THE WALL STREET JOURNAL EUROPE

Scientists find gene sites linked to obesity

By Ron Winslow

A research consortium says it has identified 18 new gene sites linked to obesity and 13 others associated with how fat is distributed in the body, advances that shed new light on the complex biology underlying one of the world's most pressing public-health problems.

The findings are based on studies involving nearly 250,000 people, making the effort the largest so far to unravel the genetic basis of common human traits, researchers said. They were published online Sunday in two papers by the journal Nature Genetics.

All told, researchers found 32 genetic sites associated with obesity, including 14 previously associated with the condition. Only one of the sites linked to body-fat distribution was previously known.

More than 25% of U.S. adults are obese, as measured by a ratio of height and weight known as body mass index, or BMI. Around the world, developed nations and emerging economies alike have seen alarming rises in the prevalence of obesity, which is associated with higher risks of heart disease, cancer and other serious ailments as well as soaring health-care costs.

Weight gain generally results when people burn fewer calories than they consume. Genes, personal choices and an environment that encourages fast food and discourages exercise all play roles. But many people keep weight off without close attention to diet and exercise, while others put on pounds despite healthy habits. Researchers believe genetic differences may explain some of this variation.

"If we could understand a lot more about why people are resistant to our environment and stay lean despite all the pressures there are to gain weight, we'd have a better shot at getting better therapies than we have now," said Joel Hirschhorn a physician and researcher at Children's Hospital Boston who was involved in both studies.

But translating the findings into tools for clinicians and consumers is, for the most part, many years off, researchers said. The new studies identify specific areas of the human genome—not actual genes. More research is needed to determine what genes are involved and, in many cases, to figure out their function before work might begin on new therapies.

Moreover, combining the effects of all 32 variants explained only a small fraction of the differences in BMI.

"If you just ask people whether their parents were obese or not, the ability to predict whether a person is going to be obese is better" than all the gene sites the researchers identified, said Michael Jensen, an obesity expert in the endocrine research unit at Mayo Clinic, Rochester, Minn.

Dr. Jensen, who wasn't involved in the study, said the new findings might help in finding new drug targets for obesity, but measuring variability in DNA "doesn't seem to be where the money is if you want to explain human obesity."

Most of the genes previously linked to obesity are involved in appetite regulation and energy balance. Thus the new results "are pretty exciting" because they indicate that much of the biology underlying obesity is uncharacterized, said Elizabeth K. Speliotes, a physician and researcher at Massachusetts General Hospital, Boston who was involved with both studies.

The 32 gene sites related to overall obesity indicate that people could have up to 64 genetic variants (one from each parent) affecting their risk, Dr. Speliotes said. The study found that people with more than 38 variants associated with increasing body mass index were 15 to 20 pounds heavier than those who had fewer than 22 such variants.

The study related to body-fat distribution found that women were much more likely than men to have genetic variants predicting development of fat in the hips and thighs as opposed to the abdomen. Abdominal fat is associated with a higher risk of diabetes and heart disease. Studies show that fat around the hips and thighs may provide protection against such diseases.


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