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Published in:
Nutrition, Metabolism and Cardiovascular Diseases

DOI:
10.1016/j.numecd.2023.04.013

Publication date:
2023

Document version:
Final published version

Nutrition, Metabolism & Cardiovascular Diseases (2023) **33**, 1316–1322

Available online at www.sciencedirect.com

Nutrition, Metabolism & Cardiovascular Diseases

journal homepage: www.elsevier.com/locate/nmcd

SYSTEMATIC REVIEWS AND META-ANALYSES

Milk consumption and the risk of type 2 diabetes: A systematic review of Mendelian randomization studies

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Received 13 July 2022; received in revised form 14 April 2023; accepted 18 April 2023
Handling Editor: A. Siani
Available online 28 April 2023

KEYWORDS
Milk;
Dairy;
Type 2 diabetes;
Nutrition;
Mendelian randomization;
Systematic review

Abstract *Aims:* Previously, no relationship between milk consumption and the risk of type 2 diabetes has been found in prospective cohorts. However, Mendelian randomization allows researchers to almost bypass much residual confounding, providing a more precise effect estimate.

This systematic review aims to investigate the risk of type 2 diabetes and levels of HbA1c by assessing all Mendelian Randomization studies investigating this subject matter.

Data synthesis: PubMed and EMBASE were searched from October 2021 through February 2023. Inclusion and exclusion criteria were formulated to filter out irrelevant studies. Studies were qualitatively assessed with STROBE-MR together with a list of five MR criteria. Six studies were identified, containing several thousand participants. All studies used the SNP rs4988235 as the main exposure and type 2 diabetes and/or HbA1c as the main outcome. Five studies were graded as "good" with STROBE-MR, with one graded as "fair". For the six MR criteria, five studies were graded "good" in four criteria, while two studies were graded "good" in two criteria. Overall, genetically predicted milk consumption did not seem to be associated with an increased risk of type 2 diabetes.

Conclusions: This systematic review found that genetically predicted milk consumption did not seem to increase the risk of type 2 diabetes. Future Mendelian randomization studies concerning this topic should consider conducting two-sample Mendelian Randomization studies, in order to derive a more valid effect estimate.

1. Introduction

Type 2 diabetes is a non-communicable disease, defined as a glycated hemoglobin value over 7.0 mmol/L or a 2-h blood sugar over 11.1 mmol/L [1]. Globally, type 2 diabetes has been steadily increasing for several years, with a global prevalence of 476 million people with type 2 diabetes in 2017 [2]. Milk consumption plays an important role in many parts of the world, providing key nutrients, such as calcium, protein, and vitamin B12 [3]. However, milk remains a controversial subject in nutritional epidemiology, mostly due to diverging health outcomes [4].

The link between the consumption of milk and the risk of type 2 diabetes has been researched over years, with

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https://doi.org/10.1016/j.numecd.2023.04.013

most prospective cohort studies finding no relationship between milk consumption and type 2 diabetes, although finding an inverse relationship for total dairy consumption, as well as other types of dairy products [5].

However, despite strenuous efforts to adjust for confounding factors, even well-designed prospective cohort studies may still be prone to residual confounding [6], reducing validity. This has compelled researchers to pursue an alternative approach to dietary exposure; Mendelian randomization (MR) [7], dictating that genetic variants are allocated randomly [6,8]. Therefore, exposed study participants are a function of an instrumental variable (IV), yielding a very low risk of confounding [6], making Mendelian randomization studies strongly equipped to infer causation between exposure and outcome [6]. Several single nucleotide polymorphisms (SNPs) correlating with higher consumption of specific foods have been found, one being milk consumption [9]. Lactose, the naturally occurring sugar found in dairy, has to be broken down into glucose and galactose to be absorbed into the bloodstream [7]. For this process, the enzyme lactase is needed, which all humans produce in infancy, and which most individuals stop producing after weaning [7]. However, in some European populations, this enzyme is still being produced into adulthood in most populations, creating the opportunity to investigate this natural randomization [7]. The production of lactase is determined by the gene coding for lactase enzyme, LCT [7] An intron variant rs4988235 in the neighboring MCM6 gene is associated with transcriptional activation of LCT and can influence lactase intolerance. Homozygous bearers of cytosine (CC) become lactose intolerant, whereas heterozygous and homozygous bearers of thymine-cytosine (TC) and thymine−thymine (TT) can digest lactose in adulthood, as lactase is still being produced [7].

