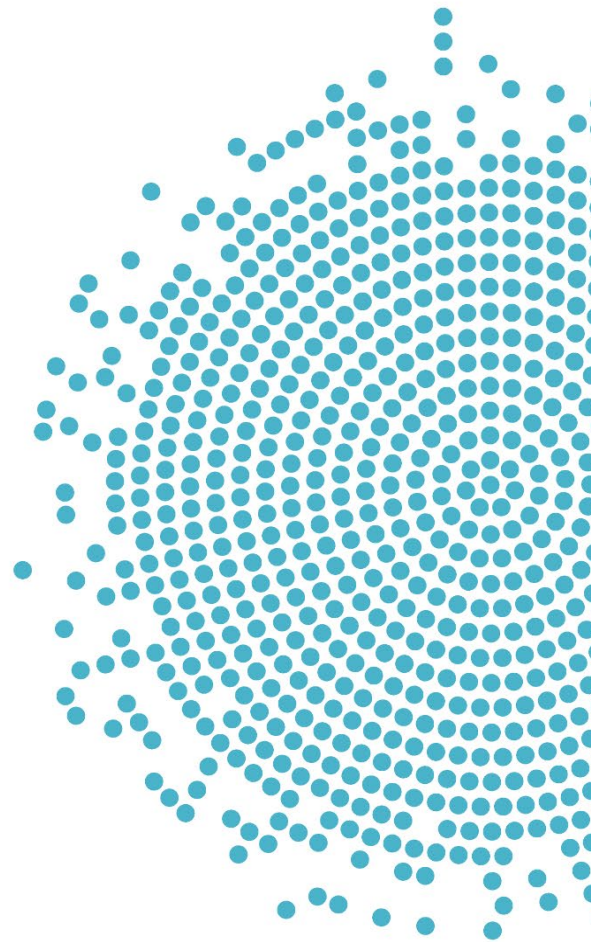


# Sugar-Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review

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# Table of contents

<b>Table of contents</b> .....	<b>3</b>
<b>Plain language summary</b> .....	<b>5</b>
<b>Abstract</b> .....	<b>6</b>
<b>Introduction</b> .....	<b>8</b>
<b>Methods</b> .....	<b>9</b>
Develop a protocol .....	9
Develop an analytic framework .....	10
Develop inclusion and exclusion criteria .....	11
Search for and screen studies .....	13
Extract data and assess the risk of bias.....	14
Synthesize the evidence .....	14
Develop conclusion statements and grade the evidence .....	14
Recommend future research.....	16
Peer review .....	16
Health equity considerations .....	16
<b>Results</b> .....	<b>16</b>
Literature search and screening results .....	16
Infants and young children .....	18
Description of the evidence .....	18
Conclusion statement and grade .....	18
Children and adolescents.....	18
Description of the evidence .....	18
Synthesis of the evidence.....	19
Conclusion statement and grade .....	20
Adults and older adults.....	21
Description of the evidence .....	21
Synthesis of the evidence.....	24
Conclusion statement and grade .....	25
<b>Summary of conclusion statements and grades</b> .....	<b>27</b>
Research recommendations .....	27
<b>Acknowledgments and funding</b> .....	<b>85</b>
<b>References of the articles included in the systematic review</b> .....	<b>86</b>
Appendix 1: Abbreviations .....	89
Appendix 2: Literature search strategy.....	90
Appendix 3: Excluded articles .....	96
Table 1. Review history .....	8
Table 2. Protocol revisions .....	9
Table 3. Inclusion and exclusion criteria.....	11
Table 4. Definitions of NESR grades.....	15
Table 5. Conclusion statement and grade for sugar-sweetened beverage consumption in infants and young children and risk of type 2 diabetes.....	18

Table 6. Conclusion statement and grade for sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes..... 20

Table 7. Conclusion statement and grade for sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes..... 25

Table 8. Evidence examining the relationship between sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes ..... 28

Table 9. Risk of bias for observational studies examining sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes ..... 34

Table 10. Intervention studies examining the relationship between sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes..... 35

Table 11. Observational studies examining the relationship between sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes..... 42

Table 12. Risk of bias for randomized controlled trials examining sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes..... 80

Table 13. Risk of bias for observational studies examining sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes ..... 81

Figure 1. Analytic framework for the systematic review question: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes? ..... 10

Figure 2. Literature search and screen flowchart ..... 17

## Plain language summary

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### **What is the question?**

The question is: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes? The populations of interest for this question include infants and young children up to age 24 months, children and adolescents, and adults and older adults.

### **Why was this question asked?**

This systematic review was conducted by the 2025 Dietary Guidelines Advisory Committee as part of the process to develop the *Dietary Guidelines for Americans, 2025-2030*.

### **How was this question answered?**

The Committee conducted a systematic review to answer this question with support from the USDA Nutrition Evidence Systematic Review team.

### **What is the answer to the question?**

#### Infants and young children up to age 24 months

- A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by infants and young children up to age 24 months and risk of type 2 diabetes because there is no evidence available.

