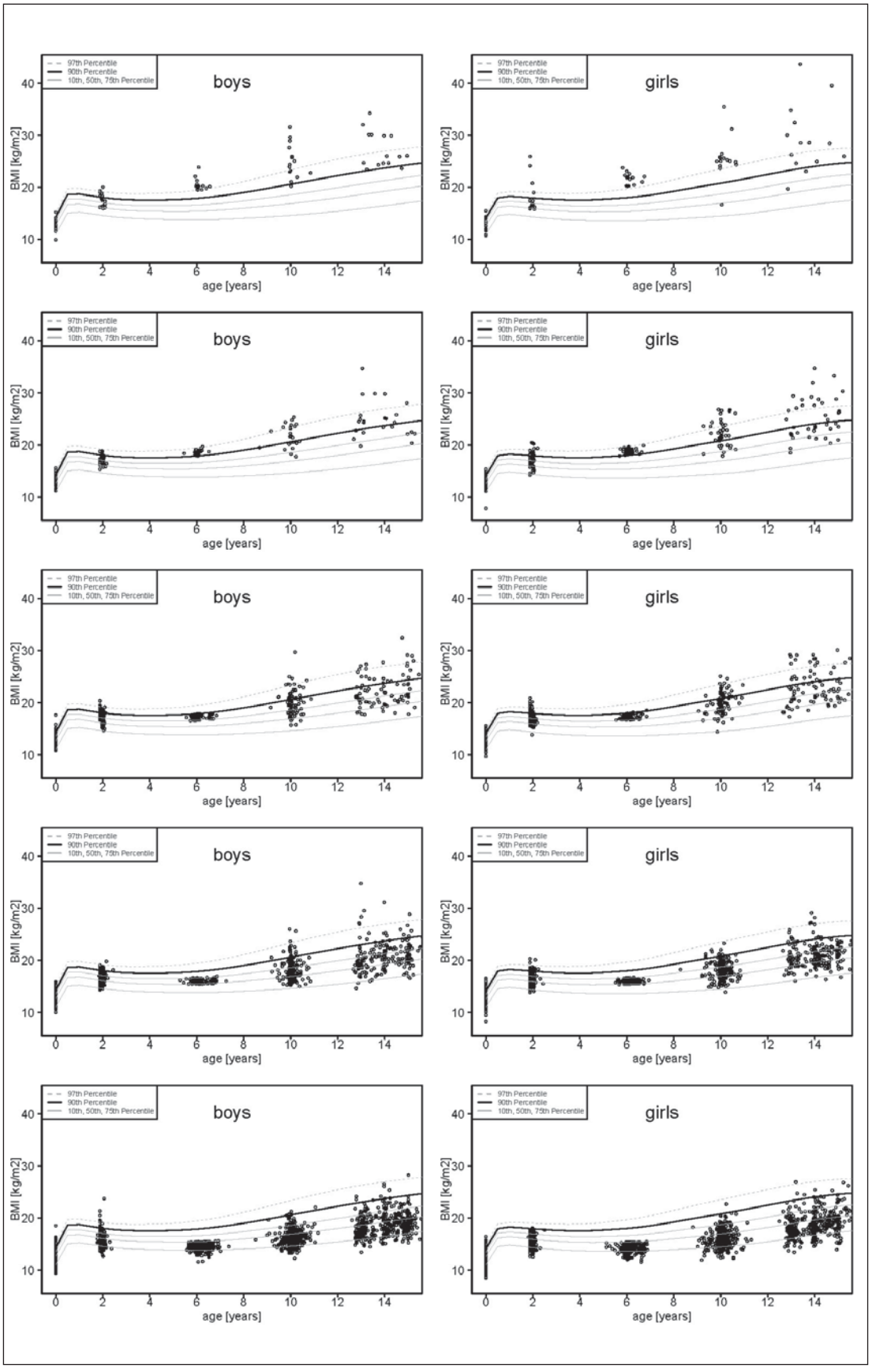

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potential to do so exists by avoiding four risk factors identified in the VIVA project. These four risk factors, all of which may be amenable to intervention, include smoking in pregnancy, excessive gestational weight gain, breastfeeding for less than 12 months and sleeping less than 12 h/day during infancy. While the presence of all four risk factors was associated with an obesity prevalence of 29%, in the absence of all four risk factors the prevalence of obesity was only 6%.

