Separating Milk Fats from Fiction

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Take Home Messages

- The saturated and trans fatty acids in milk fat are associated with perceived negative effects on human health, especially cardiovascular disease.
- Recent epidemiological studies and dietary intervention trials challenge the negative perception of milk fat and available evidence does not support the concept that consumption of saturated fats or dairy products adversely affects the risk of coronary heart disease.
- Trans fats found in dairy products are consumed in very low amounts and do not appear to have the negative health effects associated with the consumption of industrial-sources of trans fat.
- Milk fat is an excellent source of oleic acid that originates mainly by endogenous synthesis from stearic acid.
- Long chain omega-3 fatty acids and conjugated linoleic acids found in low concentrations in milk fat have potential beneficial effects in health maintenance and the prevention of chronic diseases.
- Individuals do not just consume milk fat-derived fatty acids on their own, but rather as components in dairy foods, which are highly complex and may contain many beneficial ingredients.
- Educating consumers about inaccurate and inappropriate generalizations about milk fat remains a primary challenge.

Introduction

The importance of animal-derived foods in meeting the food security needs of the global population is well recognized (Demment and Allen 2003, Randolph *et al* 2007). Dairy products are an important source for many key nutrients including high quality protein, energy, and many essential minerals and vitamins. The recent "Dietary Guidelines for Americans" emphasized that

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dairy products provide critical amounts of 3 of the 5 “nutrients of concern” for children and 4 of the 7 nutrients of concern for adults (USDA 2005). Fat is the most variable component of milk and accounts for many of the physical properties, manufacturing characteristics and organoleptic qualities of dairy products. Milk fat content and fatty acid (FA) composition can be significantly altered through nutrition of the dairy cow, and this has been extensively reviewed elsewhere (e.g. Chilliard *et al* 2000, Lock and Shingfield 2004). This research has often involved studies designed to achieve shifts in the ratio of saturated FA (SFA) to polyunsaturated FA (PUFA). While modest changes have been achieved, this can often negatively affect cow performance and lead to challenges relating to the quality and stability of dairy products.

Consumers are increasingly aware of the connection between diet and health. Many consumers consider milk fat “bad”, and scientists are being asked to clarify the role of specific foods and food components in health maintenance and chronic diseases. From this research it is increasingly clear that broad generalizations about fat can be misleading and often inaccurate (Lock *et al* 2008; Parodi 2009). Rather one must consider the biological effects and nutritional value on the basis of individual FA. In the following sections we will first provide background on rumen lipid metabolism and milk fat synthesis and then address the state of knowledge regarding milk FA and human health implications.

■ Lipid Metabolism in the Dairy Cow

Dietary lipid is extensively metabolized in the rumen and this has a major impact on the profile of FA available for absorption and subsequent use by body tissues. FA in dietary lipids are mainly esterified (fatty acids are bound to glycerol) and two major transformations occur when they enter the rumen (Palmquist et al., 2005). The first is hydrolysis of the ester linkages to yield free FA and the second is the biohydrogenation of unsaturated FA (UFA). Biohydrogenation involves only a few of the rumen bacteria species, and they carry out these reactions as a protective mechanism against the toxic effects of PUFA. Linoleic acid (18:2 n-6) and linolenic acid (18:3 n-3) are the main PUFA in ruminant diets and their biohydrogenation results in stearic acid (18:0) being the major FA in outflow from the rumen and absorption from the small intestine. During the biohydrogenation process, transitory FA intermediates are also produced and some escape via rumen outflow and are subsequently absorbed. Two such FA that were initially identified are *trans*-11 18:1 (vaccenic acid; VA) and *cis*-9, *trans*-11 conjugated linoleic acid (rumenic acid; RA), but improvements in analytical techniques established that rumen biohydrogenation processes were considerably more complex than first thought, and a remarkable range of *trans* 18:1 and conjugated linoleic acid (CLA) isomers have been identified in rumen outflow (Bauman and Lock, 2006). Thus, in addition to the pathway involving the formation of VA and RA as intermediates, there must be many minor pathways of biohydrogenation.

Furthermore, modifications in diet and rumen environment impact the relative importance of these biohydrogenation pathways resulting in substantial changes in FA intermediates produced in the rumen and ultimately incorporated into milk fat.