#### Children and adolescents

- A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by children and adolescents and risk of type 2 diabetes because of substantial concerns with directness in the body of evidence.

#### Adults and older adults

- Sugar-sweetened beverage consumption by adults and older adults may be associated with higher risk of type 2 diabetes. This conclusion statement is based on evidence graded as moderate.

### **How up-to-date is this systematic review?**

Conclusion statements from this review are based on articles published between January 2000 and January 2024.

# Abstract

## Background

This systematic review was conducted by the 2025 Dietary Guidelines Advisory Committee as part of the process to develop the *Dietary Guidelines for Americans, 2025-2030*. The U.S. Departments of Health and Human Services (HHS) and Agriculture (USDA) appointed the 2025 Dietary Guidelines Advisory Committee (Committee) in January 2023 to review evidence on high priority scientific questions related to diet and health. Their review forms the basis of their independent, science-based advice and recommendations to HHS and USDA, which is considered as the Departments develop the next edition of the *Dietary Guidelines*. As part of that process, the Committee conducted a systematic review with support from USDA's Nutrition Evidence Systematic Review (NESR) team to answer the following question: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?

## Methods

The Committee conducted a systematic review using the methodology of the USDA NESR team. The Committee first developed a protocol. The intervention/exposure was sugar-sweetened beverage consumption in infants and young children up to age 24 months, children, adolescents, adults, and older adults, the comparators were consumption of a different amount of sugar-sweetened beverages (including no consumption and versions diluted with water), water, or low- and no-calorie sweetened beverages, and the outcomes were fasting blood glucose, fasting insulin, glucose tolerance/insulin resistance, hemoglobin A1C, prediabetes, and risk of type 2 diabetes in infants and young children, children, adolescents, adults, and older adults. Additional inclusion criteria were established for the following study characteristics: a) use randomized or non-randomized controlled trial, prospective or retrospective cohort, nested case-control, or Mendelian randomization study designs, b) be published in English in peer-reviewed journals, c) be from countries classified as high or very high on the Human Development Index, and d) enroll participants with a range of health statuses. The review excluded intervention studies less than 12-week duration for hemoglobin A1C, prediabetes, and type 2 diabetes, and intervention studies less than 4-week study duration for fasting blood glucose, fasting insulin, and glucose tolerance/insulin resistance.

NESR librarians conducted a literature search in PubMed, Embase, CINAHL, and Cochrane to identify articles published between January 2000 and January 2024. Two NESR analysts independently screened all electronic results, and the reference lists of included articles based on the pre-determined criteria.

NESR analysts extracted data, from each included article, with a second analyst verifying accuracy of the extraction. Two NESR analysts independently conducted a formal risk of bias assessment, by study design, for each included article, then reconciled any differences in the assessment. The Committee qualitatively synthesized the evidence, according to the synthesis plan, with attention given to the overarching themes or key concepts from the findings, similarities and differences between studies, and factors that may have affected the results. The Committee developed conclusion statements and graded the strength of evidence based on its consistency, precision, risk of bias, directness and generalizability.

## Results

### Infants and young children up to age 24 months

**Conclusion statement and grade:** A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by infants and young children up to age 24 months and risk of type 2 diabetes because there is no evidence available. (Grade: Grade Not Assignable)

#### *Summary of the evidence:*

- No articles met the inclusion criteria for this review in infants and young children up to age 24 months.
- The Committee was not able to draw a conclusion because no evidence was available.

### Children and adolescents

**Conclusion statement and grade:** A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by children and adolescents and risk of type 2 diabetes because of substantial concerns with directness in the body of evidence. (Grade: Grade Not Assignable)

#### *Summary of the evidence:*

- Five articles from prospective cohort study designs met the inclusion criteria for this review in children and adolescents.
- The Committee was not able to draw a conclusion due to substantial concerns with directness, particularly with no studies examining the primary outcome of interest, and some concerns with risk of bias for potential confounding.

### Adults and older adults

**Conclusion statement and grade:** Sugar-sweetened beverage consumption by adults and older adults may be associated with higher risk of type 2 diabetes. This conclusion statement is based on evidence graded as moderate. (Grade: Moderate)

*Summary of the evidence:*

- Forty-three articles met the inclusion criteria for this review in adults and older adults. Six were randomized controlled trials, 1 was a nested case control study, and 36 were prospective cohort studies.
- The direction of results and size of effects were similar across studies.
- The size of study groups was large across studies. Most studies examined enough cases of type 2 diabetes. Variation around the effect estimates were narrow across studies.
- Some studies were designed and conducted well.
- The populations, interventions/exposures, comparators, and outcomes that were examined directly represent those of interest in this review.
- The evidence applies to the U.S. population but may not apply to diverse subgroups based on socioeconomic position and race and/or ethnicity.