Here is great interest for epidemiologists to investigate the magnitude of effects between milk consumption and the risk of type 2 diabetes with MR, as the estimate yields high validity, and may benefit the public through more valid information concerning the risk of type 2 diabetes.

This systematic review aims to review published MR studies that investigate genetically predicted milk consumption and the risk of type 2 diabetes and levels of HbA1c and assess them qualitatively.

2. Methods

This systematic review was conducted in alignment with the current Preferred Reporting for Systematic Reviews and Meta-Analysis Statement 2020 (PRISMA) [10] (Table S1). This systematic review was registered in PROSPERO (CRD42022287050) [11].

One author (C.F.J.) performed the literature search on PubMed and Embase between the 24th of October 2021 to the 14th of February 2023, with the second author (G.B.B.) checking the sample of included articles from the literature search. The details of all search strategies are provided in Supplementary Table 4.

From the 24th of October 2021 to the 14th of February, 2023, one author (C.F.J.) performed the data extraction, with the second author (G.B.B.) checking the data extraction. Both authors performed the qualitative assessments of the included studies. Any disagreement concerning the grading of a study was resolved by discussions between both authors until an agreement was made.

To retrieve potentially relevant studies, a snowball sampling was performed by screening the references of all studies that were retrieved in full text. The following inclusion criteria were determined [1]: MR design [2]; milk consumption as the only exposure [3]; type 2 diabetes as one of the study outcomes [4]; HbA1c as a secondary outcome. To remove irrelevant studies, the exclusion criteria were [1]; study design not being based on an MR design [2]; exposure not exclusively being milk consumption [3]; type 2 diabetes not being a possible outcome. To assess the quality of the included studies, the STROBE checklist for MR studies (STROBE-MR) was used [12] with adaptations from Burgess et al. (2020) [13], as well as a list of six MR criteria in which the fulfillment for each study was assessed [14] (Table S2). The STROBE-MR checklist contains 20 items related to study design, reporting, and analysis [12], with further four items from the MR criteria by Burgess et al. [13]. The grading of included studies follows these criteria; studies that adhered to >15 questions were graded as of "good" quality, studies that adhered to >10 items were graded as of "fair" quality, and studies that adhered to <10 items were graded as of "poor" quality (Table S3). However, studies not adhering to items related to sensitivity analysis (item 8) cannot be graded higher than "fair" (Table S3). Furthermore, a second qualitative assessment of the included studies was conducted, to assess the fulfillment of six MR criteria, which have been described elsewhere [14] with adaptations from Burgess et al. [13]. In brief, studies were rated "good", "moderate" or "poor" regarding the authors handled the following MR checklist for MR analyses; 1) use of instrumental variable analysis; 2) relevance assumption; 3) independence assumption; 4); exclusion restriction assumption 5) test of non-linearity; 6) type of main MR analysis [14] (Table S3).

For the data synthesis, the following characteristics from all studies were extracted and synthesized through a table; author(s), publication year, location (country), number of participants, SNP, type of MR study (one- or two-sample), milk exposure (g/day), allele score, statistical analysis, type of MR analysis, and the outcome variable (odds ratio (OR), beta-coefficient, hazard ratio (HR), risk ratio (RR), with corresponding 95% confidence intervals (95% CI), HbA1c status, and the grading of the STROBE-MR checklist [12].

