Milk fats: A different perspective

J. Bruce German¹,²

SUMMARY
The recent history of public recommendations for dietary intakes of macronutrients have targeted total fat, cholesterol and saturated fat intake as the principle means to improve human health. Such recommendations have been translated into a long term agricultural objective of eliminating these components from human foods. Agricultural change requires changes at many points over many years to eliminate these components. Once accomplished, such changes would be equally difficult to reverse. Furthermore, such recommendations fall disproportionately on a few commodities most notably dairy fats. Dramatic alterations in dairy consumption carry much more impact on dietary intakes than simply total fat, saturated fat and cholesterol. While it is becoming possible to alter the basic production of milk and dairy products and to envision a dramatically different milk fat composition, such steps will be difficult and expensive. Hence, it is appropriate to ask whether the data in support of this conclusion are complete and if indeed a finite intake of milk fats are beneficial to overall health. Mammalian milks, including human milk contain 50% of their total fatty acids as saturated fatty acids. Adding a single double bond is a relatively straightforward process already active within the mammary gland. Darwinian selective pressure therefore chose to maintain a significant content of saturated fatty acids in milk. Is it possible evolution found benefits to saturated fatty acids that current recommendations do not consider? Furthermore, milk does not contain simply saturated triglycerides as a bulk fat, but rather a conspicuous range of different fatty acids varying in chain length and unsaturation. Milk is present as highly complex globules with structural properties distinct from other biological sources of fats. In particular, there are complex phospholipids making up a highly glycosylated and protein embedded plasma membrane around each milk fat globule. Milkfat is thus a source of bioactive lipids provided as highly absorbable ensembles also serving as an important delivery medium for nutrients, including the fat-soluble vitamins. While very few studies have examined the nutritional consequences of these structures and compositions, their emergence through evolution implies that they provide distinct benefits to individuals consuming them.

Keywords
diet saturated fats metabolism.

¹. From the Nurit Argov Department of Food Science & Technology – University of California – Davis – CA 95616 – USA.
². Nestlé Research Center – Lausanne. Switzerland.
1 – INTRODUCTION

1.1 Diet, fatty acids and lipoproteins

Lipids and their major structural elements, the fatty acids, are still poorly understood in biology. Nutrition is also still developing a rudimentary understanding of lipids. Fat in the diet is well recognized to provide the essential fatty acids and to dissolve and aid in the absorption of fat-soluble vitamins. Fats in the diet also produce metabolic effects that are a complex consequence of the absolute and relative fat content, the fatty acid composition, the structure of other components in the foods, the timing of consumption and individual variation in those consuming them. Fatty acids are required for membrane synthesis, for modifications of proteins and carbohydrates, for construction of various structural elements in cells and tissues, for the production of signaling compounds, and for fuel. As ensemble structures lipids also act to solubilize a variety of non-polar and poorly soluble cellular and extracellular constituents and transport such molecules within and between cells and tissues.

Part of the rationale for the lack of importance of saturated fat in the diet, is the routine ability of organisms including humans to synthesize them. In the absence of sufficient dietary fat, the body is apparently capable of synthesizing all of the saturated fatty acids that it needs from the ubiquitous building block acetate as a precursor. This does not mean to infer that all saturated fatty acids are biologically indistinguishable. Compositional analyses reveal remarkable specificities for particular saturated fatty acids in different lipid classes, cellular compartments and tissues (Watkins et al., 2002). Unfortunately, the functional reasons to explain these specificities of structure are not yet known.

The complexity of structure and the diversity of functions of fatty acids, both unsaturated and saturated, remains poorly understood and in only a few biological situations have distinct actions of fatty acids been described. Research on fatty acids consumed in the diet has focused principally on their role in lipoprotein metabolism. Even for lipoprotein metabolism which literally billions of dollars have been invested in research little is actually known. Only in 2006 was the basic mechanistic link between saturated fatty acids and cholesterol metabolism revealed (Lin et al., 2005). In fact the relationship between saturated fat in the diet and cholesterol metabolism was one of the greatest scientific challenges of the 20th century. How could such a ubiquitous, non-essential component of diets and tissues – saturated fat, cause an increase in the accumulation of cholesterol rich LDL in blood? As scientific research on cholesterol metabolism proceeded through the 20th century, the question became even more perplexing. Brown and Goldstein won the Nobel prize for identifying the LDL receptor on the liver as the major determinant of serum cholesterol (Brown and Goldstein 1986). They then went on to show that cholesterol levels in the liver cell simultaneously regulated the expression of LDL receptor and cholesterol biosynthesis both by controlling gene expression and both using the identical transcription factor protein, sterol response factor binding protein (SREBP) (Brown and Goldstein 1997). While these studies made sense of cellular cholesterol regulation: if the cell needs more cholesterol, it simultaneously turns on the genes to make more (cholesterol biosynthetic enzymes) and to take more from blood (LDL receptor), they made even less sense for diet.