## Introduction

To prepare for the development of the *Dietary Guidelines for Americans, 2025-2030*, the U.S. Departments of Health and Human Services (HHS) (Appendix 1) and Agriculture (USDA) identified a proposed list of scientific questions based on relevance, importance, potential federal impact, and avoiding duplication, which were posted for public comment.\* The Departments appointed the 2025 Dietary Guidelines Advisory Committee (Committee) in January 2023 to review evidence on the scientific questions. The Committee's review of the evidence forms the basis of the Scientific Report of the 2025 Dietary Guidelines Advisory Committee,† which includes independent, science-based advice and recommendations to HHS and USDA and is considered during the development of the next edition of the *Dietary Guidelines*.

The proposed scientific questions were refined and prioritized by the Committee for consideration in their review of the evidence. As part of that process, the following systematic review question was prioritized: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes? The Committee conducted a systematic review to address this question, with support from USDA's Nutrition Evidence Systematic Review (NESR) team (**Table 1**).

**Table 1. Review history**

Date	Description	Citation
May 2023	Systematic review protocol for the 2025 Dietary Guidelines Advisory Committee published online	Hoelscher DM, Anderson CAM, Booth SL, Deierlein AL, Fung TT, Gardner CD, Giovannucci E, Raynor HA, Stanford FC, Talegawkar SA, Taylor CA, Tobias DK, Obbagy J, Cole NC, Kingshipp B, Webster A, Higgins M, Butera G, Terry N. Sugar-Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review Protocol. May 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <a href="https://nesr.usda.gov/protocols">https://nesr.usda.gov/protocols</a>
February 2024	Revisions to the systematic review protocol for the 2025 Dietary Guidelines Advisory Committee published online	Hoelscher DM, Anderson CAM, Booth SL, Deierlein AL, Fung TT, Gardner CD, Giovannucci E, Raynor HA, Stanford FC, Talegawkar SA, Taylor CA, Tobias DK, Obbagy J, Cole NC, Kingshipp B, Webster A, Higgins M, Butera G, Terry N. Sugar-Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review Protocol. May 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <a href="https://nesr.usda.gov/protocols">https://nesr.usda.gov/protocols</a>

\* Dietary Guidelines for Americans: Learn About the Process. 2022. Available at: <https://www.dietaryguidelines.gov/work-under-way/learn-about-process>

† 2025 Dietary Guidelines Advisory Committee. 2024. Scientific Report of the 2025 Dietary Guidelines Advisory Committee: Advisory Report to the Secretary of Health and Human Services and Secretary of Agriculture. U.S. Department of Health and Human Services. <https://doi.org/10.52570/DGAC2025>



## Methods

The Committee used NESR’s methodology to conduct this systematic review. NESR’s methodology is described in detail in its methodology manual,\* as well as in the Committee’s Scientific Report.† This section presents an overview of the specific methods used to answer the systematic review question: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?

### Develop a protocol

A systematic review protocol is the plan for how NESR’s methodology will be used to conduct a specific systematic review and is established by the Committee, *a priori*, before any evidence is reviewed. The protocol is designed to capture the most appropriate and relevant body of evidence to answer the systematic review question. Development of the protocol involves discussion of the strengths and limitations of various methodological approaches relevant to the question, which then inform subsequent steps of the systematic review process. The protocol describes all of the methods that will be used throughout the systematic review process. Additionally, the protocol includes the following components, which are tailored to each systematic review question: the analytic framework, the inclusion and exclusion criteria, and the synthesis plan.

The protocol was posted online (<https://nesr.usda.gov/protocols>) for the public to view and comment on. Revisions to the systematic review protocol were made during the review process. These amendments are documented in **Table 2**.

**Table 2. Protocol revisions**

Date	Protocol revision	Description
January 2024	The inclusion and exclusion criteria for study design were revised to exclude intermediate outcomes (fasting blood glucose, fasting insulin, glucose tolerance/insulin resistance, hemoglobin A1C, and prediabetes) from observational studies in adults.	This change was made to focus on a stronger body of evidence and was made before any evidence was synthesized. Trial data in adults examining intermediate outcomes will still be included, as will both trial and observational intermediate outcome data in children.
January 2024	Inclusion and exclusion criteria for publication date were updated to document that the review will include studies published through January 2024.	This revision was made to document the final publication date range covered by the literature search.

\* USDA Nutrition Evidence Systematic Review Branch. USDA Nutrition Evidence Systematic Review: Methodology Manual. February 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <https://nesr.usda.gov/methodology-overview>

† 2025 Dietary Guidelines Advisory Committee. 2024. Scientific Report of the 2025 Dietary Guidelines Advisory Committee: Advisory Report to the Secretary of Health and Human Services and Secretary of Agriculture. U.S. Department of Health and Human Services. <https://doi.org/10.52570/DGAC2025>

## Develop an analytic framework

An analytic framework visually represents the overall scope of the systematic review question and depicts the contributing elements that were examined and evaluated. It presents the core elements of each systematic review question, including the **P**opulation (i.e., those who experience the intervention/exposure and/or outcome), **I**ntervention and/or exposure (i.e., the independent variable of interest), **C**omparator (i.e., the alternative being compared to the intervention or exposure), and **O**utcome(s). The Committee identified key confounders based on their knowledge of nutrition and health research and experience as subject matter experts. Key confounders are participant characteristics, such as demographics, health status, and diet and lifestyle behaviors, and/or other factors related to both the intervention/exposure and the outcome of interest that may impact the relationships of interest. Key confounders were considered during review and evaluation of the evidence, particularly during the risk of bias assessment of non-randomized and observational studies.

