Healthy Growth and Development

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There is a remarkably consistent association between rapid infancy weight gain and higher risks for obesity in later childhood and adult life. Most recently, 45 of 46 studies reported positive associations in settings across the world [1]. The shape of this association shows a continuous influence of rising infancy weight gain on the risk of childhood obesity, plus a substantially higher risk in those infants who crossed upwards by >1.33 z-scores [2]. Those findings indicate potential benefits for both population-wide and targeted high-risk preventive strategies.

Feeding for Healthy Infancy Weight Gain

The (typical) slower trajectory of weight gain in breastfed infants underlies the rationale for the WHO 2006 Growth Standards. Adoption of these WHO growth charts has substantial (and likely beneficial) impact on the classification of more young children being in overweight and obese categories, and far fewer having underweight.

Within formula milk-fed infants, higher protein composition promotes faster infancy weight gain. There has been significant lowering of protein contents of most formula milks [3]. Recent observational studies of the (substantially more variable) nutrient composition of human breast milk supports a positive effect of milk protein on infant weight and suggests a weight-limiting role of milk fat content [4].

There is a linear relationship between total energy intake and infancy weight gain. Effective shifting downwards of the widespread excessive infant calorie intakes requires a sensitive understanding of parental attitudes and aspirations for their infant's growth, which are often higher than optimal.

There is an inconsistent association between age at the introduction of solid foods ('weaning') and the risk of obesity [1]. A recent review shed doubt on the underlying premise that early weaning promotes faster infant weight gain. Conversely, early age at weaning may be a parental response to larger infant size, rapid growth and weight gain, or even higher infant hunger signals [5].

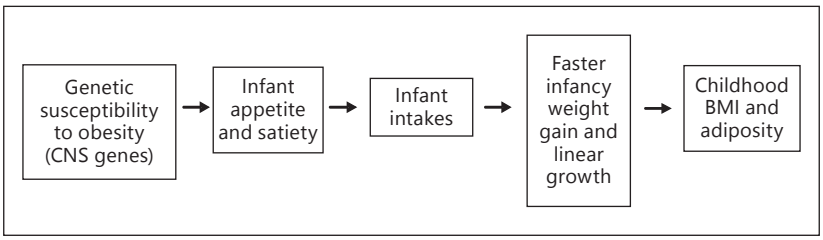

Fig. 1. Hypothesized mechanism linking adult genetic obesity susceptibility variants to infant and childhood eating behavior, dietary intakes and growth.

Infant Control of Infant Weight Gain

Infant feeding is the result of complex interaction and signaling between mother and infant. Monogenic studies of severe early-onset obesity have identified rare mutations that delineate the 'central' regulation of appetite and satiety. Affected children have extremely high and insatiable appetites ('hyperphagia') from infancy.

The heritability of BMI is high even in young children. Longitudinal studies show that common 'obesity susceptibility genetic variants' affect weight gain during infancy and childhood – the higher attained weight persists through life with adverse consequences such as diabetes and other obesity-related metabolic disorders [6]. These obesity susceptibility variants also support a predominantly 'central' genetic regulation of weight gain and BMI. Furthermore, they act across the spectrum of infancy weight gain, being protective against 'inadequate' levels of weight gain while also predisposing to overweight and obesity.

Llewellyn and Wardle [7] identified distinct infant appetitive constructs relating to food responsiveness (appetite) or satiety responsiveness. These traits are influenced by obesity susceptibility variants and are predictive of infant weight gain. Therefore, it appears that centrally acting obesity susceptibility variants contribute to the wide interindividual differences in infant appetite, satiety, food intake and weight gain, and they provide a biological link between early growth and feeding behaviors and later childhood pubertal timing and adult metabolic disease risks (fig. 1).

Future Perspectives

The long-term relevance of infant body composition and fat distribution (and also other detailed measures of infant metabolism) are yet unknown. While we await longer-term follow-up studies, research studies that categorize the infant phenotypes associated with low versus high

genetic obesity susceptibility might inform which infant profiles are indicative of future health. Recent findings indicate that, in infants with rapid weight gain, rapid gains in lean body mass are not necessarily protective for future obesity and that the emergence of higher percent body fat may be a relatively late occurrence [6].

References

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