A chemical switch in the brain tells the body to burn fat after a meal, a new study has revealed.

Scientists at Monash University in Australia have found a mechanism in the brain that specifically links eating with using energy.

Laboratory models show that the mechanism, which is called browning, turns on after a meal to burn energy and turns off in between meals to conserve it.

The discovery provides a potential novel target for the treatment of obesity, a major risk factor for things like heart disease, diabetes, liver disease and multiple forms of cancer.

"What our studies have shown is that there is a fundamental mechanism at play that normally ensures that energy expenditure is matched with energy intake," explained lead author Professor Tony Tiganis.
'When this is defective, you put on more weight. Potentially we may be able to rewire this mechanism to promote energy expenditure and weight loss in obese individuals.'

Researchers examined brain scans of mice and found that eating a meal controls the browning of fat.

Browning refers to the conversion of energy-storing fat (white fat) into energy-expending fat (brown).

Fat is stored in specialized cells in the human body called adipocytes, which change from white to brown, storing and expending energy, then back again throughout the day.

OBESE CHILDREN HAVE A GREATER RISK OF HEART ATTACKS AS ADULTS

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<th>Being obese in childhood raises the risk of heart attacks and strokes much later in life, a major study has found.</th>
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<td>Children who were obese at the age of 10 were shown to have damaged arteries 25 years later - even if they lost weight in the intervening years.</td>
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<td>The findings, by scientists at the University of Surrey, found obese children were more likely to develop pre-diabetes, thickened arteries and high blood pressure as adults - all problems which raise the risk of heart disease, strokes and other cardiovascular problems later in life.</td>
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<td>The study suggests that being obese - even fleetingly - can have a lasting impact on the human body.</td>
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<td>Officials are increasingly worried that obesity has become 'normalized' in Britain and the US because so many children are overweight.</td>
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<td>A study by Newcastle University found only 30 percent parents with an overweight child correctly identified them as having a weight problem.</td>
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The study, published in Cell Metabolism today, shows that after a meal, the brain responds to circulating insulin.

Insulin levels rise when blood glucose rises, which happens after eating. That insulin causes the brain to send signals to start the browning of fat so energy is expended.

In contrast, when someone stops eating their brain tells these browned adipocytes to convert into white, so they will store energy.
Both processes prevent excessive weight gain and loss and help body weight remain relatively stable over time.

The research team showed that the brain's ability to sense insulin and coordinate meal time with energy expenditure by browning the cells is controlled by a switch.

The switch is turned on after a fast to inhibit response to insulin, repressing any energy sent to conserving energy. After the meal is over, the mechanism is turned off so insulin will flow and promote browning to expend energy.

When people are obese, that switch stays on all the time so the body never expends energy or burns fat.

'What happens in the context of obesity is that the switch stays on all the time - it doesn't turn off during feeding,' Professor Tiganis explained.

'As a consequence, browning is turned off all the time and energy expenditure is decreased all the time, so when you eat, you don't see a commensurate increase in energy expenditure - and that promotes weight gain.'

The researchers hope now that they've found the switch, they could inhibit it for therapeutic purposes to promote the shedding of excess fat, potentially tackling the world's obesity epidemic.

'Obesity is a major and leading factor in overall disease burden worldwide and is poised, for the first time in modern history, to lead to falls in overall life expectancy,' Professor Tiganis said.

'For a long time, the missing piece to the puzzle was always why this occurred in the body,' Dr Garron Dodd, another author of the study, said.

'We've not only shown why this occurs but also the fundamental mechanism involved. It's very exciting.'