

Does a high fat diet cause diabetes t2 in mice? - Yes if it contains hydrogenated coconut fat!

A paper has been published [2] followed by some articles, for example [3] interpreting it as supposedly proving that a high fat diet (using high fat chow D12331, see [1]) causes diabetes in certain breed of mice that are genetically susceptible.

References:

1)

Product Data

D12331



Description

58 kcal% fat w/sucrose Surwit Diet

Used in Research

Obesity

Diabetes

Packaging

Product is packed in 12.5 kg box.

Each box is identified with the product name, description, lot number and expiration date.

Lead Time

IN-STOCK. Ready for next day shipment.

Gamma-Irradiation

Yes. Add 10 days to delivery time.

Form

Pellet, Powder

Shelf Life

Most diets require storage in a cool dry environment. Stored correctly they should last 3-6 months. Because of the high fat content is best if kept frozen.

Control Diets

D12329

Formula

Product #	D12331	
	gm%	kcal%
Protein	23.0	16.4
Carbohydrate	35.5	25.5
Fat	35.8	58.0
	Total	100.0
	kcal/gm	5.56
Ingredient	gm	kcal
Casein, 80 Mesh	228	912
DL-Methionine	2	0
Maltodextrin 10	170	680
Corn Starch	0	0
Sucrose	175	700
Soybean Oil	25	225
Coconut Oil, Hydrogenated	333.5	3001.5
Mineral Mix S10001	40	0
Sodium Bicarbonate	10.5	0
Potassium Citrate, 1 H2O	4	0
Vitamin Mix V10001	10	40
Choline Bitartrate	2	0
FD&C Red Dye #40	0.1	0
Total	1000.1	5558.5

Professor Richard Surwit designed these diets with us for his diet-induced obesity studies at Duke University.

Diets match 10/27/92 telephone specifications of R. Surwit, Ph. D., Duke University. Formulated by E. A. Ulman, Ph.D., Research Diets, Inc. November 6, 1992.

- 2) “Dietary and Genetic Control of Glucose Transporter 2 Glycosylation Promotes Insulin Secretion in Suppressing Diabetes”

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http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WSN-4HXBRPG-P&_coverDate=12%2F29%2F2005&_alid=528508319&_rdoc=1&_fmt=&_orig=search&_qd=1&_cdi=7051&_sort=d&_view=c&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=ada61e28d7ccb8554a5c03df972840bf

Summary

Pancreatic β cell-surface expression of glucose transporter 2 (Glut-2) is essential for glucose-stimulated insulin secretion, thereby controlling blood glucose homeostasis in response to dietary intake. We show that the murine GlcNAcT-IVa glycosyltransferase is required for Glut-2 residency on the β cell surface by constructing a cell-type- and glycoprotein-specific N-glycan ligand for pancreatic lectin receptors. Loss of GlcNAcT-IVa, or the addition of glycan-ligand mimetics, attenuates Glut-2 cell-surface half-life, provoking endocytosis with redistribution into endosomes and lysosomes. The ensuing impairment of glucose-stimulated insulin secretion leads to metabolic dysfunction diagnostic of type 2 diabetes. Remarkably, the induction of diabetes by chronic ingestion of a high-fat diet is associated with reduced GlcNAcT-IV expression and attenuated Glut-2 glycosylation coincident with Glut-2 endocytosis. We infer that β cell glucose-transporter glycosylation mediates a link between diet and insulin production that typically suppresses the pathogenesis of type 2 diabetes.

- 3) “Researchers discover how a high-fat diet causes type 2 diabetes”

Jim Keeley, Howard Hughes Medical Institute
Howard Hughes Medical Institute

http://www.eurekalert.org/pub_releases/2005-12/hhmi-rdh122105.php

Researchers discover how a high-fat diet causes type 2 diabetes
Howard Hughes Medical Institute researchers have discovered a molecular link between a high-fat, Western-style diet, and the onset of type 2 diabetes. In studies in mice, the scientists showed that a high-fat diet disrupts insulin production, resulting in the classic signs of type 2 diabetes.

In an article published in the December 29, 2005, issue of the journal Cell, the researchers report that knocking out a single gene encoding the enzyme GnT-4a glycosyltransferase (GnT-4a) disrupts insulin production. Importantly, the scientists showed that a high-fat diet suppresses the activity of GnT-4a and leads to type 2 diabetes due to failure of the pancreatic beta cells.