BMJ open
accessible medical research

Does cheese intake blunt the association between soft drink intake and risk of the metabolic syndrome? Results from the cross-sectional Oslo Health Study

Arne Torbjørn Høstmark,[1] Anna Haug[2]

[1]Section of Preventive Medicine and Epidemiology, University of Oslo, Oslo, Norway
[2]Department of Animal and Aquacultural Sciences, The Norwegian University of Life Sciences, Ås, Norway

Correspondence to
Arne T Høstmark;
a.t.hostmark@medisin.uio.no

ABSTRACT

Objectives: A high soft drink intake may promote, whereas intake of cheese may reduce risk of the metabolic syndrome (MetS), but will cheese intake blunt the soft drink versus MetS association?

Design: Cross-sectional study.

Setting: The Oslo Health Study.

Participants: Among the 18 770 participants of the Oslo Health Study there were 5344 men and 6150 women having data on cheese and soft drink intake and on risk factors for MetS, except for fasting glucose. The *MetSRisk* index=the weighted sum of triglycerides (TG), systolic blood pressure, diastolic blood pressure, waist circumference and body mass index (BMI) divided by high-density lipoprotein (HDL) were used as a combined risk estimate to examine the cheese/soft drink versus MetS interaction, and the *SumRisk* index was used to assess whether increasing intake of soft drinks/cheese would include an increasing *number* of MetS factors being above the cut-off values. We analysed the data using non-parametric correlation and analysis of covariance (ANCOVA).

Results: In all three groups of soft drink intake (seldom/rarely, 1–6 glasses/week, ≥1 glass/day), there was a negative cheese versus MetSRisk correlation (p≤0.003), but in the highest intake group the influence of cheese seemed to level off, suggesting interaction. However, there was no interaction between cheese and soft drinks within the fully adjusted models. Conversely, at all four levels of cheese intake, MetSRisk increased with an increasing intake of soft drinks (p≤0.001 at all cheese levels). Similar associations were found with the SumRisk index. When controlling for a large number of covariates (eg, sex, age group, smoking, education, physical activity, intake of fruits/berries and vegetables), the above associations prevailed.

Conclusions: Cheese intake blunted the association between soft drink intake and MetS, an influence possibly related to fatty acid desaturation, or to undetected covariates.

ARTICLE SUMMARY

Article focus

- High soft drink intake may promote metabolic syndrome (MetS), a prediabetic condition involving increased waist circumference, elevated fasting blood glucose, increased blood pressure and dyslipidaemia.
- There are reports suggesting that cheese intake may reduce the risk of MetS, a view apparently not in line with current diet guidelines which recommend a reduced cheese intake to reduce saturated fat intake.
- It is not known whether the association between soft drink intake and MetS may be counteracted by increased cheese intake.

Key message

- In the cross-sectional Oslo Health Study, increased intake of cheese seemed to blunt the association between soft drink intake and MetS, also when controlling for a large number of covariates.

Strengths and limitations of this study

- The cheese versus soft drink antagonism upon the MetS prevailed when controlling for many covariates.
- Cross-sectional data are not suitable for assessing causal relationships.

INTRODUCTION

Metabolic syndrome (MetS) is a prediabetic condition characterised by a cluster of risk factors, including central obesity, raised serum glucose levels, triglycerides (TG) and blood pressure and reduced serum high-density lipoprotein (HDL) cholesterol.[1] Persons with MetS are at high risk not only for developing type 2 diabetes (T2D), but also for coronary heart disease.[1]

The MetS definition implies cut-off levels of the various risk factors. However, we would

expect alterations in the levels of one or more MetS-related components, preceding the appearance of complete MetS. We previously hypothesised that such metabolic alterations might quite possibly be more easily detected by an index summing up the contribution of individual MetS factors. Furthermore, we anticipated that there might also be variation among subjects concerning the extent to which a certain MetS factor, for example, blood pressure, serum lipids or waist circumference, would be altered before the appearance of complete MetS. If so, we should expect associations between dietary factors influencing MetS and the *number* of MetS variables being above the threshold value (eg, serum lipids and blood pressure), and such associations were recently observed.[2][3]

Soft drink intake seems to increase the MetS risk.[2–6] On the other hand, cheese intake can be positively associated with HDL and negatively associated with TG,[7] and with indices estimating the level of several MetS components, and also with the complete MetS.[3] These findings raise the question of whether an increased intake of cheese might counteract the negative association between soft drinks and MetS. The aim of the present work was to elucidate this question, using data from the cross-sectional Oslo Health Study, keeping in mind that this type of material does not allow clarification of whether the associations are causal ones.

