Does coconut fat have beneficial effects on blood cholesterol in healthy adults?

- a systematic review

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Abstract

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Background
Coconut fat has gained a lot of attention for its claimed beneficial effects concerning health. Its fat content is made up of approximately 90% saturated fatty acids. The intake of saturated fats affects blood cholesterol levels. Cholesterol can be measured through total cholesterol (TC), Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL), Very Low Density Lipoprotein (VLDL) and LDL/HDL ratio. Research has been done on single fatty acids from coconut fat and positive results regarding health have been reached. However it is important to elucidate that conclusions based on studies made on single fatty acids may differ from coconut fat as a whole.

Objective
The aim of this systematic review was to investigate how a diet rich in coconut fat would affect blood cholesterol levels when it came to TC, LDL, HDL and the LDL/HDL-ratio.

Search strategy
The databases PubMed and Scopus were used in the literary search process for relevant articles. Search words of importance for the systematic reviews' research question were applied.

Selection criteria
RCT, CCT and cohort studies from 1990 to present, both investigating coconut fat vs. other fats as well as coconut fat vs. placebo were considered. Studies including pregnant women, participants using drugs that influenced blood lipid metabolism or studies on single fatty acids were excluded.

Data collection and analysis
Appropriate materials were selected and three final studies were chosen, two RCT and one CCT. The study quality and the strength of evidence of the endpoints were based upon the validation template *Granskningsmall för randomiserade studier, Granskningsmall för observationsstudier och icke-randomiserade kontrollerade studier* and *Sammanfattande Evidensformulär*.

Main results
The test diets rich in coconut fat (SFA diets) led to significantly higher TC and LDL levels compared to diets rich in HUFA (safflower oil, soya-bean oil or a HUFA mix). Only one study showed significantly higher HDL levels in all its subjects after a coconut fat diet. TC and LDL were significantly higher on a butter diet compared to a coconut fat diet (SFA versus SFA). The LDL/HDL ratio was lower after the HUFA mix diet compared to the SFA diets.

Conclusions
Coconut fat as the main fat in the diet significantly increases TC and LDL levels, which is today not considered beneficial regarding blood cholesterol in relation to health. The outcomes on HDL levels however differ between the studies. The LDL/HDL ratio is higher when comparing coconut fat diets with a HUFA diet. The strength of evidence regarding coconut fat's effect on TC, LDL and HDL is measured mediate-high (+++) and for LDL/HDL ratio low (++).

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Sammanfattning

Titel:	Har kokosfett gynnsamma effekter på blodkolesterol hos friska vuxna? - en systematisk översiktsartikel
Författare:	Arghanoon Farhikhtah och Eddie Grahn
Handledare:	Lena Hulthén
Examinator:	Frode Slinde
Linje:	Dietistprogrammet, 180/240 hp
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Bakgrund
Kokosfett har fått mycket uppmärksamhet för sina påstådda positiva hälsoeffekter. Fettsammansättningen i kokos består av cirka 90 % mättade fettsyror. Mättat fett påverkar kolesterolhalten i blodet. Kolesterol kan mätas genom totalkolesterol (TC), Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL), Very Low Density Lipoprotein (VLDL) och kvoten mellan LDL och HDL. Forskning har gjorts på enskilda fettsyror från kokosfett och resultat på gynnsamma hälsoeffekter har visats. Det är dock viktigt att belysa att slutsatser baserade på studier gjorda på enskilda fettsyror kan skilja sig från kokosfett i helhet.

Syfte
Syftet med denna översiktsartikel var att undersöka hur en kost rik på kokosfett påverkar kolesterolhalten i blodet gällande TC, LDL, HDL och LDL/HDL-kvoten.

Sökväg
Databaserna PubMed och Scopus användes i litteratursökningsprocessen för att få fram lämpliga artiklar. Sökord av betydelse för översiktsartikelns frågeställning användes.

Urvalskriterier
RCT, CCT och kohortstudier från 1990 och framåt som undersökte kokosfett vs. andra fetter eller kokosfett vs. placebo inkluderades. Studier med gravida kvinnor samt deltagare som använde läkemedel som påverkade blodfettsmetabolismen eller studier på enskilda fettsyror exkluderades.

Datainsamling och analys
Ett urval av adekvat material gjordes och tre studier valdes, två RCT och en CCT. Studiekvalitén samt graden av effektmåttens evidensstyrka bedömdes utifrån *Granskningsmall för randomiserade studier, Granskningsmall för observationsstudier och icke-randomiserade kontrollerade studier* och *Sammanfattande Evidensformulär*.

Resultat
Testdieterna rika på kokosfett (SFA-kost) gav signifikant högre TC- och LDL-nivåer jämfört med testdieterna rika på HUFA (tistelolja, sojabönsolja eller HUFA-mix). Endast en studie visade på signifikant högre nivåer av HDL i gruppen som helhet efter en kost med kokosfett. TC och LDL var signifikant högre med smör som testfett jämfört med kokosfett (SFA vs. SFA). LDL/HDL-kvoten var lägre efter en HUFA-diet jämfört med SFA-dieterna.

Slutsats
Kokosfett som det dominerande fettet i kosten ger en signifikant ökning av TC och LDL, något som idag anses vara ofördelaktigt gällande blodkolesterolhalten relaterat till hälsan. Effekten på HDL skiljer sig åt mellan studierna. LDL/HDL-kvoten är högre när man jämför en kost rik på kokosfett med en kost rik på HUFA. Evidensstyrkan för kokosfettets påverkan på TC, LDL och HDL bedöms vara måttlig (+++) och för LDL/HDL-kvoten låg (++).

Abbreviations and explanations

ANOVA	- Analysis of Variance
CCT	- Clinical Controlled Trial
CVD	- Cardiovascular Disease
E%	- Energy Percentage
HDL	- High Density Lipoprotein
HSFA	- High Saturated Fatty Acids
HUFA	- High Unsaturated Fatty Acids
LDL	- Low Density Lipoprotein
LSFA	- Low Saturated Fatty Acids
MCT	- Medium-Chain Triglyceride
NNR	- Nordic Nutrition Recommendations
PUFA	- Poly Unsaturated Fatty Acids
RCT	- Randomized Controlled Trial
SFA	- Saturated Fatty Acids
TC	- Total Cholesterol
VCO	- Virgin Coconut Oil/Fat
VLDL	- Very Low Density Lipoprotein
WC	- Waist Circumference

Analysis of Variance (*ANOVA*)	- Provides a statistical test showing if the means of more than two groups are equal or not
Bias	- Method error
Bonferroni	- Statistical analysis method used to face the issue of multiple comparisons
Diet record	- Retrospective food journal, often used as a dietary assessment method
Dyslipidemia	- A state where the blood lipid levels are too high or too low
Latin-square design	- An experimental design implied to control the randomization in trials
P-value	- Indicates the probability in which a certain outcome would have occurred by chance
Polyphenolic	- A type of antioxidant
Primary prevention	- A measure to prevent the onset of diseases
Run-in period	- An amount of time set to give all participants the same base before the trial starts
Secondary prevention	- A measure to stop or slow down an already diagnosed condition or risk factors as well as preventing re-injury
Student's t-test	- A statistical test used to examine whether there is a difference between two normal populations where the standard deviation is not given
Virgin Coconut Oil	- Extracted directly from coconut under controlled temp
Washout period	- A period in between two intervention episodes with the aim to prevent the previous intervention to affect the next one

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Introduction

Background

Coconut has been consumed for a long time, especially within Asian cuisine. The supply and interest in coconut consumption has increased during the last few years. It is promoted as a dietary supplement said to optimize health(1). The edible part of the coconut consists of the white flesh (copra) and coconut milk (also called coconut water). It is from the flesh coconut fat is extracted(2).

