

Výživová hodnota rostlinných tuků

Dětská výživa a obezita v teorii a praxi 2015

20.11.2015

Praha

Jiří Brát



Média ovlivňují spotřebitele ne vždy správným směrem

7 důvodů, proč je kokosový olej zázračný

Američtí experti zrušili varování o cholesterolu

Experti americké vlády, kteří sestavují potravinová doporučení, se rozhodli odvolat varování před požíváním pokrmů s vysokým obsahem cholesterolu. Cholesterol nepovažují za „znepokojivou látku“, napsal deník The Washington Post.



úterý 17. února 2015, 3:30

▲ ilustrační foto
FOTO: Profimedia.cz

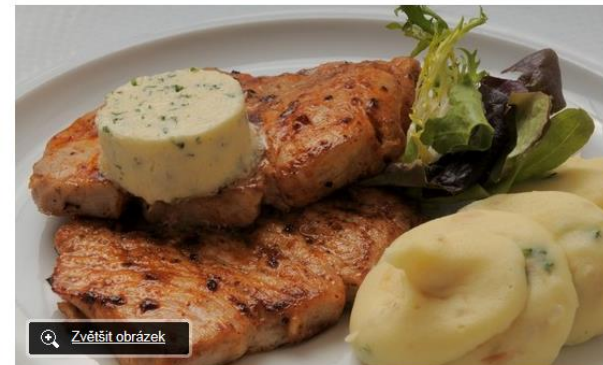


**Lékaři a vědci obrátili na pěníku:
Cholesterol je důležitý pro zdraví!**



Vědci se mýlili, zabijákem srdce jsou cukry, ne tuky

V posledních letech byli lidé neustále upozorňováni, aby omezili konzumaci nasycených živočišných tuků, které mohou za vysokou hladinu cholesterolu v krvi, a tedy ucívání cév. Podle amerických, britských a švédských kardiologů jsou ale pro srdce mnohem větším nebezpečím jednoduché sacharidy, a především bílý cukr.



dle

Jezte tučná jídla. Budete zdravější a zhubnete

17.2.2014



**Zkusíte jídelníček
z pravěku?**



Kolik tuků jiných živin konzumovat?

- tuky
 - WHO 2003 15 – 30 % z celkového příjmu energie
 - WHO 2010 20 – 35 % z celkového příjmu energie
 - Skandinávie 2012 25 – 40 % z celkového příjmu energie
 - SAFA a TFA stejné (10% a 1%),
 - PUFA horní hranice + 1% (10→11%), nárůst na úkor MUFA
 - ω 3 z 1-2% na 0,5-2%
 - ω 6 z 5-8% na 2,5-9%
- sacharidy
 - WHO 2003 55 – 75 % z celkového příjmu energie
 - Skandinávie 2012 45 – 60 % z celkového příjmu energie
 - přidaný cukr 10 % z celkového příjmu energie
 - WHO 2015 prospěšné snížení na 5 % z celkového příjmu energie
- bílkoviny
 - WHO 2003 10 – 15 % z celkového příjmu energie
 - Skandinávie 2012 10 – 20 % z celkového příjmu energie

Jaké údaje jste sledovali při nákupu balených potravin v průběhu minulého roku?

Spotřebitel se nevyzná v mastných kyselinách

	Snažím se jim zcela vyhnout	Snažím se je omezovat	Snažím se zvyšovat jejich obsah	Snažím se zařazovat co nejvíce	Je mi to jedno
Sodík / sůl	5.1%	59.8%	6.9%	1.9%	26.3%
Tuky / oleje	4.6%	59.6%	6.7%	1.9%	27.2%
Cukry obecně	12.1%	52.5%	5.8%	2.7%	26.9%
Cholesterol	13.1%	44.5%	7.6%	3.9%	30.9%
Nasyčené tuky (nasyčené mastné kyseliny)	7.3%	40.5%	10.9%	3.5%	37.8%
Nízkokalorická sladidla	31.7%	15.1%	15.4%	5.7%	32.1%
Komplexní sacharidy	3.1%	28.2%	21.6%	5.4%	41.7%
Trans tuky (trans mastné nenasycené kyseliny)	9.7%	21.6%	21.2%	6.2%	41.3%
Nenasycené tuky (mono- a polynenasycené mastné kyseliny)	3.5%	26.3%	18.9%	4.2%	47.1%
Bílkoviny	0.4%	8.5%	32.5%	20.8%	37.8%
Celozrnnost výrobku	3.9%	3.9%	39.4%	27.7%	25.1%
Vláknina	1.9%	2.3%	35.5%	33.3%	27.0%
Vápník	0.7%	0.8%	46.7%	20.5%	31.3%
Vitamíny / minerální látky	0.2%	0.4%	39.5%	37.1%	22.8%

Spotřebitel nezná složení tuků

- Jen ¼ správných odpovědí, Praha je na tom o něco lépe

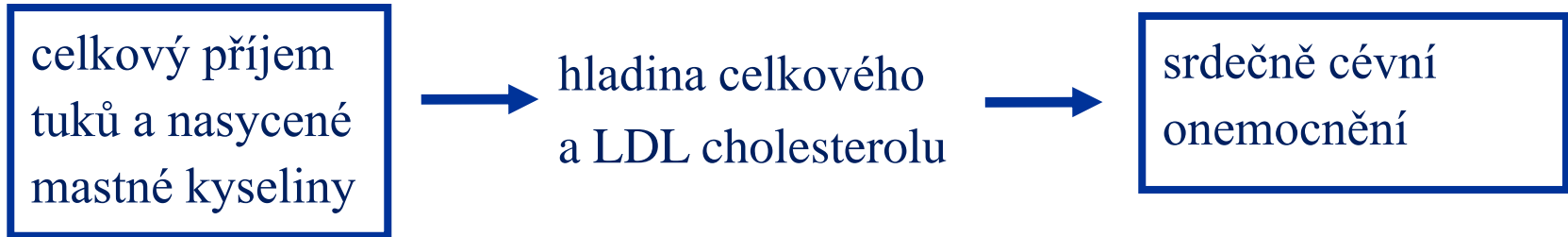
Nejméně nasycených mastných kyselin podle vás obsahuje:

	Česká republika			Praha
	Muži	Ženy	Muži i ženy	Muži i ženy
Řepkový olej	26,4 %	28,6 %	27,5%	42,1 %
Kokosový tuk	8,3 %	12,1 %	10,2 %	10,5 %
Sádlo	8,1 %	7,3 %	7,7 %	5,3 %
Palmojádrový tuk	5,9 %	4,3 %	5,1 %	10,5 %
Nevím	51,3 %	47,7 %	49,5 %	31,6 %

Vliv výživy na chronická onemocnění



tradiční pohled



moderní, komplexní přístup

omega 3 MK, transmastné kyseliny, ostatní MK, různé typy sacharidů, ořechy, luštěniny, zelenina, ovoce, vitaminy, minerální a biologicky aktivní látky, zpracování potravin, příprava potravin

složení krevních lipidů (HDL-cholesterol, triacylglyceroly, apolipoprotein B, apolipoprotein A1, Lipoprotein a), arytmie, zánětlivé procesy, endoteliální funkce, citlivost na inzulin, tvorba krevních sraženin, pocit nasycenosti a tělesná hmotnost

