Short communication: Daily intake of 125 g of cheese for 2 weeks did not alter amount or distribution of serum lipids or desaturase indexes in healthy adults in an exploratory pilot study

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ABSTRACT

Regular cheese contains saturated fat, consumption of which may negatively influence the amount of serum lipids. The American Dietary Guidelines (<https://health.gov/dietaryguidelines/2015/guidelines/>) recommend consumption of low-fat food. However, we observed a negative association between cheese intake and serum triglycerides and a positive association with high-density lipoprotein cholesterol. Cheese intake was also inversely related to metabolic syndrome and blunted the harmful association of intake of soft drinks with serum lipids. Cheese contains calcium and factors that may inhibit desaturases, thereby partly explaining why cheese might not have negative effects on serum lipids. Thus, opposing forces seem to govern the cheese effect but will any of these prevail? In an exploratory pilot study, 17 healthy subjects participated in a 4-wk crossover trial without washout. During the first 2 wk, 9 subjects were randomly assigned to add 125 g/d of regular cheese to their habitual diet. After 2 wk, cheese intake was discontinued and the subjects were instructed to return to their habitual diet. The other 8 subjects followed their habitual diet during the first 2 wk, and then added 125 g/d of cheese for the next 2 wk. Mean values (mmol/L) before and after 2 wk on habitual (cheese) diet were as follows: serum triglycerides: 0.91 (0.89) and 0.95 (0.91); total cholesterol: 5.25 (5.16) and 5.08 (5.24); low-density lipoprotein cholesterol: 3.18 (3.17) and 3.09 (3.22); and high-density lipoprotein cholesterol: 1.71 (1.64) and 1.61 (1.66). The fatty acid pattern in total serum lipids and desaturase indexes did not change significantly in response to high cheese intake. Thus, an appreciable increase in daily cheese

intake for 2 wk may not alter concentrations of serum lipids, estimates of desaturases, or the distribution of serum fatty acids.

Key words: cheese, serum lipids, desaturase index, human

Short Communication

Saturated fat may negatively influence the amount and distribution of serum lipids, thereby promoting atherosclerosis and risk of cardiovascular diseases. Dietary guidelines in many Western countries recommend decreased intake of saturated fat to prevent such diseases. Regular cheese is a major food item providing saturated fat, and the *American Dietary Guidelines 2015–2020* (USDA-DHHS, 2015) recommend that fat-free or low-fat dairy products should be consumed rather than regular cheese.

Some studies, however, have questioned whether cheese intake is harmful for cardiovascular diseases. For example, in the Swedish Malmö Diet and Cancer cohort, Sonestedt et al. (2011) reported that cheese ingestion was negatively related to such diseases in women. Furthermore, in the cross-sectional Oslo Health Study, involving 18,770 subjects of both sexes and 4 age groups, we found a negative association between cheese intake and serum triglycerides, and a positive association with high-density lipoprotein (HDL) cholesterol (Høstmark et al., 2009). We subsequently reported (Høstmark and Tomten, 2011) that cheese intake was negatively associated with metabolic syndrome, a condition predisposing for diabetes and coronary heart disease. Furthermore, cheese intake seems to attenuate the harmful association between soft drink intake and the risk of metabolic syndrome (Høstmark and Haug, 2012). Based upon epidemiological findings and experiments with liver cells in cultures, we suggest that cheese may contain factors that inhibit Δ^9 -desaturase (Høstmark and Lunde, 2012) and formation of MUFA, which are the preferred fatty acids for triglyceride synthesis (Nakamura and Nara, 2004). Through desaturase inhi-

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bition, cheese intake might decrease hepatic fatty acid synthesis and formation of triglycerides, and decrease hepatic fatty acid incorporation into very low density lipoproteins (**VLDL**), followed by decreased VLDL output from liver to the circulation, and decreased concentration of serum triglycerides.

Thus, opposing forces seem to govern the final influence of cheese upon serum lipids, raising the question of which, if any, will prevail. In the present work, we investigated whether adding an appreciable amount of cheese to the habitual diet might alter the amount and distribution of serum lipids and estimates of fatty acid desaturases.