Coconut fat consists mainly of saturated fatty acids, this being about 90%. The major fatty acids present in coconut fat are lauric acid (12:0) with 46%, myristic acid (14:0) with 17% and palmitic acid (16:0) with 9%. Only 10% are unsaturated fatty acids in contrast to olive oil for example, which consists of 87% unsaturated fatty acids. Even when comparing coconut fat with other foods high in saturated fats, for example butter, coconut fat has a higher proportion of saturated fatty acids (90%). than butters 66%. However they differ in the percental distribution of their diverse saturated fatty acids(3).

According to the Nordic Nutrition Recommendations (NNR), the intake of saturated fatty acids including trans fats should be limited to a maximum of 10 E%. It has been found that a reduction in consumption of saturated fatty acids can lead to lowered levels of blood cholesterol(4).

In the diet, the intake of saturated fats is among the factors that affect the cholesterol levels the most. Cholesterol in the blood can be measured in TC, LDL, HDL, VLDL and the ratio of LDL and HDL(5, 6). TC covers the total values of LDL, HDL and VLDL. LDL is usually referred to as the "bad" cholesterol because of the fact that high levels of it can accumulate in the blood vessel walls and form plaque, which in its turn can lead to a narrowing of the blood vessels. A major consequence of this process is that the speed of the blood flow is reduced, resulting in an increased risk of CVD. HDL is known as the "good" cholesterol because of its association with a lower risk of cardiovascular disease and its anti-oxidative properties. The function of HDL is to pick up excess cholesterol from the body tissues back to the liver to be utilized(6, 7). Therefore the recommendations are set for the population to have high HDL levels and low LDL levels. When assessing the risk of “good” and “bad” cholesterol, a valid method to use is the ratio between LDL/HDL(5). The aim is to have as low ratio as possible, as this presents a lower level of LDL and a higher level of HDL(6).

The primary prevention target values for CVD are for TC < 5.0 mmol/L, LDL < 3.0 mmol/L, HDL > 1.0 mmol/L (for men) and HDL > 1.3 mmol/L (for women) according to what is currently practiced within Swedish healthcare. These values also apply when taking into account other risk factors and the patient's overall health. For secondary prevention and high-risk groups the target values are for TC < 4.5 mmol/L and LDL < 2.5 mmol/L(5). With an altered diet, a change in blood cholesterol can be seen already after about two weeks. A maximal effect is reached after about four weeks(8).

Lauric acid, myristic acid and palmitic acid are among the saturated fats that raise LDL cholesterol and are proven to be major risk factors in the development of cardiovascular diseases. Myristic

acid and palmitic acid have also shown to raise HDL levels in the blood. As brought up previously, these three fatty acids are present in large amounts in coconut fat(3, 4, 7).

There is a broad range of current research, discussions and publications about the various health aspects of coconut, including its cholesterol-lowering effect. A study examining the effects of virgin coconut oil (VCO) on lipid profile in healthy males concluded that an intake of VCO caused a significant decrease in HDL levels(1). Another study was made on women between 20 to 40 years old with abdominal obesity (WC > 88 cm) investigating the effects of dietary fat on coconut profiles regarding weight, WC and blood lipid levels. This study showed that coconut fat was not involved in the causation of dyslipidemia and contributed to a reduction of abdominal obesity in the study population(9). Furthermore, tests on rats have shown beneficial effects from an intake of VCO. A study on rats from 2003 comparing VCO and "regular" coconut fat (copra), ie refined oil, presented this. The results showed lowered TC and LDL blood cholesterol levels and increased HDL levels with VCO. This was explained by the biologically active polyphenolic components present in VCO(10).

The main fatty acids in coconut fat belong to the group of fatty acids called MCT, i.e. medium-chain fatty acids. Over 50% of the fatty acids in coconut fat are MCT fats. These are absorbed intact from the small intestine and do not undergo degradation and reesterification processes. They are directly used in the body to produce energy. In a study investigating possible health effects of coconut constituents, it was claimed that MCT fats were, unlike other fatty acids, not stored in fat deposits and therefore of great interest for weight reducing diets. Many studies investigate separate MCT fatty acids and not the complete composition of coconut fat. It is important to highlight the fact that a single fatty acid cannot be compared to coconut fat as a whole since it consists of a various range of different fatty acids being short, medium and long chained(11).

Problem formulation

The current recommendations on fat intake, according to NNR, are to keep intakes of saturated fats including trans fats below 10 E%. It has been found that a reduction of the consumption of saturated fatty acids leads to a reduction of blood cholesterol. Coconut fat is composed mainly of saturated fatty acids (90%). Despite the high content of saturated fats, it is alleged that coconut fat has beneficial effects on blood cholesterol, on the basis of a different fatty acid composition compared with other foods rich in saturated fats, such as butter. The parted views have led to an interest of further investigation surrounding coconut fats effects on blood cholesterol in humans.

Objective

The aim of this systematic review is to investigate how an intake of coconut fat would affect blood cholesterol levels when it comes to TC, LDL, HDL and the LDL/HDL-ratio.

Research question

Does an oral intake of coconut fat have beneficial effects regarding blood cholesterol levels (TC, LDL, HDL and the LDL/HDL-ratio) in healthy adults?

Method

Inclusion- and exclusion criteria

The following inclusion search criteria for this systematic review was to be on human studies with a healthy population between 18 – 79 years old. Studies based on coconut fat versus other fats as well as coconut fat versus placebo were included in the inclusion criteria. The study designs RCT, CCT and cohort published from 1990 and on were considered. There were no restrictions on the sum of participants, study duration or amount of coconut fat consumed during the study. The research substance was to be exclusively coconut fat and not single fatty acids extracted from coconut fat. Studies involving participants using drugs known to influence blood lipid metabolism were excluded, as well as studies with pregnant women.

Data collection method

The literary search was done within the databases PubMed and Scopus using a range of search words to gather the most relevant information. Search functions AND and/or OR were used to collect appropriate articles. Keywords mentioned in found articles were of great help when it came to further literature searches. Databases, search words, limitations, matches found and articles chosen are presented in Table 1.

Data processing

During the literary reviews search in the two databases, the study titles were looked upon. In cases where the titles were considered of relevance for the research question or where the aim of the study was not apprehensive enough by only reading the title, the abstract would also be read. Abstracts not being under the chosen inclusion criteria were left out. The first elimination round left behind 19 studies where the abstracts of these were read once again. They were thoroughly analysed in order to determine whether they definitely fell under the inclusion criteria or not. The 19 studies mentioned above can be found in Table 1. A total of eleven studies remained after the detailed abstract analysis. All of these were read in full text. Out of eleven articles read in full text, eight were excluded because they were judged to have a risk of bias. Examples of bias included reported data which relied on participants subjective answers, that a single fatty acids derived from coconut fat was analysed instead of coconut fat as a whole, the overall food intake was not controlled, there was no control group, no measured description of the endpoints, no washout periods between the test oils or a simultaneous start of regular physical activity parallel to the intervention. A final of three studies, two RCT and one CCT, remained. However, some of the excluded articles would be used within the introduction and discussion section.

