## Matfett och diabetes

*Sammanfattning:*

*- Totala mängden fett i kosten har liten eller ingen betydelse för diabetes.*

*- Av de olika typerna av fett, tycks mättat och enkelomättat fett inte påverka diabetessjukdomen.*

*- Fleromättat fett kan försämra diabetes hos äldre. I andra studier kan det förbättra sjukdomen. Skillnaden kan bero på vilket fleromättat fett man väljer. Intag av fett med en stor andel omega6 fettsyror och ett högt förhållande mellan omega6 och omega3 bör undvikas.(T ex solrosolja). Möjligen kan skillnaden också bero på om det fleromättade fettet - känslig för oxidation - är berövat sina antioxidanter genom kemiska processer, eller icke processat och då åtföljs av sina naturliga antioxidanter?*

*- Diabetiker bör inte äta oxiderade fetter. Processade fleromättade fetter härsknar (oxiderar) lätt.*

*- Härdade fetter (transfetter), som bl a finns i vissa margariner, bröd, kakor, pulversåser, buljongtärningar, choklad m m visar klarast samband med diabetes, men även andra sjukdomar.*

*- Konjugerade linolsyror (CLA), som finns i smör och mjölk, tycks förbättra diabetessjukdom.*

Det är sedan länge känt att metabolismen av fett i kroppen och diabetes hör samman.

*Vessby m fl (1992)* har visat att typ-2 diabetes (åldersdiabetes) försämras vid intag av fleromättade fetter hos åldringar. (Bilaga 1). Margariner innehåller förhållandevis mycket processat fleromättat fett.

*Kuller (1993)* har visat att de som konsumerade härdat vegetabiliskt fett hade högre nivå av insulin som svar på en glykosbelastning. Vissa svenska margariner m m innehåller härdat vegetabiliskt fett. (Bilaga 2)

*Simopoulus (1994)* har uttryckt behovet av att utvärdera hur transfettsyror (finns i härdat vegetabiliskt fett) i vår diet påverkar insulinresistens. (Bilaga 3)

*Mann (1994)* förutser en utveckling av insulinresistens från transfettsyror. (Bilaga 4).

*Barnard m fl (1990)* visar att transfettsyror påverkar insulinbindningen i apor (Bilaga 5).

*Martha Belury m fl (2000)* har hos American Chemical Society rapporterat att komponenten CLA (konjugerade linolsyror) i mjölkfett tycks skydda mot typ-II diabetes. Två grupper av åldersdiabetiker fick dagligen CLA respektive safflowerolja. Fastevärdet för blodsocker var lägre för CLA-gruppen, men även halten triglycerider i blodet var sänkt, vilket också upprepats i råttförsök. Dessa personer hade också lägre halt av leptin i blodet, något som i annan forskning har satts i samband med ansamling av fett i kroppen och övervikt.

Ett annat fynd var att man kunde fördröja utbrottet av diabetes hos råttor, som var disponerade för detta, med hjälp av CLA. CLA finns i smörfett men inte i margariner.
(Bilaga 6)

*Salmerón et al. (2001)* undersökte samband mellan intag av fett och typ 2-diabetes i en stor studie med 84.000 kvinnor. Varken totala intaget eller mättade fetter hade samband med uppkomst av diabetes, men ett stort intag av transfetter ökar risken betydligt. Byte från härdade till icke härdade fleromättade fetter kan väntas förbättra läget. (Bilaga 7). I andra studier har fleromättade fetter visat samband med försämrad diabetessjukdom.

Denna skillnad kan ha att göra med vilket fleromättat fett man använder. *Sircar S et al.(1998)* säger i en rapport:
”Dagens data när det gäller matfett indikerar att det inte bara är närvaron av fleromättat fett, utan vilken typ av fleromättat fett det är frågan om som är viktigt. Ett högt innehåll av omega6 samt ett högt omega6/omega3-förhållande i matfett har visat sig kunna ge upphov till både hjärt- och kärlsjukdom samt diabetes. De nya ”hjärtvänliga” oljorna som solrosolja och saffloreolja har detta oönskade innehåll av fleromättade fetter. Det finns ett stort antal studier som indikerar att ensamt intag eller överdrivet stora intag av dessa nya vegetabiliska oljor i själva verket är skadliga för hälsan. Ett byte till en kombination av olika typer av fett, inkluderande traditionella matfetter som smör, kokosfett, senapsfröolja skulle minska risken för fel när det gäller kroppens fetter, hjärt- och kärlsjukdom samt typ-II diabetes.” (Bilaga 8).