It is standard practice in clinical trials to have primary and secondary outcome measures and power calculations to obtain, a priori, an estimate of the sample size needed to detect a significant effect. For this crossover trial, we need to know the effect size (**E**) and its standard deviation, **S** (Δ), as well as the Type I error rate (α) and the Type II error rate (β). With our aim we encountered 2 problems: (1) it was hard to prioritize between lipid variables when choosing a primary outcome measure; total cholesterol, low-density lipoprotein (**LDL**) cholesterol, HDL, and triglycerides all deserved the same focus; and (2) for all of these variables, **E** and **S** (Δ) were unknown under the conditions of our trial. Therefore, we performed an exploratory pilot study to obtain the required data. The trial showed the following **S** (Δ) values (mmol/L): triglycerides 0.28, total cholesterol 0.43, LDL cholesterol 0.31, and HDL cholesterol 0.23. However, for all of the variables, the mean **E** ("before" – "after" values) turned out to be below clinical interest; delta values (mmol/L) were as follows: triglycerides –0.02; total cholesterol –0.09; LDL cholesterol –0.05; and HDL cholesterol –0.02. Nevertheless, we carried out post hoc calculations to estimate the hypothetical number of subjects that would be needed to obtain a clinically interesting **E**. With **E** = 0.5 mmol/L for total cholesterol (used as the primary outcome measure), and accepting α = 0.05, β = 0.2, and **S** (Δ) = 0.43, 6 subjects would suffice (<http://sample-size.net/CalcF2v2.php>). For **E** = 0.3 mmol/L, we would need 16 subjects; for **E** = 0.2 mmol/L, 36 subjects; for **E** = 0.1 mmol/L (an effect of questionable clinical interest), 145 subjects. According to these calculations and assuming a hypothetical value for **E** of 0.3 mmol/L (i.e., a 6% change; before – after), 17 subjects would be sufficient.

Thus, we conducted an exploratory pilot study and recruited 18 healthy adults through announcements in employee announcement boards at the Medical Faculty, University of Oslo, Norway, where the data were collected. Complete measurements were obtained in 17 subjects (12 women, 5 men) who participated in a

4-wk crossover trial without washout. We had one test substance only (cheese) and decided by drawing lots (by authors MSHL and VTH) which subjects would be assigned to the cheese period for the first or last 2 wk of the 4-wk trial period. During the first 2 wk, we randomly assigned 9 subjects (2 men, 7 women) to add 125 g/d of cheese (Norwegian Jarlsberg, a Swiss-type cheese) to their regular diet. This cheese contains per 100 g: 1,458 kJ (351 kcal) of energy, 27 g of protein, 27 g of total fat, 0.91 g of C12:0, 2.75 g of C14:0, 7.22 g of C16:0, 2.88 g of C18:0, 0.6 g of *trans*-MUFA, 6.1 g of *cis*-MUFA, 0.36 g of C16:1, 5.5 g of C18:1, 0.6 g of *cis*-PUFA, 0.44 g of C18:2n-6, 0.11 g of C18:3n-3, 0.02 g of C20:3n-6, 0.02 g of C20:4n-6, 0.01 g of C20:5n-3, 0.02 g of C22:5n-3, and 72 mg of cholesterol (Norwegian food table; Matvaretabellen, 2017). Note that values for short-chain fatty acids C4 to C10 are not included in the Norwegian food table.

After 2 wk, cheese intake was discontinued and the subjects were instructed to return to their habitual diet. The rest of the participants (3 men, 5 women) followed their habitual diet during the first 2 wk of the trial and then added 125 g of Jarlsberg cheese daily to their habitual diet for the next 2 wk. The cheese was portioned so that each participant received 14 packages, each containing 125 g of cheese to be used for 1 d. The participants were instructed to keep the cheese refrigerated (2–8°C) throughout the trial, maintain their habitual diet during the 4-wk trial period, and make no changes in level of physical activity or other lifestyle habits. They had to fill in a brief questionnaire for control of cheese intake and other diet habits. This trial had one test substance (cheese) only; the control substance was their habitual diet. A washout period after 2 wk on the habitual diet seemed inappropriate but could have been included after the cheese period. If so, we would have missed control for possible time-related influences during this period. We accordingly chose to omit a washout period. Separate post hoc analyses for each of the 2 control periods showed that there were no significant effects related to the control periods. All 17 subjects received the intended treatment, and results from all subjects were included in the analysis.