MATERIALS/SUBJECTS AND METHODS
Main project
In 2000–2001, the Oslo Health Study was conducted under the joint collaboration of the Norwegian Institute of Public Health, the University of Oslo and the Municipality of Oslo. The study population included all individuals in Oslo County born in 1970, 1960 and 1955, 1940–1941 and 1924–1925. At the time of the data collection, the subjects were 30, 40, 45, 59–60 or 75–76 years of age. A total of 18 770 individuals (45.9% of the invited) participated. The responders consisted of 8404 men (42.4% of the invited) and 10 366 women (49.3% of the invited) who attended the physical examination and/or filled in at least one of the questionnaires. The response rate did not seem to be related to self-reported health, smoking, body mass index (BMI) or mental health as the participants differed only slightly from the estimated prevalence values in the target population.[8]

At the screening station, a simple physical examination was conducted: A venous non-fasting blood sample was analysed for serum total cholesterol, HDL cholesterol, glucose and TG. Automatic device (DINAMAP) measured pulse, systolic blood pressure and diastolic blood pressures. Body weight, height and waist–hip ratio (cm) were measured with a standard procedure according to the protocol. For further details, see: http://www.fhi.no/hubro-en. The study protocol was placed before the Regional Committee for Medical Research Ethics and approved by the Norwegian Data Inspectorate. The

study has been conducted in full accordance with the World Medical Association Declaration of Helsinki.

Population sample in the present investigation
This study involved three age groups: 30; 40 and 45 years; and 59–60 years at inclusion. Subjects aged 75–76 years could not be included since they had not been asked about the intake of colas. It was required that all the subjects had data on the frequency of cola or non-cola soft drink intake, on cheese intake and on serum TG and HDL cholesterol. Of the 18 770 participants in the complete Oslo Health Study there were 11 494 respondents (5344 men and 6150 women) having all the required data, and this population sample was used in the present analyses. The number of subjects with missing data were 3302 for soft drinks, 3191 for cheese, 3256 for TG and HDL cholesterol, 3225 for blood pressure and 3135 for waist circumference.

Questions about intake frequency of food items and beverages had 6 and 5 response levels, respectively. The specific question about soft drink intake was: 'How often do you usually drink ..?', with the response alternatives: seldom/never; 1–6 glasses/week; 1 glass/day; 2–3 glasses/day and 4 glasses or more per day. The question about cheese intake was: 'How often do you usually eat cheese (all kinds), with the response alternatives: seldom/never, 1–3 times/month, 1–3 times/week, 4–6 times/week, 1–2 times/day and 3 times or more per day. To obtain a reasonable number of subjects, for soft drinks we made three approximate intake groups: 0.1 (for never/rarely), 3.5 and 7 glasses/week, and for cheese four groups: intake: 0.5, 2, 5 and 11 times/week.

Statistical methods
Calculation of indices used to estimate risk of MetS
First, we calculated the prevalence of subjects with complete MetS, using the International Diabetes Federation (IDF) definition,[1] except for fasting glucose which was unavailable. According to the IDF,[1] complete MetS requires: waist circumference ≥80 cm for women and ≥94 cm for men, and two of the following risk factors: TG >1.7 mmol/l, HDL cholesterol <1.03 mmol/l for men and <1.29 mmol/l for women, systolic blood pressure >130 mm Hg, diastolic blood pressure >85 mm Hg or fasting glucose >5.6 mmol/l. Then, we used the previously reported compound MetS risk estimates, MetSRisk and SumRisk.[2][3] In brief, *MetSRisk*=TG (mmol/l)+ 0.01*SBP (systolic blood pressure) (mm Hg)+0.01*DBP (diastolic blood pressure) (mm Hg)+0.01*waist (cm)+ 0.05*BMI (kg/m^2) divided by HDL (mmol/l). Thus, the index is made as a sum of weighted values of variables indicating an increased risk of MetS, diabetes and cardiovascular diseases (CVD) (ie, BMI, waist circumference, diastolic and systolic blood pressure and TG) divided by HDL cholesterol, which protects against MetS. The adjustments of factors in the MetSRisk expression were weighted to give similar numeric values so as

to prevent some of the components from dominating the final score.