Möjligen kan skillnaden också bero på om de fleromättade fetterna - känslig för oxidation - är berövade sina antioxidanter genom kemiska processer, eller icke processad och då åtföljs av sina naturliga antioxidanter?

*Mary G. Enig* Ph.D, den kanske internationellt mest kunniga när det gäller transfetter (härdade fetter), säger i en intervju i Dr Mercolas nättidning Nr 157, 10/6 2000:

”Både studier på apor och människor har visat en ogynnsam påverkan på blodsockret: Transfettsyror minskar röda blodkropparnas svar på insulin, på så sätt en potentiellt oönskad effekt för diabetiker. Forskningen på apor gjordes i Maryland i samarbete med U. S. Department of Agriculture och National Institutes of Health medan forskningen på människor gjordes nyligen vid Universitetet i Pittsburgh.”

I sin bok ”Know Your Fats” (www.bethesdapress.com) säger Mary Enig:
”Många läkare tror att diabetes försämras av ”mättat fett”. Denna uppfattning grundar sig på den rådande föreställningen om orsakerna till hjärtsjukdom. Forskning har visat att transfetter har en allvarlig påverkan på diabetes, vilket inkluderar störningar på insulinbindningen. Det finns ingen visad mekanism som skulle förklara negativa effekter från konsumtion av mättade fetter. Människor som har dålig kontroll över sin diabetes är mycket känslig för väntade effekter från oxiderade fetter. När fleromättade fetter och oljor inte är ordentligt skyddade ökar andelen oxiderade fetter. Diabetiker bör undvika oxiderade fetter och oljor.”

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2001-06-21

Gunnar Lindgren
Starrkärr 210, 446 95 Älvängen
Tel 0303-745 155 el 070-567 90 54
gunnar.lindgren@ale.mail.telia.com

## **Bilagor** (Endast sammanfattningar)

Bilaga 1

*Diabet Med* 1992 Mar;9(2):126-33 

### **Polyunsaturated fatty acids may impair blood glucose control in type 2 diabetic patients.**

**Vessby B, Karlstrom B, Boberg M, Lithell H, Berne C**

Department of Geriatrics, Uppsala University, Sweden.

Fifteen patients with Type 2 diabetes were given two diets rich in either saturated fat or polyunsaturated fat in alternate order over two consecutive 3-week periods on a metabolic ward. Both diets contained the same amount of fat, protein, carbohydrates, dietary fibre, and cholesterol. The proportions of saturated, monounsaturated and polyunsaturated fatty acids in the saturated fat diet were 16, 10, and 5%-energy and in the polyunsaturated fat diet (PUFA) 9, 10, and 12%-energy. The PUFA diet contained a high proportion of n-3 fatty acids. Metabolic control improved significantly in both dietary periods, due to both qualitative dietary changes and a negative energy balance. The serum lipoprotein concentrations decreased on both diets but the serum lipids were significantly lower after the PUFA diet (serum triglycerides -20%, p = 0.001; serum cholesterol -5%, p = 0.03; VLDL-triglycerides -29%, p less than 0.001; and VLDL-cholesterol -31%, p = 0.001) than after the saturated fat diet. Average blood glucose concentrations during the third week were significantly higher fasting (+15%, p less than 0.01), and during the day at 1100 h (+18%, p less than 0.001) and 1500 h (+17%, p = 0.002) on PUFA than on the saturated fat diet. Significantly higher blood glucose levels were also recorded with a standard breakfast, while the sum of the insulin values was lower (-19%, p = 0.01). HbA1c did not differ significantly between the two dietary periods.

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Bilaga 2

THE LANCET VOL 341: APRIL 24, 1993

SIR, - There seem to be three possible explanations for Professor Willet and colleagues findings, First, *trans* fatty acids, as previously shown, result in an increase in total or low-density-lipoprotein cholesterol, or possibly, a small decrease in high-densitylipoprotein (HDL) cholesterol similar to saturated fatty acids; second, they have an independent effect on the risk of cardiovascular disease; and third, *trans* fatty acid intake is a marker for other nutrients or behavioural characteristics that are associated with the risk of coronary heart disease. The Nurses Study provides no independent measurements of blood lipoprotein concentrations, blood pressure, or other risk factors. Thus, the relation between the *trans* fatty acids and risk factors related to cardiovascular disease could not be measured.