**Figure 1** is the analytic framework for the systematic review. The intervention or exposure of interest is sugar-sweetened beverage (SSB) consumption in infants and young children (birth up to age 24 months), children and adolescents (2 up to 19 years), and adults and older adults (19 years and older). The comparators are consumption of a different amount of SSB (including no consumption and versions diluted with water), water, or low- and no-calorie sweetened beverages. The outcomes include fasting blood glucose, fasting insulin, glucose tolerance/insulin resistance, hemoglobin A1C, and prediabetes (from all included study designs in infants, children, and adolescents up to 19 years; and from interventions only in adults and older adults), and risk of type 2 diabetes (from all study designs and age groups). The key confounders are sex, age, race and/or ethnicity, socioeconomic position, anthropometry, physical activity, and family history of diabetes in all populations, and smoking and alcohol intake in adults and older adults.

**Figure 1. Analytic framework for the systematic review question: What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?**

<i>Population</i>	<i>Intervention/ exposure</i>	<i>Comparator</i>	<i>Outcome</i>	<i>Key confounders</i>
Infants and young children (birth up to 24 months)	Sugar-sweetened beverage (SSB) consumption	Consumption of a different amount of SSB (including no consumption and versions diluted with water)  SSB vs. water  SSB vs. low- and no-calorie sweetened beverages	All included study designs in children (up to 19 years) and interventions only in adults (19 years and older):  <ul style="list-style-type: none"> <li>Fasting blood glucose</li> <li>Fasting insulin</li> <li>Glucose tolerance/insulin resistance</li> <li>Hemoglobin A1C</li> <li>Prediabetes</li> </ul>	<ul style="list-style-type: none"> <li>Sex</li> <li>Age</li> <li>Race and/or ethnicity</li> <li>Socioeconomic position</li> <li>Anthropometry</li> <li>Physical activity</li> <li>Family history of diabetes</li> <li>Smoking (adults, older adults)</li> <li>Alcohol intake (adults, older adults)</li> </ul>
Children and adolescents (2 up to 19 years)				
Adults and older adults (19 years and older)				
			All included study designs in all included age groups:  <ul style="list-style-type: none"> <li>Type 2 diabetes</li> </ul>	

**Synthesis organization:**

- I. **Population:** Infants and young children up to age 24 months; Children and adolescents; Adults; Older adults
  - a. **Outcome:** Fasting blood glucose; Fasting insulin; Glucose tolerance/insulin resistance; Hemoglobin A1C; Prediabetes; Type 2 diabetes

## Develop inclusion and exclusion criteria

The inclusion and exclusion criteria provide an objective, consistent, and transparent framework for determining which articles to include in the systematic review (**Table 3**). These criteria ensure that the most relevant and appropriate body of evidence is identified for the systematic review question, and that the evidence reviewed is:<sup>\*</sup>

- Applicable to the U.S. population of interest
- Relevant to Federal public health nutrition policies and programs
- Rigorous from a scientific perspective

**Table 3. Inclusion and exclusion criteria**

Category	Inclusion Criteria	Exclusion Criteria
Study design	<ul style="list-style-type: none"> <li>• Randomized controlled trials</li> <li>• Non-randomized controlled trials<sup>†</sup></li> <li>• Prospective cohort studies</li> <li>• Retrospective cohort studies</li> <li>• Nested case-control studies</li> <li>• Mendelian randomization studies</li> </ul>	<ul style="list-style-type: none"> <li>• Uncontrolled trials<sup>‡</sup></li> <li>• Case-control studies</li> <li>• Cross-sectional studies</li> <li>• Ecological studies</li> <li>• Narrative reviews</li> <li>• Systematic reviews</li> <li>• Meta-analyses</li> <li>• Modeling and simulation studies</li> </ul>
Publication date	<ul style="list-style-type: none"> <li>• January 2000 – January 2024</li> </ul>	<ul style="list-style-type: none"> <li>• Before January 2000, after January 2024</li> </ul>
Population: Study participants	<ul style="list-style-type: none"> <li>• Human</li> </ul>	<ul style="list-style-type: none"> <li>• Non-human</li> </ul>
Population: Life stage	<ul style="list-style-type: none"> <li>• At intervention or exposure and outcome:               <ul style="list-style-type: none"> <li>○ Infants and young children (up to 24 months)</li> <li>○ Children and adolescents (2 up to 19 years)</li> <li>○ Adults and older adults (19 years and older)</li> </ul> </li> <li>• At intervention or exposure:               <ul style="list-style-type: none"> <li>○ Individuals during pregnancy</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• At intervention or exposure:               <ul style="list-style-type: none"> <li>○ N/A</li> </ul> </li> <li>• At outcome:               <ul style="list-style-type: none"> <li>○ Individuals during pregnancy</li> </ul> </li> </ul>

