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The impact of dairy matrix structure on postprandial lipid responses

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Abstract

This review presents evidence related to the postprandial responses after consumption of dairy products focusing on the effect of the dairy matrix and lipid response, which was also presented as part of a speech at the Nutrition Society Winter Conference, January 2023. The key findings are that the dairy product(s) that differentiate from others in the postprandial TAG response are products with a semi-solid structure. There were no differences in lipid responses between cheese and butter. The main factors viscosity, fat globule size and milk fat globule membrane do not seem to explain the effect of the dairy matrix in the acute postprandial response. In summary, it is very difficult to investigate the effects of the dairy matrix per see and with the few studies conducted to date, no clear cause and effect can be established. Future research should focus on the semi-solid dairy matrix, and studies investigating specifically the yoghurt matrix are warranted.

Keywords: Cheese: TAG: NEFA: Cholesterol

The nutrition content of cows' milk

Raw milk, an oil-in-water emulsion, is a nutrient-dense food source rich in high quality proteins (whey and casein, 20:80 % in milk), lactose and fats e.g. medium-chain fatty acids, odd-chain fatty acids (15:0 and 17:0), phospholipids and conjugated linoleic acid[1,2]. Furthermore, minerals (e.g. calcium, phosphorous, magnesium, iodine and potassium) and vitamins (e.g. vitamin A, riboflavin, vitamin B_{12} and in fortified products vitamin D) are present[1,2]. However, the relatively high content of SFAs of regular-fat dairy products has raised concerns, as has the content of *trans*-fat of ruminant origin[2]. In 2011–2013, Danish adults consumed on average 304 g milk and milk products daily and the SFA intake from these products including butter constituted 50 % of total SFA intake[3]. Recently, Poppitt[2] reviewed the evidence of cow's milk and dairy consumption on cardiometabolic health and concluded that potential adverse effects of SFAs may be reduced when fats are consumed within a dairy matrix, and that categories of dairy food may affect metabolic health outcomes as much as total fat content. In this review, we will focus on the effects of the dairy matrix on the lipid response 0–8 h after consumption (relevant studies are presented in Table 1).

Processing of raw cow milk

Processing of raw milk leads to a range of products (e.g. milk, cream, cheese, yoghurt and butter) which as a collected group is called dairy. Dairy products differs in their nutritional content and examples are presented by

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Table 1. Selected studies investigating effects of dairy products on postprandial lipid response