3. Results

The literature search on PubMed and EMBASE yielded 14 results. After removing nine non-eligible studies and retrieving one article due to snowball sampling, six studies were included in the final synthesis (Fig. 1). The qualitative

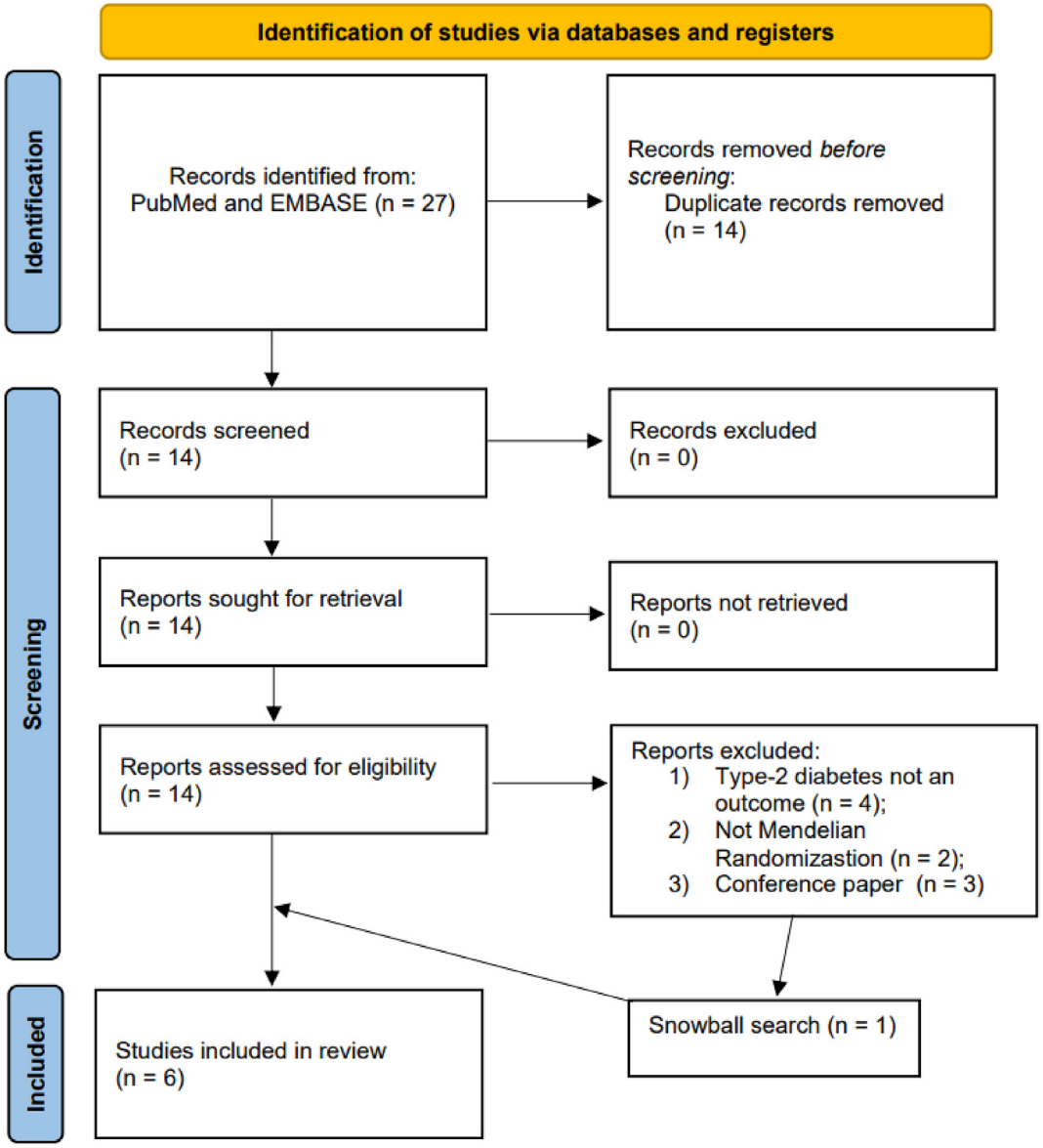

Figure 1 PRISMA flowchart of the literature search.

assessment, using STROBE-MR with adaptations from Burgess et al., revealed that five included studies were graded "good" [9,15–17], whereas one study was graded "fair" (18) (Table 1). The included six studies were investigating genetically predicted milk exposure in participants from 12 different countries; Denmark [9,15], Finland [17,19], France [9], Germany [9], Greece [9], Italy [9], The Netherlands [9], Norway [9], Spain [9], Sweden [9], Australia [17], The United Kingdom [18,19], and the United States [17]. Four out of five studies were one-sample MR studies [9,15,17,18], with two studies being two-sample MR studies [16,19] (see Table 2).