<sup>\*</sup>USDA Nutrition Evidence Systematic Review Branch. USDA Nutrition Evidence Systematic Review: Methodology Manual. February 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <https://nesr.usda.gov/methodology-overview>

<sup>†</sup> Including quasi-experimental and controlled before-and-after studies

<sup>‡</sup> Including uncontrolled before-and-after studies

Category	Inclusion Criteria	Exclusion Criteria
Population: Health status	<ul style="list-style-type: none"> <li>• Studies that <u>exclusively</u> enroll participants not diagnosed with a disease*</li> <li>• Studies that enroll <u>some</u> participants:                             <ul style="list-style-type: none"> <li>○ diagnosed with a disease;</li> <li>○ with severe undernutrition, failure to thrive/underweight, stunting, or wasting;</li> <li>○ born preterm,<sup>†</sup> with low birth weight,<sup>‡</sup> and/or small for gestational age;</li> <li>○ with the outcome of interest;</li> <li>○ receiving pharmacotherapy to treat obesity;</li> <li>○ pre- or post-bariatric surgery;</li> <li>○ and/or hospitalized for an illness, injury, or surgery</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• Studies that <u>exclusively</u> enroll participants:                             <ul style="list-style-type: none"> <li>○ diagnosed with a disease;<sup>§</sup></li> <li>○ with severe undernutrition, failure to thrive/underweight, stunting, or wasting;</li> <li>○ born preterm,<sup>†</sup> with low birth weight,<sup>‡</sup> and/or small for gestational age;</li> <li>○ with the outcome of interest (i.e., studies that aim to treat participants who have already been diagnosed with the outcome of interest);</li> <li>○ receiving pharmacotherapy to treat obesity;</li> <li>○ pre- or post-bariatric surgery;</li> <li>○ and/or hospitalized for an illness, injury, or surgery**</li> </ul> </li> </ul>
Intervention/ Exposure	<ul style="list-style-type: none"> <li>• Sugar-sweetened beverage (SSB) consumption</li> <li>• Multi-component intervention in which the isolated effect of the intervention of interest on the outcome(s) of interest is provided or can be determined despite multiple components</li> </ul>	<ul style="list-style-type: none"> <li>• Infant milk, infant formula, toddler formula/milks</li> <li>• Other beverage types, such as nutritional beverages (e.g., protein shakes, smoothies)</li> <li>• Studies focusing on specific nutrients added to beverages instead of a beverage as a whole (i.e., studies where beverages are the delivery mechanism for a nutrient)</li> <li>• Beverages that are not commercially available (e.g., experimentally manipulated beverages)</li> <li>• Supplements</li> <li>• Alcohol</li> <li>• Soups</li> <li>• Multi-component intervention in which the isolated effect of the intervention of interest on the outcome(s) of interest is not provided or cannot be determined due to multiple components</li> </ul>
Comparator	<ul style="list-style-type: none"> <li>• Consumption of a different amount of SSB (including no consumption and versions diluted with water)</li> <li>• SSB vs. water</li> <li>• SSB vs. low- and no-calorie sweetened beverages</li> </ul>	<ul style="list-style-type: none"> <li>• No comparator</li> </ul>

\* Studies that enroll participants who are at risk for chronic disease were included

† Gestational age <37 weeks and 0/7 days

‡ Birth weight <2500g

§ Studies that exclusively enroll participants with obesity were included

\*\* Studies that exclusively enroll participants post-cesarean section were included

Category	Inclusion Criteria	Exclusion Criteria
Outcome(s)	<p>All included study designs in children (up to 19 years) and interventions only in adults (19 years and older):</p> <ul style="list-style-type: none"> <li>• Fasting blood glucose</li> <li>• Fasting insulin</li> <li>• Glucose tolerance/insulin resistance</li> <li>• Hemoglobin A1C</li> <li>• Prediabetes</li> </ul> <p>All included study designs in all included age groups:</p> <ul style="list-style-type: none"> <li>• Type 2 diabetes</li> </ul>	<ul style="list-style-type: none"> <li>• Urinary measures of glucose</li> <li>• Non-fasting blood glucose</li> <li>• Non-fasting insulin</li> <li>• Gestational diabetes</li> </ul>
Study duration*	<ul style="list-style-type: none"> <li>• Intervention length <math>\geq 12</math> weeks for hemoglobin A1C, prediabetes, and type 2 diabetes; <math>\geq 4</math> weeks for fasting blood glucose, fasting insulin, and glucose tolerance/insulin resistance</li> </ul>	<ul style="list-style-type: none"> <li>• Intervention length <math>&lt; 12</math> weeks for hemoglobin A1C, prediabetes, and type 2 diabetes; <math>&lt; 4</math> weeks for fasting blood glucose, fasting insulin, and glucose tolerance/insulin resistance</li> </ul>
Publication status	<ul style="list-style-type: none"> <li>• Peer-reviewed articles published in research journals</li> </ul>	<ul style="list-style-type: none"> <li>• Non-peer-reviewed articles, unpublished data or manuscripts, pre-prints, reports, editorials, retracted articles, and conference abstracts or proceedings</li> </ul>
Language	<ul style="list-style-type: none"> <li>• Published in English</li> </ul>	<ul style="list-style-type: none"> <li>• Not published in English</li> </ul>
Country†	<ul style="list-style-type: none"> <li>• Studies conducted in countries classified as high or very high on the Human Development Index the year(s) the intervention/exposure data were collected</li> </ul>	<ul style="list-style-type: none"> <li>• Studies conducted in countries classified as medium or low on the Human Development Index the year(s) the intervention/exposure data were collected</li> </ul>