Caution with respect to such a large promise is justified because of the possibility of residual confounding and the limited chances for implementation. An association between smoking in pregnancy and childhood overweight was confirmed in two meta-analyses [6, 7], and an association between nicotine exposure and higher BMI in the offspring has also been confirmed in animal studies [8–10]. However, it was also shown that children exposed to paternal or household smoking in utero or infancy also had an increased risk of being overweight and obese. In mutually adjusted models this risk was similar in magnitude to that of children with intrauterine exposure to maternal smoking [11–14]. Because the intrauterine exposure to nicotine is about fourfold higher in fetuses of smoking mothers as compared to the dose of exposure related to paternal or household smoking [15], substantially larger effect estimates for maternal smoking in pregnancy than for paternal smoking would have been anticipated. Further doubt regarding a causal association arises from sibling studies in which one sibling was exposed to maternal smoking in pregnancy and the other was not [16, 17]. In an analysis stratified by maternal smoking habits across the first and second pregnancies, an increased risk for overweight in young men could only be detected if the mother smoked during both pregnancies. Smoking in either pregnancy only was not associated with overweight in the exposed son. Similar findings were reported by Gilman et al. [16] who assessed the effects of maternal smoking during pregnancy on children's growth and development in 16,619 siblings using conditional likelihood methods. The BMI in offspring of mothers who had been smoking during pregnancy was significantly higher in the unconditional analyses. However, with adjustment for unmeasured family conditions by analyses conditional on family-specific intercepts providing effect estimates free from bias due to potentially confounding factors to which both siblings were exposed, the effects of maternal smoking in pregnancy decreased and were no longer significant. So although there are good reasons to avoid smoking in pregnancy, the presumed effect of avoiding smoking in pregnancy to reduce likelihood of childhood overweight might reflect an artifact in epidemiological research.

Similarly the notion that there is a protective effect from breastfeeding against childhood obesity is not yet definitely proven despite a myriad of observational studies [18]. Pediatricians spend a great deal of office time consulting parents about sleeping problems in infancy. Unfortunately, there are no evidence-based concepts to solve children's sleep problems, and proof for an efficacious population advice to change sleep habits is definitely lacking.

Avoidance of excessive gestational weight gain (GWG) may be a viable option in preventing childhood obesity [19, 20]. Excessive GWG can be identified early in pregnancy, and reversal of excessive GWG early in pregnancy [21] was found to reverse the risk for overweight in the

Fig. 1. Previous and subsequent course of BMI of boys (left panel) and girls (right panel) at different BMI categories at the age of 6 years. From above to below: First row: Obese boys and girls at the age of 6 years; Second row: Overweight boys and girls at the age of 6 years; Third row: Boys and girls lying between the 90th and 75th BMI percentile at the age of 6 years; Fourth row: Boys and girls lying between the 75th and 50th BMI percentile at the age of 6 years; Fifth row: Boys and girls lying below the 50th BMI percentile at the age of 6 years. The data were generated by merging three German birth cohort studies (KOPS [2], DONALD [3] and MAS [4]).

offspring [22]. We are awaiting results from randomized trials on the impact of avoiding excessive GWG on later overweight. These trials require long-term follow-up: As depicted in figure 1, the proportion of children with high or low BMI at the age of 2 among those overweight, obese or with a BMI below the 50th percentile at the age of 6 years was almost identical. As previously shown, this reflects a high remission rate from overweight at the age of 2 years [23, 24]. Even if such long-term effects for avoidance of excessive GWG are confirmed, their potential contribution to a successful strategy for obesity prevention might prove limited. In an analysis of the attributable risk fraction excessive GWG accounted for only 1.9% of the risk for childhood overweight [14].

Early prevention of childhood obesity: a promise that may prove hard to keep.

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