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Published Version

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Vimaleswaran, K. S. ORCID: <https://orcid.org/0000-0002-8485-8930> and Mohan, V. (2026) Dairy intake and risk of type 2 diabetes and metabolic syndrome: a narrative review. *Nutrition*, 144. 113064. ISSN 1873-1244 doi: 10.1016/j.nut.2025.113064 Available at <https://centaur.reading.ac.uk/128282/>

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To link to this article DOI: <http://dx.doi.org/10.1016/j.nut.2025.113064>

Publisher: Elsevier

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Review

Dairy intake and risk of type 2 diabetes and metabolic syndrome: A narrative review

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ARTICLE INFO

Article History:

Received 4 September 2025

Received in revised form 19 November 2025

Accepted 8 December 2025

Keywords:

Dairy intake
Milk consumption
Type 2 diabetes
Metabolic syndrome
RCTs
Omics
Gene expression

ABSTRACT

Dairy products are important components of human health. While some studies claim that dairy increases the risk of type 2 diabetes (T2D) and metabolic syndrome (MetS), several large studies have shown the beneficial impact of dairy consumption. The objective of this review is to highlight the effect of dairy intake on T2D and MetS using recent evidence (published within the last decade) from large epidemiological studies, meta-analyses, randomized controlled trials (RCTs), and Mendelian randomization (MR) and multi-omics studies, and to provide plausible underlying biological mechanisms linking dairy consumption with the risk of T2D and MetS. Given the increasing prevalence of T2D and MetS, it is important to understand the benefits and/or risks of milk and dairy products in the diet. Based on all available evidence from large-scale epidemiological studies, MR analyses, and RCTs, the beneficial impact of dairy products as part of a healthy diet plan appears to be an additional way of mitigating the risk of T2D and MetS. The evidence for a protective effect appears to be undisputed for fermented dairy products like yogurt. For milk, most studies were either protective or neutral, with very few showing a deleterious effect, and with respect to cheese and butter, there were studies showing a deleterious effect, but the grade of evidence was weak. Further mechanistic studies combined with large prospective studies and RCTs in ethnically diverse populations, taking into account sufficient dose and duration, are warranted to get a more complete understanding of dairy consumption and T2D risk.

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Introduction

The prevalence of type 2 diabetes (T2D) and metabolic syndrome (MetS) has reached an epidemic level [1,2]. In 2024, 589 million adults globally had diabetes, and by 2050 this is projected to increase to 853 million (12.69%) of the global population. In India alone there are currently 101 million people with diabetes [3]. The International Diabetes Federation Obesity Task Force has also stated that 1.7 billion people globally are at risk of weight-related chronic diseases such as T2D [3]. A recent systematic review of 29 195 individuals reported a global prevalence of 30.3% for MetS [4]. T2D and MetS have a multifactorial origin where genetic factors may determine the risk to some extent, but they cannot explain the dramatic increase in prevalence over the

last several decades, which can be attributed to lifestyle factors such as sedentary lifestyle, physical inactivity, and unhealthy dietary patterns.

Even though dairy intake is featured in many dietary guidelines, the role of dairy products in the risk of T2D and related traits remains highly debated. Dairy products are nutrient-rich foods that consist of vitamins, minerals, proteins, and phospholipids, but it is feared by some that the saturated fat in dairy could have adverse effects [5]. However, several studies have demonstrated a wide range of beneficial properties of milk [6–12]. In fact, milk is a primary source of nutrition for newborns and an excellent source of nutrients for the growth of children. While dairy is an essential part of the diet in several parts of the world such as Nordic countries, dairy consumption is significantly lower in low- and middle-income countries [13]. Consumption of dairy products such as milk, cheese, butter, and yogurt lags behind Western countries in Asian countries where T2D is highly prevalent [14–16].