## Search for and screen studies

NESR librarians, in collaboration with NESR analysts and the Committee, used the analytic framework and inclusion and exclusion criteria to develop a comprehensive literature search strategy. The literature search strategy included selecting and searching the appropriate bibliographic databases, translating search using syntax appropriate for the databases being searched, and employing search refinements, such as search filters. The full literature search is documented in **Appendix 2**.

The results of all electronic database searches, after removal of duplicates, were screened independently by 2 NESR analysts using a step-wise process by reviewing titles, abstracts, and full-texts to determine which articles meet the inclusion criteria. Manual searching was conducted to find peer-reviewed published articles not identified through the electronic database search. These articles were also screened independently by 2 NESR analysts at the abstract and full-text levels.

\* Study duration criteria were developed to enable focus on a stronger body of evidence.

† The classification of countries on the Human Development Index (HDI) is based on the UN Development Program Human Development Report Office (<http://hdr.undp.org/en/data>) for the year the study intervention occurred or data were collected. If the study does not report the year(s) in which the intervention/exposure data were collected, the HDI classification for the year of publication is applied. Studies conducted prior to 1990 are classified based on 1990 HDI classifications. If the year is more recent than the available HDI values, then the most recent HDI classifications are used. If a country is not listed in the HDI, then the current country classification from the World Bank is used (The World Bank Country and Lending Groups, available from: <https://datahelpdesk.worldbank.org/knowledgebase/articles/906519-world-country-and-lending-groups>)

## Extract data and assess the risk of bias

NESR analysts extracted all essential data from each included article to describe key characteristics of the available evidence, such as the author, publication year, cohort/trial name, study design, population life stage at intervention/exposure and outcome, intervention/exposure and outcome assessment methods, and outcomes. One NESR analyst extracted the data and a second NESR analyst reviewed the extracted data for accuracy. Each article included in the systematic review underwent a formal risk of bias assessment, with 2 NESR analysts independently completing the risk of bias assessment using the tool that is appropriate for the study design.\*†‡

## Synthesize the evidence

The Committee described, compared, and combined the evidence from all included studies to answer the systematic review question.§ Synthesis of the body of evidence involved identifying overarching themes or key concepts from the findings, identifying and explaining similarities and differences between studies, and determining whether certain factors impact the relationships being examined, which includes potential causes of heterogeneity across all included evidence.

Extracted data and risk of bias assessments for all included studies were tabulated to visually display results and facilitate synthesis. During synthesis, the Committee considered the effect direction, magnitude, and statistical significance of the results reported across the articles included in the body of evidence. The evidence was synthesized qualitatively without meta-analysis of effect estimates, statistical pooling or conversion of data, or quantitative tests of heterogeneity.

The synthesis plan for this review was designed with the end-use in mind, to inform the Committee's advice to HHS and USDA regarding dietary guidance across life stages. The first level of synthesis organization was by population. Then, within each of the population groups, the evidence was organized by similar outcome based on the available evidence.

## Develop conclusion statements and grade the evidence

After the Committee synthesized the body of evidence, they drafted conclusion statements. A conclusion statement is one or more summary statements carefully constructed to answer the systematic review question. Each conclusion statement reflects the evidence reviewed, as outlined in the analytic framework (e.g., PICO elements) and synthesis plan, and does not take evidence from other sources into consideration. Conclusion statements do not draw implications and should not be interpreted as dietary guidance. The Committee reviewed, discussed, and revised the conclusion statements until they reached agreement on wording that accurately reflected the body of evidence.

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\* Sterne JAC, Savović J, Page MJ, et al. RoB 2: a revised tool for assessing risk of bias in randomised trials. *BMJ* 2019; 366: i4898.doi: 10.1136/bmj.i4898

† Sterne JAC, Hernán MA, Reeves BC, et al. ROBINS-I: a tool for assessing risk of bias in non-randomized studies of interventions. *BMJ* 2016; 355: i4919; doi: 10.1136/bmj.i4919

‡ Higgins JPT, Morgan RL, Rooney AA, et al. A tool to assess risk of bias in non-randomized follow-up studies of exposure effects (ROBINS-E). *Environment International* 2024 (published online Mar 24); doi: [10.1016/j.envint.2024.108602](https://doi.org/10.1016/j.envint.2024.108602).