We have analysed the relation between reported margarine intake and risk factors among 540 premenopausal women at baseline in the Healthy Women Study. Dietary information was obtained from food frequency and 24 h recall. Frequent users were defined as having margarine four or more times per week (n= 259, 48 %). Frequent users weighed about 2,3 kg more (p=0,05) than infrequent users; they had significantly lower HDL2 cholesterol (0,53 vs 0,59 mmol/L) and lower total HDL cholesterol concentrations. They also had higher total cholesterol (4,84 vs 4,71 mmol/L; p=0,07); higher triglyceride (0,99 vs 0,90 mmol/L; p=0,04); and higher apo B (l,75 vs 1,66 mikromol/L; P = 0,04); and the log of 2 h blood insulin after glucose load was also higher (0,31 vs 0,28 nmol/L). Despite the fact that margarine consumers weighed over 2 kg more than infrequent consumers, they reported roughly the same caloric intake (1700 calories) and similar levels of physical activity. The Nurse Study had previously reported only 1200 –1400 calorie intakes for similarly aged women

The food frequency questionnaire, as used in the Nurses and other studies, provides a poor estimate of individual dietary intake of fat and specific fats or caloric intake. There is a substantial

underestimation of caloric intake compared with body size. The Nurses Study previously noted that the mean fat intake (75g) was the same for nurses with body mass index (BMI) of 21 or more and for those with BMI of 29 or more. It is highly unlikely that this difference is attributable to exercise. We suspect that most of the missing calories are fat calories. It is obvious that women who consume more margarine; baked goods, and cookies, are eating more fat, have higher saturated fat calories, are probably fatter; and, obviously, are consuming more *trans* fatty acids.

Without better dietary data and risk factor measurements, it cannot be determined in the Nurses Study whether there is causal link between *trans* fatty acids and cardiovascular risk factors. Second, - because there are no measurements of the other cardiovascular risk factors, whether the *trans* fatty acids have an independent effect on cardiovascular disease cannot be established. Third, because of the weakness of the instrument in measuring dietary intake, it is impossible to determine whether the results are related to *trans* fatty acids or some other measures of dietary intake.

Department of Epidemiology
Graduate School of Public Healt
University of Pittsburgh
Pittsburgh, Pennsylvania 15261, USA
Lewis H. Kuller

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Bilaga 3

Free Radic Biol Med 1994 Oct;17(4):367-72 

## Is insulin resistance influenced by dietary linoleic acid and trans fatty acids?

**Simopoulos AP.**

Center for Genetics, Nutrition and Health, Washington, DC.

The incidence of obesity, noninsulin-dependent diabetes mellitus (NIDDM), hypertension, and coronary artery disease has increased in the developed world. At the same time, major changes in the type and amount of fatty acid intake have occurred over the past 40-50 years, reflected in increases in saturated fat (from both animal sources and hydrogenated vegetable sources), trans fatty acids, vegetable oils rich in linoleic acid, and an overall decrease in long chain polyunsaturated fatty acids (arachidonic acid, eicosapentaenoic acid, and docosahexaenoic acid--C20-C22). Recent findings that C20-C22 in muscle membrane phospholipids are inversely related to insulin resistance, whereas linoleic acid is positively related to insulin resistance, suggest that diet may influence the development of insulin resistance in obesity, insulin-dependent diabetes mellitus (IDDM), hypertension, and coronary artery disease (including asymptomatic atherosclerosis and microvascular angina). These conditions are known to have genetic determinants and have a common abnormality in smooth muscle response and insulin resistance. It is proposed that the current diet influences the expression of insulin resistance in those who are genetically predisposed. Therefore, clinical investigations are needed to evaluate if lowering or preventing insulin resistance through diet by increasing arachidonic acid, eicosapentaenoic acid, and docosahexaenoic acid, while lowering linoleic acid and decreasing trans fatty acids from the diet, will modify or prevent the development of these diseases.

Lancet 1994 May 21;343(8908):1268-71 

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Bilaga 4

**Metabolic consequences of dietary trans fatty acids.**

**Mann GV.**

The epidemic of coronary heart disease in the western world followed the introduction of partially hydrogenated fats in food. Exposure to trans fatty acids (TFA) in those foods can explain the observed sex and age differences in serum cholesterol concentrations and coronary heart disease (CHD), the cholesterolaemic response to pregnancy, and national differences in rates of CHD. There is evidence that TFA can be innocuously used for muscular work. I propose that the TFA in partially hydrogenated fats impair lipoprotein receptors during energy surfeit, leading to hypercholesterolaemia, atherogenesis, obesity, and insulin resistance. A series of feasible experiments is proposed to examine this hypothesis.