Změny poměrů jednotlivých živin

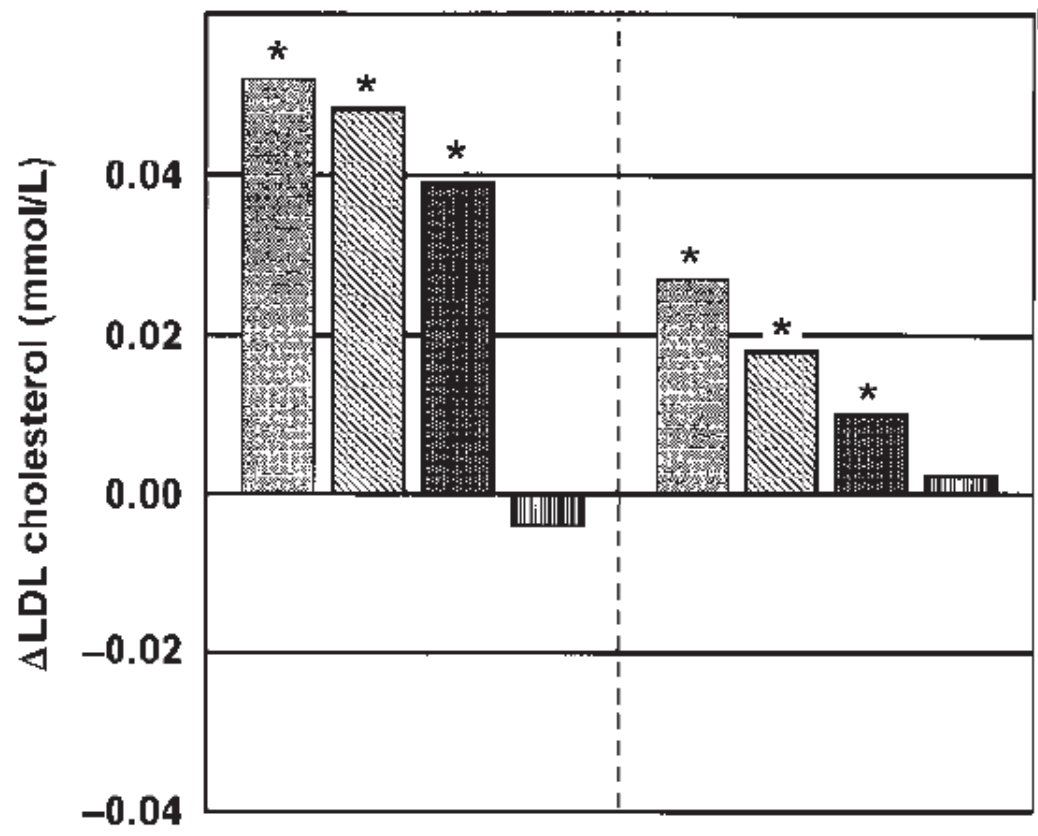
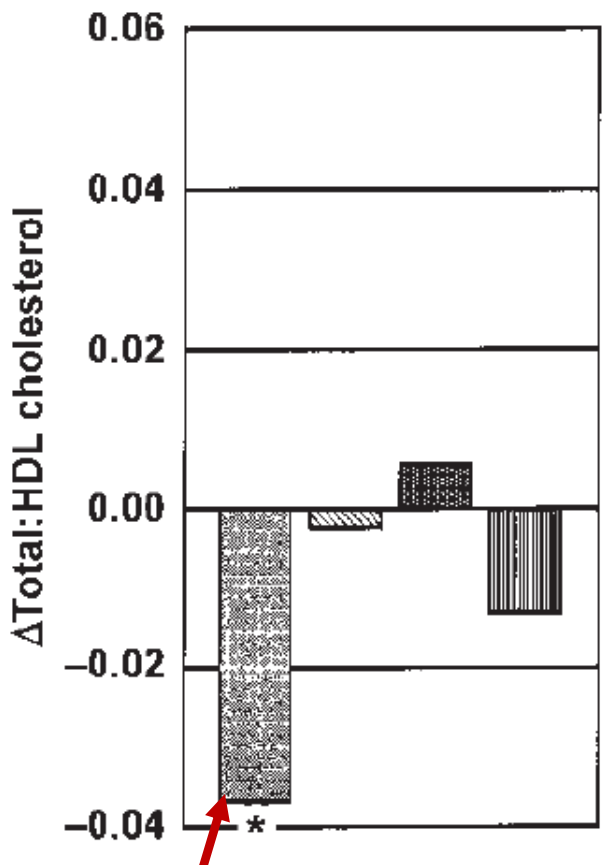
- záleží nejen na tom, co jíme, ale i co nejíme
- základní pravidlo vyvážený příjem a výdej energie
- tuky vs. sacharidy – nejde jen o příjem energie
 - nasycené MK vs. nenasycené MK
 - jednoduché cukry, komplexní sacharidy, vláknina
 - nízký glykemický index vs. vysoký glykemický index
- snižování příjmu bílkovin – redukce svalové hmoty

Mastné kyseliny a lipidy v krvi

Nahrazení sacharidů Nahrazení SAFA

	SAFA	MUFA	TFA	ω 3 PUFA	ω 6 PUFA	MUFA	TFA	ω 3 PUFA	ω 6 PUFA
TC	↑↑	↔	↑↑	↔	↓↓	↓↓	↔	↓↓	↓↓
LDL	↑↑	↔ ↓	↑↑	↔ ↓	↓↓	↓↓	↔	↓↓	↓↓
HDL	↑↑	↑↑	↓↓	↑↑	↑↑	↔ ↓	↓↓	↔ ↓	↔ ↓
TG	↓↓	↓↓	↓↓	↓↓	↓↓	↔	↑↑	↔ ↓	↔ ↓

Vliv složení tuků na poměr celkový / HDL cholesterol



ΔHDL cholesterol (mmol/L)

7 důvodů, proč je kokosový olej zázračný



-  Lauric acid
-  Palmitic acid

-  Myristic acid
-  Stearic acid

Palmový olej zabiják, kokosový tuk superpotravinina?

- Palmový olej je laciný, „skrytý zabiják“, ničí se kvůli němu pralesy
- není vždy vybírán kvůli ceně, ale jeho funkčním vlastnostem
- v potravinách vhodnější variantou oproti částečně ztuženým tukům
- palmu olejnou lze pěstovat udržitelným způsobem
- vliv na hladinu krevních lipidů je lepší než u másla
- kokosový tuk není lepší než tekuté oleje

Srovnání jednotlivých tuků a olejů z hlediska rizikových faktorů kardiovaskulárních onemocnění

	Relativní index aterogenity [4]	Relativní index trombogenity [4]	Vliv na poměr celkový/HDL cholesterol [5]	
nejvyšší ----- nejnižší	kokosový tuk	kokosový tuk	máslo	nejvyšší ----- nejnižší
	mléčný tuk	mléčný tuk	pokrmový tuk	
	skopový lůj	palmový olej	margarin ve folii	
	palmový olej	skopový lůj	palmový olej	
	hovězí lůj	vepřové sádlo	kakaové máslo	
	vepřové sádlo	hovězí lůj	kokosový tuk	
	margariny rostlinné	margariny rostlinné	margarin v kelímku	
	kuřecí tuk	kuřecí tuk	palmojádrový tuk	
	margariny s PUFA*	margariny s PUFA*	majonéza	
	olivový olej	slunečnicový olej	sójový olej	
	slunečnicový olej	tuk z makrely	řepkový olej	

*PUFA – polynenasycené mastné kyseliny.

Klinické, observační studie, metaanalýzy

- léky
 - aktivní látka versus placebo
 - méně vedlejších vlivů
- potraviny
 - soubor živin s pozitivními a negativními účinky konkrétní rizikový faktor v rámci jedné potraviny - nelze oddělit
 - různé vlivy na různé rizikové faktory (např. LDL vs. HDL chol.)
 - skladba stravy
 - lze částečně vyřešit u experimentálních randomizovaných studií s jednotnou stravou
 - méně u observačních studií
 - metaanalýzy – statistické zpracování dat
zdrojová data, způsob zpracování, výběr studií

2 metaanalýzy o vlivu SAFA na rizika KVO

Siri – Tarino et al 2010

Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease¹⁻⁵

Patty W Siri-Tarino, Qi Sun, Frank B Hu, and Ronald M Krauss

ABSTRACT

Background: A reduction in dietary saturated fat has generally been thought to improve cardiovascular health.

Objective: The objective of this meta-analysis was to summarize the evidence related to the association of dietary saturated fat with risk of coronary heart disease (CHD), stroke, and cardiovascular disease (CVD; CHD inclusive of stroke) in prospective epidemiologic studies.

Design: Twenty-one studies identified by searching MEDLINE and EMBASE databases and secondary referencing qualified for inclusion in this study. A random-effects model was used to derive composite relative risk estimates for CHD, stroke, and CVD.

