Vitamin D improves insulin sensitivity in mice

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New research presented at this year's European Association for the Study of Diabetes (EASD) meeting in Munich, Germany (12-16 Sept) shows that giving vitamin D improves insulin sensitivity in mice that have become insulin resistant due to a chronic high fat high sugar diet. Vitamin D also reduces the accumulation of fat in muscles (myosteatosis), another sign of improving metabolism. The study is by Dr Elisa Benetti and colleagues from the University of Turin, Italy.

Human studies indicate a strong association between vitamin D deficiency and type 2 diabetes. In particular, epidemiological evidence shows that a poor vitamin D status increases the risk of insulin resistance, however the mechanisms underlying this effect are still not completely understood. In addition, early clinical description reported that severe vitamin D deficiency is associated to myopathy, thus suggesting a potential association between vitamin D and muscle function. The aim of this new study was to evaluate the effect of vitamin D administration in a mouse model of diet induced insulin-resistance, focusing on skeletal muscle.

A total of 40 male mice were provided with a standard diet or high fat-high sugar diet (HFHS) for 4 months. Subsets of animals were treated with Vitamin D (7 micrograms per kg, 3 times per week) for the last 2 months. Body weight and food intake were recorded weekly. At the end of the treatment, a glucose tolerance test was performed. The expression of markers of fat generation and insulin signalling were analysed.

In comparison to standard diet, HFHS diet induced body weight increase (24.8g vs 31.8 g), hyperglycemia (108 vs 145mg/dl) and impaired glucose tolerance. At the muscle level, HFHS animals showed fat accumulation and a significant increase of triglycerides and these effects were correlated to an impaired insulin responsiveness. Vitamin D administration reduced body weight and improved the oral glucose tolerance test. Vitamin D also reversed myosteatosis by decreasing the fatty acid synthesis.

In addition, animals treated with vitamin D showed an improved muscle insulin responsiveness. HFHS diet increased the expression of both CML, one of the main AGEs (Advanced glycation end products) and its receptor RAGE. AGEs are proteins or lipids that become glycated after exposure to sugars and lipids and are implicated in the pathogenesis of several diseases, including diabetes. These effects were significantly reduced by vitamin D treatment.

The authors conclude: "Our data clearly demonstrate that vitamin D administration improves insulin resistance due a chronic exposure to a high fat high sugar diet. Reduction of myosteatosis and muscle CML/RAGE expression can significantly contribute to the beneficial effects of vitamin D."

They add: "Some clinical studies in humans have been conducted to evaluate the effect of vitamin D supplementation on the incidence or progression of type 2 diabetes, but the results are not conclusive at the moment. Further investigations are necessary to better clarify the role of vitamin D in this context."

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