Table 1. Data collection method

Database	Date	Search word	Limitations	Matches found	Chosen articles	Authors, Year
PubMed	2013-05-01	”coconut oil” OR ”coconut fat” AND ”cholesterol”	Human, Year 1990 to present	46	12	Feranil et al. 2011**(12) Cox et al. 1995(13) Cox et al. 1998**(14) Mendis et al.1990(15) Kumar et al. 1997**(16) Assuncao et al. 2009**(9) Karupaiah et al. 2011*(17) Müller et al. 2003(18) Ng K.W. et al. 1992**(19) Schwab et al. 1995**(20) Mendis et al. 2001*(21) Lu et al. 1997*(22)
PubMed	2013-05-01	”coconut oil” OR ”coconut fat” AND ”blood lipids”	Human, Year 1990 to present	2	0	
PubMed	2013-05-01	”coconut oil” OR ”coconut fat” AND ”LDL”	Human, Year 1990 to present	30	5 [5]	
PubMed	2013-05-01	”coconut oil” OR ”coconut fat” AND ”HDL”	Human, Year 1990 to present	26	6 [6]	
PubMed	2013-05-01	”coconut oil” OR ”coconut fat” AND ”health”	Human, Year 1990 to present	37	4 [1]	Sircar et al. 1998*(23) Hebert et al. 1990*(24) Amarasiri et al. 2006*(11)
Scopus	2013-05-01	”coconut oil” OR ”coconut fat” AND ”cholesterol”	Human, Year 1990 to present	52	16 [13]	Sabitha et al. 2009**(25) Woo et al. 1998*(26) Paz et al. 2010**(1)
Scopus	2013-05-01	”coconut oil” OR ”coconut fat” AND ”blood lipids”	Human, Year 1990 to present	4	0	
Scopus	2013-05-01	”coconut oil” OR ”coconut fat” AND ”LDL”	Human, Year 1990 to present	27	10 [10]	
Scopus	2013-05-01	”coconut oil” OR ”coconut fat” AND ”HDL”	Human, Year 1990 to present	23	10 [10]	
Scopus	2013-05-01	”coconut oil” OR ”coconut fat” AND ”health”	Human, Year 1990 to present	51	7 [6]	Lipoeto et al. 2001*(27)

[ ] = duplicates * = excluded after a closer abstract review ** = excluded after a closer full text review

Relevance and quality review

The validation templates *Granskningsmall för randomiserade studier* and *Granskningsmall för observationsstudier och icke-randomiserade kontrollerade studier* designed by the Swedish Council on Health Technology Assessment (SBU) were used in the quality controls of the three selected studies (see appendix 1 and 2). The four selected endpoints were TC, LDL, HDL and LDL/HDL ratio. The strength of evidence ranged from one (+) to four (++++) plus and was decided using the template *Sammanfattande Evidensformulär* developed by Gothenburg University (see appendix 3).

Results

The three studies included in the systematic review with the aim of investigating the effects of coconut fat intake on blood cholesterol levels are described initially under results. Table 2 briefly concludes all the selected studies. The results section is finalized with a study quality summary (Table 3) as well as a presentation of the evidence strength levels (Table 4). As the included studies intervention length ranged from three to eight weeks it should be highlighted that only short-term effects on blood cholesterol levels are portrayed here.

Study 1: Cox et al. 1995 (13)

Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels

Method

A total of 28 subjects, 13 men and 15 women, 29 to 67 years old were included in the study. Body weight was recorded at baseline and at week 4 and 6 in each diet period. Cholesterol levels of the participants ranged from 5.5 to 9.7 mmol/L. The study duration was held for a total of six months, which started with a six-week run-in period where a 5-day food record was to be filled in by the subjects in order to collect information about the participants' food and nutrient intakes.

The study was built up by three separate dietary fat intervention periods, each of them being six weeks. The participants were randomized into one of three dietary periods based on the randomizing design Latin-square. This design can be explained as a run-in period with a normal diet during six weeks followed by a randomized order of the test fats being either:

1. Coconut fat diet - Butter fat diet - Safflower oil diet
2. Butter fat diet - Safflower oil diet - Coconut fat diet
3. Safflower oil diet - Coconut fat diet - Butter fat diet

During the run-in period, participants were asked to report their everyday dietary intake and from this information their individual energy intake was calculated. These calculated personal energy intake levels were used in all three intervention periods. The fats provided from the three different diets were approximately 36 E%. In the Coconut fat diet, 46% of the fat content was from coconut. The same ratio of butter was given in the Butter fat diet. In the Safflower oil

diet, 29% of the fat content was from safflower oil. A 5-day diet record measured dietary compliance in each intervention period. Participants were regularly checked upon through interviews and telephone calls. A continuous habitual physical activity pattern and further lifestyle features was encouraged throughout the study.

Kits and calibrators from Boehringer-Manheim and Roche Diagnostics were used to assess TC, LDL and HDL from the participants' blood samples. Blood cholesterol was measured at week 4 and 6 in each dietary period. The results were statistically analyzed using ANOVA and paired Student's t-test.

Results

TC and LDL levels were the highest on the butter fat diet, intermediate on the coconut fat diet and lowest on the safflower oil diet, all of these being statistically significant. HDL levels did not vary significantly when it came to the group as a whole. Looking only at the women's results HDL levels were significantly higher on the butter and coconut fat periods compared to the safflower oil diet (see appendix 4). The amount of SFA and PUFA were equivalent in the butter diet and in the coconut fat diet. Total energy intake and percental energy proportions remained constant during the study. Regarding the mean body weight of the subjects, this remained unchanged throughout the study(13).

Study 2: Mendis et al. 1990 (15)

The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidaemic men

Method

A total of 25 subjects, all men, aged 20 – 26 years were included in the study. All subjects were within the range of normal body weight. The study consisted of two intervention periods, each being eight weeks. There was a washout period of three weeks between the two diet episodes. The aim of this washout period was to minimize the impact of the previous test fat on the next intervention episode. All participants started out with eating the soya-bean diet followed by a washout period and subsequently the coconut fat diet.

The study was built up by two diets based on different fats, coconut fat respectively soya-bean fat, served in their meals as the main source of fat for lunch and supper. Both dietary interventions were of same fat energy distribution. The percental amount of the different intervention fats was the same, this being around 70%. Also, the carbohydrate intake and composition were of equal amount in the two diets. Alcohol consumption was not allowed during the study. Interviews were conducted with the subjects to keep track of their compliance levels.

Blood cholesterol was measured in the beginning and at the end of each intervention period. During the two last weeks of the intervention period, two consecutive blood samples were taken to determine the level of TC and calculate the definite lipid change. Statistical analyses were made using paired Student's t-test.

Results

There were no significant differences when it came to the mean values of TC, LDL and HDL at the end of the coconut fat diet period compared to the baseline values. Mean results of TC after the coconut fat diet differed significantly from the soya-bean fat diet, as the TC levels were lower during the soya-bean fat diet (see appendix 4). Both HDL and LDL were lowered after the soya-bean intervention compared to baseline. It was judged to be a good compliance among the participants based on their interview answers. Their body weight did not differ considerably during the study(15).

Study 3: Müller et al. 2003 (18)

The serum LDL/HDL cholesterol ratio is influenced more favourably by exchanging saturated with unsaturated fat than by reducing saturated fat in the diet of women

Method

A total of 31 subjects, all women, aged 20 – 40 years participated and 25 of them completed the study. BMI of the participants ranged from 21.3 to 27.7 kg/m^2. Their body weight was checked twice a week.

The participants were randomized into one of three groups based on the randomizing design Latin-square. This design compromises a randomized order of the test fats being either:

1. High SFA – Low SFA –HUFA
2. Low SFA –HUFA – High SFA
3. HUFA – High SFA – Low SFA

This design also determined in which sequence the test fats would be received. Period 1 and 2 were 22 days each and period 3 was 20 days. Between two different test periods, a washout period of one week took place where they would go back to their normal eating habits.

The study compared the effects on blood cholesterol of a low amount of fat diet based on SFA (the main source being coconut fat, 80%), a high amount of fat diet based on SFA (the main source being coconut fat, 80%) and a high amount of fat diet based on HUFA (the sources being a mixture of sunflower oil, rapeseed oil, coconut oil and palm oil). Equal proportions of fat were presented in the High SFA and HUFA diets (42 E%). The Low SFA diet was based on 22 E% fat. The High SFA and the Low SFA diets had the same combination of fats. All weekday meals were prepared under supervised means. No foods other than the test foods were to be consumed during the intervention periods and neither were alcoholic beverages. Continuous habitual physical activity patterns as well as other lifestyle features were encouraged throughout the study period.