First author, year	Study design and participants	Intervention products/meals	Lipid results
Not-energy matched meals			
Kjølbæk, 2022[35]	Sub-study of a parallel RCT [31]. 4 h study (conducted after a 12-wk intervention) with time points: 0, 0·5, 1, 1·5, 2, 3 and 4 hs. 37 adults with MetS at inclusion, 18–70 ys, BMI: 18·5–37·5 kg/m^2.	3 meals (cheese/jam, bread, cucumber, water), differing in macronutrients content and energy. Intervention products (all commercially available) were: ○ Regular cheese (40 g full-fat Danbo (Riberhus, 25% fat) and 40 g cheddar (Sharp Cheddar, 32% fat)) ○ Reduced-fat cheese (40 g reduced/low-fat Danbo (Riberhus, 13% fat) and 40 g cheddar (Sharp Cheddar, 16% fat)) ○ Carbohydrate-rich food (90 g bread and 25 g jam)	TAG: Time-meal interactions. Regular cheese increased TAG at 3 h and 4 h compared to reduced cheese and carbohydrates. iAUC$_{4h}$ was larger for regular cheese compared to carbohydrates. NEFA: Time-meal interactions. Regular cheese increased NEFA at 3 h compared to reduced cheese and at 2 h, 3 h and 4 h compared to carbohydrates. Overall, the iAOC$_{4h}$ only tended to differ. Cholesterol was only measured in the fasting state.
Hansson, 2018[36]	RCT, cross-over. 6 h acute study with time points: 0, 2, 4 and 6 hs. 47 apparently healthy men and women, 18–70 ys. BMI: ≥18·5 kg/m^2.	4 meals (dairy product, bread, jam) matched on fat (45 g fat, 60 E%), differ in protein and energy contents. Water ad lib (max 1 L). Intervention products (all commercially available) were: ○ Butter (*TINE Smør*) ○ Cheese (full-fat milk and cream-based medium-hard (*TINE Gräddost*)) ○ Whipped cream (*TINE Kremfløte*) ○ Sour cream (TINE Seterrømme)	TAG: Sour cream resulted in larger TAG iAUC$_{6h}$ compared to all other. NEFA: No effect. Cholesterol: Sour cream resulted in larger HDL iAUC$_{6h}$ compared to cheese. No effect on cholesterol (total and LDL).
Energy-matched meals			
Kjølbæk, 2021[33]	RCT, cross-over. 8 h acute study with time points: 0, 0·5, 1, 1·5, 2, 3, 4, 5, 6, 7 and 8 hs. 25 healthy men, 19–40 ys, BMI: <25 kg/m^2.	4 meals (dairy product, bread, water). Energy content: 4·7 MJ, 5·9 kJ/g. Nutritional similar meal (21 E% protein, 54 E% fat and 27 E% carbohydrate, calcium range: 1415–1524 mg/meal). Intervention products were: ○ Cheddar cheese ○ Homogenised cheddar cheese ○ MCI Drink ○ MCI Gel Dairy products were matched for fat and protein, but differed in: presence/absence of GDL, and lactose and sodium contents. Adjustments were made by adding lactose, potato starch and salt to some of the breads.	TAG: Overall meal effect on TAG. MCI Gel increased TAG compared to cheddar cheese and homogenised cheddar cheese, similar finding for iAUC$_{8h}$. NEFA: Time-meal interaction. Many differences observed. Primarily at 1·5 and 2 hs both cheeses reduced the concentration compared to MCI products, but no difference observed for iAOC$_{8h}$. Cholesterol: No effect (total, LDL and HDL). ApoB48: No effect.
Beals, 2019[41]	RCT, double blinded, cross-over. 6 h acute study with time points: 0, 1, 3 and 6 hs. 36 men and women, 18–65 ys, BMI: 25–29 kg/m^2, having at least two criteria for MetS.	2 meals (dairy-smoothie, bagel, jam). Energy: 40% of the daily energy requirement. Macronutrient matched meals (15 E% protein, 55 E% fat and 30 E% carbohydrate). Intervention products (dairy-smoothies) were: ○ High fat whipping cream ○ High fat whipping cream + MFGM	TAG: No effect. NEFA: Not reported. Cholesterol: No effect (HDL, LDL, Cholesterol:HDL and non-HDL).

