Key link between obesity and type 2 diabetes discovered

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(Medical Xpress)—New research published in the journal *Cell Metabolism* has identified a key mechanism in the immune system involved in the development of obesity-linked type 2 diabetes. The findings open up new possibilities for treatment and prevention of this condition, which is becoming increasingly prevalent worldwide. The study is by Dr Jane Howard and Professor Graham Lord, King's College London, and colleagues, and is funded by the UK Medical Research Council.

There are an estimated 371 million people with diabetes in the world and around 90 per cent of these cases are type 2 diabetes. By 2030, there will be some 550 million with the condition based on current trends. Cases of diabetes have more than doubled since 1980, with 70 per cent of the trend due to ageing populations worldwide and the other 30 per cent

estimated to be due to increasing prevalence of risk factors including obesity.

The association between obesity and diabetes has long been recognised but the molecules responsible for this association are unclear. Dr Jane Howard, lead author in this research and colleagues from King's, studied mice genetically engineered to lack T-bet, a protein which regulates the differentiation and function of immune cells. They found that the mice had improved insulin sensitivity despite being obese.

'When T-bet was absent this altered the relationship between fat and insulin resistance; the mice had more intra-abdominal fat but were actually more sensitive to the glucose lowering effects of insulin,' said Dr Howard. 'As fat accumulation in the abdomen is typically associated with worsening insulin resistance and other features of the metabolic syndrome, the findings seen were both unusual and unexpected.'

It turned out that the intra-abdominal fat of these mice contained fewer immune cells and was less inflamed than that of normal mice. The researchers then went on to discover that by transferring immune cells lacking T-bet to young, lean mice they were able to improve insulin sensitivity. 'It appears that T-bet expression in the adaptive immune system is able to influence metabolic physiology,' added Professor Lord.

Although human obesity is often associated with insulin resistance and diabetes, this is not always the case. 'Our data suggests that obesity can be uncoupled from insulin resistance, through the absence of T-bet,' said Dr Howard. Several of the main drugs currently used to treat type 2 diabetes work by improving insulin sensitivity. Further studies are needed to identify other molecules in the pathway of action of T-bet which could pave the way for future drug development in the treatment of Type 2 diabetes. The administration of specific immune cells as immunotherapy to improve insulin resistance may also one day become a

therapeutic possibility. 'This is just the start,' said Dr Howard, 'the idea that the immune system can impact on metabolism is very exciting, but more research needs to be done before we can bring this work from the bench to the bedside for the benefit of patients.'

Provided by King's College London

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