Results: During 5–23 y of follow-up of 347,747 subjects, 11,006 developed CHD or stroke. Intake of saturated fat was not associated with an increased risk of CHD, stroke, or CVD. The pooled relative risk estimates that compared extreme quintiles of saturated fat intake were 1.07 (95% CI: 0.96, 1.19; $P = 0.22$) for CHD, 0.81 (95% CI: 0.62, 1.05; $P = 0.11$) for stroke, and 1.00 (95% CI: 0.89, 1.11; $P = 0.95$) for CVD. Consideration of age, sex, and study quality did not change the results.

Conclusions: A meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD. More data are needed to elucidate whether CVD risks are likely to be influenced by the specific nutrients used to replace saturated fat. *Am J Clin Nutr* doi: 10.3945/ajcn.2009.2772.5.

INTRODUCTION

Early animal studies showed that high dietary saturated fat and cholesterol intakes led to increased plasma cholesterol concentrations as well as atherosclerotic lesions (1). These findings were supported by associations in humans in which dietary saturated fat correlated with coronary heart disease (CHD) risk (2, 3). More recent epidemiologic studies have shown a positive (4–10), inverse (11, 12), or no (4, 13–18) associations of dietary saturated fat with CVD morbidity and/or mortality.

A limited number of randomized clinical interventions have been conducted that have evaluated the effects of saturated fat on risk of CVD. Where as some studies have shown beneficial effects of reduced dietary saturated fat (19–21), others have shown no effects of such diets on CVD risk (22, 23). The studies that showed beneficial effects of diets reduced in saturated fat replaced saturated fat with polyunsaturated fat, with the implication that the CVD benefit observed could have been due to an increase in polyunsaturated fat or in the ratio of polyunsaturated

fat to saturated fat (P:S), a hypothesis supported by a recent pooling analysis conducted by Jakobsen et al (24).

The goal of this study was to conduct a meta-analysis of well-designed prospective epidemiologic studies to estimate the risk of CHD and stroke and a composite risk score for both CHD and stroke, or total cardiovascular disease (CVD), that was associated with increased dietary intakes of saturated fat. Large prospective cohort studies can provide statistical power to adjust for covariates, thereby enabling the evaluation of the effects of a specific nutrient on disease risk. However, such studies have caveats, including a reliance on nutritional assessment methods whose validity and reliability may vary (25), the assumption that diets remain similar over the long term (26) and variable adjustment for covariates by different investigators. Nonetheless, a summary evaluation of the epidemiologic evidence to date provides important information as to the basis for relating dietary saturated fat to CVD risk.

SUBJECTS AND METHODS

Study selection

Two investigators (QS and PS-T) independently conducted a systematic literature search of the MEDLINE (<http://www.ncbi.nlm.nih.gov/pubmed/>) and EMBASE (<http://www.embase.com>) databases through 17 September 2009 by using the following search terms: (“saturated fat” or “dietary fat”) and (“coronary” or “cardiovascular” or “stroke”) and (“cohort” or “follow up”).

¹From the Children’s Hospital Oakland Research Institute, Oakland, CA (PWS-T and RMK), and the Department of Nutrition (QS and PHT) and Epidemiology (PHT), Harvard School of Public Health, Boston, MA.

²PWS-T and QS contributed equally to this work.

³The contents of this article are solely the responsibility of the authors and do not necessarily represent the official view of the National Center for Research Resources (<http://www.ncrr.nih.gov>) or the National Institutes of Health.

⁴Supported by the National Dairy Council (PWS-T and RMK) and made possible by grant UL1 RR024131-01 from the National Center for Research Resources, a component of the National Institutes of Health (NIH), and NIH Roadmap for Medical Research (PWS-T and RMK). QS was supported by a Postdoctoral Fellowship from Unilever Corporate Research. PHT was supported by NIH grant HL60712.

⁵Address correspondence to RM Krauss, Children’s Hospital Oakland Research Institute, 5700 Martin Luther King Junior Way, Oakland, CA 94609. E-mail: rkrauss@chori.org.

Received March 6, 2009. Accepted for publication November 25, 2009. doi: 10.3945/ajcn.2009.2772.5.

Chowdhury et al 2014

REVIEW

Annals of Internal Medicine

Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk

A Systematic Review and Meta-analysis

Rajiv Chowdhury, MD, PhD; Samantha Wamukala, MPH¹; Setor Kunutsor, MD, MSc²; Francesca Crowe, PhD; Heather A. Ward, PhD; Laura Johnson, PhD; Oscar H. Franco, MD, PhD; Adam S. Butterworth, PhD; Nita G. Forouhi, MRCP, PhD; Simon G. Thompson, FMedSci; Kay-Teo Khaw, FMedSci; Darush Mozaffarian, MD, DrPH; John Danesh, FRCP³; and Emanuele Di Angelantonio, MD, PhD⁴

Background: Guidelines advocate changes in fatty acid consumption to promote cardiovascular health.

Purpose: To summarize evidence about associations between fatty acids and coronary disease.

Data Sources: MEDLINE, Science Citation Index, and Cochrane Central Register of Controlled Trials through July 2013.

Study Selection: Prospective, observational studies and randomized, controlled trials.

Data Extraction: Investigators extracted data about study characteristics and assessed study biases.

Data Synthesis: There were 32 observational studies (530 525 participants) of fatty acids from dietary intake; 17 observational studies (25 721 participants) of fatty acid biomarkers; and 27 randomized, controlled trials (103 052 participants) of fatty acid supplementation. In observational studies, relative risks for coronary disease were 1.02 (95% CI, 0.97 to 1.07) for saturated, 0.99 (CI, 0.89 to 1.09) for monounsaturated, 0.93 (CI, 0.84 to 1.02) for long-chain ω -3 polyunsaturated, 1.01 (CI, 0.96 to 1.07) for ω -6 polyunsaturated, and 1.16 (CI, 1.06 to 1.27) for trans fatty acids when the top and bottom thirds of baseline dietary fatty acid intake were compared. Corresponding estimates for circulating fatty acids

were 1.06 (CI, 0.86 to 1.30), 1.06 (CI, 0.97 to 1.17), 0.84 (CI, 0.63 to 1.11), 0.94 (CI, 0.84 to 1.06), and 1.05 (CI, 0.76 to 1.44), respectively. There was heterogeneity of the associations among individual circulating fatty acids and coronary disease. In randomized, controlled trials, relative risks for coronary disease were 0.97 (CI, 0.69 to 1.36) for α -linolenic, 0.94 (CI, 0.86 to 1.03) for long-chain ω -3 polyunsaturated, and 0.89 (CI, 0.71 to 1.12) for ω -6 polyunsaturated fatty acid supplementations.

Limitation: Potential biases from preferential publication and selective reporting.

Conclusion: Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats.

Primary Funding Source: British Heart Foundation, Medical Research Council, Cambridge National Institute for Health Research Biomedical Research Centre, and Gates Cambridge.

Ann Intern Med 2014;160:398–406.

www.annals.org

For author affiliations, see end of text.

*Ms. Wamukala and Dr. Kunutsor contributed equally to this work.

†Danesh and Di Angelantonio also contributed equally to this work.

