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Obesity gene's role revealed in mice study

Researchers believe they have identified why a mutation in a particular gene can lead to obesity.

Mouse experiments suggested the body's message to "stop eating" was blocked if the animals had the mutation.

The study, published in *Nature Medicine*, said the brain's response to appetite hormones was being disrupted.

The Georgetown University Medical Center researchers hope their findings could lead to new ways to control weight.

Many genes have been linked to obesity, one of them - brain-derived neurotrophic factor gene - has been shown to play a role in putting on weight in animal and some human studies.

However, scientists at the Georgetown University Medical Center said the explanation for this link was unknown.

Overeating

In studies on mice which had been genetically modified to have the mutation, the mice consumed up to 80% more food than normal.

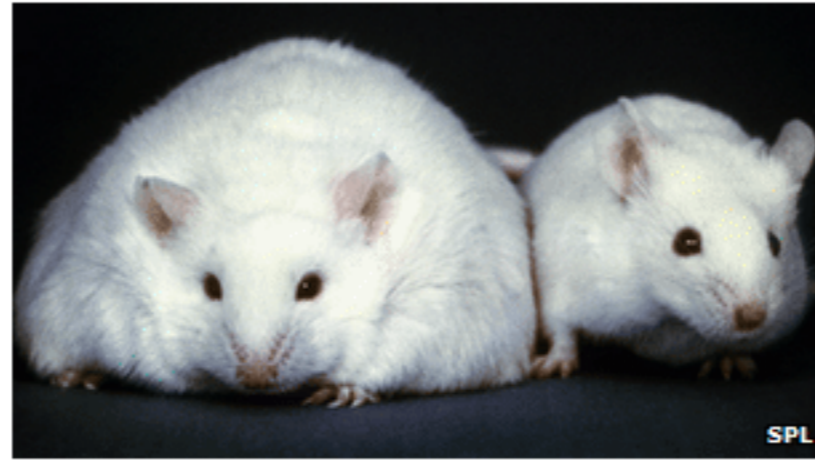
After a meal, hormones such as insulin and leptin should tell the brain that the body is full and should stop eating. The researchers showed that in the mutated mice the message was not being passed on from the hormones in the blood to the correct part of the brain.

One of the researchers Prof Baoji Xu said: "If there is a problem with the BDNF gene, neurons can't talk to each other, and the leptin and insulin signals are ineffective, and appetite is not modified."

He said the discovery "may open up novel strategies to help the brain control body weight" such as finding a "drug that can stimulate BDNF expression".

Prof Sadaf Farooqi, who studies the relationship between genes and obesity at the University of Cambridge, told the BBC: "Genes have a surprisingly large role, it's often underestimated. Between 40 and 70% of the difference in weight between two individuals is due to genetics."

She said completely disrupting the brain-derived neurotrophic factor gene had been shown to lead to severe obesity. However, she cautioned that the study was "entirely in mice" and the mutation was "very rare" in people.



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