Blood cholesterol of the subjects was measured at baseline and at the end of each intervention period. Results were statistically analyzed using ANOVA and the Bonferroni method, the later one for pair wise comparisons between the three diets. A confidence limit of 95% was used for statistical assessment.

Results

The results were the following:

- TC was higher after the High SFA diet than after the Low SFA diet.
- TC and LDL after the HUFA diet were significantly lower compared to the two SFA diets.
- LDL showed a slight difference when comparing the High with the Low SFA diet.
- HDL was after the High SFA diet significantly higher compared to the Low SFA diet or the HUFA diet.
- The LDL/HDL ratio was significantly higher after the Low SFA diet compared to the High SFA diet. The ratio was also higher after both SFA diets than after the HUFA diet (see appendix 4).

There was a general high level of compliance. Body weights of the participants at the end of the diet periods had not differed significantly (18).

Final results

Table 2. Final results of studies chosen after intervention

Author, Year	Study design	Study population	Intervention	TC (mmol/L)	LDL (mmol/L)	HDL (mmol/L)	LDL/HDL ratio (mmol/L)	Study quality
Cox et al. 1995(13)	RCT	n: 28 f: 15 m: 13	**Test fats** • CF 6 w • BF 6 w • SO 6 w In a randomized order Run-in period 6 w Test fats: 29-46% of fat E%	• CF: 6.4 • BF: 6.8 • SO: 6.1 All values: (p<0.001) CF, BF, SO in order: **f:** 6.3, 6.7, 5.9 All values (p<0.01) **m:** 6.6, 7.0, 6.2 All values (p<0.01)	• CF: 4.2 • BF: 4.5 • SO: 3.9 BF v. CF or SO (p<0.001) CF v. SO (p<0.004) CF, BF, SO in order: **f:** 4.0, 4.4, 3.8 BF v. CF or SO (p<0.005) CF v. SO (nsd) **m:** 4.4, 4.7, 4.0 SO v. BF or CF (p<0.01) BF v. CF (nsd)	• CF: 1.5 • BF: 1.4 • SO: 1.4 All values: (nsd) CF, BF, SO in order: **f:** 1.8, 17, 1.6 SO v. BF or CF (p<0.02) CF v. BF (nsd) **m:** 1.2, 1.2, 1.2 (nsd)		High-mediate high
Mendis et al. 1990(15)	CCT	n: 25 m: 25	**Test fats** • SbF 8 w • CF 8 w Washout period 3 w Test fats: 70% of fat E%	**B: 4.64** • SbF: 3.68 (p<0.001 from B) • CF: 4.61 (p<0.001 to SbF)	**B: 2.95** • SbF: 2.27 (p<0.001 from B) • CF: 2.84 (nsd)	**B: 1.10** • SbF: 0.94 (p<0.05 from B) • CF: 1.14 (nsd)		High
Müller et al. 2003(18)	RCT	n: 25 f: 25	**Test fats** • LSFA 20-22 d • HSFA 20-22 d • HUFA 20-22 d In a randomized order Washout period 1 w LSFA 22 E% fat* HSFA 42 E% fat* HUFA 42 E% fat**	**B: 4.95** • LSFA: 5.13 • HSFA: 5.38 • HUFA: 4.41 HUFA v. HSFA or LSFA (p<0.01)	**B: 2.99** • LSFA: 3.12 • HSFA: 3.20 • HUFA: 2.52 HUFA v. HSFA or LSFA (p<0.01)	**B: 1.42** • LSFA: 1.44 • HSFA: 1.69 • HUFA: 1.44 HSFA v. LSFA or HUFA (p<0.01)	**B: 2.31** • LSFA: 2.43 • HSFA: 2.10 • HUFA: 1.92 LSFA v. HSFA or HUFA (p<0.01)	High-mediate high

*: main source coconut fat (80%)
**: a mix of sunflower, rapeseed, coconut and palm oil

n: subjects
m: male
f: female

d: days
w: weeks
nsd: no significant difference (p>0.05)

B: Baseline (mean)
CF: Coconut fat (mean)
BF: Butter fat (mean)

SO: Safflower oil (mean)
SbF: Soya-bean fat (mean)

Study quality judgement

The studies chosen were judged to have a low risk of bias. A valid randomization design was used in two studies, all three studies were of cross-over design and the study duration was of appropriate time, i.e. more than two weeks(8). Every participant received the same treatment, there was a good general compliance, endpoints had a low risk of bias, valid methods of endpoint measurements and a low risk for conflicts of interest. All the aspects mentioned above motivate the fact that the studies were quality judged between high to mediate high. All three studies lacked information about side effects as well as blinding.

Table 3. Study quality

Author, Year	Study design	Study quality	Comments
Cox et al. 1995	RCT	High-mediate high	There was no information about whether the participants were blinded or not. In addition to this, meals were prepared by the subjects themselves at home, which could contribute to a higher risk of bias. There was no washout period between the intervention diets. From this information it was concluded that the study was of high to mediate high quality.
Mendis et al. 1990	CCT	High	Even though the study did not mention if the participants were blinded or not, it did not affect the judgement of the study's overall quality. Mainly due to the fact that the meals given were ready-made.
Müller et al. 2003	RCT	High-mediate high	Study quality was measured as high to mediate high because no information was given about the impact of the dropouts on the final results. Neither was any information about blinding presented.

Evidence grading

The four endpoints investigated are presented below. Out of these four, all three studies looked into TC, LDL and HDL whilst only one investigated the LDL/HDL ratio. Since the studies lacked information on blinding, *internal validity* was judged to have some limitations. When it came to *correspondence*, there was some heterogeneity because of various intervention durations in between the examined studies. All studies involved had a low amount of participants and the external validity was judged to *uncertainty*. The LDL/HDL ratio was only mentioned in one study and therefore judged as having 'some problems' when it came to *vague basic data*.

Table 4. Strength of evidence

	Endpoints			
	TC	**LDL**	**HDL**	**LDL/HDL ratio**
Number of studies	3	3	3	1
Study design – internal validity	Some limitations	Some limitations	Some limitations	Some limitations
Correspondence	Some heterogeneity	Some heterogeneity	Some heterogeneity	Some heterogeneity
Study population – external validity	Uncertainty	Uncertainty	Uncertainty	Uncertainty
Vague basic data	No problems	No problems	No problems	Some problems
Strength of evidence	Mediate-high (+++)	Mediate-high (+++)	Mediate-high (+++)	Low (++)

Discussion

Main findings

After investigating the three studies, different effects on blood cholesterol from an oral intake of coconut fat have been shown. Beneficial effects on blood cholesterol can be defined, according to the practiced healthcare guidelines, as lowering levels of TC and LDL, increasing levels of HDL and having a low LDL/HDL ratio(5). Seen in this systematic review, a diet rich in coconut fat led to significantly higher TC and LDL levels compared to a diet rich in HUFA (safflower oil, soya-bean oil or a HUFA mix). Only one study showed significantly higher HDL levels in all its subjects after a coconut fat diet compared to a HUFA diet.