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Bilaga 5

J. Nutr. Biochem., 1990, vol. 1, April

# Dietary *trans* fatty acids modulate erythrocyte membrane fatty acyl composition and insulin binding in monkeys

**Dennis E. Barnard, (1,2) Joseph Sampugna, (1) Elliott Berlin, (3) Sam J. Bhathena, (3) and Joseph J. Knapka (2)**

(1) Department of Chemistry and Biochemistry, University of Maryland, College Park, MD, USA
(2) Veterinary Resources Branch, Division of Research Services National Institutes of Health, U.S.
Department of Health and Human Services Bethesda MD, USA
(3) Beltsville Human Nutrition Research Center, Agricultural Research Service, U.S. Department
of Agriculture, Beltsville, MD, USA

*The substitution of trans- for half of the cis-monounsaturated fatty acids in the diet of Macaca fasicularis monkeys resulted in alterations in erythrocyte fatty acid composition and insulin receptor properties but not in membrane fluidity. Both cis and trans diets contained 10% fat and similar fatty acid compositions, except that approximately 50% of the cis-octadecenoate (c-18:1) in the cis diet was replaced with trans-octadecenoate isomers (t-l8:1) in the trans diet. Compared with the cis diet, the trans diet resulted in the incorporation of approximately 11 % t-18:1, an approximately 16 % decrease in total saturated fatty acids, and an approximately 20 % increase in 18: 2(n-6) in erythrocyte membrane lipids. The increase in 18:2(n-6) may reflect on homeostatic mechanisms designed to maintain overall membrane fluidity, as no diet-related changes in fluidity were observed with diphenylhexatriene steady state fluorescence polarization. Values observed for insulin binding and insulin receptor*

*number were higher and binding affinity was lower in monkeys fed the cis diet. In the absence of an effect on overall membrane fluidity, altered receptor activity suggests that insulin receptor activity is dynamic, requiring specific, fluid membrane subdomains or highly specific fatty acid-protein interactions.*

**Keywords**: *trans* Fatty acids: insulin binding: membrane fluidity

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Bilaga 6

Belury, M.A., A. Mahon, and L. Shi. Role of conjugated linoleic acid (CLA) in the management of type 2 diabetes: Evidence from Zucker diabetic (fa/fa) rats and human subjects (Abstract AGFD 26). American Chemical Society 220th National Meeting. August 20-24. Washington, D.C.

(Se även Science News, March 3, 2001; Vol. 159, No. 9)

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Bilaga 7

**Trans Fatty Acid Consumption May Increase Risk for Type 2 Diabetes**

Salmerón, Jorge, et al. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr 2001;73:1019-26.

Salmerón et al. studied the relationship between the consumption of saturated or unsaturated fats and the onset of type 2 diabetes. The 84,000 women subjects from the Nurse's Health Studies were aged 35-59 years old at the inception of the study in 1980. Over the 14-year course of the study, 2,507 cases of type 2 diabetes were diagnosed. Neither total fat intake nor saturated fat intake was associated with the incidence of diabetes, but high consumption of trans fatty acids strongly increased the risk for the disease. The authors estimate that replacement of 2% of calories in the American diet represented by trans fatty acids with similar amounts of calories as polyunsaturated fats could result in a 40% reduction in the incidence of type 2 diabetes. In an accompanying editorial, Clandinin and Wilke cautioned against extrapolating the results from this large population study to individuals, especially since trans fatty acids represent a relatively small part of the American diet.

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Bilaga 8

**1**: J Indian Med Assoc 1998 Oct;96(10):304-7 Related Articles, Books, LinkOut

## Choice of cooking oils--myths and realities.

**Sircar S, Kansra U.**

Department of Medicine, Safdarjang Hospital, New Delhi.

In contrast to earlier epidemiologic studies showing a low prevalence of atherosclerotic heart disease (AHD) and type-2 dependent diabetes mellitus (Type-2 DM) in the Indian subcontinent, over the recent years, there has been an alarming increase in the prevalence of these diseases in Indians--both abroad and at home, attributable to increased dietary fat intake. Replacing the traditional cooking fats condemned to be atherogenic, with refined vegetable oils promoted as "heart-friendly" because of their polyunsaturated fatty acid (PUFA) content, unfortunately, has not been able to curtail this trend. Current data on dietary fats indicate that it is not just the presence of PUFA but the type of PUFA that is important--a high PUFA n-6 content and high n-6/n-3 ratio in dietary fats being atherogenic and diabetogenic. The newer "heart-friendly" oils like sunflower or safflower oils possess this undesirable PUFA content and there are numerous research data now available to indicate that the sole use or excess intake of these newer vegetable oils are actually detrimental to health and switching to a combination of different types of fats including the traditional cooking fats like ghee, coconut oil and mustard oil would actually reduce the risk of dyslipidaemias, AHD and Type-2 DM.