Cows' milk contains approximately 3 to 5% fat, depending on breed, nutrition and stage of lactation. Milk fat is predominantly triglycerides (95-98%) which forms the core of the milk fat droplet, with the remainder of the lipids composed of small amounts of phospholipids, cholesterol and cholesterol esters found in the globule membrane surrounding the milk fat droplet (Lock and Bauman, 2004). Milk fat contains over 400 different FA, with most originating as intermediates formed during lipid metabolism by the rumen bacteria. These FA, however, are mainly present in trace amounts with only about a dozen FA making up the majority of milk fat. The typical FA composition of bovine milk fat is represented in Figure 1; the specific FA profile will, however, depend on diet, rumen fermentation and physiological variables. Milk FA originate from two sources, *de novo* synthesis and the uptake of preformed FA (Bauman and Griinari, 2003). Substrates for *de novo* synthesis are acetate and β-hydroxybutyrate produced during rumen fermentation. They are used by mammary epithelial cells for the synthesis of short and medium chain FA (4:0 to 14:0) plus a portion of the 16 carbon FA in milk fat. The remainder of the 16 carbon and all of the longer chain FA are derived from mammary uptake of circulating long-chain FA that arise from digestive tract absorption and the mobilization of body fat reserves. In the dairy cow *de novo* synthesis and the uptake of preformed FA contribute approximately equally to milk FA (molar basis), although this can vary according to physiological state (Bauman and Griinari, 2003). For example, the contribution of FA from body fat reserves can vary from about 5% of milk FA in a well-fed animal to over 20% in early lactation when cows are in negative energy balance.

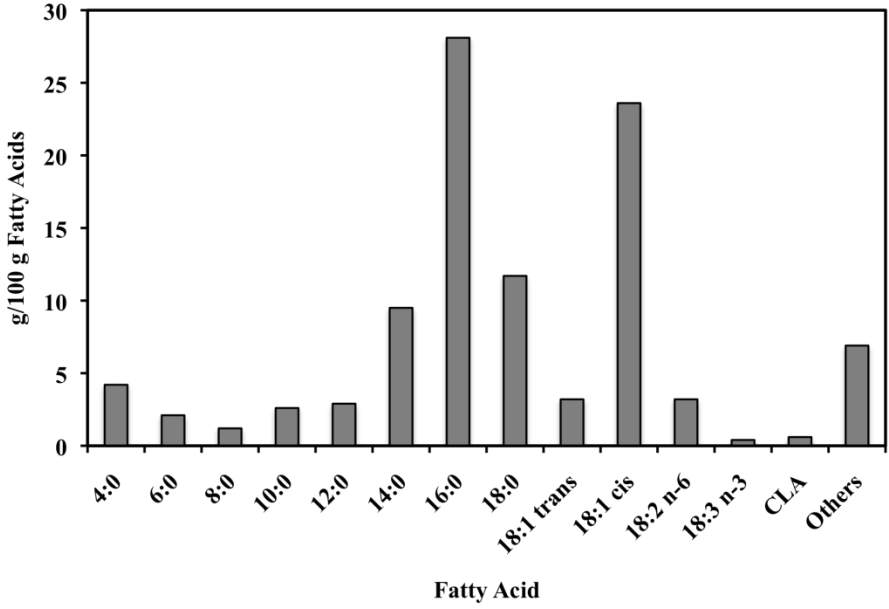

Figure 1. Representative example of the fatty acid composition of milk fat from dairy cows. Constructed using data from O'Donnell-Megaro et al. (2011).

■ Saturated Fatty Acids

Saturated fatty acids (SFA) comprise about 60% of milk FA (Figure 1) and thus milk fat is considered a saturated fat. For over a half-century the concept of healthy eating has become synonymous with avoiding dietary fat, especially saturated fat, and on a population basis a low saturated fat diet remains at the heart of nutritional advice for lowering plasma cholesterol and reducing the risk of coronary heart disease (CHD). The 2009 American Heart Association Pediatrics and Adult Nutrition Guidelines re-affirmed a target of reducing saturated fat intake to <7% of total energy intake (Gidding et al., 2009). One means to achieve this is by selecting fat-free (skim) and 1% fat milk and low-fat dairy products, and this has been a central public health recommendation in many countries. Recent estimates indicate that approximately 30% of our dietary intake of saturated fat comes from dairy products with cheese being the major source (Figure 2).