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While several studies have shown the beneficial effect of dairy consumption on reducing the risk of MetS and components of cardiometabolic risk [17–21], some studies claim that dairy increases the risk of T2D and MetS. Hence, there is increasing skepticism among the public about the health consequences of consuming dairy products, which is largely driven by vegan groups via social media. Given the increasing prevalence of T2D and MetS [1,14,22,23], it is important to understand the implications of consuming milk and dairy products. There are several review papers that have focused on the effect of dairy products on metabolic diseases [24–26]; thus, this review will highlight the effect of dairy consumption on T2D and MetS using recent evidence (within the last 10 y) from large epidemiological studies, meta-analyses and systematic reviews, randomized controlled trials, Mendelian randomization (MR) studies, gene expression and multi-omics studies, and underlying biological mechanisms.

Approach

Literature searches were conducted using PubMed and Google Scholar databases for studies published between 2015 and 2025. The search terms included dairy intake, milk, cheese, fermented, unfermented, low fat, full fat, omics, Mendelian randomization, genetic variants, single nucleotide polymorphism (SNP), metabolomics, epigenetics, microbiome, type 2 diabetes, metabolic syndrome, CVD, obesity, lipids, ethnicity, body mass index (BMI), prospective studies, randomized controlled trials (RCTs), systematic reviews, meta-analysis, intervention studies, gene expression, proteomics, transcriptomics, genomics, and mechanisms. Articles retrieved were evaluated on their relevance to the topic of this review, with studies on non-humans and those published in non-English languages excluded. Additional relevant studies were included where identified from the reference lists of primary papers, review articles, and meta-analyses.

Evidence from large epidemiological studies

Several large epidemiological studies have examined the effect of dairy consumption on the risk of cardiometabolic disease. While the majority of large-scale studies have shown an inverse association between dairy consumption and risk of T2D, a few studies failed to demonstrate the protective effect of dairy intake. In a prospective study using data from 21 countries (in up to 147 812 participants), higher intake of total dairy (at least 2 servings/d compared with no intake) was associated with a lower incidence of hypertension, T2D, and MetS [18]. This finding was further supported by another study from the same cohort (n = 136 384), where it was shown that dairy consumption (<2 servings/d compared with no intake) was associated with a lower risk of mortality and major cardiovascular disease events [19]. In 15 512 individuals from the Chinese Health and Nutrition Survey, it was shown that consuming 30–80 g/d of dairy was associated with a decreased risk of T2D among the ≤ 2000 kcal/d energy intake group [27]. However, a recent study in 7521 participants from the prospective UK Fenland cohort [28] showed that intake of high-fat dairy products was associated with higher prediabetes risk and that low-fat dairy consumption was associated with lower risk, yet when investigating dietary changes over time, increases in high-fat milk were associated with reduced risk of progressing from normoglycemia to T2D. A similar finding was observed in the Lifelines Cohort study (n = 112 086), where full-fat dairy and non-fermented dairy products were positively associated with newly diagnosed T2D, and skimmed dairy and fermented dairy were inversely associated with prediabetes [29]. These findings show that fat content and

aspects of fermentation should be taken into consideration when consuming dairy products. Further large, prospective studies are required to confirm the beneficial role of dairy products in mitigating the risk of T2D and MetS.

