Family ties point to recessive 'obesity gene'

An estimated 5 percent of the U.S. population may have inherited a tendency toward obesity, according to a new study examining family weight patterns. The research suggests obesity follows a classic model of recessive inheritance, with some individuals receiving a copy of a so-called obesity gene from each parent. People born with this genetic double whammy who fulfill their fat potential are among the heaviest in the nation and run a high risk of cardiovascular disease, the researchers say.

Other studies have indicated that obesity has a genetic component (SN: 1/25/86, p.56), but the new work points to the existence of one major obesity gene that can cause excessive weight under the right conditions. Trudy L. Burns of the University of Iowa in Iowa City described the research this week at the American Heart Association's scientific sessions in New Orleans.

Burns and her colleagues studied 277 high school students and 1,303 of their siblings, parents, aunts, uncles and first cousins. The scientists calculated each person's body mass index as a measure of obesity, then used statistical methods to see whether genes appeared to control obesity in the families studied.

They found that family weight patterns fit a model in which one highly influential gene codes for either obesity or slimness, while other genes play lesser roles. In that model, the gene coding for obesity is recessive, manifesting itself when inherited from both parents. While people who inherit two copies of the obesity gene are not condemned to a life of flab, they stand a greater chance of gaining excessive weight than do people who inherit two slimness genes or one slimness gene plus one obesity gene, Burns says. The model predicts that 34 percent of the U.S. population carries both gene types and 61 percent carries two slimness genes.

People with the genetic odds stacked against them can minimize their weight gain by altering environmental risk factors such as diet or a sedentary lifestyle, Burns says. Conversely, people genetically predisposed to slimness can become obese if they eat too much or maintain a low activity level. Burns says further studies are needed to clarify the interaction between environmental and genetic factors in the development of obesity.

Compared with their slimmer peers, obese individuals face a host of cardiovascular risk factors, including significantly higher blood pressure and lower levels of high density lipoprotein, the transport molecule that carries cholesterol from the blood to the liver for excretion.

The Iowa researchers have not identified any specific obesity gene, but one candidate is the gene coding for lipoprotein lipase, an enzyme that helps the body store fat. At the same meeting, Todd G. Kirchgessner at the University of California, Los Angeles, presented new findings about the structure and evolution of the lipoprotein lipase gene. Variation in this gene may cause obesity in some people, he speculates. Kirchgessner and scientists at the Johns Hopkins University in Baltimore plan to study the inheritance of this gene in families with a tendency toward obesity.

In the future, scientists may use genetic information to identify children at high risk of obesity. Early identification and intervention may help such people avoid obesity and reduce their risk of developing heart disease, Burns says.

– K.A. Fackelmann

CMV a risk in child care

Women of childbearing age who care for children below 3 years of age in day-care centers face a serious occupational risk: exposure to a virus that can cause birth defects. A study of 610 women working at 34 day-care centers in the Richmond, Va., area shows that these individuals, compared with 565 female hospital workers their age, face five times the risk of acquiring cytomegalovirus (CMV) infection. In adults and toddlers, this herpesvirus usually causes nothing more than flu-like symptoms. But in babies whose mothers become infected during the first six months of pregnancy, it can cause hearing loss and retardation.

Studies have shown that 25 to 60 percent of children in U.S. day-care acquire CMV, many without ever showing symptoms. They can then shed the virus in saliva for four weeks after infection and in urine for up to two years.

Stuart Adler, a virologist at the Medical College of Virginia in Richmond, reasoned that because toddlers require such intimate care, their caregivers might face a higher-than-usual CMV risk. In the Nov. 9 NEW ENGLAND JOURNAL OF MEDICINE, he confirms that the women who cared for the youngest children were significantly more likely to have been infected (46 percent) than women caring for children 3 years and older (35 percent). Moreover, during the two years these women were studied, the rate of new infection was 11 percent among the caretakers of these youngest children, compared with 2.5 percent among the hospital workers.

Adler advises day-care workers who are pregnant or anticipating pregnancy to take special care to wear plastic gloves when handling diapers, to wash their hands after wiping runny noses, and to avoid kisses in the nose and mouth area.

– J. Raloff

Manic depression: Suspect gene acquitted

A 1987 study of manic-depressive illness among six generations of an Amish family showed strong evidence that a single gene – its presence inferred from two "marker" genes – carried the disorder. Further study of the family now casts serious doubt on that particular link, while leaving open the possibility that manic depression has some yet-unidentified genetic basis.

The 1987 study analyzed the correlation between manic depression, also known as bipolar affective disorder, and two genes on chromosome 11 (SN: 2/28/87, p.132). Researchers found that family members with manic depression were likely to have particular forms of an insulin gene and of a gene associated with benign tumors. Neither of these marker genes appeared to cause manic depression, but the correlation suggested that a gene involved in manic depression lay near them on the same chromosome.

The new study, reported in the Nov. 16 NATURE, adds 39 people from five generations to the original 81-member pedigree. It also incorporates new data on some family members studied earlier. Since the original study, one participant has become manic-depressive and another has developed major depressive disorder. In 10 other original subjects, investigators had analyzed only one of the two marker genes.

When the researchers took these factors into account in reexamining the link between manic depression and the two marker genes, they found that the likelihood of a single gene on chromosome 11 carrying the disorder in this family dropped 1,000-fold, says study leader Steven M. Paul of the National Institute of Mental Health in Bethesda, Md. Although the updated study does not absolutely rule out this possibility, it makes it so unlikely as to be "unpublishable," Paul says.

The revised pedigrees still indicate manic depression has some sort of genetic basis, he says. It may involve more than one gene or a single gene on another chromosome, he suggests.

The Old Order Amish, members of a religious sect in Pennsylvania, number approximately 15,000 in large, well-documented families descended from 30 pioneer couples. Consequently, scientists view them as ideal subjects for genetic studies. And because very few Amish abuse alcohol or drugs, researchers can study their mental illnesses without these confounding factors. *– A. McKenzie*

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