Dietary fats mainly comprise triacylglycerols consisting of 3 individual fatty acids, each linked by an ester bond to a glycerol backbone (1, 2). Based on the number of double bonds they contain, fatty acids are classified as saturated, monounsaturated, or polyunsaturated. Specific fatty acids within these categories tend to have different biological effects and physical properties (3). Nutritional guidelines generally encourage low consumption of saturated fats, high consumption of ω -3 polyunsaturated fatty acids from fish or plant sources, and avoidance of trans fats, particularly those from partially hydrogenated fat, to promote cardiovascular health (4, 5). However, there is considerable variation in international guidelines about optimum amounts and types of fatty acid consumption (6–11). This variation reflects, at least in part, uncertainties in

the available evidence. For example, prospective observational studies have questioned whether there really are associations between saturated fat consumption and cardiovascular disease (12). Interpretation has been complicated by potential misclassification in the self-report questionnaires used to assess fatty acid consumption (13–15), which also lack the ability to compute intake of specific fatty acids (16). In contrast, fatty acid biomarkers may provide more accurate assessment of consumption, such as for polyunsaturated fatty acids (17), and of metabolism, such as for saturated and monounsaturated fatty acids (17–20). However, earlier analyses have generally not assessed the consistency between findings from dietary self-report and biomarker measures of fatty acids in relation to coronary disease. With respect to randomized trials of fatty acid supplements for preventing coronary disease, interpretation of results has been complicated by the differences in dietary habits of various trial populations, the absence or presence (and type) of preexisting vascular disease at entry, the composition of supplementation regimens, trial duration and power, and apparent differences in reported efficacy for coronary prevention. Furthermore, previous meta-analyses of randomized trials were only focused on ω -3 and ω -6

See also:

Web-Only
Supplements
CME quiz

Obě metaanalýzy nesledovaly skladbu stravy

- Důležitý celkový příjem tuků a jeho složení
- Vedle nasycených mastných kyselin konzumujeme různé množství
 - polynenasycených mastných kyselin
 - sacharidů
 - přidaných cukrů
 - komplexních sacharidů s vysokým podílem vlákniny
- Některé potraviny obsahují složky mající synergický nebo antagonický vliv na některé rizikové faktory

Leden 2010 SAFA nemají vliv na KVO

Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease¹⁻⁵

Patty W Siri-Tarino, Qi Sun, Frank B Hu, and Ronald M Krauss

ABSTRACT

Background: A reduction in dietary saturated fat has generally been thought to improve cardiovascular health.

Objective: The objective of this meta-analysis was to summarize the evidence related to the association of dietary saturated fat with risk of coronary heart disease (CHD), stroke, and cardiovascular disease (CVD; CHD inclusive of stroke) in prospective epidemiologic studies.

Design: Twenty-one studies identified by searching MEDLINE and EMBASE databases and secondary referencing qualified for inclusion in this study. A random-effects model was used to derive composite relative risk estimates for CHD, stroke, and CVD.

Results: During 5–23 y of follow-up of 347,747 subjects, 11,006 developed CHD or stroke. Intake of saturated fat was not associated with an increased risk of CHD, stroke, or CVD. The pooled relative risk estimates that compared extreme quantiles of saturated fat intake were 1.07 (95% CI: 0.96, 1.19; $P = 0.22$) for CHD, 0.81 (95% CI: 0.62, 1.05; $P = 0.11$) for stroke, and 1.00 (95% CI: 0.89, 1.11; $P = 0.95$) for CVD. Consideration of age, sex, and study quality did not change the results.

Conclusions: A meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD. More data are needed to elucidate whether CVD risks are likely to be influenced by the specific nutrients used to replace saturated fat. *Am J Clin Nutr* doi: 10.3945/ajcn.2009.2772.5.

INTRODUCTION

Early animal studies showed that high dietary saturated fat and cholesterol intakes led to increased plasma cholesterol concentrations as well as atherosclerotic lesions (1). These findings were supported by associations in humans in which dietary saturated fat correlated with coronary heart disease (CHD) risk (2,3). More recent epidemiologic studies have shown a positive (4–10), inverse (11,12), or no (4,13–18) associations of dietary saturated fat with CVD morbidity and/or mortality.

A limited number of randomized clinical interventions have been conducted that have evaluated the effects of saturated fat on risk of CVD. Where as some studies have shown beneficial effects of reduced dietary saturated fat (19–21), others have shown no effects of such diets on CVD risk (22,23). The studies that showed beneficial effects of diets reduced in saturated fat replaced saturated fat with polyunsaturated fat, with the implication that the CVD benefit observed could have been due to an increase in polyunsaturated fat or in the ratio of polyunsaturated

fat to saturated fat (P:S), a hypothesis supported by a recent pooling analysis conducted by Jakobsen et al (24).

The goal of this study was to conduct a meta-analysis of well-designed prospective epidemiologic studies to estimate the risk of CHD and stroke and a composite risk score for both CHD and stroke, or total cardiovascular disease (CVD), that was associated with increased dietary intakes of saturated fat. Large prospective cohort studies can provide statistical power to adjust for covariates, thereby enabling the evaluation of the effects of a specific nutrient on disease risk. However, such studies have caveats, including a reliance on nutritional assessment methods whose validity and reliability may vary (25), the assumption that diets remain similar over the long term (26) and variable adjustment for covariates by different investigators. Nonetheless, a summary evaluation of the epidemiologic evidence to date provides important information as to the basis for relating dietary saturated fat to CVD risk.

SUBJECTS AND METHODS

Study selection

Two investigators (QS and PS-T) independently conducted a systematic literature search of the MEDLINE (<http://www.ncbi.nlm.nih.gov/pubmed/>) and EMBASE (<http://www.embase.com>) databases through 17 September 2009 by using the following search terms: ("saturated fat" or "dietary fat") and ("coronary" or "cardiovascular" or "stroke") and ("cohort" or "follow up").

¹From the Children's Hospital Oakland Research Institute, Oakland, CA (PWS-T and RMK), and the Department of Nutrition (QS and PS-T) and Epidemiology (PS-T), Harvard School of Public Health, Boston, MA.

²PWS-T and QS contributed equally to this work.

³The contents of this article are solely the responsibility of the authors and do not necessarily represent the official view of the National Center for Research Resources (<http://www.ncrr.nih.gov>) or the National Institutes of Health.

⁴Supported by the National Dairy Council (PWS-T and RMK) and made possible by grant UL1 RR024131-01 from the National Center for Research Resources, a component of the National Institutes of Health (NIH), and NIH Roadmap for Medical Research (PWS-T and RMK). QS was supported by a Postdoctoral Fellowship from Unilever Corporate Research. PS-T was supported by NIH grant HL60712.

⁵Address correspondence to RM Krauss, Children's Hospital Oakland Research Institute, 5700 Martin Luther King Junior Way, Oakland, CA 94609. E-mail: rkrauss@chori.org.

Received March 6, 2009. Accepted for publication November 25, 2009. doi: 10.3945/ajcn.2009.2772.5.

Carbohydrate Rep (2010) 12:934–939
DOI 10.1093/ajcn/83-5-934-939

Saturated Fatty Acids and Risk of Coronary Heart Disease: Modulation by Replacement Nutrients

Patty W. Siri-Tarino • Qi Sun • Frank B. Hu • Ronald M. Krauss

Published online: 14 August 2010
© The Author(s) 2010. This article is published with open access at [Springerlink.com](http://springerlink.com)

Abstract Despite the well-established observation that substitution of saturated fats for carbohydrates or unsaturated fats increases low-density lipoprotein (LDL) cholesterol in humans and animal models, the relationship of saturated fat intake to risk for atherosclerotic cardiovascular disease in humans remains controversial. A critical question is what macronutrient should be used to replace saturated fat. Substituting polyunsaturated fat for saturated fat reduces LDL cholesterol and the total cholesterol to high-density lipoprotein cholesterol ratio. However, replacement of saturated fat by carbohydrates, particularly refined carbohydrates and added sugars, increases levels of triglyceride and small LDL particles and reduces high-density lipoprotein cholesterol effects that are of particular concern in the context of the increased prevalence of obesity and insulin resistance. Epidemiologic studies and

randomized clinical trials have provided consistent evidence that replacing saturated fat with polyunsaturated fat, but not carbohydrates, is beneficial for coronary heart disease. Therefore, dietary recommendations should emphasize substitution of polyunsaturated fat and minimally processed grains for saturated fat.