If the same transcription factor turns on both cholesterol biosynthesis and the LDL receptor, how can saturated fat uncouple these two and simultaneously make more cholesterol but shut down the receptor? Bruce Spiegelman found that the liver contains an additional gene control system, the peroxisome proliferator activating receptor (PPAR) and it is in turn controlled by a higher order protein complex which he termed (logically) the PPAR Gene transcription Coactivator (PGC-1) (Puigserver and Spiegelman, 2003). This transcription factor coactivator family recruits entire complexes of proteins into transcriptional regulatory units controlling such multi-faceted properties as mitochondrial biogenesis (Wu et al., 1000). In a striking result (Lin et al., 2005), his group discovered that when exposed to high levels of saturated fatty acids, liver cells, in vivo and in vitro...
actively turned on PGC-1b, and even more astonishingly this coactivator simultaneously turned on cholesterol biosynthesis but turned off the LDL receptor. Thus in one bold study the basic target linking dietary saturated fat and serum cholesterol was revealed. An obvious question is: what are the benefits to this biochemical response that would have caused it to be selected through evolution?

The basis for recommendations that the population decrease their intake of saturated fats was not mechanistic understanding but years of observational evidence that dietary saturated fats generally increase blood cholesterol concentrations in animals and humans (Food and Nutrition Board 2002). This alteration of risk factors does not necessarily lead to increases in actual heart disease and is certainly not universally true in all populations studied. Some studies of human populations evaluating the effects of saturated fat diets do not show the predicted elevation in heart disease, in fact some see the reverse (Mozaffarian et al., 2004). Recent studies are beginning to assign genetic or physiological explanations to these varying outcomes for example low birth weight appears to have an effect on subsequent responses to dietary fat (Robinson et al., 2006).

1.2 Are recommendations to lower total fat intakes justified for everyone?

Are the justification for broad recommendations to lower total fat intakes in all individuals supported by scientific evidence? In 1977 the US population was first recommended to reduce the intake of fat, with some recommendations being to reduce total fat to below 30% of calories (Dietary Goals for the United States 1977). The American Heart Association recommended that the percentage of calories be 28.6 and 25.3% total fat and 9 and 6.1% saturated fat, in respectively Step 1 and Step 2 diets for treatment of high blood cholesterol. This recommendation had unanticipated effects. Framingham Heart Study data showed that people with high triacylglycerol concentrations (>1.7 mmol/L) and low HDL cholesterol concentrations (<1.03 mmol/L) run a significantly higher rate of coronary artery disease (Castelli 1992). The long-term health benefits of consuming a low-fat diet – particularly taking into account the variation in human responses – have not been proven and to the contrary, some individuals move their risk profile even for heart disease in an adverse direction (Dreon et al., 1994, Krauss and Dreon 1995). In one study healthy, non-diabetic volunteers consumed diets that contained – as a percentage of total calories – either 60% carbohydrate, 25% fat and 15% protein, or 40% carbohydrate, 45% fat and 15% protein (Abbasi et al., 2000). Those consuming the 60% carbohydrate diet had higher fasting plasma triacylglycerol, remnant lipoprotein and remnant lipoprotein triacylglycerol, and lower HDL cholesterol without changing LDL cholesterol concentration. The low-fat diet caused both lowered HDL cholesterol and included a persistent elevation in remnant lipoproteins (Abbasi et al., 2000) both factors increasingly recognized to be important independent risk factors for heart disease and other metabolic diseases. These findings led these investigators to publish the question whether it is wise to recommend that all Americans replace dietary saturated fat with carbohydrate. Perhaps the most devastating study to the basic principle that a lower fat diet improves the health of the entire population was the prospective study (The Women’s Health Initiative Randomized Controlled Dietary Modification Trial) selected approximately 48,000 women to compare low fat diets and increased fruit and vegetable consumption and saw no statistical improvements in heart disease outcomes (Howard et al., 2006). Two possible interpretations could be drawn from this study, first that lower fat intakes have no effect on any women or second that some are benefited and some are adversely affected and the net numbers of each are relatively close leading to a conclusion in this trial of no effect. Do individuals vary in their response to fat?