Comparing coconut fat with safflower oil or soya-bean oil (the diet being mainly saturated fat respectively unsaturated fats) TC and LDL levels were lower after the soya-bean and safflower interventions(13, 15). Comparing coconut fat with butter (their main source consisting of saturated fats), it was seen that TC and LDL levels were significantly higher on the diet based on butter than on the one based on coconut fat(13). TC and LDL were significantly higher in the HSFA and LSFA diets (based on coconut fat) compared to the HUFA diet. However, HDL was significantly higher after the HSFA diet compared to the LSFA diet or the HUFA diet(18). Apart from Müllers study, the overall HDL cholesterol levels, in the remaining two studies, did not differ significantly between the different intervention diets in the group as a whole. On the other hand, in one study, the women's HDL levels were significantly higher on the butter and coconut fat diets compared to the safflower oil diet(13). The LDL/HDL ratio was in total lowered after the HUFA diet compared to both SFA diets but only significantly lower after the LFSA diet. The study measuring the ratio concluded that the amount of fat in the diet was not of main importance, but the composition of the fat quality being crucial. Since this study only involved women, the results can only be based on women as well(18).

Coconut fat – not single fatty acids

There is a possibility that different conclusions are reached when investigating the impact of single fatty acids, such as MCT, compared to a complete fat source, as for example in this paper, being coconut fat. The possible differences could be since coconut fat is known to have a high amount of MCT fats, but also has other fatty acids and components that can affect the outcome. Studies that have examined MCT fats beneficial effects on health in general, positive effects have been demonstrated in, for example, weight loss. These studies results claimed that an intake of coconut fat has a positive impact on weight and health(28-30). The question is; can one conclude that coconut fat has beneficial effects when only individual fatty acids have been investigated?

Coconut fat – Cholesterol – Cardiovascular disease

In this systematic review the relationship between the consumption of coconut fat and blood cholesterol levels, and not the relationship between coconut fat and CVD, has been examined. It has not been investigated in what way an intake of coconut fat may increase the risk of CVD,

but only the extent to which it affects cholesterol levels in the blood. To be able to express how coconut fat would affect the incidence of CVD, research has to be divided into two steps: the first being coconut fats effect on blood cholesterol and the second being cholesterols effect on CVD. Regarding the second step, a comprehensive international study, named the INTERHEART study, concluded dyslipidemia as a major risk factor for CVD(31).

Study design – dietary composition

In the different intervention diets, the percentages of test fats of the total fat intake differ. Levels were 46% in study 1 (Cox et al.), 70% in study 2 (Mendis et al.) and 80% in study 3 (Müller et al.). Consuming the test fat as half of the daily fat intake compared to eating three quarters of the daily fat intake as test fat may possibly play a role in how big the impact on the cholesterol levels will be. The difference in coconut fat percentage in the diet can thus make a difference in the final results. Common in all three studies is that the participants consume more saturated fat than NNRs recommended level. Despite the percental differences of consumed test fat, the results of the studies all follow the same pattern in blood cholesterol measures.

In study 1 (Cox et al.), the participants were moderately hypercholesterolemic at baseline. In study 2 (Mendis et al.) and study 3 (Müller et al.) their baseline cholesterol levels were within recommended levels. In this systematic review, the focus has been comparing the effects on blood cholesterol between coconut fat with either another SFA fat or a HUFA fat as the main fat in the diet. Less focus has been put on comparing the cholesterol levels from baseline. This due to the fact that it is assumed that some kind of fat will most probably be included in a person's daily diet and therefore of broader interest to examine the possible contrasting effects of the different kinds of fat rather than to baseline values. To be noted is that despite slight differences in baseline values, the results in blood cholesterol levels all follow similar patterns.

It should be mentioned that conclusions drawn in this systematic review are presenting values, which are based on having coconut fat as the main fat in the diet and not as a food that is rarely eaten. Therefore no statements are pronounced on the amount of consumed coconut fat needed for a potential impact on blood cholesterol levels.

Study design – length, gender and species

The results are based on short-term interventions (three to eight weeks) and can therefore only speak for short-term effects on blood cholesterol. To see an alternation in the blood cholesterol levels from a changed diet, a minimum of two weeks is needed. A maximal effect is reached after about four weeks(8). The studies investigated in this systematic review were spread from three to eight weeks. Due to these facts, the intervention periods in the included studies were of adequate study duration and are therefore judged to show reliable results. Would the study period have stretched over a very long time, for example several years, other factors such as illnesses occurred within the study period could have had an impact on the outcomes. Opposite to this, an intervention period being very short, i.e. less than two weeks, the time would not have been sufficient enough to be able to see any differences in blood cholesterol levels.

As seen in Table 2, the study where women and men participated under the same circumstances, the women's cholesterol values tended to result in lower levels in TC and LDL

and higher in HDL compared to the men. The study's results show that the women had higher HDL levels compared to the men regardless of which fat was being tested. These results go hand in hand with the already existing primary prevention target values for CVD which present higher values on HDL for women compared to men(5).

In the present systematic review, animal studies have been excluded as they represent other species than humans. To be able to conclude results on coconut fat's effects on humans, it was decided that only studies based on people would be used. Additionally, studies including pregnant women and individuals taking blood lipid lowering medication were excluded. This was because pregnant women can have changed metabolic features during their pregnancy, which can portray a risk of possible bias. The same hypothesis applied for studies involving participants using drugs known to influence blood lipid metabolism.

Study design – strengths and weaknesses

Due to various aspects of power in the chosen studies, it can be concluded that the overall judgement of the study quality is high. Specific strengths include randomization designs(13, 18), run-in period(13), washout periods(15, 18), regular checkups, equal E% proportions of fat and prepared meals(15, 18). The randomization design helps to increase the validity and minimize the risk of bias in the studies. Run-in periods alleviate all the participants in these studies to start from more similar baseline conditions. With a washout period, the risk of the previous intervention intervening with the following one is reduced. Regular checkups in different forms such as telephone calls, diet records and interviews during an intervention period will provide more reliable results than for example if no check up was to be done. In two of the selected studies, the meals were prepared and served under controlled conditions, which gives more credibility to results found(15, 18). In studies not controlling the total energy intake or E% proportions the risk of self-handeledy altering the results are higher and were therefore excluded(1).

Weaknesses in the selected studies could be that all three studies lacked information about side effects as well as blinding. No side effects were mentioned which can be interpreted as if on one hand there were no side effects, and on the other as if they were not exposed. The importance of blinding should not be neglected as it provides credibility for results found. For example, if a participant is aware of which test fat he or she is going to eat and wants this to show a certain result, hypothetically speaking, the participant could unreported eat foods that are known to have a certain effect on cholesterol levels. Under the same reasoning, a participant could eat lipid-lowering drugs during an intervention period to affect their cholesterol, without reporting the usage of drugs. All the facts and examples mentioned above could contribute to possible risks of bias.

Some of the studies excluded had a high risk of bias due to, among others, starting parallel sequences of physical activity during their intervention period, instructions to increase the intake of fruits and vegetables and reduce simple carbohydrates and animal fat. Such alternations in an individual's daily life can effect blood cholesterol levels and contribute to biased results. Apart from these aspects, studies with self reported data and retrospective materials gave subjective results and were therefore eliminated in the selection of studies (5, 9, 12, 25).

Types of coconut fat

Studies have shown both positive and negative effects on blood cholesterol levels from an intake of VCO. In a study where VCO was compared with regular coconuts fat on rats, beneficial results on cholesterol were seen from an intake of VCO. The reason for this was explained to be because of the biologically active polyphenol components present in the fat(10). In addition to this, another previously published systematic review within the subject of coconut fat has also discussed polyphenol components having a possible beneficial role in positively altered cholesterol levels(11). Contradictory to this, a study as recent as 2010 presented results where decreased HDL levels were seen from a diet based on VCO. It should be mentioned that this study was judged to have a high risk of bias(1). The data can be interpreted as being vague and not consistent enough for a definite conclusion to be drawn surrounding VCO.

In media today, when portraying the beneficial aspects of coconut fat, it is often the VCO type that is referred to. Due to this fact, findings reached in this systematic review are not completely applicable to what is said specifically about VCO. However, the final results of this systematic review can be connected to what is said about coconut fat in general. Future large scale and widespread interventions are encouraged, as coconut fat is a current topic within the world of nutrition. Investigating possible beneficial effects of VCO on humans and differences between regular and virgin coconut fat is desirable.

