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The Connection Between Dairy Consumption and Inflammatory Stress: A Comprehensive Review

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Abstract. Emerging studies challenge the popular view that dairy products unanimously promote inflammation. This review synthesizes current research on how variations in fermentation status and fat content influence the inflammatory potential of dairy. Through enrichment with probiotics and postbiotics, fermented dairy products demonstrate dual regulatory effects: modulating gut microbiota composition while suppressing pro-inflammatory signaling pathways. Conversely, non-fermented and high-fat dairy products exhibit heterogeneous inflammatory responses—particularly among individuals with metabolic dysfunction—where outcomes range from neutral to markedly pro-inflammatory. The inconsistencies arise from variations in bacteria strains, fat composition, processing methods, and host metabolic status. This review underscores the need for more standardized interventions and broader participant diversity in the future to clarify dairy's role in inflammation.

Keywords: Dairy products, Fermented dairy, Gut microbiota, Metabolic syndrome

1. Introduction

With the increasing interest in dietary strategies to improve health outcomes, the role of dairy in modulating inflammation has gained increasing attention. Traditionally valued for their calcium and vitamin D content, dairy products are now under scrutiny for their influence on inflammatory pathways. Inflammation occurs when the body initiates a physiological immune response, releasing pro-inflammatory biomarkers such as TNF-α, IL-6, and MCP-1 [1]. While some observational studies suggest that dairy exacerbates inflammation, emerging clinical trials and mechanistic research indicate that specific types of dairy may exert anti-inflammatory effects, particularly in individuals with metabolic disorders such as type 2 diabetes and obesity [2]. Yet not all dairy products reduce inflammation equally. The anti-inflammatory potential of dairy depends mainly on processing method (fermented vs. non-fermented) and fat content (full-fat vs. low-fat): Fermented dairy contains beneficial microbes and bioactive compounds that regulate the gut microbiota through pathogen suppression and production of short-chain fatty acids and postbiotics, which actively inhibit pro-inflammatory pathways like NF-κB and TLR4 signaling [3,4]. Non-fermented dairy lacks these bioactive components, resulting in its limited anti-inflammatory effect [5,6]. In addition, full fat dairy (e.g., whole milk, cheese) contains anti-inflammatory bioactive lipids like conjugated linoleic acid (CLA] and omega-3 fatty acids, which may offset the inflammation caused

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by saturated fat [7,8]. Thus, RCTs often present neutral or even reduced inflammation in healthy subjects [2]. By contrast, low-fat dairy such as skim milk and low-fat yogurt is often rich in vitamin D, suppressing TNF-α and reducing inflammation [9]. "Low-fat dairy products are often effective for metabolically vulnerable individuals due to their low caloric density and reduced saturated fat content [10]. Hence, the primary aim of this review is to critically synthesize current evidence on how fermentation and fat content modulate inflammatory effects of dairy, with a focus on their metabolic health implications.

2. Fermented dairy

Fermented dairy products, such as yogurt and kefir, are produced through microbial fermentation, enriching them with probiotics and postbiotics. These components modulate the gut microbiota, enhance intestinal barrier function, and suppress pro-inflammatory pathways, suggesting stronger anti-inflammatory effects than non-fermented dairy products. Several studies support these benefits and the following subsections examine specific fermented dairy products—yogurt, kefir, and cheese—to assess their respective anti-inflammatory properties.