1.3 Individual response to dietary diet intakes

Studies of response to dietary fats have found variation among individuals and differences between men and women in their response to dietary fat changes. (Cox et al., 1995, Weggemans et al., 1999, Velez-Carrasco et al., 1999). There are also differential responses in individuals that consume low-fat diets (Aztolos et al., 2000). A series of stud-
ies showed that very-low-fat (10%), high-carbohydrate diets enriched in simple sugars increased the synthesis of fatty acids, especially palmitate, and that the individual differences in increased triglyceride levels varied considerably (Hudgins 2000). These fluctuations seen time after time have propelled the field of Nutrigenomics and scientists are now pursuing more detailed analyses of individuals, their responses to diet and the mechanistic basis for variations in diet and risk (Or dov as 2007).

Controversy still remains high as to the roles that dietary fat and cholesterol play in the risk of heart disease and the wealth of confounding factors demonstrate that saturated fat is not an overwhelming input variable for any populations studied to date. Dietary saturated fats are not the only variables associated with heart disease – the causes are multi-factorial. The results of studies on the etiology of heart disease are inclusive and sometimes contradictory. It is time to take a broader view to the multiple actions and functions of each of the different saturated fats and a more individual view to assessment of diet and risk.

1.4 Biological Activities of Saturated Fatty Acids

The overwhelming emphasis on the role of saturated fats in the diet and risk of CHD has distracted investigators from studying other effects that individual saturated fatty acids may provide to the body. One striking observation is the presence in mammalian milks of a wide range of saturated fatty acids with different chain lengths. It therefore seems prudent in view of the Darwinian selective pressure on milk and all of its constituents to at least ask how they may affect growth, development and survival of mammalian offspring.

Fatty acids are parts of all body tissues, where they are a major part of the phospholipid component of the cell membrane and contribute to the structural diversity within the membrane now recognized to be a key aspect of membrane functions (Fielding 2007). Fatty acids anchor proteins to particular regions of cell membranes, participate in signaling activities, transport cellular components and provide fuel. Saturated fatty acids have been suggested to be the preferred fuel for the heart (Lawson and Kumerow 1979). In the absence of sufficient fat from the diet, the body synthesizes fatty acids from carbohydrates. When present in the diet, different structures of fatty acids appear to have differing effects on a variety of metabolic and physiological processes.

1.5 Butyric acid

Short-chain fatty acids are hydrolyzed preferentially from triacylglycerols and absorbed from the intestine to the portal circulation without resynthesis of triacylglycerols. These fatty acids serve as a ready source of energy. Butyric acid (C:4) is the shortest saturated fatty acid and is present in ruminant milk fat at 2–5% by weight (Smith and German 1995), which on a molar basis is about one-third the amount of palmitic acid. Human milk contains a lower percentage (ca. 0.4%) of butyric acid. No other common food fat contains this fatty acid directly, however the consumption of a wide range of fermentable carbohydrates can lead to the synthesis of butyric acid by endogenous microflora in the lower intestine. Butyrate is a well-known modulator of genetic regulation and its ability to promote differentiation has led various investigators to pursue this mechanism as a means to alter the risk and development of cancer (Smith et al., 1998, Young, et al., 2005).

1.6 Caproic caprylic and capric acids

In bovine and human milk, respectively, caproic acid (C:6) is present at ca. 1 and 0.1%, and caprylic acid (C:8) and capric acid (C:10) are present at ca. 0.3 and 1.2%, respectively, of the milk fat. Goat milk contains the highest percentage of caprylic acid, at 2.7% of milk fat. Studies to date have documented that three fatty acids have similar biological activities as antimicrobial agents. Caprylic and capric acid have antiviral activity, its monoglyceride form, monocaprin has been shown in vivo in animals to possess antiviral activity against retrovirus infection (Neyts et al., 2000).
1.7 Lauric acid

Lauric acid (C:12) is present in human and bovine milk at ca. 5.8 and 2.2%, respectively, of the milk fat. Studies have shown antiviral and antibacterial activities (Hornung et al., 1994). Release of lauric acid in the stomach may have direct antimicrobial activities towards *H. pylori* either as the fatty acid or monoacylglycerols produced by pre-intestinal lipase(s) acting on triacylglycerols milk fat (Sun et al., 2002, 2003). Interestingly lauric acid may provide oral health benefits according to anticaries and antiplaque activities (Schuster et al., 1980). The overall antimicrobial effects of the medium-chain saturated fatty acids and their monoacylglycerol derivatives on various microorganisms, including bacteria, yeast, fungi and enveloped viruses, were originally suggested to be acting through the lipid membranes of the organisms (Thormar et al., 1987). Support for this deactivation process has been shown using human and bovine milk (Isaacs et al., 1995). Monolaurin released from milk lipids lipases may account for milk’s anti-protozoal activities (Reiner et al., 1996).