Evidence from systematic reviews and meta-analysis

Most large-scale systematic reviews and meta-analyses have demonstrated the protective effect of dairy consumption on T2D and MetS [30–34]. In a meta-analysis of 22 cohort studies (n = 579 832), dairy intake (≥ 3 servings/d) was associated with a 2–15% decreased risk of T2D [32]. A recent meta-analysis comprising 6653 individuals with prediabetes from 6 studies demonstrated a quadratic inverse association between total dairy intake and prediabetes risk, with the lowest risk at 3.4 servings/d [33]. Another meta-analysis of data from 21 studies (a total of 560 869 participants and 36 281 cases) showed that total dairy and yogurt intake was associated with a 3% and 7% lower risk of diabetes per 200 g/d and 50 g/d intake increase, respectively, in a dose–response analysis [31]. A similar inverse association with hypertension was shown for low-fat dairy in the same study. A large-scale meta-analysis of 29 prospective cohort studies with data on 938 465 participants and 28 419 patients with coronary heart disease found an inverse association between total fermented dairy products and cardiovascular disease risk, where an increase in consumption of 20 g/d was associated with a 2% reduction in risk [30]. The same study also reported that total yogurt consumption was associated with a 26% decreased risk of MetS. Although the mechanisms showing the beneficial effect of yogurt intake are unknown, some studies have demonstrated that yogurt leads to an increase in the levels of glucagon-like peptide 1 (GLP-1), which has an anorexiogenic effect [35] and could contribute to the protective effects of yogurt intake on T2D and related traits. Key milk components that stimulate L cells in the small intestine to secrete GLP-1 include proteins and peptides, amino acids, and calcium [36]. A recent umbrella review comprising 41 meta-analyses [34] showed that higher milk intake was inversely associated with T2D risk, obesity, and MetS, and a 200 g/d increment of milk was related to a 13% lower risk of MetS and a 16% lower risk of obesity. In addition, two large meta-analyses showed that dairy consumption was inversely associated with the risk of T2D [26] and MetS [25], respectively.

In contrast, a meta-analysis of data from three large cohorts (34 224 men in the Health Professionals Follow-Up Study, 76 531 women in the Nurses' Health Study, and 81 597 women in the Nurses' Health Study II) found that increasing cheese consumption by >0.5 serving/d was associated with a 9% higher risk of T2D, and it also showed that substituting this with 1 serving/d of yogurt or reduced-fat milk was associated with a 16% or 12% lower risk of T2D, respectively. While a cross-sectional study in 38 735 participants of the Henan Rural Cohort study demonstrated that high dairy intake was positively associated with T2D, in the same study (a meta-analysis of 23 studies that included multiple ethnicities) a marginal protective effect of dairy against T2D was shown [37]. These findings raise the possibility that these differences might depend on the country, ethnicity, dietary pattern, and—more importantly—the type of dairy consumed.

Evidence from interventional studies

Within the past decade, only a few RCTs have examined the effect of dairy intake on metabolic disease-related outcomes. A recent meta-analysis of data from 19 RCTs (≥ 12 wk intervention) with 1427 participants showed that high dairy intake, irrespective of fat content, had no adverse effects on cardiometabolic health-

related outcomes [38]. However, while dairy intake improved systolic blood pressure, waist circumference, triglycerides, and high-density lipoprotein cholesterol, it impaired glycemic control (fasting glucose and glycated hemoglobin levels), suggesting that there is little robust evidence that a higher dairy intake has adverse effects on cardiometabolic health outcomes. In accordance with the aforementioned meta-analysis, a RCT in 111 participants with T2D also showed that, irrespective of fat content, increasing dairy consumption to ≥ 3 servings/d compared with <3 servings/d within the same caloric intake had no effect on glycated hemoglobin, body weight, body composition, lipid profile, or blood pressure [39]. In contrast, two previously published meta-analyses demonstrated a beneficial effect of dairy intake. The first meta-analysis of 14 RCTs including 883 adults [40] found that increased dairy consumption was associated with a 0.72 kg greater reduction in fat mass, 0.58 kg gain in lean mass, and 2.19 cm reduction in waist circumference compared with controls. The second meta-analysis of 29 RCTs with 2101 participants [41] also showed a significant beneficial effect of dairy intervention on body fat in energy-restricted diets or short-term trials. Another meta-analysis of data from 30 RCTs (n = 413) reported that low-fat dairy intake was inversely associated with the homeostasis model assessment of insulin resistance (HOMA-IR), waist circumference, and body weight [42]. These discrepancies could be attributed to intervention duration and type, participant age group, sample size, ethnicity, genetic heterogeneity, and type of dairy studied.