The overall exposure in all studies was the SNP coding for lactase persistence (rs4988235) [9,15–19]. Four studies ascertained this exposure by an increment in genetically predicted milk consumption [9,16,18,19], pairing information on the distribution of lactase alleles with actual dairy consumption in food-frequency questionnaires. Two studies used a combination of both genetically predicted milk consumption and an increment in lactase allele score [15,17]. Four studies chose to adjust for potential confounding factors [9,15,18,19] for the risk of type 2 diabetes. In studies looking at levels of HbA1c, three studies adjusted for potentially confounding factors [9,17,18].

Concerning the risk of type 2 diabetes, four studies did not find statistically significant associations between genetically predicted milk consumption and type 2 diabetes [9,15,18,19], whereas one study found a statistically significant increased risk of developing type 2 diabetes [16]. However, in one of three two-sample MR analyses, using the DIAGRAM and UK Biobank, with adjustment for BMI, Yuan et al. also found a decreased risk of type 2

Table 1 Table of the included studies and their characteristics.

Author	Year	Country	Study population	(*n*)	SNP	STROBE-MR Grading	Type of MR study
Bergholdt et al. [15]	2015	Denmark	General population aged 47−67	97,811	rs4988235	Good	One-sample
Mendelian Randomization of Dairy Consumption Working Group et al. [17]	2019	Denmark, Finland & The United States	Data from 17 cohort studies and one case control study	60,016	rs4988235	Good	One-sample
Vimaleswaran et al. [18]	2021	United Kingdom	British birth cohort from age 45+ Health and retirement study US: age 50+ UK Biobank 40−69	372,195	rs4988235	Fair	One-sample
Vissers et al. [9]	2019	EPIC interact case cohorts[b]	Volunteers aged 30+	21,830	rs4988235	Good	One-sample
Yang et al. [16]	2021	European descent[c]	The following databases were used: GEFOS, CARDIoGRAMplusC4D, DIAGRAM, GIANT, GLGC and MAGIC	149,361	rs4988235	Good	Two-sample
Yuan et al. [19]	2022	Finland and United Kingdom	The following databases were used: FinGenn, UK Biobank, and DIAGRAM	336,252	rs4988235	Good	Two-sample

[a]The sample did not fulfill the criteria for being a one-sample study, as there were no gene-exposure data.
[b]The countries from the European Investigation into Cancer (EPIC) study are: Denmark, France, Germany, Greece, Italy, The Netherlands, Norway, Spain, Sweden, and the United Kingdom [14].
[c]The data source is a collection of gene-exposures and gene-outcomes from participants of European descent, including some of the cohorts used in Bergholdt, Nordestgaard Ellervik [12], Vimaleswaran et al. [14] and Yuan et al. [18].

diabetes per every 50 g daily increment in genetically predicted milk consumption [19]. All four studies investigating HbA1c found that there was not a statistically significant difference in HbA1c levels, stratified by lactase allele type [9,16−18].

4. Discussion

This is the first systematic review to assess published MR studies regarding genetically predicted milk consumption and the risk of type 2 diabetes, containing several hundred thousands of participants, albeit with several overlaps of the included cohorts between the included studies. The main finding is that genetically predicted milk consumption, overall, does not seem to be associated with the risk of type 2 diabetes. These findings are in alignment with the findings of previous prospective cohorts [5]. These results and underlying methodological quality between the published mendelian randomization studies are very constant using the method suggested by van de Luitgaarden et al., 2022 (Table S3). Particular BMI and lipid metabolism seems to be a potential confounder in the association between genetic variant and type 2 diabetes. In addition, it was also found that genetically predicted milk consumption was not associated with levels of HbA1c. These findings are in contrast to the findings of RCTs, conducted on low-fat dairy, finding improvements in biomarkers related to type 2 diabetes, albeit not entirely focused on milk [20]. This may be attributed to calcium, vitamin K2, and flavonoids, which may be beneficial in the prevention of type 2 diabetes [3]. An explanation for the null relationship between milk consumption and the risk of type 2 diabetes could be the high water content of milk, yielding a lower nutrient density, compared to other dairy products. In contrast, other dairy products, such as yogurt, may decrease the risk of type 2 diabetes [21]. Another explanation may be due to the variety of milk consumed in the cohorts, as milk exposure did not factor in different types of milk, causing nutrient intakes, e.g. intake of saturated fat, to vary slightly across cohorts.

