

Diabetes gene raises odds of lower birth weight

July 29 2009

Pediatric researchers have found that a gene previously shown to be involved in the development of type 2 diabetes also predisposes children to having a lower birth weight. The finding sheds light on a possible genetic influence on how prenatal events may set the stage for developing diabetes in later childhood or adulthood.

Researchers from The Children's Hospital of Philadelphia and the University of Pennsylvania School of Medicine published the study July 10 in the online version of the journal <u>Diabetes</u>.

"It's a bit unusual to find a gene linked to both prenatal events and to a disease that occurs later in life," said study leader Struan F.A. Grant, Ph.D., a researcher at the Center for Applied Genomics of The Children's Hospital of Philadelphia. "This gene variant carries a double whammy, in raising the risk of both lower birth weight and the development of type 2 diabetes in later life."

Type 2 diabetes occurs either when the pancreas produces too little insulin or when the body cannot efficiently use the insulin that is produced. Formerly called adult-onset diabetes and still most common in adults, type 2 diabetes has been increasing sharply among children.

Grant and study co-leader Hakon Hakonarson, Ph.D., director of the Center for Applied Genomics at Children's Hospital, investigated 20 gene locations previously reported to be associated with type 2 diabetes. Drawing on a cohort of some 5,700 Caucasian children in an ongoing



genome-wide association study of childhood obesity at Children's Hospital, the researchers compared birth weights with the occurrence of the 20 gene variants.

They found that one of the gene variants, called CDKAL1, had a strong association with lower birth weight—a finding that supports the so-called fetal insulin hypothesis. Previous studies by European diabetes researchers, said Grant, had suggested that CDKAL1 was implicated in both lower birth weight and type 2 diabetes, and the current study, using a large sample size, reinforced that association.

Under the fetal insulin hypothesis, a slight underproduction of insulin, an important fetal growth factor, during the prenatal period may cause a baby to be born smaller. Low birth weight is already known to increase the risk of disease later in life, and the fetal insulin hypothesis proposes that the same gene that causes lower <u>birth weight</u> also increases the risk of developing type 2 diabetes.

"The mechanisms by which CDKAL1 may act are not well understood, but it is believed to reduce insulin secretion, and that underproduction contributes to type 2 diabetes," said Grant. He added that further research may investigate biological pathways on which the gene functions, and may also study whether it may influence the risk of developing other diseases in later life.

Source: Children's Hospital of Philadelphia (<u>news</u>: <u>web</u>)

Citation: Diabetes gene raises odds of lower birth weight (2009, July 29) retrieved 26 April 2024 from https://medicalxpress.com/news/2009-07-diabetes-gene-odds-birth-weight.html

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