§ USDA Nutrition Evidence Systematic Review Branch. USDA Nutrition Evidence Systematic Review: Methodology Manual. February 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <https://nesr.usda.gov/methodology-overview>

The Committee then graded the strength of the evidence underlying each conclusion statement. They did this using NESR's predefined criteria, based on 5 grading elements: consistency, precision, risk of bias, directness and generalizability of the evidence. Study design and publication bias were also considered.\*

- **Consistency:** Consistency considers the degree of similarity in the direction and magnitude of effect across the body of evidence. This element also considers whether differences across the results can be explained by variations in study designs and methods.
- **Precision:** Precision considers the degree of certainty around an effect estimate for a given outcome. This element considers measures of variability, such as the width and range of confidence intervals, the number of studies, and sample sizes, within and across studies.
- **Risk of bias:** Risk of bias considers the likelihood that systematic errors resulting from the design and conduct of the studies could have impacted the accuracy of the reported results across the body of evidence.
- **Directness:** Directness considers the extent to which studies are designed to directly examine the relationship among the interventions/exposures, comparators, and outcome(s) of primary interest in the systematic review question.
- **Generalizability:** Generalizability considers whether the study participants, interventions and/or exposures, comparators, and outcomes examined in the body of evidence are applicable to the U.S. population of interest for the review.

The Committee assigned a grade to each conclusion statement (i.e., strong, moderate, limited, or grade not assignable). The grade communicates the strength of the evidence supporting a specific conclusion statement to decision makers and stakeholders. A conclusion statement can receive a grade of Strong, Moderate, or Limited, and if insufficient or no evidence is available to answer a systematic review question, then no grade is assigned (i.e., Grade Not Assignable) (**Table 4**). The overall grade is not based on a predefined formula for scoring or tallying ratings of each element. Rather, each overall grade reflects the expert group's thorough consideration of all of the grading elements, as they each relate to the specific nuances of the body of evidence under review.

**Table 4. Definitions of NESR grades**

Grade	Definition
Strong	The conclusion statement is based on a strong body of evidence as assessed by consistency, precision, risk of bias, directness, and generalizability. The level of certainty in the conclusion is strong, such that if new evidence emerges, modifications to the conclusion are unlikely to be required.
Moderate	The conclusion statement is based on a moderate body of evidence as assessed by consistency, precision, risk of bias, directness, and generalizability. The level of certainty in the conclusion is moderate, such that if new evidence emerges, modifications to the conclusion may be required.
Limited	The conclusion statement is based on a limited body of evidence as assessed by consistency, precision, risk of bias, directness, and generalizability. The level of certainty in the conclusion is limited, such that if new evidence emerges, modifications to the conclusion are likely to be required.
Grade Not Assignable	A conclusion statement cannot be drawn due to either a lack of evidence, or evidence that has severe limitations related to consistency, precision, risk of bias, directness, and generalizability.

\* Spill MK, English LK, Raghavan R, et al. Perspective: USDA Nutrition Evidence Systematic Review Methodology: Grading the Strength of Evidence in Nutrition- and Public Health-Related Systematic Reviews. *Adv Nutr.* 2022 Aug 1;13(4):982-991. doi: 10.1093/advances/nmab147

## Recommend future research

The Committee identified and documented research gaps and methodological limitations throughout the systematic review process. These gaps and limitations are used to develop research recommendations that describe the research, data, and methodological advances that are needed to strengthen the body of evidence on a particular topic. Rationales for the necessity of additional or stronger research are also provided with the research recommendations.

## Peer review

This systematic review underwent external peer review in a process coordinated by staff from the National Institutes of Health (NIH). NIH staff identified potential peer reviewers through outreach to a variety of professional organizations to select academic reviewers from U.S. colleges and universities across the country with a doctorate degree, including MDs, and expertise specific to the questions being reviewed. All peer reviewers were external to the *Dietary Guidelines* process, and therefore, current Committee members or Federal staff who supported the Committee or the development of the *Dietary Guidelines* were not eligible to serve as peer reviewers.

The peer review process was anonymous and confidential in that the peer reviewers were not identified to the Committee members or NESR staff, and in turn, the reviewers were asked not to share or discuss the review with anyone. Peer reviewers were made aware that per USDA, Food and Nutrition Service (FNS) agency policy, all peer reviewer comments would be summarized and made public, but comments would not be attributed to a specific reviewer.

Peer review occurred after draft conclusion statements were discussed by the full Committee at its third, fourth, fifth, and sixth public meetings. NIH staff assigned and distributed the reviews to at least 2 peer reviewers based on area of expertise. Following peer review, the Committee reviewed and discussed comments and made revisions to the systematic review, as needed, based on the discussion.