Next, we calculated the variable *SumRisk* so as to provide increasing number of individual risk *requirements* for MetS. Thus, if a MetS risk factor was above the IDF-cut-off value, it was given the value 1 and 0 if not present. We calculated SumRisk so as to identify the presence of 0, 1, 2, 3, 4 or 5 MetS requirements. Note that the SumRisk definition does not strictly correspond to the MetS definition, since each of the five single requirements will contribute equally to the SumRisk score (ie, TG, HDL cholesterol, systolic blood pressure, diastolic blood pressure and waist circumference). The associations were first studied using non-parametric correlations (Spearman). For multiple comparisons, we used one-way analysis of variance (ANOVA), with Bonferroni correction.

With analysis of covariance (ANCOVA), we studied the association between the dependent variables, MetSRisk and SumRisk, and the frequencies of soft drink and cheese intake, using three models: Model 1 controlling for sex and age; Model 2=Model 1+time since the last meal (h), made to control for an effect of the meal upon serum TG; Model 3=Model 2+adjustments for the frequency of intake of fatty fish (six levels), coffee (cups/day), alcohol (eight levels), smoking (never, current/previous), leisure time physical activity (four levels), years at school and birthplace. Finally, we used logistic regression to study the association between complete MetS as defined above, and soft drink (cheese) intake, controlling for the same factors as in the ANCOVA analyses, and also adding to the list of independents of the product of intake frequency of soft drinks and cheese. SPSS V.18.0 was used for the statistical analyses, and the significance level was p<0.05.

RESULTS
Characteristics of the study sample
Background characteristics of the study population are given in table 1.

With increasing age group, years at school decreased, whereas BMI, percentage of subjects reporting alcohol intake 1–7 times/week, percentage of subjects with MetS

Table 1 Characteristics of the study sample

	Men			**Women**		
Age (years)	30	40+45	59–60	30	40+45	59–60
N	1628	2345	1371	1892	2824	1434
Education*	15	14	13	16	14	12
BMI (kg/m^2)	25.7	26.4	27.2	23.9	25.2	26.2
Daily smokers†	23.3	32.4	25.1	24.2	34.1	24.3
Alcohol users†	54.4	59.8	65.3	35.7	48.5	49.7
Physical activity†	29.4	32.3	31.4	27.4	30.4	32.4
Percentage with MetS‡	14.8	26.1	36.6	6.0	16.1	29.0
MetSRisk§	5.1	5.5	5.2	3.3	3.6	3.7
SumRisk¶	1.8	2.2	2.6	0.7	1.2	1.9
Diabetes**	0.4	1.6	5.3	0.5	1.9	2.4
Hypertension††	9.5	31.0	59.0	2.0	11.4	35.7
Antihypertensives‡‡	1.2	4.3	20.4	1.0	3.9	16.9
Cholesterol lowering§§	1.1	3.6	14.4	0.7	1.8	8.8
Coronary artery disease¶¶	0.1	0.4	3.4	0.0	0.3	2.4
Lipid disturbances***	46.6	52.1	50.4	24.8	31.0	36.6
Serum glucose (mmol/l)	5.1	5.4	5.8	4.9	5.2	5.4
Triglycerides (TG) (mmol/l)	1.8	2.0	1.9	1.1	1.2	1.5
Total chol (mmol/l)	5.1	5.7	6.0	4.9	5.3	6.1
HDL chol (mmol/l)	1.3	1.3	1.4	1.6	1.6	1.7

*Years at school, mean values.
†Percentages; for smoking, those reporting daily use; for alcohol, intake 1–7 times per week; for physical activity, leisure time physical activity 1–2 h/week.
‡Calculation was based upon the IDF[1] definition, but fasting glucose values were not available.
§MetSRisk = (TG+0.01*SBP+0.01*DBP+0.01*waist+0.05* BMI)/HDL, in which BMI, body mass index (kg/m^2); HDL, high-density lipoprotein cholesterol (mmol/l); SBP and DBP, systolic and diastolic blood pressure (mm Hg), respectively; TG, serum triglycerides (mmol/l); waist, waist circumference in cm.
¶For women, *SumRisk*=1 (if TG≥1.7 mmol/l)+1 (if HDL<1.29 mmol/l)+1 (if SBP>130 mm Hg)+1 (if DBP>85 mm Hg)+1 (if waist ≥ 80 cm). For men, *SumRisk*=1(if TG≥1.7 mmol/l)+1 (if HDL<1.03 mmol/l)+1(if SBP>130 mm Hg)+1 (if DBP>85 mm Hg)+1(if waist ≥ 94 cm).
**% reporting diabetes.
††% with blood pressure>130/85 mm Hg.
‡‡% using antihypertensives.
§§% using cholesterol-lowering drugs.
¶¶% with myocardial infarction or angina pectoris.
***% with lipid disturbances; that is, for men TG≥1.7 or HDL<1.03 mmol/l (men), and for women TG≥1.7 or HDL<1.29 mmol/l.