One major strength of this systematic review is the Mendelian randomization study design itself. By using genetic exposures, this may carry a higher precision than prospective cohort studies, although depending on the fulfillment of MR assumptions and sample size [8]. Another strength is that all included studies used rs4988235 as the genetic variant, the most consistent SNP correlating with milk consumption [7]. However, several limitations are still present. Although one of the main assumptions of MR is the absence of horizontal pleiotropy, this is limited to current knowledge about the physiological effects of lactase persistence. However, the current knowledge of these effects may be explained by milk consumption through several physiological pathways [18], which then likely qualifies as vertical pleiotropy [6]. Therefore, adjusting for these covariates may, at worst, induce collider bias given the variables are affected by milk consumption and type 2 diabetes or HbA1c levels [6].

Table 2 Outcomes of included studies.

Author	Year	Exposure	Instrument characteristics P value, R-square, F statistics, Power	Statistical analyses	Primary outcome: Risk of type-2 diabetes	Secondary outcome: HbA1c	Statistical adjustment(s)
Bergholdt, Nordestgaard & Ellervik [15]	2015	250 g daily increase in genetically predicted milk consumption & distribution of lactase alleles	$P = 9 \times 10^{-36}$, 2%, NA, NA	Main analysis: Wald estimate Sensitivity analyses: 1) CC vs TC; 2) CC vs. TT; 3) CC vs. TC/TT; 4) Multiplicative generalized method of moments estimator 5) Extreme genotype score instrumental variable analysis; 6) 2-stage least square regression	Main analysis: HR: 0.99 (0.93−1.06) Sensitivity analysis: *1)* OR: 0.97 (0.86−1.09) *2)* OR: 0.98 (0.87−1.10) 3) OR: 0.97 (0.87−1.09) 4) OR: 0.99 (0.93−1.06) 5) OR: 1.00 (0.94−1.07) 6) OR: 1.01 (0.94−1.08)	NA	Main analysis: None. Sensitivity analysis:*1), 2), and 3):* Adjusted for sex, age, population, and height. *4), 5), and 6):* None.
Mendelian Randomization of Dairy Consumption Working Group et al. [17]	2019	One serving increase in genetically predicted milk consumption	$P = 6.8 \times 10^{-6}$, NA, NA.	Wald estimate (no sensitivity analysis)	NA	β: 0.005 ± 0.044	None.
Vimaleswaran et al. [18]	2021	50 g daily increase in genetically predicted milk consumption	$P = 2 \times 10^{-7}$ (For EPIC InterAct, per g/day), NA, NA, NA $P = 7.2 \times 10^{-14}$ (For UK Biobank, consumers vs non consumers), 2.5%,1055, NA P = 0.111 (for 1958 BBC milk drinkers vs non-drinkers, NA, NA, NA, NA	Primary outcome: Wald estimate (no sensitivity analysis) Secondary outcome: Linear regression (no sensitivity analysis)	OR: 0.89 (0.82−0.97 (UK Biobank) OR 1.06 (0.97−1.16) (DIAGRAM))	β: −0.001 ± 0.001	Primary outcome: None. Secondary outcome: Gender, BMI, assessment centre, SNP array and region of residence or principal components, lipid-lowering medication, and anti-diabetic mediation.
Vissers et al. [9]	2019	15 g daily increase in genetically predicted milk consumption	$P = 2 \times 10^{-7}$, NA, F = 74, NA	Primary outcome: Main analysis: Wald estimate Sensitivity analyses: 1) Percentile bootstrap (n = 10,000); 2) Excluding HbA1c >6.5% (48 mmol/mol); 3) Only illumine 660 W quad chip; 4) Only countries in HWE; 5) Lactase persistence model. Secondary outcome: Linear regression	Main analysis: HR: 0.99 (0.93−1.05) Sensitivity analyses: 1) Percentile bootstrap (n = 10,000); HR: 0.99 (0.94−1.04) 2) Excluding HbA1c >6.5% (48 mmol/mol); HR: 0.98 (0.92−1.05) 3) Only Illumina 660 W quad chip; HR: 0.97 (0.90−1.05) 4) Only countries in HWE; HR: 1.04 (0.81−1.33) 5) Lactase persistence model HR: 0.99 (0.92−1.07	Per allele: β: −0.07 (−0.22 to 0.07, 95% CI)	Primary outcome: Sex, genetic variability, study center, and genotyping platform. Secondary outcome: Age, sex, genetic variability, study center, and genotyping platform.