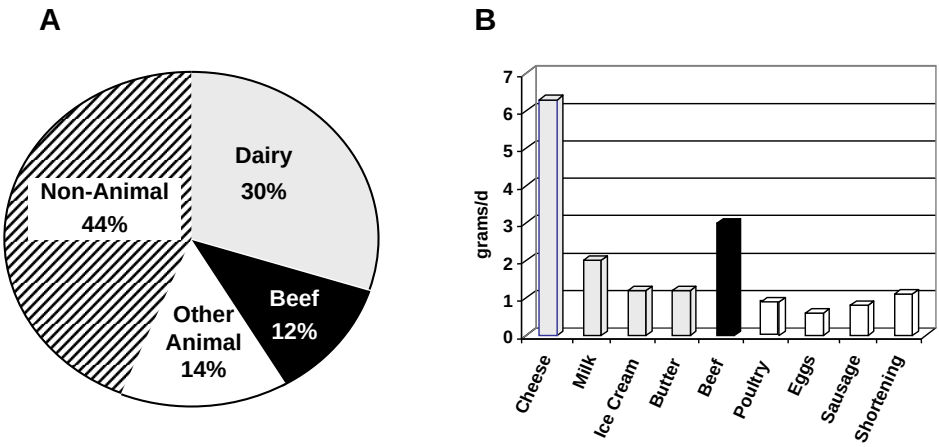

Figure 2. Saturated fatty acid intake in the US population presented as source (Panel A) and quantity from different animal-derived foods (Panel B). Constructed using data from Ervin et al. (2004).

Initial research indicated that SFA intake was the major determinant of circulating cholesterol. However, the relationship of fats, cholesterol and health is far more complex than initially thought and today many dietary, genetic and secondary factors causing hypercholesterolemia have been identified (Grundy and Vega, 1990). In reviewing the history and politics behind the diet-heart hypothesis, Taubes (2001) concluded that after 50 years of research, there was little evidence that a diet low in saturated fat prolongs life. This conclusion is reinforced by recent results from several large-scale investigations. For example, the Women's Health Initiative, an 8-year randomized dietary modification trial involving ~50,000 women, represents the largest dietary intervention ever undertaken; results demonstrated no differences in risk of CHD, stroke or cardiovascular disease (CVD) for the group in which the dietary intervention reduced total fat intake and increased intakes of vegetables, fruits and grains (Howard et al., 2006). Likewise, a recent meta-analysis of 21 prospective epidemiologic studies concluded, "there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD" (Sira-Tarino et al., 2010). Clearly, the relationship of fats including saturated fats, cholesterol and CHD is more complex than initially thought and the risk of CVD is multifaceted (Parodi, 2009).

The Nutrition Committee of the American Heart Association has emphasized the diversity of the biological effects of individual FA and the need to evaluate specific FA with respect to a range of variables related to the risk of CHD (Kris-Etherton et al., 2001). The SFA in milk vary in structure and most have no effect on circulating cholesterol and no negative implications with regard to

human health. Of the SFA in milk fat, only lauric (12:0), myristic (14:0) and palmitic (16:0) acids have been shown to increase blood levels of total cholesterol and LDL-cholesterol when added as dietary supplements, but these represent only one-quarter to one-third of total milk FA (see Figure 1). Further advances in this area have established that lauric, myristic, and palmitic acids also result in increases in circulating HDL-cholesterol, a change that is associated with a reduced risk of CHD (Mensink et al., 2003). Thus, the pattern of changes of circulating cholesterol in different lipid fractions is an important consideration, and several recent investigations suggest that comparisons of the ratio of total cholesterol:HDL-cholesterol is among the best indicators of atherogenic risk.

A meta-analysis by Mensink et al. (2003) provides convincing evidence of differences in the effects of individual FA on risk factors for CHD, but also challenges conventional wisdom about the atherogenic risk from specific saturated FA; using data from 60 clinical studies, they evaluated CHD risk on the basis of the plasma ratio of total cholesterol:HDL-cholesterol. As illustrated in Figure 3, ratios for 12:0, 14:0 and 16:0 provide little or no evidence for an atherogenic effect when compared to an isoenergetic substitution with carbohydrate; in fact the ratio for lauric acid was significantly decreased. When compared by FA group, the meta-analysis revealed no effect of saturated fat when compared to carbohydrate substitution on an isoenergetic basis (Figure 3). However, changes in ratio were indicative of the well-established anti-atherogenic effect of monounsaturated and polyunsaturated fats and the increased atherogenic risk of *trans* fatty acids (TFA).

Despite the large number of studies on the effects of changing dietary lipid type it is worth noting that only a very limited number of intervention studies have examined the benefits of reduced-SFA dairy products on CVD risk factors in humans. Those that have been done were of fairly short duration and relied almost entirely on effects on plasma cholesterol (see review by Givens and Minihane, 2009).