as well as the MetSRisk index increased. As expected, there was also an age-related increase in percentage subjects with diabetes and high blood pressure and treatment for these. For smoking, physical activity and the MetSRisk index there were no consistent age-related changes. In all age groups women had lower BMI, percentage alcohol users, percentage of subjects with MetS and lower mean SumRisk and MetSRisk values as compared with men. Women also had lower percentage of subjects with diabetes and hypertension, and a lower relative number of subjects treated for these conditions. In the cut-off levels used by the IDF[1] there was a great proportion with lipid disturbances, especially among men. Mean values for serum glucose and lipids were considered within normal limits in non-fasting samples.

Bivariate correlations in the whole material
As shown in figure 1, there was an inverse relationship between the MetSRisk index and cheese intake frequency, observed at all three levels of soft drink intake. In subjects with the highest level of soft drink intake (1 glass or more per day), the influence of cheese seemed to level off. Rs values appear in the figure text. Similar observations were
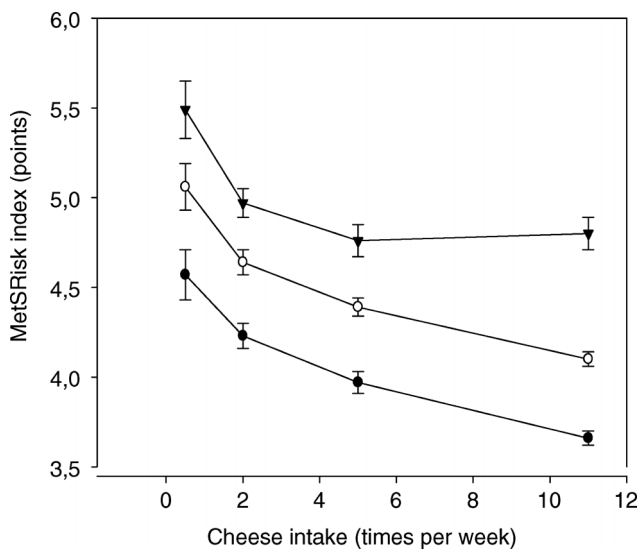

Figure 1 Relationship between the intake frequency of cheese and the MetSRisk index, as influenced by three levels of soft drink intake. The MetSRisk index is defined in the legend to table 1. Numbers of men with cheese intake 0.5, 2, 5 and ≥11 times/week were, in subject reporting soft drink intake never/rarely (filled circles): 303, 768, 942 and 1627; in subjects with soft drink intake 1–6 glasses/week (open circles): 427, 1418, 1686, 2004; and in those with soft drink intake ≥1 glass/day (filled triangles): 280, 691, 656 and 692. Rs values for cheese vs MetSRisk in the three soft drink intake groups: −0.151(p<0.001, n=3640); −0.128 (p<0.001, n=5535); −0.083 (p<0.001, n=2319). Conversely, Rs values for soft drink intake versus MetSRisk for the four cheese groups are: 0.132 (p=0.001, n=1010), 0.112 (p<0.001, n=2877), 0.129 (p<0.001, n=3284) and 0.205 (p<0.001, n=4323), respectively. Mean values±SEM; Note broken y-axis.
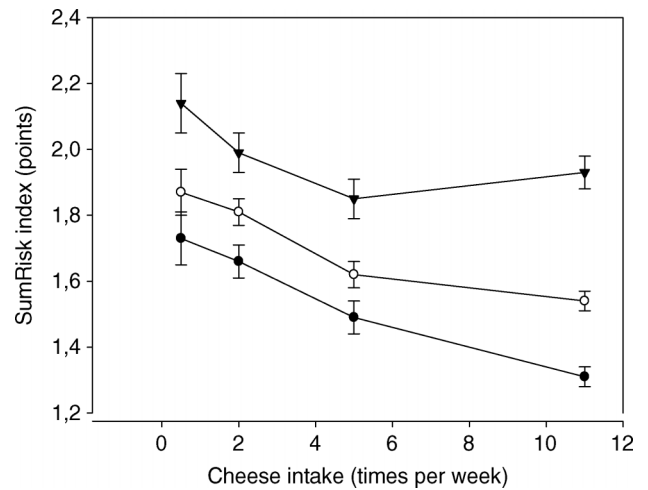