A recent continuous glucose monitoring study from our team investigated the effects of dairy and non-dairy products on the glycemic and metabolite profiles of 30 healthy adults following lacto-vegetarian and vegan diets using a parallel randomized feeding trial [43]. Our continuous glucose monitoring data showed that individuals who consumed a vegan diet had significantly higher levels of mean glucose concentrations and post-prandial phenylalanine, and lower levels of acetyl carnitine compared with those who consumed a lacto-vegetarian diet, suggesting that increased phenylalanine levels in the vegan group may be explained by a hypothetical mechanism in which higher glucose induces oxidative stress [44], whereas the increased acetyl carnitine from dairy in the lacto-vegetarian group may protect against oxidative stress, contributing to lower glucose concentrations [45]. Even though the sample size was modest, this is one of the first studies to compare the effects of dairy consumption using a metabolomics study. Given that the majority of the RCTs focused on total dairy consumption, future RCTs should focus on comparing the effects of specific dairy products. In addition, further mechanistic studies combined with large, prospective studies and large RCTs in adequate populations of sufficient dose and duration are warranted to obtain a complete understanding of dairy consumption and T2D risk.

Evidence from MR studies

Given that observational studies are prone to reverse causation and residual confounding, large MR studies, which are analogous to RCTs, are required to investigate the causality between dairy consumption and the components of MetS. A two-sample MR analysis in up to 1 904 220 individuals demonstrated a causal association between genetically instrumented milk intake and lower serum cholesterol levels, and a lower risk of coronary artery disease [46]. In the same study, genetically instrumented higher milk intake was associated with decreased risk of T2D and lower HbA1c concentration in the UK Biobank cohort (n = 404 648). This finding was supported by a recent two-sample MR analysis using DIAGRAM (DIabetes genetics replication and meta-analysis) (n = 74

124) and UK Biobank (n = 339 197), where a decreased risk of T2D per every 50 g daily increment in genetically predicted milk consumption after adjusting for BMI was reported [47]. Furthermore, a two-sample MR analysis in 123 579 individuals showed an inverse causal association between genetically instrumented cheese intake and gestational hypertension and diabetes, respectively [48]. Another MR analysis of 182 041 participants from 18 studies reported that genetically higher dairy intake was associated with increased lean mass after correction for multiple testing [49], which reflects the role of dairy protein in promoting the maintenance of lean mass. In addition, data from a 10 y longitudinal study of the Cardiovascular Disease Risk Factor Two-township Study (n = 1644) and Taiwan Biobank (n = 10 000) showed that genetically instrumented milk intake was associated with lower odds of being obese [50].

In contrast, a recent systematic review of six MR studies in European populations [51] failed to demonstrate a significant association of genetically predicted milk consumption with the risk of T2D and glycated hemoglobin levels. This lack of association could be attributed to differences between the included studies, where one study was a two-sample MR. These findings are also supportive of the MR study in 97 811 Danish individuals, which showed that high milk consumption was not associated with a low risk of T2D or overweight-obesity, both observationally and genetically [52]. Another MR study in 21 820 European individuals from the EPIC-InterAct study also failed to provide evidence for an association of milk intake with diabetes [53]. However, an MR study in 420 090 East Asians showed that both whole milk and skim milk were significantly associated with an increased risk of developing T2D [54]. These findings indicate that the association of dairy intake with the risk of T2D and related metabolic traits could be ethnic-specific and depend on various factors such as sample size and participant characteristics; for example, those with or without obesity. Future MR studies assessing the association of dairy with T2D should consider these aspects.