It is important to recognize that individuals do not consume SFA as a dietary entity, but rather as fats in food. The appropriateness of the AHA recommendations regarding the intake of dairy products is also challenged by conclusions from a number of recent meta- analyses and data summaries including those by German et al. (2009) and Elwood et al. (2010). In general these reviews have observed no association between the intake of milk and dairy products with variables related to the risk of CHD and in some cases a slight beneficial effect was reported. Overall, available evidence does not support the concept that consumption of dairy products adversely affects the risk of CHD. Indeed, there appears to be an enormous disconnect between the evidence from long-term prospective studies and perceptions of harm from the consumption of dairy products (Elwood et al., 2010). It is unfortunate

that due to a focus on the small rise in blood cholesterol with milk drinking, the debate on milk has never achieved a reasonable balance in the evaluation of risks and benefits. In addition, as will be discussed later, several bioactive FA found in milk fat have potential benefits for health maintenance and the prevention of chronic diseases, and this reinforces the need for the dietetic community to reconsider current recommendations on dairy fat and human health.

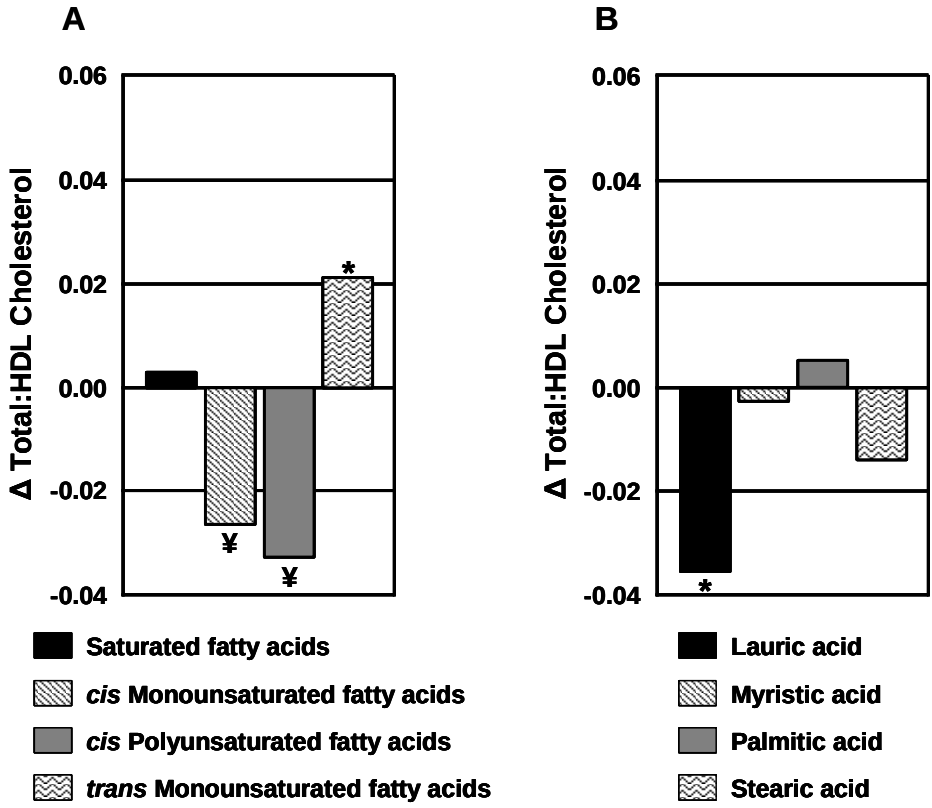

Figure 3. Predicted changes in the ratio of serum total to HDL cholesterol when carbohydrates constituting 1% of energy are replaced isoenergetically with fatty acids; A meta-analysis of 60 Trials. Panel A: saturated, *cis* monounsaturated, *cis* polyunsaturated, or *trans* monounsaturated (* = $P < 0.05$; ¥ = $P < 0.001$). Panel B: lauric acid (12:0), myristic acid (14:0), palmitic acid (16:0), or stearic acid (18:0) (* = $P < 0.001$). Adapted from Mensink *et al* (2003).

■ *Trans* Fatty Acids

Milk fat contains about 2-4% TFA, mainly *trans* 18:1 with minor amounts of *trans*-PUFA such as CLA. The double bond in UFA present in foods is typically of a *cis* configuration, but TFA have been of considerable interest in recent years due to their association with increased risk of CHD and other chronic diseases. *Trans* double bonds are introduced into FA by one of two means, chemical processes during the formation of partially hydrogenated vegetable oils (PHVO; industrial sources) or as FA intermediates formed during rumen biohydrogenation (natural sources) as discussed earlier. PHVO have been used extensively in many prepared foods such as bakery products, cooking fats, margarine and fried products.