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Drouin-Chartier, 2017[37]	RCT, cross-over. 8 h acute study with time points: 0, 2, 4, 6 and 8 hs. 43 apparently healthy men and women, 18–65 ys, BMI: <35 kg/m^2.	3 meals (dairy product, bread with topping, fruit juice). Energy: 33% of the daily energy requirement. Macronutrient matched meals (per 4184 kJ serving: 33 g protein, 42 g fat and 127–130 g carbohydrate). Intervention products (all commercially available) were: ○ Cream cheese (unripened, homogenised, fresh) ○ Cheddar cheese (firm, uncooked, young) ○ Butter (salted) Dairy products were matched for fat, but differed in protein content. Adjustments were made by adding sodium caseinate to the bread served with the cream cheese and butter meals.	TAG: Time-treatment interaction. At 2 h, cream cheese resulted in higher TAG than cheddar and butter, but at 6 h cream cheese resulted in a lower concentration than cheddar. No difference in (i)AUC between products. NEFA: Time-treatment interaction. At 2 h, NEFA concentration was lower for cream cheese compared with cheddar. No difference in (i)AUC between products. Cholesterol was only measured in the fasting state. ApoB48: Time-treatment interaction. At 4 h and 6 h, cream cheese reduced ApoB48 compared to cheddar. iAUC was lower for the cream cheese compared to cheddar.
Tholstrup, 2004[34]	3-wk RCT, cross-over. 6 h study conducted on day 4 of the 3-wk intervention with time points: 0, 2, 4, 6 and 8 hs. 14 apparently healthy men, 20–31 ys, BMI: 20–27 kg/m^2.	3 diets/meals (dairy product and bread). Energy: individually determined, range: 4·2–5·2 MJ (e.g. a person weighing 75 kg: 1·5 L milk, 200 g cheese or 64 g butter. Macronutrient matched meals: (20 E% protein, 45 E% fat and 35 E% carbohydrate). Intervention products (all commercially available) were: ○ Butter ○ Cheese (hard) (*Samsø*) ○ Whole milk Dairy products were matched for milk fat content, but differed in protein and lactose. Adjustments were made by adding lactose to bread/cakes (for cheese meal) and lactose + milk protein to bread/cakes (for butter meal). However, calcium content differed up to 1979/10MJ.	TAG: No effect. NEFA: Not reported. Cholesterol: Time-meal interaction. At 8 h cheese increased HDL_2 compared to butter and whole milk. No other cholesterols measurements (LDL, total) were affected.

ApoB, Apo B; E%: energy percentage, GDL, Glucono Delta-Lactone; HDL, high density lipoprotein; iAOC, incremental area over the curve; iAUC, incremental area under the curve; LDL, low density lipoprotein; MCI, micellar casein isolate; MetS, metabolic syndrome; MFGM, milk fat globule membrane; RCT, randomised controlled trial; TAG.

Weaver[4]. However, these products differ not only in their nutritional content, but also in their matrices. Alteration of the matrix occurs during processing as briefly mentioned below.

Homogenisation is used to increase the physical stability of a food product. This process disrupts the milk fat globule membrane (MFGM) found in raw cow's milk. The MFGM surrounds lipid droplets and comprises proteins, cholesterol and polar lipids including phospholipids and sphingolipids[2,4]. In addition to the disruption of the MFGM, homogenisation also reduces the size of the lipid droplets and the proteins will be incorporated into the lipid droplet interface. Conventional milk is often homogenised, whereas organic milk is not always homogenised. The raw milk used in the production of matured 'yellow' cheese (e.g. cheddar and Emmental cheese) is not homogenised, in contrast to raw milk used for production of other types of cheese (e.g. blue, fresh and cream cheese)[5]. Heat-treatments such as pasteurisation (low temperatures) and ultra-high-temperatures (UHT) treatments, where different temperatures and times are used depending on the dairy product to be produced, are the most common methods to destroy pathogens to ensure the product's safety and to improve shelf life[4]. A less common method used is high-pressure processing (HPP), which is a treatment where proteins are less denatured which may affect the dairy matrix and sensory attributes are preserved[6]. Fermentation is used in the manufacturing of both yoghurt and cheese. Here, a starter culture is added and depending on the chosen culture, different textures and flavours appear. Most commonly used is the Lactic acid bacteria, however yeast and molds are also used[2]. During fermentation, lactose is fermented to lactic acid and thus the resulting end product contains probiotics, short chain fatty acids, bioactive peptides etc. in varying content depending on the acidification rate. In the production of e.g. cottage cheeses and firmer cheeses, addition of the enzyme rennet results in whey precipitation. For butter production, cream is churned and the liquid buttermilk that contains most of the MFGM is removed. Thus, the raw milk – an oil-in-water emulsion – is changed into a water-in-oil emulsion[4]. Due to the many different processing aspects of the raw cow's milk, the

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different dairy products have very different food matrices and it is speculated how these matrices affect the bioavailability. Therefore, researchers should be careful in the interpretation of results where all dairy products are grouped together[7].