Fasting blood samples were taken from an antecubital vein at baseline, after 2 wk, and at the end of the trial in serum separating tubes provided by Furst Medical Laboratories (Oslo, Norway; <http://www.furst.no/for-legekontoret/bestilling-av-utstyr/>). Serum was frozen and kept at –20°C until analyzed. Serum triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, and fasting blood glucose were determined at Furst Medical Laboratories, Oslo, Norway. Determination of fatty acids in total lipid extract of serum was carried out as described previously (Høstmark and Lunde, 2012).

This study complied with all appropriate laws, regulations, and policies governing the use of human subjects in research (registration number S-08876a; The Norwegian National Research Ethics Committee).

Statistical significance of the “before” minus “after” values during the control and cheese periods, respectively, was evaluated using a paired Student's *t*-test. We also used a paired Student's *t*-test to compare differences between the changes within the control period and within the cheese period.

The significance level was set at $P < 0.01$. We performed 48 comparisons; if we had chosen $P = 0.05$, then 1 out of 20 tests could falsely have been classified as significant (~ 2 of 48 tests). On the other hand, with Bonferroni correction, the significance level would be $P = 0.001$ ($0.05/48$). To balance the risk of making Type 1 or Type 2 errors, we chose $P < 0.01$. The SPSS 25.0 software package (IBM Corp., Armonk, NY) was used for statistical analyses.

We described participants by intervention sequence; that is, those assigned by drawing lots to continue their habitual diet during the first 2 wk (group A; 3 men, 5 women), and those assigned to ingest 125 g of cheese daily in the first 2 wk (group B; 2 men 7 women). The mean ($\pm$ SD) age in groups A and B was 44 ± 12 and 45 ± 14 yr. Body weight and height were 82 ± 17 and 72 ± 12 kg and 1.74 ± 0.12 and 1.72 ± 0.09 m, respectively. Body mass index was 28 ± 6 and 24 ± 2 , and length of education was 18 ± 2 and 17 ± 4 yr. Two subjects in group B were smokers; the remaining subjects (of both groups) were nonsmokers. Habitual cheese intake (days per week, mean $\pm$ SD) at breakfast, lunch, and supper was 3 ± 1 , 3 ± 1 , 3 ± 1 in group A, and 4 ± 1 , 3 ± 1 , 4 ± 1 in group B, respectively. Only one of the participants (group A) reported use of regular sucrose-cola (1–6 glasses per week); the others reported sucrose cola intake of “never/rarely.”

According to the self-reported questionnaire, all participants complied with the instruction to add 125 g/d of Jarlsberg cheese to their habitual diet, and to make no changes otherwise in their diet or change their level of physical activity.

Mean values ($\pm$ SE, mmol/L) before and after 2 wk on the habitual diet for serum triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol are given in Table 1. We tested whether the significant ($P = 0.004$) 5.8% reduction in HDL in the control period might be due to a carryover effect from the preceding cheese intake period in half of the subjects. There was, however, no HDL-raising effect of cheese (see below). Analyzing each control group separately, we observed a nonsignificant 5.2% reduction in HDL cholesterol in the 9 subjects who had consumed cheese in the previous 2 wk (1.73 ± 0.12 vs. 1.64 ± 0.13 ; $t = 2.53$, $P = 0.035$).

We found a nonsignificant 7.1% reduction in HDL cholesterol during the control period in the 8 subjects with no preceding cheese intake (1.68 ± 0.18 vs. 1.56 ± 0.15 ; $t = 2.18$, $P = 0.065$).