Keywords Saturated fat • Polyunsaturated fat • Carbohydrate • Monounsaturated fat • Atherosclerosis • Coronary heart disease • Cardiovascular disease • LDL cholesterol • Obesity • Insulin resistance • HDL cholesterol • Triacylglycerol • Triglyceride • Diet • Insulin resistance • Epidemiology • Prospective cohort studies • Randomized controlled trials • Lipids • Weight loss • Nutrient replacement • Clinical studies • Dyslipidemia • Meta-analysis

P. W. Siri-Tarino • R. M. Krauss (✉)
Atherosclerosis Research,
Children's Hospital Oakland Research Institute,
5700 Martin Luther King Junior Way,
Oakland, CA 94609, USA
e-mail: rkrauss@chori.org

P. W. Siri-Tarino
e-mail: psiri@chori.org

Q. Sun
Channing Laboratory, Department of Medicine,
Brigham and Women's Hospital and Harvard Medical School,
181 Longwood Avenue,
Boston, MA 02115, USA
e-mail: qsun@hsph.harvard.edu

F. B. Hu
Departments of Nutrition and Epidemiology,
Harvard School of Public Health,
665 Huntington Avenue,
Boston, MA, USA
e-mail: fhu@hsph.harvard.edu

Introduction

Dietary saturated fat intake has been shown to increase low-density lipoprotein (LDL) cholesterol, and therefore has been associated with increased risk of cardiovascular disease (CVD). This evidence, coupled with inferences from epidemiologic studies and clinical trials, has led to longstanding public health recommendations for limiting saturated fat intake as a means of preventing CVD. However, the relationship between saturated fat and CVD risk remains controversial, due at least in part to the intrinsic limitations of clinical studies that have evaluated this relationship.

Prospective epidemiologic studies have been limited by a reliance on nutritional assessment methods of varying accuracy, the assumption that diets remain the same over the long term, and incomplete adjustments for relevant confounders, including other nutrients. Randomized clinical

Srpen 2010 pozitivní vliv záměny SAFA → PUFA

Nebývalé množství kritiky předních světových odborníků k této studii

Annals of Internal Medicine

COMMENTS AND RESPONSE

Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk

TO THE EDITOR: We appreciate that Chowdhury and colleagues (1) have corrected some of the gross errors in their original paper. Of note, the inverse association of intake of long-chain ω -3 polyunsaturated fatty acids (PUFAs) with cardiovascular disease (CVD) risk is now significant. We also appreciate the sensitivity analysis showing that with exclusion of the outlying SDHS (Sydney Diet Heart Study), the included randomized, controlled trials (RCTs) show benefit of replacing saturated fatty acids (SFAs) with PUFAs. The extreme diet used in that study was never recommended or consumed in the United States. It included a trans fat-based margarine and probably very little ω -3 PUFAs, because sunflower oil was used to replace other fats as much as possible. However, other serious problems with Chowdhury and colleagues' analysis remain. They report that the nonsignificant findings for biomarkers of long-chain ω -3 fatty acid intake are based on total long-chain ω -3 PUFAs in only 4 studies. However, in the Supplemental Tables, long-chain ω -3 PUFAs were actually examined in 13 studies, and findings for the specific long-chain PUFAs (icosapentanoic and docosahexanoic acids) were robustly and significantly inverse. Thus, the results for both intake and biomarkers for long-chain ω -3 fatty acids support benefit. Although the findings for RCTs vary, these results would be expected because many of the populations studied had relatively high intake of ω -3 fatty acids and most individuals would likely experience little benefit.

The analysis for ω -6 PUFAs still includes only 8 studies and omits others included in Jacobsen and coauthors' (2) pooled analysis of original data and other published papers. The data on intake of ω -6 PUFAs from the Kuopio Heart Study (3), the study with the most positive association, are erroneous because the denominator is almost double the number of healthy participants. Contrary to what Chowdhury and colleagues state, they apparently included persons with prevalent CVD at baseline instead of limiting the analysis to healthy persons. The original study reported a relative risk (RR) of 0.38 (95% CI, 0.20 to 0.70) for fatal CVD among those with higher intake of PUFAs.

Chowdhury and colleagues still do not acknowledge the earlier pooled analysis of primary data based on a larger number of studies, which allowed direct comparisons among different types of fat. In that analysis, substitution of SFAs with PUFAs was associated with lower risks for coronary heart disease (CHD) (2). The large body of data showing that replacing SFAs with monounsaturated fatty acids (MUFAs) or PUFAs reduce low-density lipoprotein (LDL) cholesterol is still not recognized.

Although Chowdhury and colleagues say that their conclusions did not change, a more inclusive and correct review of available evidence would support the replacement of SFAs with PUFAs.

Walter C. Willett, MD, DrPH
Meir J. Stampfer, MD, DrPH
Frank M. Sacks, MD
Harvard School of Public Health
Boston, Massachusetts

LETTERS

DISCLOSURES: Authors have disclosed no conflicts of interest. Forms can be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=114-0319.

References

1. Chowdhury R, Warnakula S, Kunjathoor S, Crowe F, Whitt EA, Johnson L, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med*. 2014;160:398-406. [PMID: 24728179] doi:10.7554/AM.11788
2. Jacobsen ML, O'Rahilly EJ, Holman RL, Perin MA, Miller K, Fraser GE, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *Am J Clin Nutr*. 2009;89:342S-52. [PMID: 19211817] doi:10.3945/ajcn.2008.27124
3. Laksonen DE, Nyyssonen K, Niskanen L, Kainanen TA, Salonen JT. Prediction of cardiovascular mortality in middle-aged men by dietary and serum linoleic and polyunsaturated fatty acids. *Arch Intern Med*. 2005;165:195-9. [PMID: 1566856]

TO THE EDITOR: Chowdhury and colleagues (1) analyzed 8 studies to assess the association between circulating blood levels of SFAs and RR for coronary outcomes (1). From our point of view, the results of NSHDs (Northern Sweden Health and Disease Study) and VIP (Västerbotten Intervention Program) have been misinterpreted, and the studies should be excluded for the following reasons.

First, data from VIP (2, 3) have been included in the evaluation of NSHDs results (4). Second, VIP and NSHDs assessed the association between high intake of SFAs from dairy products (indicated by pentadecanoic acid [C15:0] and heptadecanoic acid [C17:0] or their sum in serum lipid esters) and CVD (3, 4). In both studies, negative associations between milk fat intake and first-ever myocardial infarction were found. Neither of the 2 studies described the impact of total circulating blood levels of SFA on coronary outcomes. Of note, C15:0 and C17:0 contribute only 0.9% to 1.0% of the fatty acids in total phospholipid levels (4). In contrast, the total SFA level in plasma phospholipids ranges between 40% and 49%, which is mainly composed of palmitic acid (C16:0) and stearic acid (C18:0) with approximately 50% to 60% and 30% to 40% of the total SFA level, respectively (5). Thus, C15:0 and C17:0 are markers for milk or ruminant fat intake (3, 4) but not for total SFA intake. However, there are several SFA sources, such as baking margarine, coconut oil, and palm oil, that do not contain C15:0 and C17:0. In agreement with this, we also found that proportions of C15:0 and C17:0 in human erythrocyte membranes are between 1.0% to 2.9% of total SFA levels and show no correlation with the concentration of total SFAs (ClinicalTrials.gov: NCT01437930 and NCT01742468). When we repeated the meta-analysis after excluding VIP and NSHDs results, we found a positive association of total SFA blood levels and coronary outcomes (RR, 1.21 [CI, 1.04 to 1.40]). This finding contradicts the overall conclusion drawn by Chowdhury and colleagues (1).

Proper communication of the health risks associated with dietary habits is essential to achieve appropriate lifestyle changes and improve cardiovascular health. The results of the meta-analysis gave rise to misleading headlines, like "Animal fat is not bad for the heart" (6), in the national lay press. Consumers may continue their unhealthy dietary habits in response to such simplified messages. Because of the impact of meta-analysis on the general public, thoroughly and reasonably selection of studies and careful evaluation of data are vital to reporting accurate results and protecting people from harm.

ACP AMERICAN COLLEGE OF PHYSICIANS
INTERNAL MEDICINE | *Journal for Adults*

Annals of Internal Medicine

Reviews | 18 March 2014

Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk: A Systematic Review and Meta-analysis

Rajiv Chowdhury, MD, PhD
Samantha Warnakula, MPhil
Seter Kunjathoor, MD, MS^{1*}
Francesca Crowe, PhD
Heather A. Ward, PhD
Laura Johnson, PhD
Oscar H. Franco, MD, PhD
Adam S. Butterworth, PhD
Nita G. Forouhi, MRCP, PhD
Simon G. Thompson, FMedSci
Kay-Tee Khaw, FMedSci
Dariusz Mozaffarian, MD, DrPH
John Danesh, FRCP^{2*}
Emanuele Di Angelantonio, MD, PhD^{3*}

Background: Guidelines advocate changes in fatty acid consumption to promote cardiovascular health.

