Type 2 diabetes is a complex metabolic disorder resulting from a failure in the body's capacity to secrete enough insulin to combat the insulin resistance that has developed in skeletal muscle and liver. Contributing factors include obesity, advanced age and elevations in stress hormones that are known to promote insulin resistance in skeletal muscle and increase blood sugar production by the liver. It is now fairly well established that the development of insulin resistance is tightly coupled with the development of inflammation and oxidative stress in a numbers of tissues including adipose tissue, muscle and liver. In this paper, and in a companion paper (Am J Physiol Regul Integr Comp Physiol. 2010 Apr 14. [Epub ahead of print]), we examine some of the mechanisms by which regular exercise helps to prevent type 2 diabetes in a rodent model of this disease, the Zucker Diabetic Fatty (ZDF). Here we show that if animals remain sedentary, then the liver develops inflammation and undergoes oxidative stress that then contributes to insulin resistance and the development of Type 2 diabetes. In contrast, if animals exercise voluntarily on a rodent running wheels (averaging just a few kilometres per night), we observe that the markers of inflammation and oxidative stress are ameliorated and the animals do not develop liver insulin resistance and type 2 diabetes. This is yet another example of the powerful and beneficial effects that regular exercise has on metabolism and immune function.

Király MA, Campbell J, Park E, Bates HE, Yue JT, Rao V, Matthews SG, Bikopoulos G, Rozakis-Adcock M, Giacca A, Vranic M, **Riddell MC**. Exercise maintains euglycemia in association with decreased activation of c-Jun NH2-terminal kinase and serine phosphorylation of IRS-1 in the liver of ZDF rats. Am J Physiol Endocrinol Metab. 2010 Mar;298(3):E671-82.

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