2.1. Yogurt

Yogurt is one of the most commonly seen and consumed dairy products in our everyday life. This fermented dairy product is produced by bacterial fermentation of milk, often using Lactobacillus delbrueckii subsp. bulgaricus and Streptococcus thermophilus as starter cultures [11]. The anti-inflammatory effects of yogurt depend largely on the bacterial strains used as starter cultures. In a parallel RCT, Pei et al. observed a 19-25% decrease in TNF-α ($p<0.05$) in both obese and non-obese women after consuming 200 g/day of probiotic yogurt (Lactobacillus bulgaricus, Streptococcus thermophilus) for 12 weeks, though the study lacked a placebo group and dietary control [12]. While the findings suggest anti-inflammatory effects of probiotic yogurt, the mechanism in the study is limited by its focus on peripheral blood mononuclear cells (PBMCs) and negation of gut short-chain fatty acid (SCFA) analysis. The authors hypothesized that suppression of TLR4/NF-κB signaling, evidenced by elevated NFKBIA expression, might mediate the observed TNF-α reduction. However, the lack of changes in other NF-κB-related genes (e.g., PTGS2, TNF) implies additional pathways may be involved. One possibility is SCFA-mediated HDAC inhibition: fermentation by probiotics leads to the production of SFCAs (like butyrate), which act as histone deacetylase (HDAC) inhibitors. Functionally, as an HDAC inhibitor, it impedes chromatin remodeling required for NF-κB transcriptional activation, 'silencing' TNF-α gene expression without directly affecting NF-κB-related genes [13]. Additionally, probiotic-induced gut barrier enhancement reduces lipopolysaccharides (LPS) translocation, lowering the activation of TLR4 receptors and diminishing TNF-α production [14]. However, the results are not uniformly positive. Some studies suggest that strain and host context critically affect yogurt's efficacy. For instance, Hilimire et al. reported a 10% increase in TNF-α levels in 35 healthy adults after six weeks of probiotic yogurt consumption containing L. rhamnosus [15]. This abnormal finding underscores the importance of strain-specific immunomodulation, as L. rhamnosus may inadvertently promote TNF-α production in immunocompetent hosts by triggering TLR2/6 receptors on antigen-presenting cells [16]. Yari et al. conducted a 15-week RCT in 44 obese adults consuming 300 g/day of conventional yogurt (strain unspecified), reporting no significant changes in IL-6 or TNF-α [17]. Obesity-related factors such as leptin resistance, gut dysbiosis, and increased intestinal permeability all account for this null result. In addition, leptin (an adipokine elevated in obesity) stimulates both JAK2/STAT3 signaling in

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immune cells and NF-κB in macrophages, directly upregulating pro-inflammatory cytokines (IL-6/TNF-α). In obese individuals, these mechanisms may override the anti-inflammatory effects of probiotic yogurt, thereby amplifying IL-6 and TNF-α expression [18]. Likewise, Mazidi et al. conducted a similar trial involving 60 obese adults who consumed fermented milk (L. casei) for 8 weeks, reporting no significant changes in CRP or IL-6 [19]. The lack of dietary control likely undermined the intervention's efficacy, as high-glycemic food intake among obese participants may have impaired probiotic function. Specifically, a high-glycemic diet tends to induce postprandial hyperglycemia, increasing reactive oxygen species (ROS) production and activating the NF-κB-mediated inflammatory cascade, thereby counteracting the anti-inflammatory effects of L. casei [20]. Finally, meta-analyses by Marco et al. and Żółkiewicz et al. have confirmed that strains like L. bulgaricus and S. thermophilus can reduce TNF-α and CRP, although these effects remain strain- and population-specific[4 [21]. The inclusion of obese participants in both studies may also have contributed to the diminished efficacy of fermented dairy, as metabolic dysfunction is associated with heightened baseline inflammation and reduced probiotic responsiveness [19]. Thus, the anti-inflammatory effects of fermented dairy appear less consistent in individuals with metabolic dysfunction. Several limitations in study design may account for these inconsistencies. Many trials lacked dietary control, with obese participants often consuming pro-inflammatory foods such as processed carbohydrates. Additionally, the absence of placebo groups and the relatively short intervention periods (i.e., 12 weeks) may have limited the studies' ability to detect changes in chronic inflammation. These divergent findings highlight the significance of fat content, bacterial strains, and host metabolic status in determining the anti-inflammatory potential of fermented dairy. Taken together, these studies demonstrate both positive and null effects of yogurt on inflammatory markers, heavily impacted by strain specificity and host metabolic conditions.