1.8 Myristic acid

Bovine milkfat contains 8–14% myristic acid (C:14) and in human milk, it averages 8.6% of the milk fat. Human epidemiological studies have shown that myristic acid and lauric acid were the saturated fatty acids most strongly related to the average serum cholesterol concentrations in humans (Kromhaut et al., 1995). Nonetheless several studies have shown that myristic acid increases HDL at least as much as LDL cholesterol and further studies have demonstrated that the unique positional distribution of myristic acid in the sn-2 position of triglycerides in milkfat is responsible for its tendency to raise HDL (Dabadie et al., 2005, ).

1.9 Palmitic acid

Palmitic acid (C:16) is present in human and bovine milk at 22.6 and 26.3%, yet almost exclusively esterified at the sn-2 position of the triglyceride. This unusual stereospecific distribution appears to have important nutritional and biological implications. Human infants consuming a formula containing triacylglycerides similar to those in human milk (16% palmitic acid esterified predominantly to the sn-2 position) improve intestinal absorption not just of the palmitic acid but calcium as well (Carnielli et al., 1995, 1996).

1.10 Stearic acid

Stearic acid is present in human and bovine milk fat at 7.7 and 13.2% of fat, respectively. Stearic acid does not appear to raise serum cholesterol hence is considered neutral to heart disease risk. It may exert other effects also consistent with protection from heart disease via separate mechanisms. Healthy males who consumed dietary stearic acid (19 g/d) for 4 wk exhibited beneficial effects on thrombogenic and atherogenic risk factors as compared with the effects of dietary palmitic acid (Kelly et al., 2001).

1.11 Long-chain Unsaturated Fatty Acids

Although most dietary PUFA are desaturated in the bovine rumen, PUFA are present in bovine milk at about ~3.4% of total fatty acids and 91% of milkfat globule membrane fatty acids (Jensen, 2002). Arachidonic acid (20:4 n-6) is present at ~0.1% of fatty acids, and although DHA (22:6 n-3) is present at < 0.1%, its concentration in bovine milk can be increased by feeding fish meal (Whitlock et al., 2002). Long-chain PUFA have been shown to exert a wide variety of effects on neurological processes, cellular regulation, metabolic regulation and immune functions (Browning et al., 2002, Caplan and Jilling 2001). A variety of studies have attempted to address the importance of these PUFA in bovine and
human milk, but the lack of mechanistic knowledge of the function of membrane lipids themselves makes it difficult to interpret the specific effects of individual long-chain PUFA. It can be stated with some confidence that the next decade of life science will be the lipid decade as the basic biological properties, functions and activities are finally understood. On this background, nutrition research and the importance of dietary polyunsaturated fatty acids in milk can be finally addressed.

1.12 Delivery of Fat soluble nutrients

Fat-soluble nutrients include the essential nutrients vitamins A, D, E and K, carotenoids as vitamin A precursors, essential PUFA, and non-essential nutrients that cannot be made by humans, such as various tocopherols, phenolics, carotenoids (e.g., lycopene, lutein and zeaxanthin) and conjugated linoleic acid (CLA) isomers. Fat-soluble nutrients are considered to have biological activities beyond the simple prevention of deficiency and are consistent with many aspects of overall health (Giovannucci et al., 1995; Traber and Packer, 1995, Dietrich et al., 2006). In particular, the abundance of fat-soluble nutrients in tissues is frequently reported to be inversely correlated with a variety of chronic and degenerative diseases, including cancers (Zhu et al., 1996; Ahmed et al., 1999), cardiovascular diseases (Palace et al., 1999a; Palace et al., 1999b), diabetes (Haffner, 2000) and specific tissue degeneration such as macular degeneration (Belda et al., 1999; Delcourt et al., 1999). With the recognition that there are potential health values associated with fat-soluble nutrients, their absorption has become a key issue. In general, non-polar molecules are poorly absorbed, and it is not certain that the presence of a component in a food means that it will be absorbed and delivered to particular tissues in which it might be active (Papas, 1996).

