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Review

Milk fatty acids and potential health benefits: An updated vision

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ABSTRACT

Background: Milk fat intake is often associated with a high risk of suffering from cardiovascular disease (CVD) due to its high saturated fat content. However, not all saturated fatty acids (SFA) are equal and they present structural differences that promote distinct effects on the biological processes. In addition, there is a growing scientific consensus pointing to dairy fat as a natural source of bioactive components.

Scope and approach: The present review provides the most recent knowledge on the bioactive properties of fatty acids detected in dairy products and their potential effects on consumer health. The metabolic processes that involve these fatty acids and serious chronic diseases such as CVD, obesity, diabetes or cancer are explained and discussed throughout the text based on *in vitro*, animal and human studies. Moreover, information gaps are highlighted to inspire further research in the field.

Key findings and conclusions: Recent investigations support that milk SFA should no longer be considered as a single group in terms of metabolism or negative effects in case of excess. Even they suggest that individual SFA possess specific properties associated with important physiological functions. Whole dairy products would also promote human health due to the presence of certain bioactive fatty acids. Among them, it is worth mentioning the maintenance of gut microbiota and weight control from short and medium-chain SFA, the essential role of branched-chain SFA in gut health at birth and the prevention of chronic inflammatory diseases by vaccenic and rumenic acids.

1. Introduction

Lipids are among the most important constituents of milk for nutritional and economic reasons. They are a good source of energy and provide unique sensory and physical attributes to dairy products. Milk fat is also carrier of the naturally present fat-soluble vitamins (A, D, E and K) as well as β-carotene, a pro-vitamin A carotenoid. The main lipids in dairy fat are the triacylglycerides (TAG), accounting for more than 98% of total fat, while the remaining 2% comprises diacylglycerides, monoacylglycerides, free fatty acids (FFA), phospholipids, sterols and hydrocarbons. TAG composition is extremely complex as more than 400 different fatty acids (FA) can be esterified in the three positions (*sn*-1, *sn*-2 and *sn*-3) of the glycerol backbone at different concentrations, which mainly depend on ruminant diet and its lactation stage (Hanuš, Samková, Křížová, Hasoňová, & Kala, 2018; Jensen, 2002; Schroeder & Vetter, 2013; Shingfield, Bonnet, & Scollan, 2013). The most abundant milk FA are reported in Table 1.

It is estimated that dairy products (excluding butter) contribute to 24% of the saturated fat intake of the USA diet and 25–30% in European countries (Liang, Zhou, Amakye, Su, & Zhang, 2018). The dogma that dietary saturated fatty acids (SFA) should be minimized to reduce the metabolic syndrome and cardiovascular disease (CVD) risk has dominated nutritional guidelines for decades and the high content of SFA in milk fat (about two-thirds of total FA) has been currently employed as argument to link dairy products consumption with an increased incidence of those pathologies. However, recent scientific studies do not justify the maintenance of those recommendations in a healthy population (Astrup et al., 2016; Guo et al., 2017; Lovegrove & Givens, 2016). Firstly, up-to-date research does not support an association between biomarkers of dairy fat intake with risk of diabetes mellitus or CVD (Kleber, Delgado, Lorkowski, Marz, & Von Schacky, 2016; Liang et al., 2018; Yakoob et al., 2016). Furthermore, the observational evidence does not endorse the hypothesis that high-fat dairy foods contribute to metabolic syndrome or cardiovascular risk, and even indicate that fat dairy consumption within typical dietary patterns is inversely associated with this risk (Alexander et al., 2016; Kratz, Baars, & Guyenet, 2013; Thorning et al., 2017).

In the past, it was widely accepted that the intake of 12:0, 14:0 and 16:0 SFA, which are detected in relevant quantities in dairy fat, would seem to be unhealthy in excessive amounts. However, the matrix in which these SFA are contained may influence health outcomes. Recent research has shown that several dairy matrix components, mainly

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Table 1
Mean values of cow, sheep and goat milk fat main fatty acids (% in total fatty acid methyl esters). Data reported from De la Fuente, Ramos, Recio, and Juárez (2013)[1] and Alonso, Fontecha, Lozada, Fraga, and Juárez (1999)[2].

Fatty Acid	Cow[1]	Sheep[1]	Goat[2]
4:0	3.9	3.5	2.2
6:0	2.5	2.9	2.4
8:0	1.5	2.6	2.7
10:0	3.2	7.8	10.0
12:0	3.6	4.4	5.0
14:0	11.1	10.4	9.8
15:0	1.2	1.0	0.7
16:0	27.9	25.9	28.2
17:0	0.6	0.6	0.7
18:0	12.2	9.6	8.9
cis 18:1	17.2	18.2	19.3
trans 18:1	3.9	2.9	2.1
18:2 n-6	1.4	2.3	3.2
18:3 n-3	1.0	0.8	0.4

calcium, peptides, phosphorus and the milk fat globule membrane, modify blood lipid responses to SFA intake (Thorning et al., 2017). As a result, these FA formerly considered hypercholesterolemic (12:0, 14:0 and 16:0) would have no impact on cardiovascular health parameters when supplied in the dairy matrix. On the other hand, it is also well stablished that the amount of carbohydrates in the diet regulates the synthesis and metabolism of saturated fats in humans (Ruiz-Núñez, Dijck-Brouwer, & Muskiet, 2016). A high intake of carbohydrates would promote its initial utilization for energy generation, leading to SFA storage and promoting *de novo* lipogenesis, while a low-carbohydrate diet would cause oxidation of dietary SFA and reduce its storage.