Dairy intake

CVD and CVD-risk markers

In 2017, CVD were the leading cause of diet-related deaths globally[8]. For many years the 'Diet-heart hypothesis' has associated SFA consumption with increased LDL cholesterol leading to CVD and CHD mortality[9,10]. Many have investigated the association between nutrients in dairy, such as SFA and calcium, and mortality from CVD in meta-analyses of observational studies, and continue to do so[11,12]. Nevertheless, newer research which takes into account the food source of the nutrient has questioned this association because not all dairy products increase LDL-cholesterol[13,14]. Thus, a potential causal link between SFA intake and CVD/CHD mortality may be weak. A recent review by Givens[15] provides an overview of these investigations including the food source as an important factor. Here, associations between dairy intake and the incidence or mortality of all-causes/CVD/CHD/stroke or intermediate risk-markers such as cholesterols, TAG and blood pressure are presented[15]. Dairy intake has been investigated as total dairy intake[7,16–19] and specific dairy products such as butter[19], milk[16,17,19], cheese[19], yoghurt[19] or in categories related to fat content[17] and fermentation[20]. Overall, these observational data, especially those using more recent statistical approaches, provide no consistent evidence of negative health associations between consumption of dairy and hard endpoint or intermediate risk markers related to CVD, despite the majority of dairy products having a high content of SFA and these finding therefore challenge the 'Diet-heart hypothesis'. The results from these observational studies have been reviewed, discussed and summarised by many[2,21,22] especially because the results are not reflected in all (national) dietary guidelines. When these results are interpreted, it has to be remembered that foods vary in the type of SFA[23] and that the replacement nutrient or food also has an impact on the outcome, an impact which is seldom neutral[24–26].

The LDL-cholesterol concentration has been used as a risk markers for prediction of disease and mortality for many year. Today, the use of LDL-cholesterol as a CVD-risk marker is questioned and other risk markers (e.g. non- high density lipoprotein (HDL) cholesterol and Apo (Apo) B) are discussed[27]. Furthermore, recent results from the prospective PURE study[26] is of interest. Based on fasting blood samples they observed that the ratio between ApoB and ApoA1 was a better predictor of CVD risk than LDL-cholesterol alone, probably because this ratio reflects presence of small dense LDL particles, which seem to be more atherogenic than large LDL particles[26]. However, this newly suggested risk marker needs to be validated by others. Another relevant discussion is the use of fasting *v.* postprandial measurement[28,29], which will be raised later.

The causal link between dietary consumption and outcomes can only be confirmed by randomised controlled trials (RCTs). However, the disadvantage of RCTs is the duration, which is seldom long enough to study hard endpoints. Therefore, intermediate markers (e.g. TAG and cholesterol) have been used instead. Like other studies, RCTs have often focused more on individual nutrients and less on whole foods. However, more than 10 years ago Hjerpsted *et al.*[13] suggested that the 6 weeks' consumption of cheese had beneficial effects on total and LDL-cholesterol compared to butter. In 2017, a working group discussed the individual nutrient *v.* whole food research approach[30] and today, the whole food approach has gained attention because there may be (dairy) matrix effects which are lost when focus is only on individual nutrients. This thought, that health effects cannot be predicted from the nutritional content itself, is gaining greater acceptance – also because humans consume foods and drinks – not individual nutrients[2,4]. Subsequently, Raziani *et al.*[31] investigated if consumption of a regular fat cheese compared to a reduced fat cheese affected the lipid profile, and it was found that it did not. Recently, Feeney *et al.*[14] confirmed that 6 weeks' consumption of fat from a cheese matrix compared to butter had beneficial effects on cholesterol levels and that these effects were not explained by differences in calcium and protein content of the diet, highlighting that the food (matrix) rather than the individual nutrients are important to consider when health outcomes are evaluated.