Evidence from gene expression and multi-omics studies

The majority of the omics-related studies showed an inverse association between dairy consumption and risk of T2D. A recent study in 10 obese patients with hyperinsulinemia who consumed a high-dairy diet (4 servings/d) for 6 wk showed a differential expression of 236 genes that were involved in the signaling pathways (such as G-protein-coupled receptors) related to insulin signaling and glucose homeostasis, which reduced the risk of T2D [55]. A previous study has demonstrated that G-protein-coupled receptor signaling activates Ras signaling, which in turn activates the PI3K/AKT signaling that has been shown to play a role in glucose homeostasis [56]. While there is insufficient evidence to prove an "anti-inflammatory" effect of dairy foods, the majority of clinical research studies have demonstrated that dairy foods do not increase concentrations of biomarkers of chronic systemic inflammation. In accordance with this, a randomized crossover study conducted in 14 healthy young men that compared the effects of probiotic yogurt and non-fermented, acidified milk intake on gene expression showed that probiotic yogurt (800 g as a single dosage) and acidified milk (800 g as a single dose) reduced the expression of genes associated with inflammation and altered insulin signaling [57]. Another study in 10 hyperinsulinemic adults [58] showed that a high-dairy diet for 6 wk modified the abundance of butyrate-producing bacteria, which correlated negatively with the HOMA-IR. This could be explained by the role of butyrate-producing bacteria, which reduce intestinal pH and increase calcium

absorption, and has been shown to decrease insulin resistance and improve insulin sensitivity [59].

Using multi-omics data from the Hispanic Community Health Study/Study of Latinos (up to 3972 participants) [60], it was shown that higher milk intake was associated with lower risk of T2D in lactase non-persistent individuals. In this study [60], milk-associated *Bifidobacterium* species were found to be correlated with lower levels of adiposity measures and fasting glucose levels, respectively. In addition, milk-associated metabolites such as β -cryptoxanthin and indolepropionate were associated with lower risk of T2D, and higher levels of metabolites, which are negatively associated with milk consumption (such as glycocholate, branched chain amino acids, and γ -glutamyl valine), were also associated with increased risk of T2D [60]. In the French MONA LISA cross-sectional, multicenter, population-based study ($n = 298$), the authors showed that consumption of low-fat dairy products could reduce the risk of MetS and reported an inverse association between γ -glutamyl amino acids and low-fat dairy intake [61]. Furthermore, a metabolic profiling analysis in 659 adults from the tri-ethnic multicenter Insulin Resistance Atherosclerosis Study [62] reported that a dairy fatty acid biomarker, pentadecanoic acid, was associated with a 27% decreased incident T2D risk and positively associated with insulin sensitivity and β -cell function. The Guangzhou Nutrition and Health Study [63] comprising 1780 participants identified an inverse association between dairy-related gut microbiota and serum triglyceride levels, where 1 serving/d higher in total dairy and milk intake was associated with 0.12 and 0.19 SD lower triglyceride levels, respectively, among individuals with higher α -diversity. These studies suggest that circulating metabolite biomarkers and gut microbiota could serve as potential therapeutic targets for cardiometabolic disease.

In contrast, a recent population-based metabolomics study in 893 Swedish individuals [64] demonstrated that intake of non-fermented milk and cheese was associated with an increased risk of T2D and different subclasses of sphingomyelins, which is in accordance with previous studies [65–69] suggesting that sphingomyelins could be considered as candidate biomarkers of dairy intake. As shown by several studies, there were no metabolites that were associated with all types of dairy intake, which indicates that

different types of dairy intake might have different metabolic responses. Hence, future studies should investigate the associations between types of dairy products and the risk of T2D.

Evidence for plausible mechanisms demonstrating the beneficial effect of dairy intake

The mechanism involved in the association between dairy intake and T2D can partly be explained by the effect of dairy intake on body weight. Studies have shown that calcium from dairy products lowers insulin resistance [70] and improves insulin sensitivity by influencing β -cell function, inflammation, and insulin signaling pathways [71–73]. Furthermore, the whey protein in milk and yogurt has been shown to reduce postprandial glucose response in diabetic subjects, which could be due to the branched chain amino acids in the whey protein enabling a greater stimulation of glucose-dependent insulinotropic polypeptide [74], which is an important factor in lowering blood glucose concentrations.

