

Why tackling appetite could hold the key to preventing childhood obesity

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Juan Carreño de Miranda's "La monstrua desnuda" (The Nude Monster) painting.

A heartier appetite is linked to more rapid infant growth and to genetic predisposition to obesity, according to two papers published in *JAMA Pediatrics* today.

The studies investigated how weight gain is linked to two key aspects of appetite, namely lower satiety responsiveness (a reduced urge to eat in response to internal 'fullness' signals) and higher food responsiveness (an



increased urge to eat in response to the sight or smell of nice food).

The first paper reveals that infants with a heartier appetite grew more rapidly up to age 15 months, potentially putting them at increased risk for <u>obesity</u>.

The authors used data from non-identical, same-sex twins born in the UK in 2007. Twin pairs were selected that differed in measures of satiety responsiveness (SR) and food responsiveness (FR) at 3 months, and their growth up to age 15 months was compared. Within pairs, the infant who was more food responsive or less satiety responsive grew faster than their co-twin.

The more food responsive twin was 654g heavier (1.4lbs) than their cotwin at six months and 991g heavier (2.1lbs) at 15 months. The less satiety responsive twin was 637g heavier (1.4lbs) than their co-twin at six months and 918g heavier (2lbs) at 15 months.

"Obesity is a major issue in child health" says Professor Jane Wardle, lead author of the study from the UCL Health Behaviour Research Centre. "Identifying factors that promote or protect against weight gain could help identify targets for obesity intervention and prevention in future. These findings are extremely powerful because we were comparing children of the same age and same sex growing up in the same family in order to reveal the role that appetite plays in infant growth.

"It might make life easy to have a baby with a hearty appetite, but as she grows up, parents may need to be alert for tendencies to be somewhat over-responsive to food cues in the environment, or somewhat unresponsive to fullness. This behaviour could put her at risk of gaining weight faster than is good for her."



The second *JAMA Pediatrics* paper, in collaboration with King's College London, sheds further light on the way that appetite, particularly low satiety responsiveness, acts as one of the mechanisms underlying genetic predisposition to obesity.

The researchers accessed data from 2,258 10-year-old children born in the UK between 1994 and 1996. The team created a polygenic obesity risk score (PRS) for each child to estimate their genetic susceptibility to obesity, by adding up the number of higher-risk alleles from 28 obesityrelated genes. Higher PRS scores indicated a higher genetic predisposition to obesity.

The PRS scores were then examined to determine the correlation with the children's satiety responsiveness and adiposity (body fatness).

"As expected, we found that children with a higher PRS score (more obesity-risk' genetic variants) were likely to have larger BMI and waist circumference," says Dr Clare Llewellyn, lead author from the UCL Health Behaviour Research Centre. "But more importantly, we also found that these children were more likely to have low satiety responsiveness.

"This suggests that satiety sensitivity could be targeted for pharmacological and behavioural interventions, to prevent or treat obesity. For example, children with lower satiety sensitivity could be taught techniques that might improve their fullness signals when eating, such as slowing their eating speed. Another approach might be to provide better advice to parents and children about appropriate portion sizes, limiting access to 'second helpings' and ensuring tempting treats are out of sight between meals."

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