Conclusion

It can be concluded that coconut fat as the main fat in the diet led to significantly higher TC levels (+++) and LDL levels (+++) in adults compared to diets rich in HUFA, which is today not considered to be beneficial when it comes to blood cholesterol in relation to health. Regarding HDL, only one study shows significantly higher levels in all its subjects after a coconut fat diet compared to a HUFA based diet (+++), i.e. a beneficial effect. The LDL/HDL ratio (++), solely investigated in women, is higher when comparing two SFA diets based mainly on coconut fat with a HUFA diet, but only significantly higher comparing LFSA with HUFA. The aim is to have a ratio as low as possible.

It is of relevance to mention that the results show that TC and LDL levels are significantly higher on a butter diet compared to a coconut fat diet (SFA versus SFA). These levels are significantly lower on a HUFA diet compared to a coconut fat diet (HUFA versus SFA). The results show short-term effects on blood cholesterol levels as the intervention diets only lasted from three to eight weeks. Further research is encouraged for more specific results to be drawn surrounding the subject of coconut fats effects on blood cholesterol levels in humans.

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Appendix 1. Granskningsmall för randomiserade studier
(Validation template for RCT)

Granskningsmall för randomiserade studier

Författare	
År	
Artikelnummer	

Alternativet "uppgift saknas" används när uppgiften inte går att få fram från texten.
Alternativet "ej tillämpligt" väljs när frågan inte är relevant.

A1. Selektionsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Användes en lämplig randomiseringsmetod?				
b) Om man har använt någon form av begränsning i fördelningsprocessen (ex block, strata, minimisering), är skälen till detta adekvata?				
c) Var grupperna väl balanserade vid studiens start avseende relevanta baslinjevärden?				
d) Kan man utesluta att avhopp/exklusion efter randomisering kan snedvrida resultaten med tanke på storlek och fördelning på avhoppet över grupperna?				
Kommentarer:				
A1. Bedömning av risk för selektionsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A2. Behandlingsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Var studiedeltagare blindade?				
b) Var behandlare/prövare blindade?				
c) Är följsamheten acceptabel?				
d) Är metoden för mätning av följsamhet/exponering validerad för den undersökta parametern?				
e) Är metoden för mätning av följsamhet/exponering validerad för den aktuella populationen?				
f) Är utfallet av valideringen acceptabel?				
g) Är resultaten justerade för mätfel i metoden för registrering av exponering?				
h) Har variationer i exponering över tid tagits med i analysen?				
i) Har deltagarna behandlats/exponerats på samma sätt bortsett från interventionen?				
Kommentarer:				
A2. Bedömning av risk för behandlingsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A3. Bedömningsbias (kritiska utfallsmått)	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är det kritiska utfallsmåttet okänsligt för bedömningsbias?				
b) Var den som utvärderade resultaten blindad för vilken intervention som gavs?				
c) Är utfallet definierat på ett lämpligt sätt?				
d) Är utfallet identifierat/diagnosticerat med validerade mätmetoder?				
e) Var observatörsöverensstämmelsen acceptabel?				
f) Om det fanns obalanser i baslinjevariabler, har de korrigerats för på ett adekvat sätt i den statistiska analysen?				
g) Var tidpunkten för mätning lämplig?				
h) Är valet av mått för rapporterad effekt lämpligt?				
i) Är den analyserade populationen lämplig för den fråga som är föremål för studien?				
Kommentarer:				
A3. Bedömning av risk för bedömningsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A4. Bortfallsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är bortfallet tillfredsställande lågt i förhållande till populationens storlek?				
b) Är bortfallets storlek balanserad mellan grupperna?				
c) Är relevanta baslinjevariabler balanserade mellan avhoppare och icke avhoppare?				
d) Är den statistiska hanteringen av bortfallet adekvat (ex PP, ITT)?				
Kommentarer:				
A4. Bedömning av risk för bortfallsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A5. Summering av risk för bias	Låg	Måttlig	Hög
A1) Selektionsbias			
A2) Behandlingsbias			
A3) Bedömningsbias			
A4) Bortfallsbias			
Kommentarer:			
A5. Bedömning av risk för bias: ☐ Låg ☐ Måttlig ☐ Hög			

B1. Risk för selektiv rapportering	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Anges vilket/vilka utfallsmått som är primära respektive sekundära?				
b) Har man uppgett att man följt ett i förväg publicerat studieprotokoll?				
c) Redovisas alla i förväg angivna utfallsmått på ett fullständigt sätt?				
d) Kan man utesluta rapportering av utfallsmått som inte angivits i förväg?				
e) Var tidpunkterna för mätning angivna i förväg?				
f) Mättes biverkningar/komplikationer på ett systematiskt sätt?				
Kommentarer:				
B1. Bedömning av risk för selektiv rapportering: ☐ Låg ☐ Måttlig ☐ Hög				

B2. Intressekonflikter	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Föreligger, baserat på författarnas angivna bindningar och jäv, låg risk att studiens resultat har påverkats av intressekonflikter?				
b) Föreligger, baserat på uppgifter om studiens finansiering, låg risk att studien har påverkats av en finansiär med ekonomiskt intresse i resultatet?				
c) Föreligger låg risk för annan form av intressekonflikt?				
Kommentarer:				
B2. Bedömning av intressekonflikter ☐ Låg ☐ Måttlig ☐ Hög				

B3. Summering av risk för publikationsbias	Låg	Måttlig	Hög
B1) Risk för selektiv rapportering			
B2) Intressekonflikter			
Kommentarer:			
B3. Bedömning av risk för publikationsbias: ☐ Låg ☐ Måttlig ☐ Hög			

C. Överförbarhet	Ja	Nej	Delvis	Ej tillämpligt
a) Överensstämmer sammanhanget och kontroll-villkoren med den tänkta, svenska vårdsituationen?				
Kommentar:				
b) Överensstämmer studiedeltagarna med den tänkta, svenska målpopulationen?				
Kommentar:				
c) Kan interventionen och sammanhanget där interventionen ges i studien översättas till hur den ges/skulle ges under svenska förhållanden?				
Kommentar:				
C. Brister i överförbarhet	☐ Inga	☐ Vissa	☐ Stora	

D. Kritiska utfallsmått	Risk för bias	Risk för publikationsbias	Överförbarhet
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0

E. Viktiga utfallsmått	Risk för bias	Selektiv rapportering	Överförbarhet
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0
	0	0	0

Appendix 2. Granskningsmall för observationsstudier och icke-randomiserade kontrollerade studier
(Validation template for observation studies and non-randomized controlled studies)

Granskningsmall för observationsstudier och icke-randomiserade kontrollerade studier

Författare	
År	
Artikelnummer	

Alternativet "uppgift saknas" används när uppgiften inte går att få fram från texten.
Alternativet "ej tillämpligt" väljs när frågan inte är relevant.

