Maternal, paternal genes' tug-of-war may last well into childhood

July 28 2009

'Compared to other primates, human babies are weaned quite early, yet take a very long time to reach full nutritional independence and sexual maturity,' says David Haig, George Putnam Professor of Organismic and Evolutionary Biology. File photograph by Ruby Arguilla/Harvard News Office

(PhysOrg.com) -- An analysis of rare genetic disorders in which children lack some genes from one parent suggests that maternal and paternal genes engage in a subtle tug-of-war well into childhood, and possibly as late as the onset of puberty.

This striking new variety of intra-family conflict, described this week in

the Proceedings of the National Academy of Sciences, is the latest wrinkle in the two-decades-old theory known as genomic imprinting, which holds that each parent contributes genes that seek to nudge his or her children's development in a direction most favorable, and least costly, to that parent.

"Compared to other primates, human babies are weaned quite early, yet take a very long time to reach full nutritional independence and sexual maturity," says author David Haig, George Putnam Professor of Organismic and Evolutionary Biology in Harvard University's Faculty of Arts and Sciences. "Human mothers are also unusual among primates in that they often care for more than one child at a time. Evidence from disorders of genomic imprinting suggests that maternal and paternal genes may skirmish over the pace of human development."

Previous research has offered evidence of a genetic struggle for supremacy only during fetal development: In the womb, some genes of paternal origin have been shown to promote increased demands on mothers, leading to fetal overgrowth, while genes of maternal origin tend to have the opposite effect. This new work suggests maternal and paternal genes continue to engage in internal genetic conflict past childbirth.

"This analysis suggests that human life history, and especially humans' unusual extended childhood, may reflect a compromise between what's best for mothers, fathers, and the offspring themselves," Haig says.

Haig delved into clinical case reports on patients with four rare genetic disorders. He found evidence that children with disorders characterized by dominance of some maternal genes -- Silver-Russell syndrome, Prader-Willi syndrome, and Temple syndrome -- place fewer demands on their mothers' resources.

For example, newborns with all three disorders display a weak desire to nurse, and slower childhood growth in general. Many also show early onset of puberty, which often marks a point at which children become less dependent on their mothers' sustenance.

Conversely, babies with Beckwith-Wiedemann syndrome, in which some maternally derived genes are suppressed and paternal genes dominate, are born heavy with particularly large tongues. These individuals usually end up being tall, owing to their rapid growth both in the womb and as young children. They have a high frequency of childhood cancers.

"Clinical data from imprinting disorders suggest paternally-expressed genes promote, and maternally-expressed genes inhibit, childhood growth," Haig writes.

Haig adds that further longitudinal study of feeding and development in individuals with Silver-Russell syndrome, Prader-Willi syndrome, Temple syndrome, and Beckwith-Wiedemann syndrome is needed to more fully understand the role of genomic imprinting in such disorders.

Source: Harvard University (news : web)