Medium-chain fatty acids, trans-palmitoleic acids, and micronutrients such as vitamin D and magnesium from dairy products have been shown to be associated with lower insulin resistance [70,72,73] and a reduced risk of T2D [70]. In addition, given that insulin-like growth factor-1 has been shown to enhance insulin sensitivity and regulate glucose homeostasis [75], reduction in the risk of T2D could also be due to an increase in insulin-like growth factor-1 induced by dairy products [76]. The beneficial impact of fermented dairy products such as cheese and yogurt with regard to T2D could be due to their effect on the gut microbiome [77]. Dairy products fortified with probiotic bacteria have been shown to improve glycemic control by influencing the composition and function of the gut microbiome [78]. Similarly, vitamin D fortification in yogurt has been shown to improve glycemic status [79]. In addition, leucine in dairy has been shown to promote mitochondrial production and has beneficial effects on glucose homeostasis [80]. Figure 1 summarizes the possible mechanisms to explain the beneficial effects of dairy on T2D and MetS.

Plant-derived and milk-derived fermented products have been shown to prevent and manage diabetes as they improve glycemic

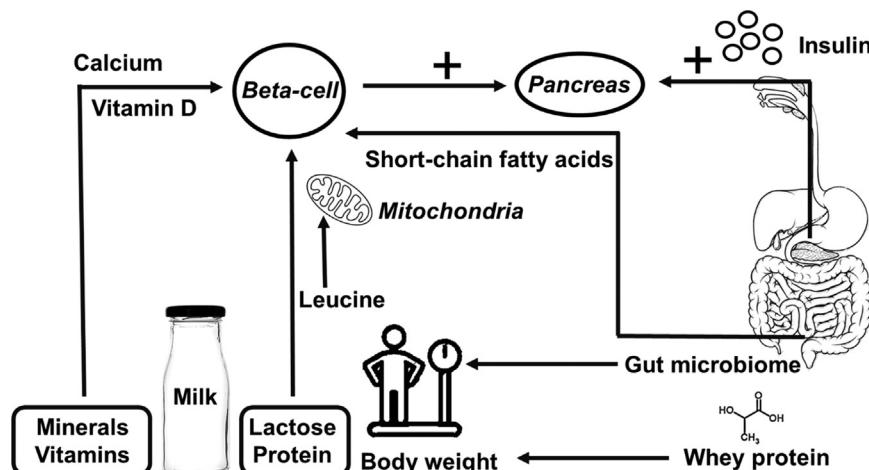


Fig. 1. Plausible mechanisms by which dairy products can have a beneficial effect in preventing type 2 diabetes and related traits. Studies have shown that calcium and vitamin D from dairy products lower insulin resistance. Leucine in dairy has been shown to promote mitochondrial production and has beneficial effects on glucose homeostasis. Short-chain fatty acids play a significant role in β -cell function and glucose homeostasis, largely through their influence on the gut microbiome and subsequent signaling pathways. Milk proteins such as whey protein and casein, which serve as abundant reservoirs of essential amino acids and bioactive components, have also been shown to have functional roles in human health. Whey protein can also produce fewer very low-density lipoproteins by inhibiting lipogenic enzymes in the adipocytes, improving energy intake balance and promoting weight loss.

control by modulating the gut microbiome, reducing inflammation, and producing beneficial bioactive compounds [81,82]. While milk-based fermented products such as yogurt and kefir have consistent long-term data supporting their preventive effects from a large body of observational studies, cohort studies, and meta-analyses [83], the research is less extensive for plant-based fermented foods where evidence primarily comes from animal studies [84].

Studies have shown that dairy foods are a heterogeneous group of products that represent complex matrices of nutrients, minerals, bioactives, food structures, and other factors, with complex effects on health and disease [85]. Different structural levels can be found within a dairy matrix, and these variations in structure and interactions define the matrices that influence the extent and kinetics of nutrient digestion and absorption, appetite regulation, and disease risk [86]. Several components within dairy matrices may play a crucial role in eliciting beneficial biological responses. For instance, the dairy matrix involves a complex system where casein proteins bind calcium into micelles to form ion clusters, which increase calcium bioavailability [87] and thereby reduce the risk of cardiometabolic disease [88]. Furthermore, the fermentation process of yogurt and cheese yields unique bioactives, such as short-chain fatty acids produced by bacteria, which have been shown to contribute to improved insulin sensitivity and reduced blood pressure [89,90].