A number of countries have established policies aimed at reducing TFA intake in the human diet. These efforts have primarily involved nutritional labelling of the content of TFA in food products and/or legislation limiting the use of PHVO in industrially prepared foods. The United States, as well as other public health agencies in North America and Europe, has enacted labelling requirements for the TFA content of food products, regardless of their TFA source (Lock et al., 2005b). The specific labelling requirements (i.e. e.g. labelling thresholds) as well as definitions for what constitutes a 'TFA' differ substantially between countries. The Danish Government chose a different approach to reduce dietary intake of TFA; they decided to use a legislative approach to limit the amount of TFA in food products with any oils and fats used as ingredients in foods currently not allowed in Denmark if they contain greater than 2% TFA. In effect this legislation banned the use of PHVO in industrially-produced foods. It is important to note that this law only applies to the use of industrially-produced oils. Ruminant-derived food products are exempt from this ruling and this decision was based their extensive review of the scientific literature (Lock et al., 2005b).

Estimates for worldwide consumption of TFA have recently been reviewed (Craig-Schmidt and Rong, 2009). Over the last decade, available data from the US indicates that mean or median adult intake of *trans* fat is approximately 2 to 3 g/d or 1 to 2% of daily calories as estimated from semi-quantitative food frequency questionnaires, and 4 to 8 g/d or 2 to 3% of daily calories based on diet recalls and diet records; these estimates are lower than those published in the mid 1990's (Craig-Schmidt and Rong, 2009). In response to the various labelling requirements for TFA, the food industry has been transitioning to alternative practices that allow for a marked reduction in the use of PHVO in processed food products. As a consequence the TFA intake from industrial sources is declining, whereas the intake of TFA from ruminant sources has remained more or less constant; the net result is a reduction in the dietary intake of TFA but the proportion of total TFA intake from ruminant sources is gradually increasing. As Craig-Schmidt and Rong (2009) highlighted, within a few years the TFA in the food supply will be

mostly limited to the 'natural' supply present in ruminant-derived fats in meat and dairy products. Thus, understanding the biological effects of TFA found in dairy products and differences among TFA isomers are of great importance.

The predominant TFA in the human diet are *trans* monounsaturated FA isomers with 18 carbons (*trans*-18:1). PHVO generally contain about 40-60% TFA and the isomer profile is a Gaussian distribution that centers on *trans*-9, *trans*-10, *trans*-11 and *trans*-12 18:1 (Figure 4). In contrast, the major TFA isomer in ruminant fat (milk and meat) is VA. Differences in isomer profile are of importance because the position of the *trans*-double bond can influence both physiological properties and the rate of biochemical reactions (Lock et al., 2005b).

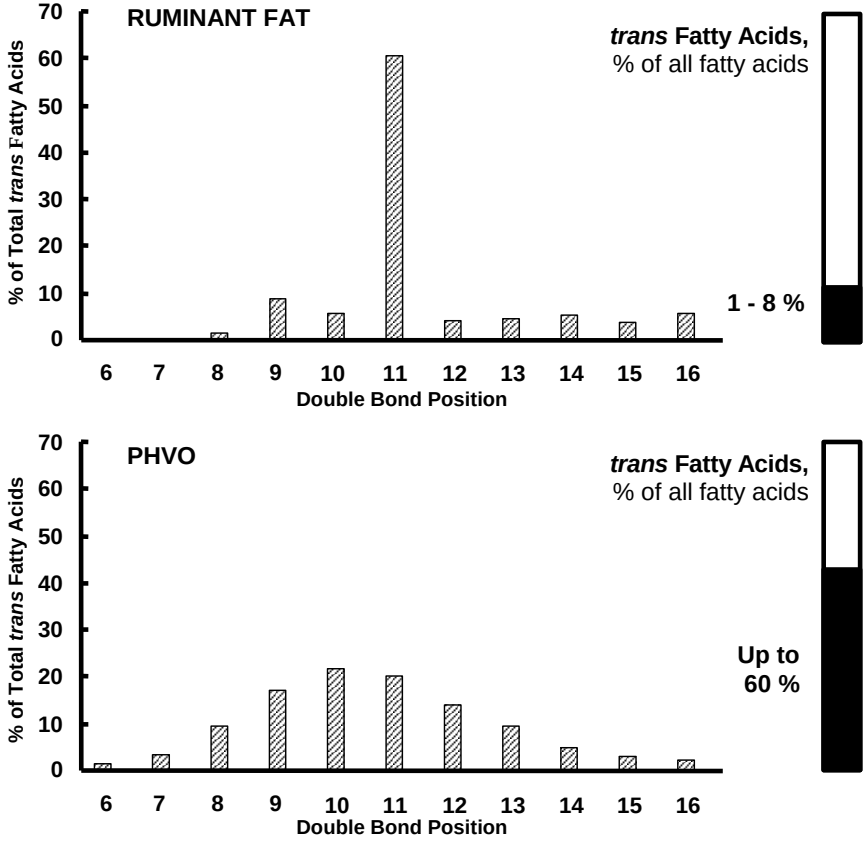

Figure 4. Typical distribution of *trans* 18:1 fatty acids in ruminant fat and partially hydrogenated vegetable oils (PHVO). Adapted from Lock *et al* (2005b).