A1. Selektionsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är de observerade grupperna rekryterade på ett likartat sätt?				
b) Var grupperna väl balanserade vid studiens start avseende relevanta baslinjevärden?				
c) Om det fanns obalanser, har de korrigerats för på ett adekvat sätt i den statistiska analysen?				
d) Har författarna tagit hänsyn till eventuella skillnader i socioekonomisk status?				
e) Är den statistiska modellen adekvat?				
Kommentarer:				
A1. Bedömning av risk för selektionsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A2. Behandlingsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är följsamheten acceptabel?				
b) Är metoden för mätning av följsamhet/exponering validerad för den undersökta parametern?				
c) Är metoden för mätning av följsamhet/exponering validerad för den aktuella populationen?				
d) Är utfallet av valideringen acceptabel?				
e) Är resultaten justerade för mätfel i metoden för registrering av exponering?				
f) Har variationer i exponering över tid tagits med i analysen?				
g) Är risken låg för att deltagarna exponerats för annat än den undersökta exponeringen (kontaminering, självmedicinering m.m.)?				
Kommentarer:				
A2. Bedömning av risk för behandlingsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A3. Bedömningsbias (kritiska utfallsmått)	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är det kritiska utfallsmåttet okänsligt för bedömningsbias?				
b) Var den som utvärderade resultaten blindad för studiedeltagarnas exponeringsstatus?				
c) Är utfallet definierat på lämpligt sätt?				
d) Är utfallet adekvat identifierat/diagnosticerat?				
e) Var observatörsöverensstämmelsen acceptabel?				
f) Är valet av mått för rapporterad effekt lämpligt (ex RR vs HR, kontinuerligt vs dikotomt, enskilda mått vs kompositmått)?				
Kommentarer:				
A3. Bedömning av risk för bedömningsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A4. Bortfallsbias	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Är bortfallet (loss to follow-up) tillfredsställande lågt i förhållande till populationens storlek?				
b) Är bortfallets storlek balanserad mellan grupperna?				
c) Är relevanta baslinjevariabler balanserade mellan bortfalls- och analysgruppen?				
d) Är den statistiska hanteringen av bortfallet adekvat?				
Kommentarer:				
A4. Bedömning av risk för bortfallsbias: ☐ Låg ☐ Måttlig ☐ Hög				

A5. Summering: risk för bias	Låg	Måttlig	Hög
A1) Selektionsbias			
A2) Behandlingsbias			
A3) Bedömningsbias			
A4) Bortfallsbias			
Kommentarer:			
A5. Bedömning av risk för bias: ☐ Låg ☐ Måttlig ☐ Hög			

B. Risk för selektiv rapportering	Ja	Nej	Uppgift saknas	Ej tillämpligt
Kan man utesluta selektiv rapportering?				
Kommentarer:				
B. Bedömning av risk för rapporteringsbias: ☐ Låg ☐ Måttlig ☐ Hög				

C. Intressekonflikter	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Föreligger, baserat på författarnas angivna bindningar och jäv, låg risk att studiens resultat har påverkats av intressekonflikter?				
b) Föreligger, baserat på uppgifter om studiens finansiering, låg risk att studien har påverkats av en finansiär med ekonomiskt intresse i resultatet?				
c) Föreligger låg risk för annan form av intressekonflikt (ex författarna har utvecklat interventionen)?				
Kommentarer:				
C. Bedömning av risk för intressekonflikter ☐ Låg ☐ Måttlig ☐ Hög				

D. Överförbarhet	Ja	Nej	Delvis	Ej tillämpligt
a) Överensstämmer sammanhanget och kontrollvillkoren med den tänkta, svenska vårdsituationen?				
Kommentar:				
b) Överensstämmer studiedeltagarna med den tänkta, svenska målpopulationen?				
Kommentar:				
c) Kan interventionen och sammanhanget där interventionen ges i studien översättas till hur den ges/skulle ges under svenska förhållanden?				
Kommentar:				
D. Brister i överförbarhet ☐ Inga ☐ Vissa ☐ Stora				

E. Effektstorlek	Ja	Nej	Uppgift saknas	Ej tillämpligt
a) Var effekten stor (t ex RR<0,5 eller >2.0)?				
b) Var effekten mycket stor (t ex RR<0,2 eller >5.0)?				
Kommentar:				

F. Dos-responssamband	Ja	Nej	Uppgift saknas	Ej tillämpligt
Finns stöd för ett dos-responssamband mellan exponering och utfall?				
Kommentar:				

G. Utfallsmått	Risk för bias	Överför-barhet	Effektstor-lek	Dos-respons
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
	0	0	0	0
Kommentar:				

Appendix 3. Sammanfattande Evidensformulär
(Evidence strength template)

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Institutionen för medicin
Avdelningen för klinisk näringslära
Dietistprogrammet, 2012/AW

Sammanfattande Evidensformulär **Effektmått:**

RCT utgår från ++++, kohortstudier utgår från ++. Sänk eller höj därefter graderingen utifrån studiekvalitet, överensstämmelse, överförbarhet, oprecisa data, risk för publikationsbias och effektstorlek.

Tillstånd:	
Åtgärd:	
Effektmått:	
Ingående studier: RCT ☐ (++++) Kohortstudier ☐ (++) Alla eller några av studierna sammanfattade i en systematisk översikt ☐ Antal studier: Antal pt:	+ 4 alt. +2
Studiedesign - Intern validitet (Randomiseringsförfarande, blindning, uppföljning, bortfall, intention-to-treat, vid kohortstudier – hantering av confounders) ☐ Inga begränsningar ☐ Vissa begränsningar (*men inte nog för nedgradering*[1]) ☐ Allvarliga begränsningar (*minska ett steg*) ☐ Mycket allvarliga begränsningar (*minska två steg*) Kommentera begränsningar eller grundvalen för nedgradering:	 ☐ 0 ☐ ? ☐ -1 ☐ -2
Överensstämmelse (Estimat av relativa effekten lika storlek och riktning mellan studierna? Överlappande konfidensintervall?) ☐ Inga problem ☐ Viss heterogenicitet (*men inte nog för nedgradering*[1]) ☐ Bekymmersam heterogenicitet (*minska ett steg*)	 ☐ 0 ☐ ? ☐ -1

Kommentera brist på överensstämmelse eller grundvalen för nedgradering:	
Studiepopulation – extern validitet(överförbarhet) Interventionen (effektmåttets relevans, relevans av jämförelsemetod, sjukvårdsmiljö, adekvat uppföljningstid)	
☐ Ingen osäkerhet	☐ 0
☐ Viss osäkerhet (*men inte nog för nedgradering*[1])	☐ ?
☐ Osäkerhet (*minska ett steg*)	☐ -1
☐ Påtaglig osäkerhet (*minska två steg*)	☐ -2
Kommentera viss osäkerhet eller grundvalen för nedgradering:	
Oprecisa data (Få händelser, vida konfidensintervall som infattar möjlig ogynnsam effekt) - kohort	
☐ Inga problem	☐ 0
☐ Vissa problem med precision (*men inte nog för nedgradering*[1])	☐ ?
☐ Oprecisa data (*minska ett steg*)	☐ -1
Kommentera viss osäkerhet eller grundvalen för nedgradering:	

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Osäkert underlag (Få och små studier från samma forskargrupp eller företag som alla visar samma sak) ☐ Inga problem ☐ Vissa problem (men inte nog för nedgradering[1]) ☐ Klar risk för publikationsbias (*minska ett steg*) Kommentera grundvalen för nedgradering	 ☐ 0 ☐ ? ☐ -1
Effektstorlek Vid stor effekt eller mycket stor effekt kan man uppgradera evidensstyrkan (Kohort) ☐ Ej relevant ☐ Stor effekt (RR<0,5 eller >2) (öka ett steg) ☐ Mycket stor effekt (RR<0,2 eller >5) (öka två steg) Kommentera grundvalen för uppgradering	 ☐ 0 ☐ +1 ☐ +2
Kommentera andra viktiga aspekter som ska beaktas vid kategorisering av evidensstyrka/bedömning av vetenskapligt underlag, t.ex. stark dos-respons, allt-eller-inget-effekter, confounders som maskerar del av effekt kan uppgradera evidensstyrkan. (kohort)	☐ +1
Räcker summan av smärre brister under flera punkter till en nedgradering med ett helt steg? (beräkna antal ? i ovanstående frågor) ☐ Ja ☐ Nej	 ☐ -1 ☐ 0
Evidensstyrka ☐ Hög (++++) ☐ Måttlig (+++) ☐ Låg (++) ☐ Mycket låg (+) (= saknas vetenskapligt underlag)	

Appendix 4. Tables showing blood lipid levels in selected studies

Study 1: Cox et al. 1995

Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels

TABLE 3. Plasma lipids and lipoproteins during coconut, butter, and safflower diets.