The mechanism of the antihypertensive effect of dairy could be explained by the role of casein hydrolysates in lowering blood pressure by blocking the activity of angiotensin-converting enzyme [91], which plays a crucial role in regulating blood pressure. Other components in dairy products such as calcium and magnesium have also been shown to reduce hypertension [92]. Dairy intake has also been shown to have a favorable effect on blood lipids, which could be due to the health benefits of conjugated linoleic acid [93]. In addition, dairy products are a source of protein, potassium, calcium, magnesium, and phosphorus, and hence these could have a role in improving cardiometabolic health [94]. Besides reducing the risk of T2D, whey protein can also produce fewer very low-density lipoproteins by inhibiting lipogenic enzymes in adipocytes [95], and improving energy intake balance and promoting weight loss (Figure 1) [96]. Milk contains medium-chain fatty acids that have been found to induce angiopoietin-like 4 gene expression, which is known to suppress lipoprotein lipase and control lipid metabolism [97].

Conclusion

While a few studies have failed to demonstrate the beneficial effect of dairy products, the majority of large-scale studies have reported an inverse association between dairy intake and the risk of T2D. Dairy products play a significant role in nutrition not only for children, but also for all age groups. Dairy products have been proposed to have a negative effect on cardiometabolic health due to the high amount of saturated fatty acids. However, this relationship has been ruled out by fermented dairy products, which have been shown to promote a beneficial gut microbiome, increase the production of short-chain fatty acids, and regulate hepatic cholesterol synthesis [98]. Furthermore, milk proteins such as whey protein and caseins, which serve as abundant reservoirs of essential amino acids and bioactive components, have also been shown to have functional roles in human health [99].

In summary, despite a few studies that have failed to show the beneficial effects of dairy products, the majority of the evidence from large-scale epidemiological studies, systematic reviews, meta-analyses, MR studies, RCTs, and multi-omics studies has demonstrated the beneficial effects of dairy intake on T2D and

MetS. However, given the wide range of mixed findings with uncertain conclusions, future studies should explore additional confounding factors such as ethnicity, geographical location, dietary patterns, food choice, food intake behavior, and lifestyle factors to ascertain the potential effects that may have had a role in leading to these disparate conclusions. Hence, readers should maintain caution when assessing new studies and consider the issues pertaining to heterogeneity, generalizability, and statistical significance when making inferences. Based on all of the available evidence from large-scale epidemiological studies, MR analyses, and RCTs, the beneficial impact of dairy products as part of a healthy diet plan appears to be an additional way of mitigating the risk of T2D and MetS. The evidence for a protective effect appear to be undisputed for fermented dairy products, in particular plain (unsweetened or unflavored) yogurt. For milk, most studies are either protective or neutral, with very few showing a deleterious effect; and with respect to cheese and butter, there are studies showing a deleterious effect, but the grade of evidence is weak. Further large systematic reviews and meta-analyses demonstrating dose-response relationships and linear/non-linear associations are required to provide further evidence for developing guidelines for dairy consumption. To implement more targeted preventive strategies and interventions, large RCTs in ethnically diverse populations using different types of dairy to validate potential underlying mechanisms are warranted so as to make global policy recommendations. These recommendations will also have ethnic variations as well as focus on the amounts of dairy to be consumed.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Karani Santhanakrishnan Vimaleswaran: Writing – review & editing, Writing – original draft, Resources, Methodology, Investigation, Data curation. **Viswanathan Mohan:** Writing – review & editing, Supervision, Resources, Methodology, Investigation, Conceptualization.

Funding

None.

Author contributions

V.K.S. and M.V. have contributed equally to the manuscript.

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