2.2. Kefir

Kefir is a fermented milk product that has a complicated microbial content, including lactic acid bacteria (e.g., Lactobacillus kefiri), yeasts (Saccharomyces spp.), and acetic acid bacteria. Unlike yogurt its impact on IL-6 and TNF-α is more variable, yet studies have demonstrated its consistent CRP lowering effect. In a study by Bourrie et al. a 0.5 mg/L reduction in CRP ($p=0.03$) among 28 adults with metabolic syndrome following 600 mL/day of kefir for 15 weeks [22]. Although the specific strains were not identified and IL-6 levels remained unchanged, kefir's microbial components (particularly yeasts and lactic acid bacteria) may indirectly reduce CRP by stimulating butyrate production and enhancing intestinal regulatory T cell activity [20]. In contrast, Ebringer et al. found no changes in CRP nor IL-6 in 60 healthy adults after consuming pasteurized kefir for 8 weeks. This null effect likely resulted from pasteurization destroying live probiotics and postbiotics, thereby compromising kefir's functional properties. These findings underscore that viable microbes are critical for kefir's anti-inflammatory activity. Ebringer's study suggests that live microbes are essential for the pro-inflammatory efficacy of keifir. All in all, while kefir demonstrates consistent CRP-lowering effects its impact on IL-6 and TNF-α is rather inconsistent. These discrepancies likely stem from differences in preparation (e.g., pasteurization), strain content, and host health status. Similarly, in a study by O'Brien et al. 67 healthy adults after consuming 454 g of kefir twice weekly in combination with endurance training exhibited a significant reduction in CRP levels ($P < 0.05$) [21]. In contrast, the control group, which consumed isocaloric carbohydrate beverages, did not show such benefits, indicating the unique anti-inflammatory properties of kefir are independent of calorie intake or exercise.

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2.3. Cheese (high-fat fermented dairy)

Cheese as a prevalently used ingredient among diets is produced by the coagulation of milk proteins through either enzymatic or acidic methods. Fermented cheese is a more concentrated form of a dairy product that is rich in saturated fats with palmitic acid (C16:0) being the predominant SFA (30–40% of total fats) [23]. Unlike yogurt and kefir, fermented cheeses, particularly those high in saturated fat, present a more variable inflammatory profile. Nestel et al. studied 50 overweight adults consuming 40 g/day of high-fat fermented cheese for 12 weeks, observing no change in CRP levels and a 15% increase in IL-6 [24]. This pro-inflammatory response likely stems from palmitic acid—abundant in high-fat dairy—activating TLR4 on macrophages and adipocytes, thereby triggering the IKK/NF-κB cascade and upregulating IL-6 transcription [25,26]. Unlike yogurt or kefir—rich in anti-inflammatory probiotics such as L. bulgaricus and B. lactis—high-fat cheeses may counteract microbial benefits via palmitic acid-mediated TLR4/NF-κB activation, particularly in metabolically compromised individuals [24]. Thus, palmitic acid predominates in high-fat dairy, overriding any potential anti-inflammatory effects. Meta-analyses by Labonté et al. further supported this association between high fat dairy and elevated levels of CRP and IL-6. While cheeses may contain probiotics, their benefits are often outweighed by their fatty acid composition, especially exhibited in metabolically compromised individuals with pre-existing inflammation [6]. This highlights the importance of differentiating low-fat vs. high-fat fermented dairy when evaluating anti-inflammatory potential.

3. Non-fermented dairy

Non-fermented dairy refers to dairy products that have not undergone microbial fermentation, including milk, cream, butter and ice cream [11]. These dairy products are staples of Western diets but lack the probiotics and postbiotics (e.g., SCFAs) found in fermented dairy (yogurt, kefir). Unlike their fermented counterparts, non-fermented dairy products generally demonstrate limited anti-inflammatory effects [27]. Lacking probiotics and postbiotics, they neither promote short-chain fatty acids (SCFAs) production—which directly inhibits NF-κB nuclear translocation through HDAC blockade—nor inhibit inflammatory signaling pathways [13]. Moreover, the high saturated fat content—notably palmitic acid–may activate TLR4, exacerbating inflammation [24,25]. Other components, such as lactose and A1 casein, in non-fermented dairy can irritate the gut in metabolically compromised or lactose-intolerant individuals, leading to dysbiosis and further inflammatory responses [28].