Values before and after 2 wk of cheese intake for serum triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol are given in Table 1. None of the “after” values differed significantly from corresponding “before” values, and “before – after” differences in the cheese period did not differ significantly from those in the control period (P_b in Table 1).

Considering all subjects together, we found that mean values (all mmol/L) at baseline, after 2 wk, and after 4 wk were as follows: triglycerides: 0.89, 0.92, and 0.95, respectively; total cholesterol: 5.15, 5.26, and 5.07; LDL cholesterol: 3.13, 3.22, and 3.09; HDL cholesterol: 1.69, 1.65, and 1.62. We found no sex differences in the concentration of corresponding variables (results not shown). Additionally, we did not find any significant effect of cheese consumption on the fatty acid pattern in total serum lipids (Table 1), indexes used to estimate fatty acid desaturases, or total sum of fatty acids. Values before and after the control period were 0.64 ± 0.02 and 0.61 ± 0.02 , respectively, for the Δ^9 -desaturase estimate ($18:1n-9$)/(18:0) ratio; 0.42 ± 0.04 and 0.46 ± 0.03 for the Δ^6/Δ^5 -desaturase estimate ($20:4n-6$)/(18:2n-6) ratio; and 1.21 ± 0.06 and 1.15 ± 0.06 g/L for the sum of fatty acids. Corresponding values before and after the cheese intake period were 0.61 ± 0.02 and 0.62 ± 0.02 for the ($18:1n-9$)/(18:0) ratio; 0.45 ± 0.03 and 0.42 ± 0.03 for the ($20:4n-6$)/(18:2n-6) ratio; and 1.17 ± 0.26 and 1.15 ± 0.25 g/L for the sum of fatty acids.

This small study suggests that addition of 125 g of regular cheese daily with the habitual diet for 2 wk may not have any effect on amount and distribution of serum lipids, as estimated by traditional lipoprotein components such as triglycerides and total, HDL, and LDL cholesterol. Furthermore, the fatty acid pattern in serum total lipids and estimates of fatty acid desaturases may not become significantly altered by high cheese intake.

Dietary guidelines recommend a decrease in intake of saturated fat (USDA-DHHS, 2015). The recommendation relates to the view that saturated fat may increase serum total and LDL cholesterol, and thereby may increase the risk of cardiovascular diseases. Our participants ingested about 34 g of extra fat from cheese daily, raising the question of whether high cheese intake influences the amount of serum lipids. In fact, we found no effect of high daily cheese intake for 2 wk on the concentration and distribution of serum lipids. Our results are in line with previous studies suggesting that the effect of cheese on blood lipids may be less consistent than expected from the dietary guidelines, especially as

Table 1. Concentration (mean $\pm$ SE) of serum lipids in healthy adults before and after 2 wk on habitual diet (control period) or on the same diet plus daily intake of 125 g of cheese (cheese period), $n = 17^1$