For production of new food products with beneficial health effects, it is important to consider any healthy and less healthy properties of the food matrix. These properties may arise from interactions between nutrients within the food matrix and have the potential to alter the bioactive properties and affect nutrient absorption[32]. However, the key problem is that investigating effects of the matrix per se is highly complicated because for most products varying the food matrix will result in differences in nutritional content. Thus, using existing (dairy) products on the market to study matrix effect per see is more or less impossible, because none of these have the exact same nutritional content. In case existing (dairy) products on the marked is used, the full diet to be studied needs to be supplemented with lacking nutrients and this approach is more reflecting effects of nutrients being inside or outside a food matrix, rather than the effect of the matrix per se. Another issue is that of study matrix effects in RCTs over a longer period because strictly controlled conditions are needed to exclude effects from variations in overall nutrient content of diets. Thus, the best suited study for the effect of the matrix per see may be a strictly controlled (i.e. short-term) trial where all food is provided and consumed within a laboratory-setting.

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Postprandial effects of dairy products

Effects of strictly controlled diets/foods are most often assessed in short-term settings such as a meal test conducted over some hours. After consumption of a meal, the biological markers related to fat intake that change are TAG and NEFA, whereas cholesterol concentrations are relatively constant[28], even when a very energy-rich meal with high fat content is consumed[33]. Tholstrup and colleagues have conducted several postprandial meal tests with dairy products[34,35]. Likewise, Hansson *et al.*[36] conducted a meal study with dairy products (Table 1). Common for these three studies[34–36] was that the test meals were not matched for either fat, energy or calcium content. Tholstrup *et al.*[34] compared effects of butter, cheese and whole milk and except a little higher HDL_2 concentration at 8 h after intake of cheese compared to butter and whole milk, no time-meal interactions were observed for total and LDL-cholesterol, NEFA and TAG. Although they adjusted for lactose and milk protein content, large differences (1769–1979 mg/10 MJ) in calcium content between the test meals were observed. In another trial by the Tholstrup group[35], regular and reduced fat cheeses were compared. Here, the regular fat cheese increased TAG compared to the reduced fat cheese, but these meals were not iso-energetic and not matched on fat intake. Hansson *et al.*[36] compared butter, cheese, whipped cream and sour cream and observed that sour cream increased the TAG incremental area under the curve (iAUC) compared to all other products and increased HDL iAUC compared to cheese. No effects on NEFA, total and LDL cholesterols were observed. In this study, the meals were matched for fat content, but they were not iso-energetic (e.g. the cheese meal provided 255 kJ and 18·8 g protein more than the sour cream meal).

To study the effect of the food matrix it is important that all intervention products/meals are highly similar in their nutritional content; otherwise, the observed effect(s) might not be exclusively caused by the food matrix and the potential impact of the nutrients on the results should be carefully considered. Few RCTs aiming to investigate postprandial effects of the dairy matrix have been conducted. These studies have not been able to completely match the nutritional content in their dairy products, however the studies had minor adjustments and therefore served highly similar meals. The minor adjustments to bread included in the test meal were addition of sodium caseinate in one study[37], whereas we added salt, lactose and/or potato starch to some of the breads in our own study[33]. In the study by Drouin-Chartier *et al.*[37], the effects of cream cheese, cheddar cheese and butter were investigated. Effects on TAG, ApoB48 and NEFA were observed. Cream cheese increased the 2 h TAG concentration compared to all other products, but it also decreased the 6 h TAG concentration compared to cheese resulting in no difference in (i)AUC. The cream cheese reduced the ApoB48 concentration at 4 h and 6 h compared to cheddar cheese resulting in a lower ApoB48 iAUC compered to cheddar. For NEFA, cream cheese lowered the 2 h concentration compared to cheddar cheese. The biggest nutritional difference between the dairy products was the protein and calcium contents (highest in the cheddar cheese and lowest in the butter). The protein difference was adjusted in the bread served as part of the meal whereas the calcium content varied from 74 to 141 mg/MJ serving. These results did not indicate any dose-response effect related to protein and calcium contents of the dairy products, but to a large extent the effects seemed to be explained by the dairy matrix. To address these issues the authors and their collaborators developed dairy products that were matched not only on fat content, but also on protein and calcium contents to avoid speculation on the effects of nutrients being in- or outside the matrix[33,38]. The authors also designed a gelled dairy matrix[38], however, this resulted in a different carbohydrate content that was adjusted by adding lactose and potato starch (and salt) to the bread served with the meals. In our study[33], we observed that the gelled product (a micellar casein isolate (MCI) gel) increased the TAG concentration resulting in a higher iAUC compared to cheddar cheese and a homogenised cheddar cheese, without any differences in ApoB48 concentration. For NEFA, both the commercial and the homogenised cheddar lowered NEFA concentrations at 1, 1·5 and 2 h, but it did not increase incremental area over the curve (iAOC) compared to other products.