3.1. Milk (low-fat non-fermented dairy)

Studies suggest that low-fat, non-fermented milk generally exhibits neutral or mildly anti-inflammatory effects in healthy adults, but its impact may differ significantly in metabolically compromised populations. In a 6-week randomized controlled trial, Ulven et al. reported no significant changes in CRP, IL-6, or TNF-α levels ($p > 0.05$) among 46 healthy adults consuming 500 mL/day of 1.5% fat milk compared to a water control [29]. Similarly, a meta-analysis by Bordoni et al. of 18 RCTs ($n = 2,100$) found no significant effect of low-fat milk (≤2% fat) on inflammatory markers in healthy individuals [2]. A modest CRP reduction (−0.2 mg/L) was only observed when low-fat milk replaced high-fat dairy, suggesting limited benefit. These null findings are mainly attributed to the biochemical properties of low-fat, non-fermented milk. Unlike fermented dairy, it lacks live probiotics and postbiotics that produce SCFAs such as butyrate that

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inhibit inflammation. Moreover, its low saturated fat content offers limited lipid signaling, failing to activate either the TLR4/NF-κB pro-inflammatory axis or the PPAR-γ anti-inflammatory pathway. Without microbial modulation, low-fat milk cannot fortify the gut barrier or restrain LPS translocation—key mechanisms by which fermented dairy exerts anti-inflammatory effects. Furthermore, healthy adults typically exhibit low baseline inflammation (e.g., CRP <1 mg/L, IL-6 <2 pg/mL), leaving little room for improvement. In contrast, obese individuals with elevated baseline inflammation appear more susceptible to the adverse effects of non-fermented dairy. In a 12-week RCT by Kratz et al., 60 obese adults with insulin resistance who consumed three servings per day of 1% low-fat milk showed significant increases in IL-6 (+15%, p=0.03) and TNF-α (+12%, p=0.04) [30]. The elevated lipopolysaccharide (LPS) levels, gut barrier dysfunction, and leptin resistance in obese individuals may have resulted in the enhanced TLR4/NF-κB signaling. Without probiotics/SCFAs to counteract this—and with leptin resistance amplifying responses—systematic inflammation is further exacerbated in obese individuals. Collectively, the evidence indicates that low-fat, non-fermented milk remains neutral in healthy adults but may shift toward a pro-inflammatory profile in metabolically compromised individuals, where LPS-driven TLR4 activation is unchecked by gut-protective microbes.

3.2. Butter+cream (high-fat non-fermented dairy)

Butter (≥80% fat) and cream (30–40% fat) are concentrated, non-fermented dairy products derived from milk fat separation. With the rising popularity of ketogenic and high-fat diets, consumption of these products has increased notably. Similar to milk, both butter and cream lack microbial content, leaving them devoid of probiotics or postbiotics (e.g., SCFAs) that modulate gut health [31]. Although both milk and butter/ cream lack the gut-microbiota interactions of fermented dairy, butter and cream are substantially higher in saturated fats—particularly palmitic acid—leaving their pro-inflammatory potential debated, especially in metabolically dysfunctional populations. In healthy individuals, however, high-fat, non-fermented dairy appears to exert neutral effects on systemic inflammation.

Similarly, Björnsson et al. studied 2,100 elderly adults and found that cream consumption of ~1–2 tbsp/day (14–28 g) also did not significantly alter CRP levels (p = 0.31). As healthy individuals, the three protective factors mentioned before ensure homeostasis after modest cream intake, regardless of the low-grade inflammation susceptible in aging individuals. In contrast, obese and metabolically dysregulated individuals demonstrate greater inflammatory sensitivity to high-fat, non-fermented dairy. In a 6-week RCT, Nestel et al. found that 45 overweight adults (BMI 27–35) consuming 40 g/day of butter (80% fat) exhibited significant increases in IL-6 (+18%, p = 0.02) and CRP (+0.9 mg/L, p = 0.01) [24]. In contrast, obese individuals frequently display gut-barrier dysfunction and LPS translocation, amplifying both TLR4/NF-κB and leptin-JAK2/STAT3 signaling pathways. Thus, the consumption of butter, high in palmitic acid, would be prone to activating TLR4 signaling on macrophages and adipocytes, driving NF-κB-mediated transcription of IL-6 and CRP. Due to chronic inflammation, hyperinsulinemia, and immune priming, diabetic individuals, like obese individuals, also show amplified NF-κB and JAK/STAT pathways when exposed to high saturated fat intake from butter.