Purpose: To summarize evidence about associations between fatty acids and coronary disease.

Data Sources: MEDLINE, Science Citation Index, and Cochrane Central Register of Controlled Trials through July 2013.

Study Selection: Prospective, observational studies and randomized, controlled trials.

Data Extraction: Investigators extracted data about study characteristics and assessed study biases.

Data Synthesis: There were 32 observational studies (512 420 participants) of fatty acids from dietary intake; 17 observational studies (25 721 participants) of fatty acid biomarkers; and 27 randomized, controlled trials (105 085 participants) of fatty acid supplementation. In observational studies, relative risks for coronary disease were 1.03 (95% CI, 0.98 to 1.07) for saturated, 1.00 (CI, 0.91 to 1.10) for monounsaturated, 0.87 (CI, 0.78 to 0.97) for long-chain ω -3 polyunsaturated, 0.98 (CI, 0.90 to 1.06) for ω -6 polyunsaturated, and 1.16 (CI, 1.06 to 1.27) for trans fatty acids when the top and bottom thirds of baseline dietary fatty acid intake were compared. Corresponding

http://wphna.org/wp-content/uploads/2014/08/2014-03_Annals_of_Int_Med_Chowdhury_et_al_Fat_and_CHD_+_responses.pdf

Cílené záměny živin



jednoduché cukry
vysoký glykemický index

zvýšení hladiny triglyceridů, snížení
hladiny HDL-cholesterolu
zvýšení poměru denzních LDL-částic

nasycené mastné kyseliny

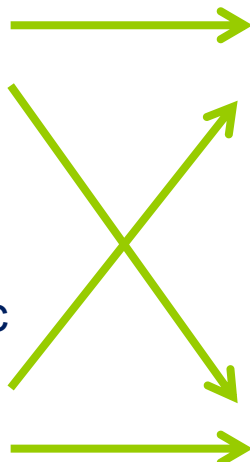
snížení počtu LDL-receptorů, zvýšení
hladiny LDL-cholesterolu

**komplexní sacharidy +
vláknina**
nízký glykemický index

snížení vstřebávání žlučových
kyselin, snížení hladiny
LDL-cholesterolu

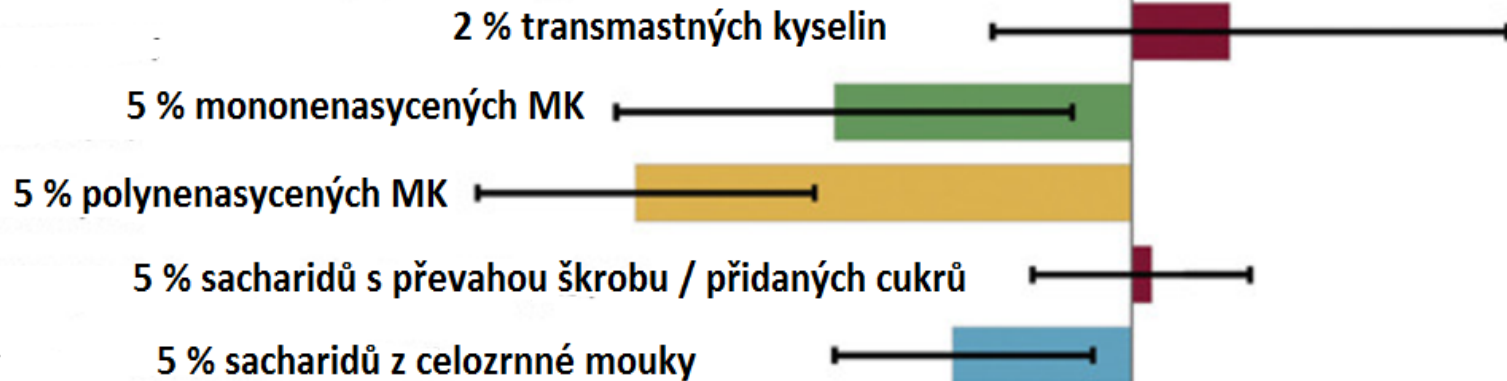
**nenasycené mastné
kyseliny**

zvýšení počtu LDL-receptorů, snížení
hladiny LDL-cholesterolu

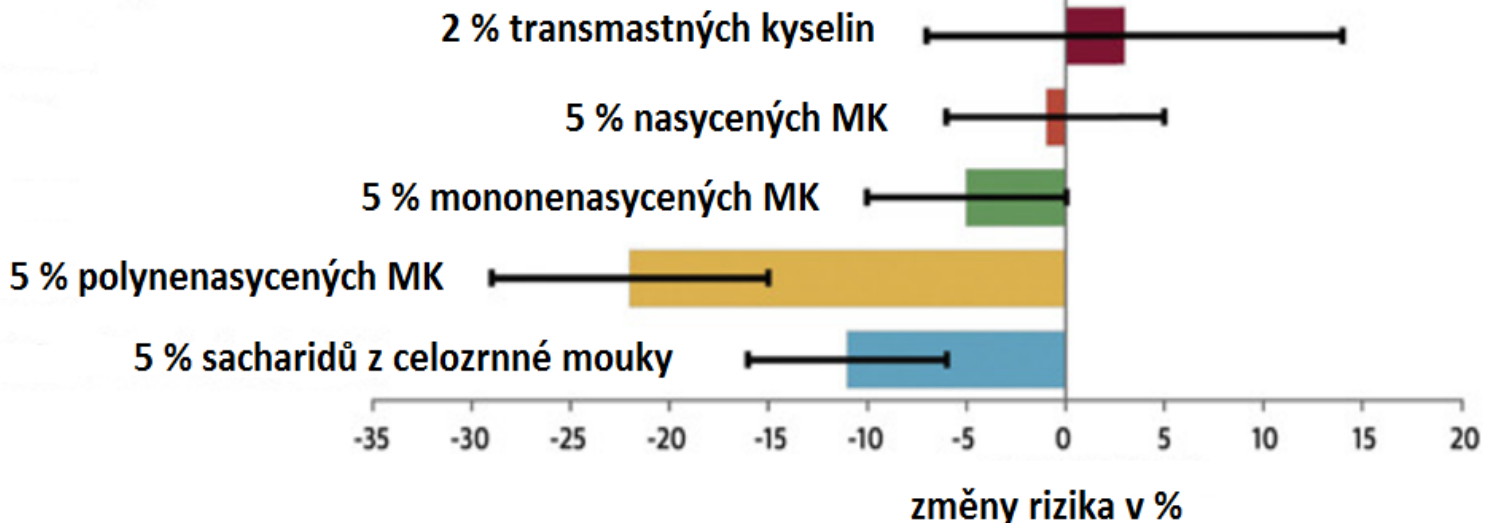


Změna rizika vzniku ICHS při substituci živin

Místo stejného množství energie dodaného prostřednictvím nasycených mastných kyselin



Místo stejného množství energie dodaného prostřednictvím sacharidů na bázi škrobu / přidaných cukrů



Spotřeba tuků a olejů v roce 2013

	spotřeba kg/os./rok	SAFA* g/den	SAFA % z RHP**
komodita oleje a tuky			
máslo v hodnotě čistého tuku	4,1	7,4	37,1
sádlo v hodnotě čistého tuku	3,9	4,4	21,9
pokrmový tuk	3,3	5,1	25,3
jedlé oleje	9,9	2,7	13,6
rostlinné tuky v čisté hodnotě tuku*	2,7	2,6	12,9
Součet		22,2	110,8

*výpočet z rostlinných tuků a olejů (15,9 g) v čisté hodnotě (pokrmový tuk a oleje brány jako stoprocentní)
**referenční hodnota příjmu pro SAFA 20 g/den