Lipid	Control period			Cheese period			
	Before	After	P_w	Before	After	P_w	P_b
Triglycerides, mmol/L	0.91 $\pm$ 0.12	0.95 $\pm$ 0.13	0.31	0.89 $\pm$ 0.10	0.91 $\pm$ 0.12	0.77	0.84
Total cholesterol, mmol/L	5.25 $\pm$ 0.27	5.08 $\pm$ 0.26	0.11	5.16 $\pm$ 0.28	5.25 $\pm$ 0.25	0.41	0.16
LDL, ² mmol/L	3.18 $\pm$ 0.22	3.09 $\pm$ 0.21	0.35	3.17 $\pm$ 0.22	3.22 $\pm$ 0.20	0.54	0.37
HDL, ³ mmol/L	1.71 $\pm$ 0.10	1.61 $\pm$ 0.10	0.00 ⁴	1.64 $\pm$ 0.10	1.66 $\pm$ 0.10	0.68	0.08
Fatty acids, ⁵ % of total							
14:0	0.2 $\pm$ 0.1	0.1 $\pm$ 0.16	0.37	0.2 $\pm$ 0.1	0.3 $\pm$ 0.1	0.28	0.23
16:0	32.2 $\pm$ 0.3	32.3 $\pm$ 0.3	0.72	32.3 $\pm$ 0.3	32.4 $\pm$ 0.2	0.56	0.96
16:1n-7	0.1 $\pm$ 0.1	0.2 $\pm$ 0.1	0.58	0.1 $\pm$ 0.1	0.0 $\pm$ 0.0	0.46	0.49
18:0	15.0 $\pm$ 0.2	14.8 $\pm$ 0.2	0.64	14.9 $\pm$ 0.3	15.0 $\pm$ 0.2	0.72	0.66
18:1n-9	9.7 $\pm$ 0.3	9.0 $\pm$ 0.3	0.08	9.0 $\pm$ 0.3	9.3 $\pm$ 0.3	0.37	0.12
18:2n-6	22.1 $\pm$ 0.6	20.8 $\pm$ 0.8	0.17	21.2 $\pm$ 0.8	22.1 $\pm$ 0.6	0.31	0.21
20:4n-6	9.1 $\pm$ 0.4	9.2 $\pm$ 0.4	0.57	9.2 $\pm$ 0.4	9.0 $\pm$ 0.4	0.46	0.49
20:5n-3	2.5 $\pm$ 0.3	3.0 $\pm$ 0.4	0.24	2.6 $\pm$ 0.3	2.4 $\pm$ 0.3	0.43	0.25
22:6n-3	9.4 $\pm$ 0.3	10.5 $\pm$ 0.5	0.01	10.4 $\pm$ 0.6	9.4 $\pm$ 0.3	0.04	0.01
(18:1)/(18:0) ⁶	0.64 $\pm$ 0.02	0.61 $\pm$ 0.02	0.20	0.61 $\pm$ 0.02	0.62 $\pm$ 0.02	0.59	0.26
(20:4)/(18:2) ⁶	0.42 $\pm$ 0.04	0.46 $\pm$ 0.03	0.26	0.45 $\pm$ 0.03	0.42 $\pm$ 0.03	0.36	0.29
Sum ⁷	1.21 $\pm$ 0.06	1.15 $\pm$ 0.06	0.23	1.17 $\pm$ 0.06	1.15 $\pm$ 0.06	0.56	0.62

¹ P_w = P -value for comparison of before and after values in either the control period or the cheese period; P_b = P -value for comparison of control and cheese periods.

²Low density lipoprotein cholesterol.

³High density lipoprotein cholesterol.

⁴HDL was significantly decreased ($P = 0.004$) during the control period. Note that some values appear as zero due to the number of decimals.

⁵14:0 = myristic acid; 16:0 = palmitic acid; 16:1n-7 = palmitoleic acid; 18:0 = stearic acid; 18:1n-9 = oleic acid; 18:2n-6 = linoleic acid; 20:4n-6 = arachidonic acid; 20:5n-3 = eicosapentaenoic acid; 22:6n-3 = docosahexaenoic acid.

⁶(18:1)/(18:0) is an estimate of Δ^9 -desaturase; (20:4)/(18:2) is an estimate of Δ^6/Δ^5 -desaturase.

⁷Sum (g/L) of all measured fatty acids.

it relates to the risk of cardiovascular diseases (Sonestedt et al., 2011). Indeed, cheese intake may beneficially influence the risk of diabetes (Sluijs et al., 2012; Struijk et al., 2013), which may be a precursor of cardiovascular diseases.