Diet	TC	LDL-C	HDL-C	VLDL-C	TAG
Butter					
Total group	6.8 ± 0.9 (263 ± 33)	4.5 ± 0.8 (175 ± 30)	1.4 ± 0.4 (56 ± 14)	0.65 ± 0.65 (24 ± 25)	2.0 ± 1.3 (177 ± 115)
Male	7.0 ± 1.0 (269 ± 38)	4.7 ± 0.9 (181 ± 35)	1.2 ± 0.2[c] (45 ± 8)	1.0 ± 0.8[b] (38 ± 31)	2.6 ± 1.6[a] (230 ± 142)
Female	6.7 ± 0.7 (258 ± 28)	4.4 ± 0.7 (170 ± 26)	1.7 ± 0.3 (66 ± 10)	0.3 ± 0.2 (13 ± 8)	1.5 ± 0.4 (133 ± 35)
Coconut					
Total group	6.4 ± 0.8 (249 ± 29)	4.2 ± 0.8 (163 ± 29)	1.5 ± 0.4 (57 ± 15)	0.54 ± 0.51 (21 ± 19)	1.8 ± 1.0 (159 ± 89)
Male	6.6 ± 0.9 (255 ± 34)	4.4 ± 0.9 (171 ± 33)	1.2 ± 0.1[c] (45 ± 6)	0.8 ± 0.6[b] (32 ± 22)	2.4 ± 1.1[c] (231 ± 97)
Female	6.3 ± 0.6 (243 ± 24)	4.0 ± 0.6 (156 ± 25)	1.8 ± 0.3 (68 ± 11)	0.3 ± 0.2 (10 ± 6)	1.3 ± 0.3 (115 ± 27)
Safflower					
Total group	6.1 ± 0.8 (233 ± 29)	3.9 ± 0.7 (151 ± 28)	1.4 ± 0.3 (54 ± 13)	0.53 ± 0.54 (20 ± 20)	1.7 ± 1.0 (151 ± 89)
Male	6.2 ± 0.8 (239 ± 30)	4.0 ± 0.6 (155 ± 21)	1.2 ± 0.3[c] (46 ± 10)	0.8 ± 0.6[b] (31 ± 25)	2.3 ± 1.2[b] (204 ± 106)
Female	5.9 ± 0.7 (228 ± 28)	3.8 ± 0.9 (148 ± 33)	1.6 ± 0.3 (62 ± 10)	0.3 ± 0.2 (10 ± 7)	1.3 ± 0.3 (115 ± 27)
Total group comparisons					
Butter v Coconut	$P = 0.001$	$P = 0.001$	$P = 0.17$	$P = 0.13$	$P = 0.01$
Butter v Safflower	$P = 0.001$	$P = 0.001$	$P = 0.25$	$P = 0.02$	$P = 0.02$
Coconut v Safflower	$P = 0.001$	$P = 0.004$	$P = 0.08$	$P = 0.75$	$P = 0.48$
Female comparisons					
Butter v Coconut	$P = 0.003$	$P = 0.005$	$P = 0.15$	$P = 0.09$	$P = 0.01$
Butter v Safflower	$P = 0.001$	$P = 0.001$	$P = 0.02$	$P = 0.06$	$P = 0.005$
Coconut v Safflower	$P = 0.01$	$P = 0.14$	$P = 0.01$	$P = 0.88$	$P = 0.78$
Male comparisons					
Butter v Coconut	$P = 0.01$	$P = 0.07$	$P = 0.83$	$P = 0.33$	$P = 0.44$
Butter v Safflower	$P = 0.001$	$P = 0.006$	$P = 0.51$	$P = 0.13$	$P = 0.16$
Coconut v Safflower	$P = 0.004$	$P = 0.01$	$P = 0.57$	$P = 0.69$	$P = 0.52$

Values are mean ± SD in mmol/l and mg/dl in brackets. Statistics by paired Student's *t*-tests.
[a]Significantly different from females ($P < 0.05$).
[b]Significantly different from females ($P < 0.01$).
[c]Significantly different from females ($P < 0.001$).

Study 2: Mendis et al. 1990

The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidaemic men

Table 2. *Concentrations of plasma lipids and lipoproteins during daily consumption of soya-bean fat or coconut fat by healthy young men*

(Mean values and standard deviations)

Experimental period‡ ...	Baseline		Soya-bean fat		Coconut fat	
	Mean	SD	Mean	SD	Mean	SD
Cholesterol (mmol/l)	4·64	0·37	3·68***	0·42	4·61†††	0·39
Triacylglycerol (mmol/l)	1·42	0·44	1·06**	0·42	1·45	0·41
HDL-C (mmol/l)	1·10	0·25	0·94*	0·26	1·14	0·27
LDL-C (mmol/l)	2·95	0·43	2·27***	0·36	2·84	0·37

HDL-C, high-density-lipoprotein cholesterol; LDL-C, low-density-lipoprotein cholesterol.
Mean values were significantly different from baseline values: * $P < 0·05$, ** $P < 0·01$, *** $P < 0·001$.
Mean values were significantly different from soya-bean fat values; ††† $P < 0·001$.
‡ For details, see p. 548 and Table 1.

Study 3: Müller et al. 2003

The serum LDL/HDL cholesterol ratio is influenced more favourably by exchanging saturated with unsaturated fat than by reducing saturated fat in the diet of women

TABLE 3

Serum lipids and lipoprotein compositions in women at baseline consuming the three test diets[1,2]

	Baseline	HSAFA diet[3]	LSAFA diet[4]	HUFA diet[5]
Total cholesterol, *mmol/L*	4.95 ± 0.71	5.38 ± 0.89[a]	5.13 ± 0.72[b]	4.41 ± 0.72[a,b]
LDL-cholesterol, *mmol/L*	2.99 ± 0.65	3.20 ± 0.77[a]	3.12 ± 0.66[b]	2.52 ± 0.65[a,b]
HDL-cholesterol, *mmol/L*	1.42 ± 0.45	1.69 ± 0.57[a,b]	1.44 ± 0.50[a]	1.44 ± 0.43[b]
VLDL-cholesterol, *mmol/L*	0.53 ± 0.24	0.49 ± 0.20[a]	0.57 ± 0.22[a,b]	0.46 ± 0.16[b]
LDL-/HDL-cholesterol	2.31 ± 0.88	2.10 ± 0.84[a,d]	2.43 ± 0.96[a,b]	1.92 ± 0.78[b,d]
Triacylglycerol, *mmol/L*	1.18 ± 0.54	1.09 ± 0.44[a]	1.27 ± 0.49[a,b]	1.02 ± 0.36[b]
ApoB, *g/L*	0.97 ± 0.20	0.97 ± 0.20[a]	0.99 ± 0.19[b]	0.83 ± 0.18[a,b]
ApoA-I, *g/L*	1.51 ± 0.31	1.68 ± 0.43[a,b]	1.50 ± 0.35[a]	1.50 ± 0.32[b]
ApoB/ApoA-I	0.67 ± 0.20	0.62 ± 0.20[c]	0.69 ± 0.21[c,a]	0.58 ± 0.17[a]

[1] Values are means ± SD, $n = 25$. Means with common superscripts differ: a and b, $P < 0.01$, c, $P < 0.02$ and d, $P < 0.04$.
[2] For fatty acid composition see Table 2.
[3] Diet high in saturated fatty acids.
[4] Diet low in saturated fatty acids.
[5] Diet high in poly- and monounsaturated fatty acids.