

New link between mother's pregnancy diet and offspring's chances of obesity found

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Scientists have discovered that a mother's nutrition during pregnancy can strongly influence her child's risk of obesity many years later.

An international study, led by University of Southampton researchers and including teams from New Zealand and Singapore, has shown for the first time that during pregnancy, a mother's diet can alter the function of her child's DNA. The process, called epigenetic change, can lead to her child tending to lay down more fat. Importantly, the study shows that this effect acts independently of how fat or thin the mother is and of child's weight at birth.

Keith Godfrey, Professor of Epidemiology and Human Development at the University of Southampton, who led the study, says: "We have shown for the first time that susceptibility to [obesity](#) cannot simply be attributed to the combination of our [genes](#) and our [lifestyle](#), but can be triggered by influences on a baby's development in the womb, including what the mother ate. A mother's nutrition while pregnant can cause important epigenetic changes that contribute to her offspring's risk of obesity during childhood."

Researchers measured epigenetic changes in nearly 300 children at birth and showed that these strongly predicted the degree of obesity at six or nine years of age. What was surprising to the researchers was the size of the effect - children vary in how fat they are, but measurement of the epigenetic change at birth allowed the researchers to predict 25 per cent of this variation.

The study was primarily funded by the UK Medical Research Council (MRC), the National Institute for Health Research, WellChild (previously Children Nationwide), Arthritis Research UK and the University of Southampton.

The epigenetic changes, which alter the function of our DNA without changing the actual [DNA](#)

sequence inherited from the mother and father, can also influence how a person responds to lifestyle factors such as diet or exercise for many years to come.

"This study indicates that measures to prevent childhood obesity should be targeted on improving a mother's nutrition and her baby's development in the womb. These powerful new epigenetic measurements might prove useful in monitoring the health of the child," adds Professor Godfrey.

Mark Hanson, British Heart Foundation Professor and Director of the University of Southampton's Human Development and Health Unit and one of the research team explains: "This study provides compelling evidence that epigenetic changes, at least in part, explain the link between a poor start to life and later disease risk. It strengthens the case for all women of reproductive age having greater access to nutritional, education and lifestyle support to improve the health of the next generation, and to reduce the risk of the conditions such as diabetes and heart disease which often follow obesity."

Research team member Sir Peter Gluckman FRS of the Liggins Institute at the University of Auckland and the Singapore Institute of Clinical Sciences comments: "This study provides the most compelling evidence yet that just focusing on interventions in adult life will not reverse the epidemic of chronic diseases, not only in developed societies but in low socio-economic populations too."

The study team are part of an international consortium involving the Universities of Southampton and Singapore, the Singapore Institute for Clinical Sciences, the Liggins Institute of the University of Auckland, AgResearch New Zealand and the Medical Research Council Lifecourse Epidemiology Unit, University of Southampton.

Professor Cyrus Cooper, who directs the MRC Lifecourse Epidemiology Unit, says: "MRC population-based studies have shown that early life factors influence risk of disease many years later. Now we can begin to see the mechanisms by which this happens, opening up new avenues for medical research and interventions."

More information: Their findings will be published next week (26 April 2011) in the printed journal *Diabetes*.

Provided by University of Southampton

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