Figure 2 Relationship between the intake frequency of cheese and the SumRisk index, as influenced by three levels of soft drink intake. The SumRisk index is defined in the legend to table 1. Filled circles=soft drink intake never/rarely; open circles=soft drink intake 1–6 glasses/week; filled triangles: soft drink intake ≥1 glass/day. Numbers in the various groups are as in figure 1. Rs values for cheese vs SumRisk in the three soft drink intake groups: −0.108 (p<0.001, n=3640); −0.084 (p<0.001, n=5535); −0.038 (p=0.065, n=2319). Conversely, Rs values for soft drink intake vs SumRisk for the four cheese groups were: 0.102 (p=0.001, n=1010), 0.082 (p<0.001, n=2877), 0.084 (p<0.001, n=3284) and 0.140 (p<0.001, n=4323), respectively. Mean values±SEM; Note broken y-axis.

made with the SumRisk index (figure 2). A 35 (40)% cheese-related reduction in the MetSRisk (SumRisk) index was found when comparing subjects with soft drink intake ≥1 glass/day and rare intake of cheese with those reporting rare intake of soft drinks and cheese intake ≥1–2 times/day.

Bivariate correlations in each sex and age group
The soft drink intake frequency correlated positively with the MetSRisk index, and also with the SumRisk index (table 2). In contrast, cheese intake correlated negatively with SumRisk (p<0.001 in all groups) and with SumRisk, except in young men and women.

ANCOVA Results
Controlling for sex and age group, there was a significant association between MetSRisk (dependent variable) and cheese (F=28.2, p<0.001), and soft drink intake (F=61.2, p<0.001) and a significant interaction between cheese and soft drinks (F=2.16, p=0.044). Adding length of education, leisure time physical activity, intake of fruit/berries, fatty fish, fruit juice, cod liver oil (used by many Norwegians) and birthplace to the covariate list, the negative associations prevailed: for MetSRisk versus cheese: F=4.9, p=0.002 and for MetSRisk versus soft drink intake: F=24.1, p<0.001). When controlling for this

Table 2 Spearman correlation coefficients for association between the intake frequency of soft drinks (cheese) and indices used to assess MetS risk

	Men			Women		
Age group (years)	30	40+45	59–60	30	40+45	59–60
n	1628	2345	1371	1892	2824	1434
Soft drinks						
MetSRisk	0.157*	0.137*	0.081*	0.114*	0.193*	0.121*
SumRisk	0.159*	0.136*	0.088*	0.045**	0.167*	0.127*
Cheese						
MetSRisk	−0.069*	−0.096*	−0.083*	−0.091*	−0.102*	0.144*
SumRisk	−0.039	−0.080*	−0.079*	−0.016	−0.104*	0.137*

The indices MetSRisk and SumRisk are defined in the legend to table 1.
*p<0.01.
**p<0.05.

large number of covariates there was no significant interaction between cheese and soft drinks (F=1.5, p=0.158).

Using SumRisk index as the dependent variable, and controlling for sex and age group, there was a significant association with cheese intake (F=15.9, p=0.004) and soft drink intake (F=66.4, p<0.001), but no significant interaction between cheese and soft drink intake (F=1.7, p=0.122). As found with MetSRisk, when controlling for the same factors, there was still an association between SumRisk and cheese (F=4.4, p=0.004), and soft drink intake (F=29.4, p<0.001), but no significant interaction between cheese and soft drinks (F=0.8, p=0.542). We also did a separate calculation with inclusion of diseases (diabetes, myocardial infarction, angina pectoris and stroke), and available data on drug treatment (lowering of cholesterol and blood pressure) among the covariates. The associations between soft drink (cheese) intake and the indices remained significant (p≤0.003). Thus, cheese and soft drinks both could influence the compound indices used to assess the risk of MetS, however, with no interaction when controlling for covariates.

Association between the intake frequency of soft drinks or cheese and complete MetS

Using logistic regression, with complete MetS[1] as the dependent variable, we found a modest positive association between soft drink intake and MetS in all the three models, with OR=1.10–1.07 (p<0.01) when going from Models 1–3, table 3). In contrast , cheese intake was negatively associated (p<0.01) with the syndrome (OR=0.96–0.97 in Models 1–3).

DISCUSSION

This study shows that the association between soft drink intake and two compound risk estimates for MetS, can be strongly and dose-dependently counteracted by cheese intake. Conversely, the apparent healthy influence of cheese on MetS risk can be blunted when the intake frequency of soft drinks increases. Similarly, opposite associations were found for cheese and soft drink intake when the dependent variable was complete MetS. All MetS-related associations with soft drinks and cheese prevailed when adjusting for a large number of possible confounding variables. It may, however, be difficult to evaluate the outcome when trying to control for a large number of factors, for example, because some of the independent variables could be in a direct relation from soft drink or cheese intake to the outcome.