Over the past 50 years the relationship between dietary TFA intake and plasma lipid levels and human health, particularly CHD, has been extensively investigated. Data from controlled human intervention studies have consistently demonstrated that diets containing TFA result in increased serum total-cholesterol and LDL-cholesterol, and decreased HDL-cholesterol (see Figure 3, Panel A). Prospective epidemiological studies have consistently supported findings from intervention studies further indicating that higher intakes of TFA are associated with increased risk of CHD. These results have been broadly extrapolated to imply that high consumption of all sources of TFA is associated with an increased risk of CHD. However, further examination of the epidemiological investigations reveals that the positive association with risk of CHD can be explained entirely by the intake of industrial TFA (Figure 5; Lock et al., 2005b). In contrast, the relationship between intake of naturally-derived TFA and risk of CHD observed for these studies is a significant negative association, an inverse non-significant association, or no association (Figure 5).

The difference between TFA sources and the risk of CHD probably relates to differences in the TFA isomer profile as discussed earlier. In addition, an important related aspect is that the VA in milk fat can be converted to RA via Δ^9-desaturase and this will be discussed in a later section. Consistent with this, biomedical studies with the hamster model demonstrated that a TFA-enriched milk fat had positive effects on plasma lipoprotein biomarkers (Lock et al., 2005a). Several studies have established that humans are capable of this conversion, with approximately 20% of VA converted to RA in humans, thereby doubling the CLA supply (Palmquist et al., 2005). Thus, this enzyme system may be key in differentiating VA from other *trans* 18:1 fatty acid isomers.

Finally, the impact of milk fat naturally enriched in TFA has been recently examined in human clinical studies (Chardigny et al., 2008; Motard-Bélanger et al., 2008). It is important to note that the *trans* fats found in dairy products are consumed in low amounts and results from these clinical studies indicate that current levels of intake have no significant impact on CVD risk factors. Recent reviews by Jakobson and Overvad (2009) and Malpuech-Brugère et al. (2009) provide additional information on the effects of TFA on CVD-risk factors based on epidemiological and clinical evidence, respectively.

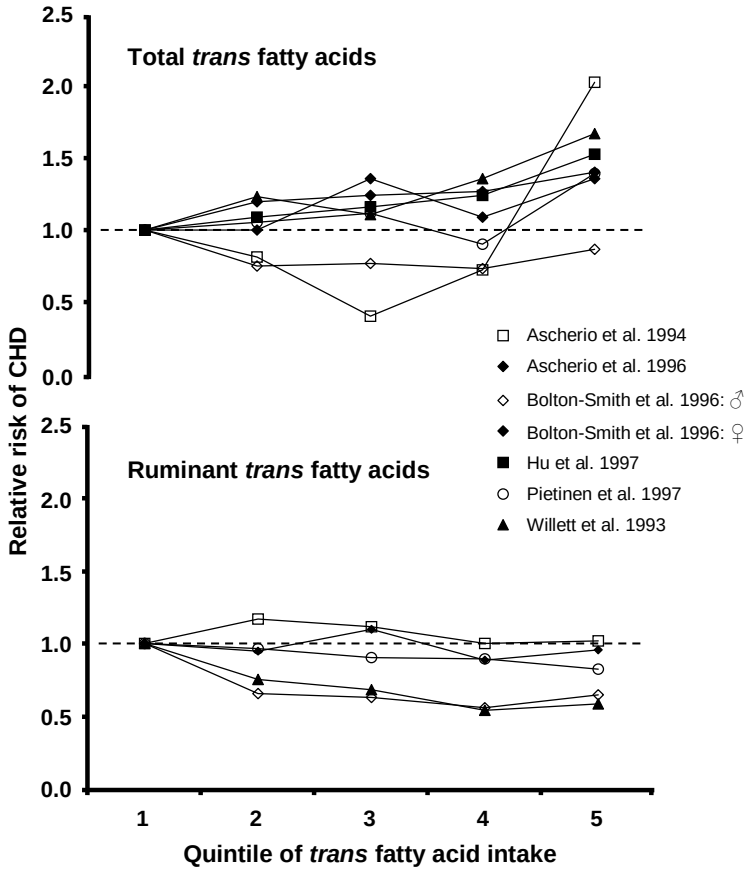

Figure 5. Relative risk of coronary heart disease with increasing relative intake (quintiles) of total and ruminant-derived *trans* fatty acids. Risks are relative to the risk in the lowest quintile of *trans* fatty acid intake; the fully adjusted model is presented for each study. Adapted from Lock *et al* (2005b).