1.13 Milkfat as a Nutrient Delivery Medium

Although adequate for prevention of overt nutrient deficiencies, the overall nutrient content of the U.S. diet is in many cases less than that considered for optimal health through an entire life span in all individuals (Surgeon General 1988). As a result, some individuals are looking to supplements to augment their nutrient intakes. However, pills are not ideal for the delivery of all nutrients. Not only are some nutrients in pill form apparently less effective than when delivered in food, but in some instances and in some individuals, the net actions of the nutrients in supplement form can even be deleterious (Patrick, 2000). Two examples of natural food nutrients being superior to supplemental nutrients are b-carotene and vitamin E. The supplement form of synthetic b-carotene is all-trans in contrast to the natural cis-trans isomeric mixtures found in most foods. In the intestinal enterocyte, the 9-cis isomer is a direct precursor to 9-cis retinoic acid (Nagao and Olson, 1994). Retinoic acid acts as a hormone in signaling processes where it binds to nuclear receptors and controls normal reproduction and maintenance of epithelial tissue (Hansen et al., 2000). 9-cis retinoic acid is formed only from 9-cis b-carotene, whereas all-trans b-carotene is transformed into only all-trans retinoic acid (Wang et al., 1994). Vitamin E is a nutrient that has greater bioactivity when provided in milk because it is better absorbed from milk, with or without fat, than it is from capsules (Perlman and Hayes, 2000).

The mechanisms underlying differences between nutrients provided in food or as a supplement are complex, and it is clear that the matrix in which a nutrient is dispersed affects its stability, digestion, absorption, metabolism, distribution, tissue and cellular uptake, turnover and excretion. Myriad actions of nutrients are influenced by the food matrix in which the nutrients are dispersed and consumed, and it is reasonable to examine the molecular basis of nutrient delivery. Bovine milkfat is easily absorbed, and in the Western diet, it is a substantial delivery vehicle of energy and micronutrients, including a-tocopherol and carotenoids. Milkfat is a food matrix that increases the net absorption of nutrients in growing mammals. Application of a dairy food matrix to other foods could enhance delivery of other fat-soluble nutrients. Milkfat has been recognized for decades to be a “solvent” for the fat-soluble vitamins present in milk. This solvent effect is impor-
tant in carrying fat-soluble vitamins from the mammary gland to the infant, and even in assisting with carrying them through the intestine. However, the more active roles that the lipid components of milkfat play in accelerating metabolism and in successfully delivering the nutrients to the appropriate tissue are unknown. Clearly, it is appropriate to investigate the solvent effect of milkfat more fully. There is a need to explore the various actions of milkfat that may impact the ultimate tissue status of particular nutrients following their consumption in a food containing milkfat rather than exploring milkfat as merely a food nutrient.

1.14 Milkfat and HDL

One of the well established properties of milkfat is the stimulation of HDL. It is well established that HDL cholesterol levels are a very strong and independent predictor of heart disease, they have not received the same attention as LDL, ironically because the levels of HDL are not as responsive to diet and drugs as LDL. Substantial epidemiological research has shown associations of HDL and heart disease, both high HDL and protection from heart disease, even in the face of elevated LDL, and low HDL and increased risk, with or without elevated triglycerides. However, it has not been possible to assign independent variables to HDL differences and studies have largely been based on HDL levels that are presumably high or low based on genetic rather than dietary determinants (Wang et al., 2004, Wang and Paigen 2005).

HDL exert beneficial effects on overall health by myriad mechanisms including binding and eliminating toxins, delivering bioactive compounds, protecting various cells and lipoproteins from damage and participating in their repair (Argraves and Argraves 2007, Rader 2006, Canturk et al., 2002). HDL is particularly important in the successful response to infection by binding and clearing bacterial endotoxin or lipopolysaccharide. Lipopolysaccharide (LPS) is the major glycolipid component of gram-negative bacterial outer membranes and is responsible for pathophysiological symptoms characteristic of infection. A wide variety of studies have documented that LPS is associated with plasma lipoproteins, suggesting that sequestering of LPS by lipid particles may form an integral part of a humoral detoxification mechanism (Feingold et al., 1995, Pajkrt et al., 1996). The binding of LPS to lipoproteins is highly specific under simulated physiological conditions, and HDL has the highest binding capacity for LPS (van Leeuwan et al., 2001, Levels et al., 2000). This basic protection mechanism may be particularly important for children (Liuba et al., 2003) and for intestinally derived endotoxin. Thus, lipoprotein-binding protein-lipoprotein complexes may be part of a local defense mechanism of the intestine against translocated bacterial toxin. Because milk fats enhance HDL concentrations, they are of potential importance in protection against bacterial LPS toxicity.

