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EMBARGOED UNTIL: Monday, June 4th 2012, at 3:00 PM U.S. Central Time *(Archives of Pediatrics & Adolescent Medicine)*

TITLE: *Polygenic risk, rapid childhood growth, and the development of obesity*

A research team led by Dan Belsky, Avshalom Caspi and Terrie Moffitt at Duke University and the University of North Carolina at Chapel Hill reports that a genetic risk for obesity leads children to grow more rapidly in the earliest years of life and that this rapid early growth leads to obesity in adulthood.

PUBLICATION SOURCE: Archives of Pediatrics & Adolescent Medicine, to appear on June 5, 2012.

THE FINDINGS:

- Obesity is a complex health problem that is influenced by the environment and by many different genes (i.e. it is "polygenic"). In our study, we followed 1,000 individuals from birth through their fourth decade of life and asked how 32 genetic markers recently discovered to predict obesity in adults influenced growth and development across the life course.
- *Genetic risks for adult obesity predicted rapid growth in early childhood*. In turn, this rapid growth made children more likely to develop obesity later on.
- Children who were at high genetic risk but who grew more slowly were *less* likely to develop obesity as compared to faster growing peers.
- Genetic risk markers added information about a child's likelihood of developing obesity *over and above* their family history.

WHY ARE THESE FINDINGS IMPORTANT?

- **Findings highlight the importance of life course longitudinal studies to understand the genetics of complex health problems like obesity:** Recent discoveries from the frontiers of genome science have uncovered some of the genetic roots of obesity. But how do these genetic factors cause obesity? Our findings suggest that they do so by accelerating growth in early life, which in turn increases the likelihood of becoming obese later on. In other words, recently discovered "obesity genes" may actually be "rapid early growth genes."
- **Findings affirm the importance of public health efforts to promote healthy early childhood growth and development:** We followed children all the way to mid-life to ask whether those who grew at a normal rate were protected from developing obesity. A more normative rate of growth in early childhood largely protected children from genetic risks.

SUPPORTING DETAILS:

Genetic Risk: We measured genetic risk using a polygenic (many gene) profile composed of 32 different genetic markers called single nucleotide polymorphisms, or "SNPs." Each of these SNPs was discovered to be related to obesity in genome-wide association studies (GWAS) of hundreds of thousands of adults. The GWAS asked whether each of millions of individual SNPs was more common in adults with higher body mass index (BMI). The 32 SNPs in the genetic profile we studied were reliably related to higher BMI in multiple GWAS.

Rapid Early Growth: We measured rapid early growth during gestation and during the early childhood years. Growth during gestation was measured as weight at birth. Growth during the early childhood years was measured as (1) weight gain between birth and age 3 years; and (2) adiposity rebound. The adiposity rebound is the point when children begin to put on body fat after losing it during early childhood, usually around age 6 years.

Obesity: We measured obesity during cohort members' teens (ages 15 and 18 years), 20s (ages 21 and 26 years) and 30s (ages 32 and 38 years), and chronically, across adulthood (ages 15-38 years).

PARTICIPANTS:

Participants were members of the Dunedin Multidisciplinary Health and Development Study, which tracks the development of a birth cohort of 1,032 children born in 1972-1973 in Dunedin, New Zealand. This birth cohort's families represent the full range of socioeconomic status and health in the general population. Follow-ups have been carried out at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and most recently at age 38 years, when 96% of the living cohort members took part. We examined all of the cohort members with European ancestry who provided DNA samples (98%).

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UNIVERSITIES INVOLVED:

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(2) Gillings School of Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA.

The study protocol was approved by the Otago Ethics Committee and university ethics review board. Parents and children gave informed consent for the research.

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