■ Unsaturated Fatty Acids

"Functional foods" is a collective term for any food or food component that provides additional health benefits beyond that associated with traditional nutrients. Several UFA are recognized as "functional food" components having beneficial effects on health maintenance and disease prevention. For milk fat, these include oleic acid, very long-chain omega-3 FA and RA. Enhancing their content in milk fat requires an understanding of the interrelationship between dietary supply of lipid, rumen fermentation and mammary synthesis (Lock and Bauman, 2004). As discussed earlier, dietary

UFA typically undergo extensive biohydrogenation in the rumen and as a consequence minimal amounts of UFA are absorbed. Thus, even though linoleic and linolenic acids are prevalent in dairy cattle feedstuffs, the milk fat content of PUFA is only about 4% of total milk FA. An alternative method to minimize their rumen metabolism is to feed a lipid supplement formulated to protect against rumen biohydrogenation. The most common method for rumen protection of lipid supplements is the use of Ca salts; while this method minimizes adverse effects of UFA on fiber digestion, recent studies have demonstrated it offers little protection against ruminal biohydrogenation (Lock et al., 2006). As proof of concept several studies have abomasally infused plant oils as a convenient means to bypass rumen biohydrogenation; results have uniformly shown that increasing the supply of UFA results in an increase in the UFA content of milk fat. However, undesirable effects are often noted whereby the abomasal infusions also resulted in a linear reduction in dry matter intake and milk production. Thus, increasing milk fat content of UFA by increasing the supply of UFA has limitations in devising UFA formulations that will bypass rumen biohydrogenation while avoiding any adverse effects on performance.

Oleic Acid

Milk fat is an excellent source of oleic acid (*cis*-9 18:1) with this monounsaturated FA representing about 25% of total milk FA (Figure 1). As a consequence of rumen biohydrogenation, dairy cows absorb very little oleic acid. Rather, the oleic acid in milk fat originates predominately from endogenous synthesis involving mammary uptake of stearic acid and its conversion to oleic acid by the enzyme Δ^9-desaturase, also known as stearoyl-CoA desaturase. In fact, about two-thirds of the stearic acid taken up by the mammary gland is converted to oleic acid, and this has major effects on the fluidity properties of milk fat. Additional milk FA synthesized endogenously by mammary Δ^9-desaturase include myristoleic acid (*cis*-9 14:1 derived from 14:0), palmitoleic acid (*cis*-9 16:1 derived from 16:0) and RA (derived from VA), albeit these desaturase products are present at much lower concentrations than oleic acid (Bauman et al., 2006). As a proxy for Δ^9-desaturase activity, researchers often calculate a desaturase index that is based on the substrate:product ratio for the Δ^9-desaturase FA pairs (Kelsey et al., 2003). Investigations have demonstrated that the desaturase index in milk fat varies considerably among individual cows and the heritability of this index is approximately 0.2 to 0.3 (Soyeurt et al., 2008; Garnsworthy et al., 2010). Thus, the opportunity exists to select cows for high mammary expression of Δ^9-desaturase or utilize nutritional management practices that increase mammary activity of this enzyme.

Omega-3 Fatty Acids

The milk fat content of omega-3 FA are of special interest because these FA

are essential for growth and development and are beneficial in the maintenance of human health and prevention of chronic diseases including CVD, inflammatory diseases and neurological disorders (Yashodhara et al., 2009). Milk fat content of omega-3 FA is generally very low, less than 0.5% of total FA, and this is mainly α-linolenic acid (ALA, Jensen 2002). α-Linolenic acid is functional for some processes, but it's conversion to very long chain omega-3 FA (eg. eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) is essential for many physiological effects including the prevention of chronic diseases. This conversion requires Δ^6-desaturase, an enzyme that is rate limiting in humans (James et al., 2003; Whelan and Rust, 2006). Efforts to increase the omega-3 content of milk fat have often involved feeding rations that have a high content of α-linolenic acid (e.g. flaxseed, linseed oil, pasture) (Lock and Bauman, 2004; Palmquist, 2009). While this dietary approach increases the milk fat content of α-linolenic acid, it has little or no effect on milk fat levels of EPA or DHA because of limited Δ^6-desaturase activity in the mammary gland of the dairy cow (Hagemeister et al., 1991); thus omega-3 enhanced dairy products produced by this approach lack the specific omega-3 FA isomers that are necessary for health maintenance and disease prevention.