Yang et al. [16]	2017	66 g daily increase in genetically predicted milk consumption*	$P = 2.5 \times 10^{12}$, NA, NA, NA	Primary outcome: Main analysis: Wald estimate. Sensitivity analysis: Per allele increase. Secondary outcome: Main analysis: Wald estimate. Sensitivity analyses: Per allele increase.	Main analysis: β: 0.921 (0.829–1.023, 95% CI) Sensitivity analysis: β: 0.978 (0.952–1.005, 95% CI)	Main analysis: β: 0.015 (−0.015 – 0.045, 95% CI) Sensitivity analysis: β: 0.004 (−0.004 – 0.012, 95% CI)	None
Yuan et al. [19]	2022	50 g daily increase in genetically predicted milk consumption	NA	Main analysis: Wald estimate. Sensitivity analyses: MR-Egger.	Main analysis: OR: 1.03 (0.97–1.09) (FinnGen) OR 0.97 (0.92–1.03) (DIAGRAM)) Sensitivity analysis: OR 0.92 (0.86–0.97) (DIAGRAM))	NA	BMI[c]

[a]Adjusted for dairy intake.
[b]Type-2 diabetic patients.
[c]Adjusted for BMI.

Moreover, as almost all populations in this review were of European descent, it is unknown if the effect of genetically predicted milk consumption on the risk of diabetes or levels of HbA1c remains the same across several ethnic groups. Another limitation is the lack of fulfillment of all MR study criteria by any included study, which may bias the true effect estimate of genetically predicted milk consumption on the risk of type 2 diabetes and HbA1c levels. Especially the fact that only one study was a two-sample MR may lower the validity, due to the risk of bias inherent in the sample, affecting the estimated causal relationship. Inherent limitations in this systematic review should also be acknowledged, as the literature search and data extraction only was done by one author, despite following a predefined PROSPERO protocol [11].

5. Conclusion

The findings of this systematic review may indicate that genetically predicted milk consumption is not associated with the risk of type 2 diabetes or HbA1c levels, aligning with previous observational findings. However, in spite of the strong association between the IV and milk consumption, rs4988235 only explains 2% of the variance in milk consumption. Future studies will benefit from better understanding of heritability of milk consumption and stronger IV based on multiple variables.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.numecd.2023.04.013.

References

[1] World Health Organization, International Diabetes Federation. Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: report of a WHO/IDF consultation [cited 2021 Oct 24]; Available from: https://apps.who.int/iris/handle/10665/43588; 2006.
[2] Cho NH, Shaw JE, Karuranga S, Huang Y, da Rocha Fernandes JD, Ohlrogge AW, et al. IDF Diabetes Atlas: global estimates of diabetes prevalence for 2017 and projections for 2045. Diabetes Res Clin Pract 2018;138:271–81.
[3] Guo J, Givens DI, Astrup A, Bakker SJL, Goossens GH, Kratz M, et al. The impact of dairy products in the development of type 2 diabetes: where does the evidence stand in 2019? Adv Nutr 2019; 10(6):1066–75.
[4] Willett WC, Ludwig DS. Milk and health. Campion EW, editor. N Engl J Med 2020;382(7):644–54.
[5] Gijsbers L, Ding EL, Malik VS, de Goede J, Geleijnse JM, Soedamah-Muthu SS. Consumption of dairy foods and diabetes incidence: a dose-response meta-analysis of observational studies. Am J Clin Nutr 2016;103(4):1111–24.
[6] Davies NM, Holmes MV, Davey Smith G. Reading Mendelian randomisation studies: a guide, glossary, and checklist for clinicians. BMJ 2018:k601.
[7] Sacerdote C, Guarrera S, Smith GD, Grioni S, Krogh V, Masala G, et al. Lactase persistence and bitter taste response: instrumental variables and mendelian randomization in epidemiologic studies of dietary factors and cancer risk. Am J Epidemiol 2007;166(5): 576–81.
[8] Davey Smith G, Holmes MV, Davies NM, Ebrahim S. Mendel's laws, Mendelian randomization and causal inference in observational data: substantive and nomenclatural issues. Eur J Epidemiol 2020; 35(2):99–111.