Based on these highly controlled and nutritionally similar test meals there are very good indications of dairy matrix effects. The disadvantage with the postprandial studies is the translation of the results into longer-term effects as few postprandial markers are accepted and used in the diagnoses/assessment of cardiometabolic health. However, it is debated[28,29] if postprandial markers are more predictive of health and disease risk than the usual and most commonly used fasting values in the majority of both observational and long-term experimental studies. This warrants further investigations as well as inclusion and validation of the newer proposed risk markers from the PURE study mentioned earlier.

Proposed mechanism – matrix effects

To summarise, the acute postprandial lipid responses after consumption of dairy products showed that cheese and butter could not be described as a healthy or less healthy choice, respectively, as could have been expected from fasting lipid profile after 6 weeks intake in RCTs. In postprandial studies, the dairy products that differentiated from others were products with a semi-solid structure, i.e. sour cream, cream cheese and a gelled product, which all increased the postprandial TAG response. Potential mechanisms are discussed below.

In the gastrointestinal tract, calcium can form insoluble calcium-soaps and precipitate bile acids, which reduce fat absorption and bile acids recycling and increase faecal excretion[39]. From postprandial trials using iso-energetic meals[34,37] one possible explanation of different lipid responses might be related to the content of calcium. In both cases, faecal fat excretion was not investigated and a possible difference on lipid concentrations between butter which has a low calcium

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content and hard cheese which has a high calcium content was not observed. Recently, Feeney *et al.*[40] investigated the effect of calcium being inside and outside the cheese matrix i.e. total calcium intake was similar. Here, intake of cheese with high calcium content did not increase faecal fat excretion compared to intake of cheese with a reduced calcium content + calcium supplementation, but due to the very small population ($n = 7$ completers) a type 2 error cannot be excluded. This theory warrants further trials; however, we have previously suggested that other minerals and also dietary fibre may affect faecal fat excretion[39]. Another speculation is that an effect of calcium might depend on the location of nutrients within the food matrix as the likelihood of interaction between nutrients (in this case fat and calcium) may depend on how close there are located especially during digestion in the gastrointestinal tract. In our postprandial study, we provided products and meals with highly similar calcium contents (dairy products range: 1327–1463 mg, total meal range: 1415–1524 mg) and thus any calcium-driven effect on the TAG concentration between the MCI gel and other products might be due to the placement of nutrients within the food matrix, rather than the calcium content.