Feeding fish oil to dairy cattle leads to modest increases of EPA and DHA in milk and in some markets speciality milk and dairy products are currently available which have been enriched with EPA and DHA through adding fish oil or fish by-products to the cow's diet. Another approach is the use of plant sources of stearidonic acid (SDA), an omega-3 FA that bypasses the Δ^6-desaturase reaction. Advances in genetic modification of oil seeds allow for the production of SDA-enhanced oils, and a recent study demonstrated that dairy cows supplied with SDA-enhanced soybean oil had markedly increased omega-3 FA in their milk fat including SDA and EPA (Bernal-Santos et al., 2010). However, as discussed earlier, feeding large amounts of UFA from oils, plant seeds or other byproducts often has undesirable effects on rumen function and the biohydrogenation processes; when this occurs there is an accumulation of *trans* FA and other biohydrogenation intermediates, some of which have negative effects on mammary fat synthesis resulting in low milk fat tests (Bauman and Griinari, 2003).

Conjugated Linoleic Acid

The major source of CLA in human diets is ruminant-derived foods, mainly fat from milk and dairy products. As shown in Figure 6, RA is the predominant CLA in milk fat (~90% of total CLA). RA was originally thought to be derived from rumen outflow of bioyhdrogenation intermediates, but studies over the last decade have unequivocally established that it originates predominantly via endogenous synthesis; this involves Δ^9-desaturase with the substrate being VA, another intermediate in rumen biohydrogenation (Bauman and Lock, 2006). Therefore, strategies to increase the milk fat content of CLA

center on enhancing rumen output of VA and increasing tissue activity of Δ^9-desaturase (Figure 6). Diet has the most influence on RA content of milk fat and this has been extensively reviewed elsewhere (Chilliard et al., 2000; Palmquist et al., 2005). By choosing combinations of feed ingredients that affect the dietary supply of PUFA and/or affect the rumen environment to alter the rate and completeness of biohydrogenation, milk fat content of RA can be markedly affected. The RA content of milk fat is also influenced by physiological factors and individual variation among cows is ~3-fold (Bauman and Lock, 2006). As discussed earlier, a genetic component for desaturase has been identified using the desaturase index (milk fat ratio 14:1/14:0) to establish heritability estimates.

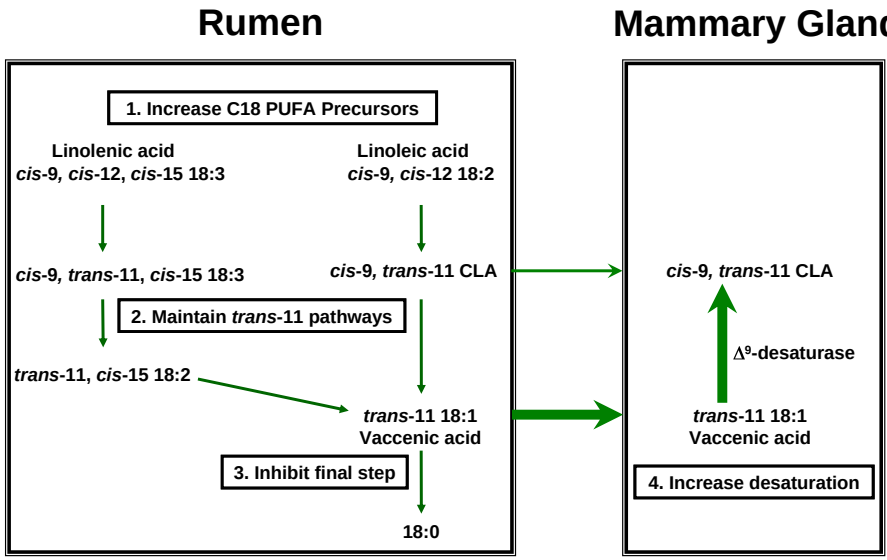

Figure 6. Pathways for ruminal and endogenous synthesis of *cis*-9, *trans*-10 CLA in dairy cows and strategies to increase milk fat content of CLA. Adapted from Bauman and Lock (2006).

Interest in RA as a functional food component relates to its anti-cancer and anti-atherogenic effects. The protective effect of RA against cancer has been consistently observed in animal and cell culture models, being especially effective in models for breast cancer (Bauman et al., 2006). In addition, VA found in milk fat is also anti-carcinogenic through its conversion to RA. The anti-atherogenic effects of RA have been established in animal models and humans, with effects due to changes in both lipoprotein and cholesterol metabolism and anti-inflammatory effects. Of special importance, butter naturally enriched in RA has been effective in biomedical studies with cancer and atherogenic models. However, extending these results to humans has

been challenging and problematic because it involves prevention of chronic diseases that often lack consensus biomarkers and there are difficulties in dietary analyses and food library values for CLA (Bauman et al., 2006).

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