At last, milk fat is a natural and almost exclusive source of certain bioactive FA with potential benefits on human health. Some of them are not found in our diets in significant amounts elsewhere and the consumption of low-fat or fat-free dairy products would limit their intake. For instance, dairy fat is almost the only source of butyric acid (4:0), conjugated linoleic acid (CLA) as well as branched-chain FA in human diet. Although these FA constitute only a minor percentage in dairy fat, small amounts may still be biologically relevant, alone or within the context of the dairy matrix (Kratz et al., 2013). The present review aims to summarize the most recent knowledge of the bioactive properties of milk FA and their potential effect on consumer health.

2. Short and medium-chain saturated fatty acids

SFA of total C number from 2 to 6 are usually classified as short-chain saturated FA (SCSFA), whereas those from 7 to 12 C atoms are defined as medium-chain saturated FA (MCSFA). SCSFA and MCSFA are easily digestible and they show a low tendency to be stored in the adipose tissue. These FA are preferentially hydrolysed from the TAG molecules and transferred directly from the intestine to the bloodstream. Afterwards, they are transported as FFA to the liver where they are rapidly metabolized via mitochondrial β-oxidation without TAG resynthesis, indicating that those SFA are a quick energy source for active cells. SCSFA and MCSFA also contribute to the regulation of cell metabolism and play an important role in intracellular signalling (Schönfeld & Wojtczak, 2016).

Milk fat is the main source of SCSFA in the human diet since most food products, including ruminant meat, present SFA of longer chain length. SCFA can exert antimicrobial activities and affect the pathogenesis of a diverse range of diseases (Tan et al., 2014). They are esterified almost entirely at the *sn*-3 TAG position, which would have some implications in the human digestion process. When dairy fat is consumed, our lingual and gastric lipases preferentially hydrolyse the FA at the *sn-3* position, and therefore there is a selective release of SCSFA in the gastrointestinal tract.

As average, a 4% of the FA esterified to cow milk TAG corresponds to butyric acid (4:0) (Table 1) which is the most important SCSFA in dairy foods and exert multiple functions in the organism (Fig. 1). 4:0 is the primary energy source for intestinal epithelial cells and it has an essential role in the maintenance of colonic homeostasis and health (Hamer et al., 2008). Butyric acid has a prominent mission in preserving the physiological functions of the colonic mucosa, a barrier consisting of mainly mucins, which is considered the first line of defence against pathogens and harmful substances. Mucin secretion positively influences adhesion of probiotics such as bifidobacteria and lactobacilli, while inhibits the incorporation of pathogenic bacteria. In this line, *in vitro* studies using human specific cell lines have shown that the presence of butyrate stimulates the production of mucins through upregulation of their gene expression, which would be beneficial for the microbiota adherence to the mucus layer (Hatayama, Iwashita, Kuwajima, & Abe, 2007; Jung, Park, Jeon, & Han, 2015).

Low concentration of 4:0 can inhibit growth in a wide range of human cancer cell lines, mainly colon (Hamer et al., 2008). Firstly it was believed that central to the anti-cancer action of 4:0 was its ability to inhibit histone deacetylases, which results in histone hyperacetylation and destabilization of chromatin structure that facilitates activation of genes associated with cell growth (Davie, 2003). Subsequent studies have reported that butyrate could also exert other effects in colon cells in different stages of cancer development (Fung, Cosgrove, Lockett, Head, & Topping, 2012). On primary chemoprevention, butyrate reduces inflammatory processes by modulating genes involved in oxidative and metabolic stress in human colon cells (Scharlau et al., 2009). Regarding secondary chemoprevention, 4:0 has shown anticancer activity through the induction of apoptosis (Zhang et al., 2010)

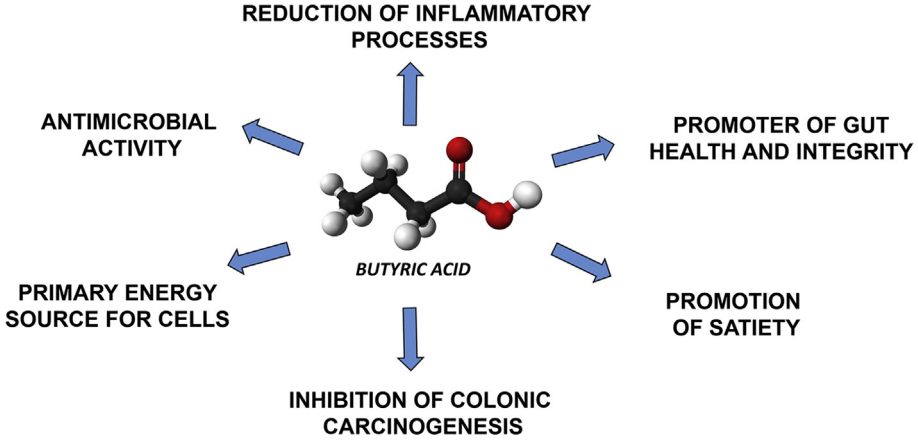

Fig. 1. The role of butyric acid in health and disease.