Collectively, evidence suggests that low-fat fermented dairy—particularly yogurt and kefir—modestly lowers CRP in metabolically healthy individuals, while effects on IL-6 and TNF-α are population- and strain-dependent. Anti-inflammatory efficacy further differs by age, sex, and body weight: women derive the greatest benefit, whereas obese adults show attenuated responses. In contrast, cheese, a high-fat fermented products, triggers inflammation due to its SFA content. On the

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other hand, non-fermented dairy, generally lacks beneficial probiotics and postbiotics while retaining pro-inflammatory components (saturated fat and casein content), further exacerbating gut permeability and systemic inflammation in sensitive populations. Future trials should address methodological gaps—brief interventions, absent placebo arms, insufficient sex- and metabolic-status stratification, and inadequate dietary monitoring—to delineate the nuanced interplay among dairy type, fermentation, and inflammation across populations.

4. Discussion

Despite the growing body of evidence, the majority of studies researching the anti-inflammatory effect of fermented dairy are heavily dependent on biomarker measurements. These biomarkers, however, could be affected by various factors, including the metabolic efficiency of participants, dietary habits, and body weight. While reductions in CRP are consistently observed across studies, changes in cytokine levels (TNF-α/IL-6) appear more variable, often requiring highly specific probiotic strains and careful participant selection. A key limitation is the prevailing practice of treating dairy as a monolithic category. Future work must rigorously distinguish subtypes by fermentation status and fat content to enhance both accuracy and translational relevance. This distinction is especially important when considering dairy's role in inflammation among individuals with metabolic syndrome. To advance the field, future RCTs should implement standardized definitions and formulations of dairy products, expand the diversity of their subjects, and stratify analysis by metabolic health status. Researchers should also place greater emphasis on evaluating the role of postbiotic, instead of focusing solely on probiotics. In addition, the inflammatory effect of dairy may depend not only on its fermentation status, but also on how it interacts with the broader dairy context. For example, fat proportion can influence probiotic viability and efficacy, while accompanying nutrients or dietary patterns may also mitigate the negative effects of saturated fats. Hence, dietary guidelines must embrace this complexity, explicitly incorporating fermentation status and fat content to tailor recommendations for heterogeneous populations.

5. Conclusion

In conclusion, the dairy impact on inflammation is largely dependent on processing method and fat content. Fermented dairy, enriched in probiotics and postbiotics, consistently demonstrates reductions in inflammatory markers (CRP, TNF-α) by modulating gut microbiota and inhibiting pro-inflammatory signaling pathways. In contrast, non-fermented dairy—lacking these bioactive components—commonly exhibits neutral or pro-inflammatory effects, particularly in high-fat forms. The inflammatory profile of full-fat dairy is relatively nuanced: while it contains anti-inflammatory lipids like CLA, its saturated fat content may counteract these benefits, especially in metabolically vulnerable populations. Low-fat fermented dairy appears to offer the most consistent anti-inflammatory benefits, particularly for individuals with obesity, diabetes, or other metabolic dysfunctions. To refine our understanding and application of these findings, future research should prioritize strain-specific interventions (e.g., L. acidophilus vs. L. lactis), investigate diverse populations including elderly and pediatric groups, and integrate with multi-omics data approaches such as gut metagenomics to predict individual responses. Ultimately, not all dairy is created equal —fermentation and fat content dictate its inflammatory effects. Tailoring dietary recommendations based on these distinctions holds promise for advancing metabolic health and reducing chronic inflammation.

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