The observed high prevalence of MetS and its age-related increase is in accordance with earlier reports.[9–11] However, we probably have underestimated true MetS

Table 3 Association between the intake frequency of soft drinks or cheese (main independent variables) and complete MetS

Logistic regression (dependent variable=complete MetS)		
Model 1	Model 2	Model 3
Main independent variable=Soft drinks		
1.10 (1.08 to 1.13)*	1.08 (1.04 to 1.11)*	1.07 (1.03 to 1.11)**
Main independent variable=Cheese		
0.96(0.95 to 0.97)*	0.94 (0.93 to 0.96)*	0.97 (0.94 to 0.99)**

Complete MetS is here defined as: waist circumference ≥80 cm for women and ≥94 cm for men, with either two of the following risk factors: triglycerides ≥1.7 mmol/l, HDL cholesterol <1.03 mmol/l for men and <1.29 mmol/l for women, systolic blood pressure ≥130 mm Hg, or diastolic blood pressure ≥85 mm Hg; fasting blood glucose values were not available. Regression Model 1=main independents, intake frequencies of soft drinks and cheese (entered simultaneously) adjusted for sex, age; Model 2=model 1+adjustment for time since the last meal, and inclusion of an interaction factor=intake frequency of soft drinks multiplied by that of cheese; Model 3=model 2+adjustments for the intake frequency of fruit/berries, fruit juice, fatty fish, coffee, alcohol, smoking, leisure time physical activity and years at school.
*p≤0.001.
**p<0.01. Mean value with 95% CI in parentheses.

prevalence in our study population since values for fasting glucose were not available. Furthermore, it seems that the observed presence of MetS and its components is largely driven by hypertension (prevalence 30–50%, table 1) and dyslipidemia (30–50%). Since the basic pathophysiology behind MetS is insulin resistance and dysglycaemia, the MetS in this cohort might not be representative of MetS in the general western population, and the results may not be generalisable.

The reason for the apparent low prevalence of diabetes in the Oslo Health Study could in part be related to the fact that the study does not reflect the current prevalence, since data collection was done several years ago, in 2000. Furthermore, the prevalence for diabetes in this study is based upon self-reported data, thereby probably underestimating the true prevalence.

That soft drinks may promote and dairy products prevent MetS is in accordance with previous findings.[3][12–17] However, although the association between the frequency of soft drink or cheese intake and MetS remained significant after adjusting for a large number of possible confounding factors, we cannot exclude the possibility of residual confounding by other dietary and lifestyle factors. On the other hand, it is hard to appreciate how the present results could have been caused by systematic errors such as information and selection bias.

After a fat meal we would anticipate an increase in serum TG concentration due to the presence of chylomicron TG. However, including 'time since last food intake' as a confounding variable did not obliterate the significance of the association between soft drinks/cheese and MetS risk estimates.

The lack of quantitative data is a weakness of this study, and there is also a possibility of reverse causation. For example, patients with MetS may have reduced their cola intake and thereby blunt the association between cola intake and MetS. In addition, all variables collected in the study, such as alcohol intake, are dependent on patients' recall, as they are collected from a questionnaire. Hence, there is subjectivity and potential for recall bias in the data. This may lead to residual confounding when the data are adjusted in the models. Also, adjustment for the use of antidiabetic medications was not possible due to lack of data.

Possible explanations of the findings

We will shortly mention some possible mechanisms, keeping in mind that cross-sectional data are not suitable for clarifying whether the associations are causal ones.

Sucrose can increase serum TG concentration and lower HDL,[4][5][18][19] but also soft drinks without sugar seemed to be associated with MetS.[2]

Furthermore, by stimulating adipose tissue lipolysis, *caffeine* in colas could possibly increase hepatic synthesis of TG and very-low-density lipoproteins (VLDL).[20] However, the soft drink versus MetS risk association

persisted after adjusting for coffee intake, making caffeine a less likely explanatory factor.

Colas also contain *phosphoric acid*.[21][22] We previously observed in a rat trial that intake of colas and other acid fluids tended to increase serum TG and lower HDL cholesterol,[23] suggesting that the phosphoric content in colas might contribute to increasing serum TG. Also, the fructose moiety of sucrose might increase acid load.[24] Furthermore, *trans* palmitoleic acid has been associated with reduced insulin resistance and a more favourable plasma lipoprotein profile.[25]

Is the antagonistic effect of soft drinks and cheese related to desaturation of fatty acids?