1.15 Milk fat Globule Structure

Lipids are mostly insoluble and hence the vast majority of lipids in cells of all organisms are not present as soluble molecules but as insoluble aggregates. The milk fat globule is a model of the extent of structural complexity possible within biological materials. The milk fat globule is primarily a core of triacylglycerides synthesized within the endoplasmic reticulum of the mammary epithelial cell. This core of triacylglyceride is bounded by a single monolayer of polar lipids such as phospholipids derived from the endoplasmic reticulum membrane. The entire globule as secreted, however, is bounded by a bilayer membrane, which enrobes the globule as it exits the epithelial cell. This membrane is composed of the lipids and proteins of the epithelial cell plasma membrane, including significant quantities of cholesterol, phosphatidylcholine and sphingomyelin. Additional components unique to the external surface of the native fat globule include glycolipids, gangliosides and significant quantities of membrane glycoproteins and mucins. The membrane helps to stabilize the fat globules in an emulsion within the aqueous environment of milk.
To date little attention has been given to the structural properties of milk lipids and their nutritional implications. The fact that these structures vary widely, but consistently, among all mammalian species and among different periods of lactation implies that structure is of functional value. However, the techniques necessary to describe the structures of lipids are not fully developed. As these techniques become available, research must address how variations in structure influence biological and nutritional properties.

2 – CONCLUSIONS

The genes and biochemical processes of lactation that produce milkfat evolved under the constant Darwinian selective pressure of nourishing mammalian infants. Lipids in milk are a source of energy for the neonate of each species. The composition and structures of lipids in milk provide bioactive components that, although not identified as "essential" nutrients by standard definitions, none-the-less serve important functions as structural building blocks, fuels, transport systems, anti-inflammatory, anti-bacterial and antiviral agents in the intestine. These lipids include triacylglycerols – which are metabolized to monoacyl- and diacylglycerides and fatty acids – and phospholipids such as sphingomyelin. The lipids in milk are also carriers of important fat-soluble vitamins such as vitamin E, vitamin A and vitamin D.

Among the 12 major fatty acids present in bovine milk fat, three saturated fatty acids – lauric, myristic and palmitic acids – have been identified as dietary factors that raise blood cholesterol levels. It is still unknown what the mechanisms by which this association with lipoproteins in blood occurs. Although lauric and palmitic acids are reported to raise LDL-cholesterol, which has been linked with an increased risk of coronary heart disease, saturated fatty acids, especially myristic acid may also significantly raise the level of blood HDL-cholesterol, which has a positive association with reduced risk of heart disease.

The unique structure of the milk fat globule membrane and its nutritional properties has been largely ignored in research on milk and its health value. The globule components provide unique constituent proteins and polar lipids, and also the phospholipids phosphatidylcholine, phosphatidylethanolamine, sphingomyelin, phosphatidylserine and phosphatidylinositol. As the science of lipid nutrition moves forward, milk with its unusual complex composition and structures, its rate of digestion and the selective absorption and delivery of fat soluble nutrients to targeted tissues should be a valuable model to guide research and establish nutritional benchmarks. While an unusual set of environmental and dietary habits led a generation of scientists to assign much of the cause of heart disease to milk fat, recent research and epidemiological outcomes are not reaching the same conclusion.
REFERENCES


CAPLAN M.S. and JILLING T. 2001. The role of polyunsaturated fatty acid supplementation in intestinal inflammation and neonatal necrotizing enterocolitis. Lipids, 36, 1053-1057.

CARNIELLI V.P., LUIJENDIJK I.H., VAN GOUDEVER J.B. et al., 1995. Feeding premature newborn infants palmitic acid in amounts and stereoisomeric position similar to that of human milk: effects on fat and mineral balance. Am J Clin Nutr, 61, 1037-42.


Milk fats: A different perspective


The absorbability by rats of various triglycerides of stearic and oleic acid and the effect of

© Lavoisier – La photocopie non autorisée est un délit.