In our opinion, this experimental approach might reflect a real-life situation encountered when a person decides to increase their intake of cheese. Mechanisms to explain the outcomes are not clear, but we propose 2 reasons why cheese might not increase blood lipids in spite of the saturated fat content. First, cheese has a high calcium content; calcium can bind fatty acids in the gut and lead to increased fecal fat excretion, thereby causing decreased fat absorption (Kjølbaek et al., 2017). Furthermore, inhibition of fatty acid desaturation by some cheese compound(s) might work to decrease serum triglycerides. Desaturases represent a key metabolic point in lipid metabolism (Dobrzyn and Ntambi, 2005a,b), and fatty acid desaturation has been reported to be associated with cardiovascular mortality (Warensjö et al., 2008). In fasted animals, triglycerides are mainly carried in VLDL (Mayes, 2000), and VLDL components such as triglycerides, cholesterol esters, and phospholipids preferably contain MUFA (Nakamura and Nara, 2004). Stearoyl-CoA desaturase is the rate-limiting enzyme for synthesis of these fatty acids,

and mice lacking the enzyme have decreased hepatic lipogenesis and lower plasma triglyceride concentrations (Dobrzyn and Ntambi, 2005a,b). In a dietary trial in rats, we found that ingestion of sucrose-cola appreciably increased desaturase estimates, thereby providing one mechanism by which soft drink intake might increase serum triglycerides (Høstmark et al., 2010). Also, cheese intake blunted the association between intake of colas and the risk of metabolic syndrome in humans (Høstmark and Haug, 2012). Additionally, our data from a cross-sectional study, supported by experiments with cultures of human hepatoma cells, suggest that cheese may contain factors that inhibit desaturases (Høstmark and Lunde, 2012).

Cheese contains CLA (Mushtaq et al., 2010), and a CLA isomer (*trans*-10,*cis*-12) has been shown to suppress Δ^9 -, Δ^6 -, and Δ^5 -desaturases in HepG2 cells (Eder et al., 2002); however, we have no data to support this inhibition of desaturases by cheese via CLA. The results of this small study in healthy subjects do not support the view that fatty acid desaturase activities are altered by cheese intake. We cannot explain the discrepancy between the present study and our previous results concerning desaturases. However, the present trial was small and of short duration. We cannot exclude the possibility of compensatory diet changes,

although compliance records do not favor this suggestion. Finally, our previous hypothesis that desaturases may be inhibited by cheese should be verified.

We found that serum HDL cholesterol levels were as high in men as in women, both before and after the cheese intake period. This observation was unexpected because men generally have lower HDL levels than women. We have no obvious explanation for this observation, but selection bias could be involved. We did not observe any harms or unintended effects of the cheese intake on the participants.

The present results seem to suggest that an appreciable increase in daily cheese intake may not have any negative effect on the concentration of serum lipids, distribution of fatty acids, or indexes estimating desaturases. However, because the trial was small and of short duration, its generalizability is limited and more extensive studies are required to verify the results.

