

The Cat as a Model for Human Obesity and Diabetes

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Abstract

Obesity is the most common nutritional disorder of cats and is a risk factor for diabetes. Similar to developments in obese people, obese cats show peripheral tissue insulin resistance and may demonstrate glucose intolerance when challenged with pharmacological amounts of glucose. However, they compensate well for the insulin resistance and do not show elevated glucose concentrations when monitored during their regular daily routine, including postprandial periods. This is possible because obese cats in the fasted and postprandial state are able to maintain hepatic insulin sensitivity and decrease endogenous glucose production, which allows them to maintain normoglycemia. Also dissimilar to what is seen in many obese humans, cats do not develop atherosclerosis and clinical hypertension. The time course for progression to overt diabetes of obese cats is unknown. One might speculate that diabetes develops when the liver finally becomes insulin resistant and/or insulin secretion becomes too low to overcome increased glucose production. In addition, amyloid, demonstrated to be deposited in islet of chronically obese cats, may contribute to a reduction in insulin secretion by reducing functional β -cell mass.

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Abbreviations: (BMI) body mass index, (DEXA) dual energy X-ray absorptiometry, (EGP) endogenous glucose production, (EHC) euglycemic hyperinsulinemic clamp, (HDL) high-density lipoprotein, (HOMA) homeostatic model assessment, (IVGTT) intravenous glucose tolerance test, (LDL) low-density lipoprotein, (VLDL) very-low-density lipoprotein

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