[9] Vissers LET, Sluijs I, van der Schouw YT, Forouhi NG, Imamura F, Burgess S, et al. Dairy product intake and risk of type 2 diabetes in EPIC-InterAct: a mendelian randomization study. Diabetes Care 2019;42(4):568–75.
[10] Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021:n71.
[11] PROSPERO. PROSPERO [cited 2021 Dec 5]. Available from: https://www.crd.york.ac.uk/prospero/; 2021.
[12] Skrivankova VW, Richmond RC, Woolf BAR, Davies NM, Swanson SA, VanderWeele TJ, et al. Strengthening the reporting of observational studies in epidemiology using mendelian randomisation (STROBE-MR): explanation and elaboration. BMJ 2021:n2233.
[13] Burgess S, Davey Smith G, Davies NM, Dudbridge F, Gill D, Glymour MM, et al. Guidelines for performing Mendelian randomization investigations. Wellcome Open Res 2020;4:186.
[14] van de Luitgaarden IAT, van Oort S, Bouman EJ, Schoonmade LJ, Schrieks IC, Grobbee DE, et al. Alcohol consumption in relation to cardiovascular diseases and mortality: a systematic review of Mendelian randomization studies. Eur J Epidemiol 2022;37(7):655–69.
[15] Bergholdt HK, Nordestgaard BG, Ellervik C. Milk intake is not associated with low risk of diabetes or overweight-obesity: a Mendelian randomization study in 97,811 Danish individuals. Am J Clin Nutr 2015;102(2):487–96.
[16] Yang Q, Lin SL, Au Yeung SL, Kwok MK, Xu L, Leung GM, et al. Genetically predicted milk consumption and bone health, ischemic heart disease and type 2 diabetes: a Mendelian randomization study. Eur J Clin Nutr 2017;71(8):1008–12.
[17] Mendelian Randomization of Dairy Consumption Working Group, Huang T, Sun D, Heianza Y, Bergholdt HKM, Gao M, et al. Dairy intake and body composition and cardiometabolic traits among adults: mendelian randomization analysis of 182041 individuals from 18 studies. Clin Chem 2019;65(6):751–60.
[18] Vimaleswaran KS, Zhou A, Cavadino A, Hyppönen E. Evidence for a causal association between milk intake and cardiometabolic disease outcomes using a two-sample Mendelian Randomization analysis in up to 1,904,220 individuals. Int J Obes 2021;45(8):1751–62.
[19] Yuan S, Sun J, Lu Y, Xu F, Li D, Jiang F, et al. Health effects of milk consumption: phenome-wide Mendelian randomization study. BMC Med 2022;20(1):455.
[20] Sochol KM, Johns TS, Buttar RS, Randhawa L, Sanchez E, Gal M, et al. The effects of dairy intake on insulin resistance: a systematic review and meta-analysis of randomized clinical trials. Nutrients 2019;11(9):2237.
[21] Alvarez-Bueno C, Cavero-Redondo I, Martinez-Vizcaino V, Sotos-Prieto M, Ruiz JR, Gil A. Effects of milk and dairy product consumption on type 2 diabetes: overview of systematic reviews and meta-analyses. Adv Nutr 2019;10(suppl_2):S154–63.