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REFERENCES

- Dobrzyn, A., and J. M. Ntambi. 2005a. The role of stearoyl-CoA desaturase in the control of metabolism. *Prostaglandins Leukot. Essent. Fatty Acids* 73:35–41.
- Dobrzyn, A., and J. M. Ntambi. 2005b. Stearoyl-CoA desaturase as a new drug target for obesity treatment. *Obes. Rev.* 6:169–174.
- Eder, K., N. Slomma, and K. Becker. 2002. *Trans*-10, *cis*-12 conjugated linoleic acid suppresses the desaturation of linoleic and alpha-linolenic acids in HepG2 cells. *J. Nutr.* 132:1115–1121.
- Høstmark, A. T., and A. Haug. 2012. Does cheese intake blunt the association between soft drink intake and risk of the metabolic syndrome? Results from the cross-sectional Oslo Health Study. *BMJ Open* 2:e001476. <https://doi.org/10.1136/bmjopen-2012-001476>.
- Høstmark, A. T., A. Haug, S. E. Tomten, D. S. Thelle, and A. Mosdøl. 2009. Serum HDL cholesterol was positively associated with cheese intake in the Oslo Health Study. *J. Food Lipids*. <https://doi.org/10.1111/j.1745-4522.2009.01134.x>.
- Høstmark, A. T., and M. S. H. Lunde. 2012. Cheese can inhibit indexes that estimate fatty acid desaturation. Results from the Oslo Health Study and from experiments with human hepatoma cells. *Appl. Physiol. Nutr. Metab.* 37:31–39.
- Høstmark, A. T., M. S. H. Lunde, and E. Eilertsen. 2010. Does an acid load promote liver desaturases and increase serum lipids? *Clin. Rev. Opin.* 2:8–16.
- Høstmark, A. T., and S. E. Tomten. 2011. The Oslo health study: Cheese intake was negatively associated with the metabolic syndrome. *J. Am. Coll. Nutr.* 30:182–190.
- Kjølbæk, L., J. K. Lorenzen, L. H. Larsen, and A. Astrup. 2017. Calcium intake and the associations with faecal fat and energy excretion, and lipid profile in a free-living population. *J. Nutr. Sci.* 6:e50. <https://doi.org/10.1017/jns.2017.55>.
- Matvaretabellen. 2017. Norwegian food table. Accessed Jan. 29, 2018. <http://www.matvaretabellen.no/ost-g1.4/jarlsberg-ost-01.035>.
- Mayes, P. A. 2000. Lipid transport and storage. Pages 268–284 in *Harper's Biochemistry*. R. K. Murray, D. K. Granner, P. A. Mayes, and V. W. Rodwell, ed. McGraw-Hill, New York, NY.
- Mushtaq, S., H. E. Mangiapane, and K. A. Hunter. 2010. Estimation of *cis*-9, *trans*-11 conjugated linoleic acid content in UK foods and assessment of dietary intake in a cohort of healthy adults. *Br. J. Nutr.* 103:1366–1374.
- Nakamura, M. T., and T. Y. Nara. 2004. Structure, function, and dietary regulation of delta6, delta5, and delta9 desaturases. *Annu. Rev. Nutr.* 24:345–376.
- Sluijs I., N. G. Forouhi, J. W. Beulens, Y. T. van der Schouw, C. Agnoli, L. Arriola, B. Balkau, A. Barricarte, H. Boeing, H. B. Bueno-de-Mesquita, F. Clavel-Chapelon, F. L. Crowe, B. de Lauzon-Guillain, D. Drogan, P. W. Franks, D. Gavrila, C. Gonzalez, J. Halkjaer, R. Kaaks, A. Moskal, P. Nilsson, K. Overvad, D. Palli, S. Panico, J. R. Quirós, F. Ricceri, S. Rinaldi, O. Rolandsson, C. Sacerdote, M. J. Sánchez, N. Slimani, A. M. Spijkerman, B. Teucher, A. Tjønneland, M. J. Tormo, R. Tumino, D. L. van der A, S. J. Sharp, C. Langenberg, E. J. Feskens, E. Riboli, N. J. Wareham, and InterAct Consortium. 2012. The amount and type of dairy product intake and incident type 2 diabetes: results from the EPIC-InterAct Study. *Am. J. Clin. Nutr.* 96:382–390.
- Sonestedt, E., E. Wirfält, P. Wallström, B. Gullberg, M. Orho-Melander, and B. Hedblad. 2011. Dairy products and its association with incidence of cardiovascular disease: The Malmö diet and cancer cohort. *Eur. J. Epidemiol.* 26:609–618. <https://doi.org/10.1007/s10654-011-9589-y>.
- Struijk, E. A., A. Heraclides, D. R. Witte, S. S. Soedamah-Muthu, J. M. Geleijnse, U. Toft, and C. J. Lau. 2013. Dairy product intake in relation to glucose regulation indices and risk of type 2 diabetes. *Nutr. Metab. Cardiovasc. Dis.* 23:822–828.
- USDA-DHHS. 2015. Dietary Guidelines for Americans 2015–2020. USDA/Department of Health and Human Services, Washington, DC. <https://health.gov/dietaryguidelines/2015/guidelines/>.
- Warensjö, E., J. Sundström, B. Vessby, T. Cederholm, and U. Risérus. 2008. Markers of dietary fat quality and fatty acid desaturation as predictors of total and cardiovascular mortality: A population-based prospective study. *Am. J. Clin. Nutr.* 88:203–209.