Elevated serum TG is one of the MetS requirements.[1] In fasted animals the serum TG concentration is mainly carried in VLDL from the liver.[20] VLDL-TG, cholesterol esters and phospholipids preferably contain monounsaturated fatty acids.[26] The rate-limiting enzyme for the synthesis of these fatty acids is stearoyl-CoA desaturase (SCD). Mice lacking SCD have reduced hepatic lipogenesis and lower plasma TG concentration.[27][28] Thus, one mechanism by which soft drink intake might increase serum TG could be stimulation of desaturase activities in the liver. Indeed, in our diet trial in rats,[23] we found that animals ingesting sucrose-cola had appreciably increased desaturase estimates, which also correlated positively with ingested amount of phosphoric acid, and with urinary acid excretion. Furthermore, cheese may inhibit desaturation of fatty acids, as indicated by our recent findings.[17] One finding was that a cheese extract added to cultures of human hepatoma cells caused a dose-dependent reduction in desaturase estimates. Cheese contains conjugated linoleic acid (CLA)[29] and a CLA isomer (t10, c12 CLA) has been shown to suppress Delta9, Delta6 and Delta5 desaturases in HepG2 cells.[30]

Thus, desaturases could be causally involved both in the effect of cheese and in the effect of soft drinks upon MetS. In this context it would seem of interest that results of a population-based prospective study[31] suggest that fatty acid desaturation may be associated with cardiovascular mortality. However, in our cross-sectional data it is hard to control for all covariates and the observed antagonistic cheese/soft drink relationship concerning MetS risk could have been caused by an as-yet unknown covariate. In addition, cross-sectional data are not suitable for assessing causality.

In line with the positive association between MetS and type 2 diabetes,[1] and the finding that cheese intake may be negatively associated with MetS, it was recently reported that cheese intake may be beneficially influencing the risk of diabetes.[32][33] In a recent review[34] it was pointed out that the majority of observational studies did not find an association between the intake of dairy products and increased risk of CVD, coronary heart disease and stroke, regardless of milk fat levels. Furthermore, compared with butter, cheese intake

seems to lower low-density lipoprotein (LDL) cholesterol, which is a major CVD risk factor.

In conclusion, the present study seems to suggest that cheese intake may blunt the promoting influence of frequent soft drink intake on MetS, irrespective of the intake level of soft drinks. Hypothetically, an effect of both diet items upon desaturation of fatty acids could be involved, but the apparent antagonism might also depend on residual confounding. In addition, cross-sectional data are not suitable for assessing causal relationships.