84,3%

Více než 4/5 tolerovaného denního příjmu pro SAFA je vyčerpáno tuky s nepříznivým složením mastných kyselin (máslo, sádlo, pokrmový tuk)

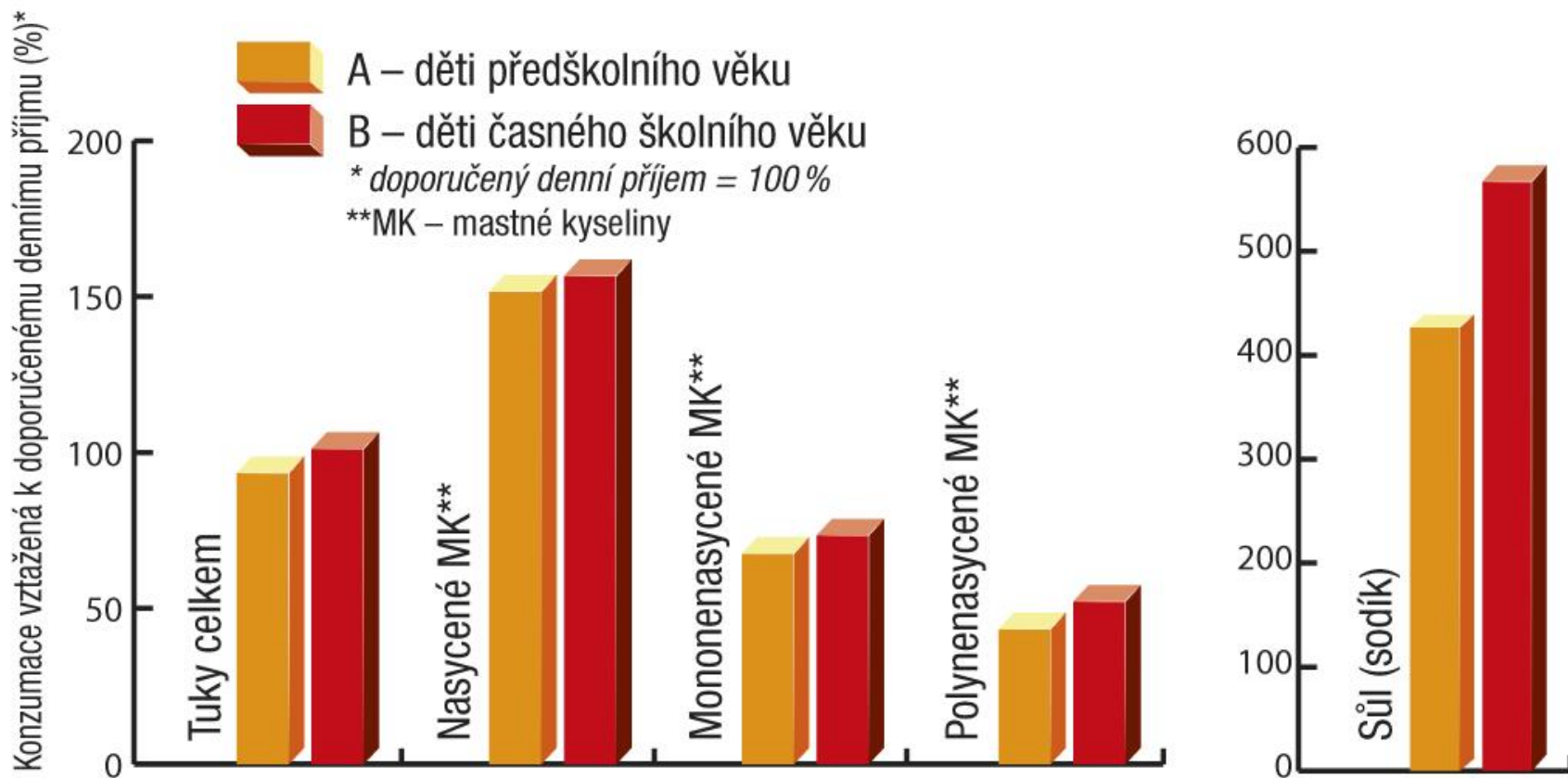
Velký potenciál pro záměnu tuků

Další tuky i nasycené mastné kyseliny konzumujeme jako skryté (maso, mléčné výrobky apod.)

Špatná skladba tuků i dětském věku

Konzumace vybraných živin v rámci dětské populace

Vychází z průzkumu stravovacích zvyklostí na vybraných školách.



Příkladné složení tuků a olejů

Tuk / olej	SAFA	TFA	MUFA	ω 3 PUFA	ω 6 PUFA	Bod tání
Řepkový olej	8	1	61	9	20	-10
Slunečnicový olej	12	1	25,5	0,5	61	-17
Lněný olej	11	1	18	53	17	-24
Sójový olej	16	1	23	7	53	-16
Olivový olej	15	0	75	1	9	-6
Palmový olej	50	0,5	40	0	9,5	35
Palmojádrový tuk	82	0	14	0	4	24
Kokosový tuk	90	0	7	0	3	25
Vepřové sádlo	41	1	49	1	8	41
Mléčný tuk	67,5	2,5	27	0,5	1,5	32-35
Hovězí lůj	50	4,5	40	0,5	5	35-40
Kuřecí tuk	41	1	37	1	20	35
Rybí tuk	28	0	52	15	5	-70 až 15
Kakaové máslo	60	0	38	0	2	34

Pravidlo 1/3

tuky 30 – 35 % z celkového příjmu energie

nasyčené MK méně než 10 % z celkového příjmu energie

=

přednostní konzumace s obsahem nasycených MK méně než jedna třetina

	Konzumujte přednostně							Konzumujte umírněně					Konzumujte velmi střídavě		
Tuk olej	Řepkový olej	Slunečnicový olej	Olivový olej	Sojový olej	Rybí tuk	Běžný kvalitní margarín		Běžný kvalitní rostlinný tuk na pečení	Vepřové sádlo	Kuřecí tuk	Palmový olej	Hovězí lůj	Kakaové máslo	Máslo a mléčný tuk	Kokosový tuk
obsah SAFA z celkového obsahu tuku (%)	8	12	15	16	28	30	30	40	41	41	50	50	60	67	90

Infografika AHA - Hodný, zlý a ošklivý

Blondák Joe (Hodný)
Clint Eastwood



Zabiják Sentenza (Zlý)
Lee Van Cleef



Bandita Tuco (Ošklivý)
Eli Wallach



✓ GOOD

Monounsaturated & Polyunsaturated Fats

- Can lower bad cholesterol levels
- Can lower risk of heart disease & stroke
- Can provide essential fats that your body needs but can't produce itself

SOURCE

Plant-based liquid oils, nuts, seeds and fatty fish

✗ BAD

Saturated Fats

- Can raise bad cholesterol levels
- Can lower good cholesterol levels
- Can increase risk of heart disease & stroke

SOURCE

Most saturated fats come from animal sources, including meat and dairy, and from tropical oils

✗ UGLY

Hydrogenated Oils & Trans Fats

- Can raise bad cholesterol levels
- Can lower good cholesterol levels
- Can increase risk of heart disease & stroke
- Can increase risk of type 2 diabetes

SOURCE

Processed foods made with partially hydrogenated oils

American Heart Association Recommendation

Eat a healthy dietary pattern that:

Includes good fats

Limits saturated fats

Keeps trans fats as LOW as possible

Kde v ČR nacházíme transmastné kyseliny a kde ne

- transmastné kyseliny můžeme najít

- v jemném a trvanlivém pečivu
- sušených sójových nápojích
- nečokoládových cukrovinkách
- tukových polevách
- čokoládových „pochoutkách“

ošklivý



- částečně ztužené tuky obsahující transmastné kyseliny se dnes nepoužívají

- při výrobě margarínů
- při smažení pokrmů ve velkých provozovnách rychlého občerstvení

Změny stravovacích návyků snížily mortalitu v ČR

Tab. 2. Spotřeba vybraných potravin v České republice a na Slovensku (kg/osoba/rok). Upraveno podle [12,13]

potravina	Česká republika			Slovensko		
	1990	2000	2012	1990	2000	2012
rostlinné oleje a tuky	12,8	16,3	16,4	11,9	17,8	15
máslo	8,7	4,1	5,2	6,4	2,7	3
sádlo	6,9	4,8	4,7	6,9	3,3	3,9
hovězí maso	28	12,3	9,4	21,8	9,1	3,6
vepřové maso	50	40,9	41,6	44,5	33,1	30
drůbeží maso	13,6	22,3	24,5	15,2	17,4	17,7
jižní ovoce	14,9	27,5	31,2	13,6	22,4	25,8

Změny stravovacích návyků ve vztahu k rizikovým faktorům a kardiovaskulární mortalitě

Jiří Brát¹, Michal Vrablík², Otto Herber³

¹ Vím, co jím a piju o.p.s., ředitelka Lucie Gonzálezová
² III. interní klinika 1. LF UK a VFN Praha, přednostka prof. MUDr. Štěpán Svoboda, DrSc., MBA
³ Ústav všeobecného lékařství 1. LF UK Praha, přednostka doc. MUDr. Bohumil Seifert, Ph.D.

