Genetic factors are predominant for the spontaneous evolution of the body mass index (BMI), but only environmental events are able to modify the BMI curve

**Key insights**

Childhood obesity can be considered a programmed disease in which several genetic and/or in utero mechanisms play a role. Constitutional susceptibility is predominantly a genetic predisposition. For example, it is likely that the growth curves of obese children are programmed at a higher level than their nonobese counterparts. Further research into gene-gene interactions and a focus on the respective contribution between genetic contribution and lifestyle in determining individual weight are needed.

**Current knowledge**

An obesogenic environment can be considered the trigger, not the primary cause, of childhood obesity. Common obesity is polygenic with no observable simple Mendelian inheritance. The discovery of rare monogenic forms of obesity and obesity-linked mutations means any preconceived notions that obesity represents an issue with no biological basis can be dispelled. Adipostat, programmed BMI curves, and some environmental factors induce the programming towards childhood obesity. An improved understanding of the key molecular and genetic mechanisms that regulate body weight should help find the ‘missing links’ in the question of heritability and susceptibility.

**Practical implications**

Providers need to recognize that treatment requires a substantial motivation to fight against the programming and that childhood obesity is not simply the result of inappropriate habits. Moreover, prevention strategies should focus only on those children with a constitutional susceptibility to become obese under the pressure of an obesogenic environment.

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**Programming towards Childhood Obesity**

by Patrick Tounian

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**Recommended reading**


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Schematic: programming towards childhood obesity and interdependent challenges (see text for details).