REFERENCES

1. IDF consensus worldwide definition of the metabolic syndrome. International Diabetes Federation 2009. http://www.medscape.com/viewarticle/514211 (accessed Mar 2012).
2. Høstmark AT. The Oslo Health Study. A Dietary Index estimating high intake of soft drinks and low intake of fruits and vegetables was positively associated with components of the metabolic syndrome. *Appl Physiol Nutr Metab* 2010;35:816–25.
3. Høstmark AT, Tomten SE. The Oslo Health Study: cheese intake was negatively associated with the metabolic syndrome. *J Am Coll Nutr* 2011;30:182–90.
4. Dhingra R, Sullivan L, Jacques PF, *et al*. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. *Circulation* 2007;116:480–8.
5. Merchant AT, Anand SS, Kelemen LE, *et al*. Carbohydrate intake and HDL in a multiethnic population. *Am J Clin Nutr* 2007;85:225–30.
6. Høstmark AT, Tomten SE. Cola intake and serum lipids in the Oslo Health Study. *Appl Physiol Nutr Metab* 2009;34:901–6.
7. Høstmark AT, Haug A, Tomten SE, *et al*. Serum HDL cholesterol was positively associated with cheese intake in the Oslo Health Study. *J Food Lipids* 2009;16:89–102.
8. Søgaard AJ, Selmer R, Bjertness E, *et al*. The Oslo Health Study: the impact of self-selection in a large, population-based survey. *Int J Equity Health* 2004;3:3–12.
9. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 2002;287:356–9.
10. Hildrum B, Mykletun A, Hole T, *et al*. Age-specific prevalence of the metabolic syndrome defined by the International Diabetes Federation and the National Cholesterol Education Program: the Norwegian HUNT 2 study. *BMC Public Health* 2007;7:220.
11. Sidorenkov O, Nilssen O, Brenn T, *et al*. Prevalence of the metabolic syndrome and its components in Northwest Russia: the Arkhangelsk study. *BMC Public Health* 2010;10:23.
12. Elwood C, Pickering JE, Hughes J, *et al*. Milk drinking, ischaemic heart disease and ischaemic stroke II. Evidence from cohort studies. *Eur J Clin Nutr* 2004;58:718–24.
13. Elwood C, Pickering JE, Fehily AM. Milk and dairy consumption, diabetes and the metabolic syndrome: the Caerphilly prospective study. *J Epidemiol Community Health* 2007;61:695–8.
14. Biong AS, Rebnord HM, Fimreite RL, *et al*. Intake of dairy fat and dairy products, and risk of myocardial infarction: a case–control study. *Int J Food Sci Nutr* 2008;59:155–65.
15. Elwood C, Pickering JE, Givens DI, *et al*. The consumption of milk and dairy foods and the incidence of vascular disease and diabetes: an overview of the evidence. *Lipids* 2010;45:925–39.
16. Fumeron F, Lamri A, Emery N, *et al*. Dairy products and the metabolic syndrome in a prospective study, DESIR. *J Am Coll Nutr* 2011;30:4545–635.
17. Høstmark AT, Lunde MSH. 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* 2012;37:31–9.
18. Høstmark AT, Blom PC. Previous exercise nullifies the plasma triacylglycerol response to repeated fructose ingestion in young men. *Acta Physiol Scand* 1985;125:553–4.
19. Archer SL, Liu K, Dyer AR, *et al*. Relationship between changes in dietary sucrose and high density lipoprotein cholesterol: the CARDIA study. Coronary Artery Risk Development in Young Adults. *Ann Epidemiol* 1998;8:433–8.
20. Murray RK, Granner DK, Mayes PA, *et al*. In Harper's biochemistry. 25th edn. New York, NY: McGraw-Hill, 2000:281–3.
21. Jensdottir T, Holbrook P, Nauntofte B, *et al*. Immediate erosive potential of cola drinks and orange juices. *J Dent Res* 2006;85:226–30.
22. Kapicloglu S, Baki AH, Tekelioglu Y, *et al*. The effect of cola consumption on oral mucosa in rats. *Dis Esophagus* 2000;13:69–71.
23. Høstmark AT, Lunde MSH, Eilertsen E. Does an acid load promote liver desaturases and increase serum lipids? *Clin Rev Opin* 2010;2:8–16.
24. Taylor EN, Curhan GC. Fructose consumption and the risk of kidney stones. *Kidney Int* 2008;73:207–12.
25. Mozaffarian D, Cao H, King IB, *et al*. *Trans*-palmitoleic acid, metabolic risk factors, and new-onset diabetes in U.S. adults: a cohort study. *Ann Intern Med* 2010;153:790–9.
26. Nakamura MT, Nara TY. Structure, function, and dietary regulation of delta6, delta5, and delta9 desaturases. *Ann Rev Nutr* 2004;24:345–76.
27. Dobrzyn A, Ntambi JM. The role of stearoyl-CoA desaturase in the control of metabolism. *Prostagl Leukotr Essent Fatty Acids* 2005;73:35–41.
28. Dobrzyn A, Ntambi JM. Stearoyl-CoA desaturase as a new drug target for obesity treatment. *Obes Rev* 2005;6:169–74.
29. Mushtaq S, Mangiapane HE, Hunter KA. 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* 2010;103:1366–74.
30. Eder K, Slomma N, Becker K. *Trans*-10, *cis*-12 conjugated linoleic acid suppresses the desaturation of linoleic and alpha-linolenic acids in HepG2 cells. *J Nutr* 2002;132:1115–21.
31. Warensjo E, Sundstrøm J, Vessby B, *et al*. 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* 2008;88:203–9.
32. Sluijs I, Forouhi NG, Beulens JW, *et al*. The amount and type of dairy product intake and incident type 2 diabetes: results from the EPIC-InterAct Study. *Am J Clin Nutr* 2012;96:382–90.
33. Struijk EA, Heraclides A, Witte DR, *et al*. Dairy product intake in relation to glucose regulation indices and risk of type 2 diabetes. *Nutr Metab Cardiovasc Dis* 2012; Jul 23. Epub ahead of print.
34. Huth PJ, Park KM. Influence of dairy product and milk fat consumption on cardiovascular disease risk: a review of the evidence. *Adv Nutr* 2012;3:266–85.