Souhrn
 Evropa je regionem s nejvyšším výskytem neinfekčních onemocnění hromadného výskytu. Kardiovaskulární onemocnění patří mezi hlavní příčiny nemocnosti a úmrtnosti. Interakce mezi skladbou stravy, životním stylem a metabolismem lipidů významně ovlivňují rozvoj aterosklerózy a jejích komplikací. Mortalita na ischemickou chorobu srdeční (ICHS) se v poslední dekádě 20. století výrazně snížila. Významné a pozitivní změny ve stravovacích návycích, zejména snížení příjmu nasycených mastných kyselin a jejich záměna za polyenesycené, přispěly ke snížení sérové hladiny cholesterolu v populaci. Pokles mortality na ICHS významně souvisel s vývojem tohoto rizikového faktoru, který nelze vysvětlit rozvojem farmakoterapie. Česká republika není jedinou zemí, v níž byly podobné změny stravovacích návyků ve vztahu k úmrtnosti na ICHS zaznamenány. Zlepšení stravovacích návyků se zdá být jednou z nejdůležitějších strategií v prevenci kardiovaskulárních onemocnění.

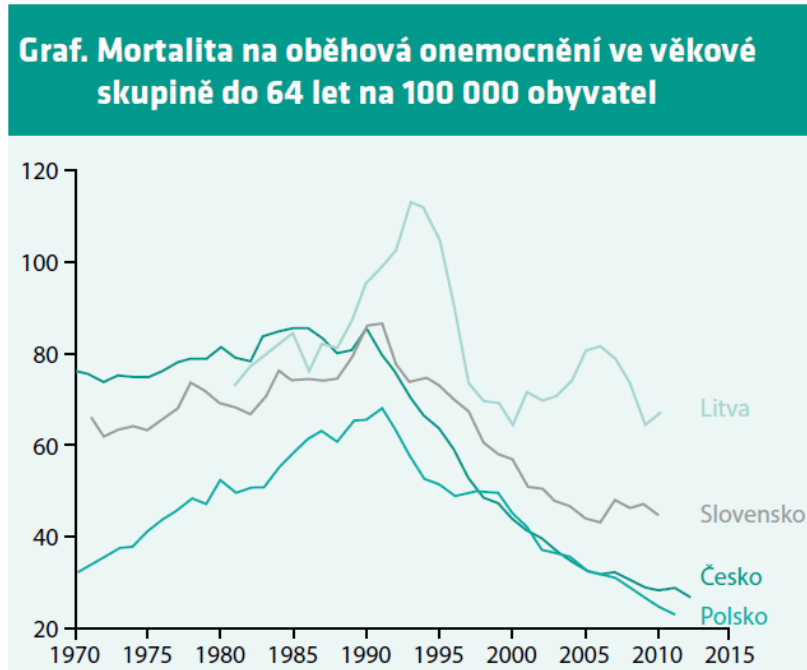
Klíčová slova: ischemická choroba srdeční – mortalita – nasycené mastné kyseliny – nenasyčené mastné kyseliny – stravovací návyky

Dietary changes in relationship to risk factors and coronary heart disease mortality

Summary
 Europe is a region, which is most severely affected by non-communicable diseases. Cardiovascular disease is the leading cause of disability and death. Complex interactions between diet, lifestyle, and lipoprotein metabolism significantly contribute to the development of atherosclerosis and its complications. Role of diet in prevention of coronary heart disease becomes sometime underestimated in comparison with pharmacological treatment. Coronary heart disease (CHD) mortality has declined substantially in the Czech Republic in the last decade of the 20th century. Significant and positive dietary changes, mainly reduction of saturated fatty acids intake and its replacement by polyunsaturated fatty acids, contributed to decline of the average serum cholesterol level in population. Fall in CHD mortality was attributable to reduction in this major cardiovascular risk factor and not driven by pharmacological intervention. The Czech Republic is not the only country where similar trends have been recorded. Improvements in dietary habits seem to be one of the most important strategies in cardiovascular disease prevention.

Key words: coronary heart disease – coronary heart disease mortality – dietary habits – cholesterol – polyunsaturated fatty acids – saturated fatty acids

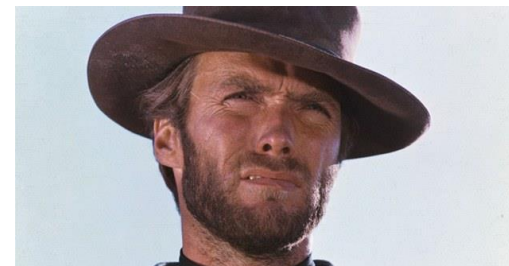
Úvod
 Nové milénium je charakterizováno nárůstem výskytu neinfekčních onemocnění hromadného výskytu. Incidence nadváhy a obezity zvláště v mužské populaci, stejně jako diabetu 2. typu, roste dramatickým způsobem a řada předpovědí do budoucna varuje před ještě zhoršujícím se trendem. Mortalita z důvodu kardiovaskulárních onemocnění (KVO) se podílí na celkové úmrtnosti zhruba polovinou. Příčin je celá řada, ale většina z nich má společného jmenovatele – špatnou, nevyváženou stravu, nevhodný životní styl provázený klesající fyzickou aktivitou, kouřením a nadměrnou konzumací alkoholu. Přitom potenciál snížení výskytu neinfekčních onemocnění hromadného výskytu je relativně vysoký. Přibližně 75 % KVO, která mají nejvyšší proporcí zastoupení mezi neinfekčními onemocněními, vzniká v souvislosti s nejčastějšími a nejdůležitějšími ovlivnitelnými rizikovými faktory – arteriální hypertenzí, dys-



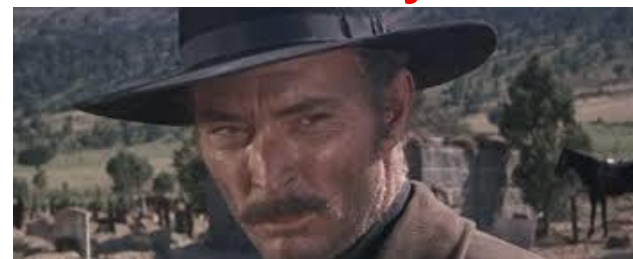
Závěr

- Jezte pestrou vyváženou stravu s vyrovnaným příjmem a výdejem energie, která obsahuje
- dostatečné množství poly- a mononenasycených mastných kyselin
- omezené množství nasycených mastných kyselin
- co nejnížší množství transmastných kyselin

hodný



zlý



ošklivý





**Nízkosacharidová
dieta**

**Špatná skladba tuků,
převaha živočišných,
málo esenciálních
mastných kyselin**

**T 32-33 % en.
S 52-53 % en.
B 15 % en.
< 10 % energ.
nasycených MK
< 10 % energ.
přidaných cukrů**

Nízkotuková dieta

**Hodně jednoduchých
cukrů, málo
komplexních
sacharidů, vlákniny**

Děkuji za pozornost