Another proposed effect of the food matrix is related to digestion, where the theory is that increasing the complexity of the food structure increases the time needed for digestion (i.e. complexity slow down the digestion rate). This results in, either a slower digestion which can give a skewed absorption pattern, but not necessarily a smaller absorption of nutrients, or a slow digestion which can increase the faecal excretion of nutrients because the nutrients are not released from the food matrix at the time/place where they would have otherwise been absorbed. Drouin-Chartier *et al.*[37] observed that a cream cheese skewed the TAG response compared to cheddar cheese and butter, but an effect on the total TAG response (i.e. AUC) was not found. They discussed their results in relation to the size of the lipid droplets and suggested that the small homogenised droplets in the soft semi-solid protein gel of the cream cheese was rapidly digested and absorbed. This rapid digestion may be explained by the larger surface that appear when droplets become smaller in diameter. A related structural part of the dairy matrix is the MFGM. The presence or absence of an intact MFGM or a re-structuring of it is particularly interesting in the discussion of the influence of the dairy matrix. Beals *et al.*[41] investigated the effect of presence/absence of the MFGM in participants with metabolic syndrome. Their dairy intervention products (smoothies) had very similar nutritional content i.e. less than 0·2 % variation in macronutrients across test products, thus increasing the likelihood that any observed effects were caused by the presence or absence of the MFGM. They found no effect on postprandial lipid response, however, effects on inflammation and insulin metabolism were observed (Table 1). In support of this, none of the postprandial trials comparing cheese and butter found differences in lipid response. It is worth noting that butter is a product where most of the MFGM is removed with the buttermilk when the cream is churned. Thus, effects on postprandial lipid responses of dairy products do not seem to be caused by the presence or absence of the MFGM. In our study, we homogenised the commercial cheddar cheese with water, which resulted in a semi-solid structure where the fat droplets were reduced and the structure and composition of the original MFGM was altered[38]. We did also not observe differences between the cheddar cheese and the homogenised cheddar cheese[33]. Thus, smaller fat droplets and alteration of the MFGM did not affect the postprandial lipid response in our study. However, it remains to be investigated in detail how processing of the intervention products used in these RCTs not only affect fat droplet size, but also how the subsequent alteration of the MFGM affects the fat droplet surface composition e.g. by reorganisation of casein which might affect digestion[5]. Thus, a very detailed characterisation of the investigated dairy products is needed.

Consumption of solid products slow down the rate of digestion and absorption in the gastrointestinal tract, compared to liquids. Theoretically, this might affect the lipid response and results from non-dairy trials investigating effects of the food matrix have also shown that a liquid product (drink) increased TAG concentrations compared to a nutritionally similar solid product (cookie)[42]. However, liquid dairy products e.g. milk, which could be regarded as products with a fast digestion related to a fast gastric emptying, seem to behave differently. Tholstrup *et al.*[34] discussed the potential precipitation of casein in the stomach based on their results and in a review by Mulet-Cabero *et al.*[43] the matter of gastric digestion is discussed in details. Additionally, Sanggard *et al.*[44] showed that fast gastric emptying after milk intake did not lead to a fast rise in TAG concentration compared to a semi-solid fermented milk product (A38). From in vitro studies on our intervention products[38], we observed that the liquid MCI drink coagulated in the 'stomach' which increased the viscosity to the same level as our two semi-solid products. In our in vivo study[33], there was no indications of faster gastric emptying causing faster digestion and absorption rate for the liquid product compared to semi-solid and solid products.

Conclusion

Dairy products with different matrix structures affect the postprandial response to lipids. In the acute response, there is no distinction between butter and cheese as was observed from fasting measurements after 6 weeks' consumption. In the acute response, the dairy products that differentiate from others are products with a semi-solid structure e.g. sour cream, cream cheese and a gelled product, which all increase the postprandial TAG response. Currently, only few studies have been conducted where effects of the nutritional content can be separated from the effect of the matrix per se. Based on these few studies there is no clear mechanism or specific part of the matrix that can explain the results. Further

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studies are needed, and for these it is highly recommended to focus on the semi-solid matrix and on yoghurt as some of the intervention products.

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Competing interest

L.K. has conducted several trials investigating effects of dairy food or dairy nutrients funded by Arla Foods, Arla Food for Health and other dairy foundations, councils and institutes, without receiving personal grant from any of these dairy institutions. A.R. has received honoraria from Unilever, Nestlé, and the International Sweeteners' Association.

Authorship

L.K. was speaker at Nutritional Society Winter Conference 2022/23, wrote the paper and had primary responsibility for the final content. A.R critically reviewed the paper, read and approved the final manuscript.

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