## Health equity considerations

The Committee was charged by HHS and USDA to review all scientific questions with a health equity lens to ensure that the next edition of the *Dietary Guidelines* is relevant to people with diverse racial, ethnic, socioeconomic, and cultural backgrounds. The Committee made a number of health equity considerations throughout the NESR systematic review process. The Committee's Scientific Report\* includes a more detailed discussion of their approach to applying a health equity lens to their review of evidence, but examples include consideration of key confounders relevant to health equity and assessment of generalizability of the evidence.

## Results

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### Literature search and screening results

The literature search was conducted to identify all potentially relevant articles for 2 systematic reviews assessing beverages and risk of type 2 diabetes: low- and no-calorie sweetened beverages<sup>†</sup> and sugar-

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\* 2025 Dietary Guidelines Advisory Committee. 2024. Scientific Report of the 2025 Dietary Guidelines Advisory Committee: Advisory Report to the Secretary of Health and Human Services and Secretary of Agriculture. U.S. Department of Health and Human Services. <https://doi.org/10.52570/DGAC2025>

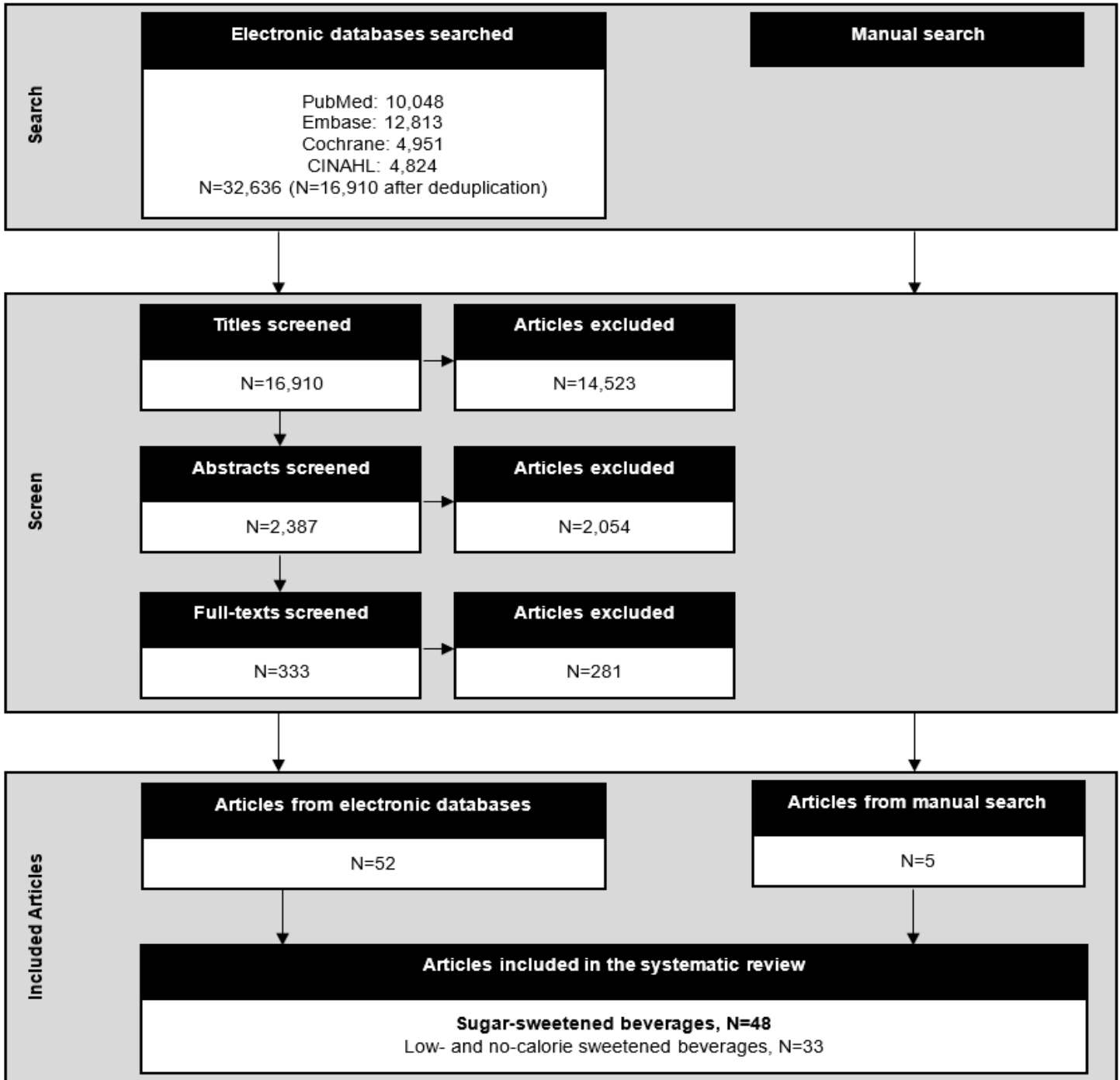
<sup>†</sup> Giovannucci E, Taylor CA, Deierlein AL, et al. *Low- and No-Calorie Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review*. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review; 2025. <https://doi.org/10.52570/NESR.DGAC2025.SR15>



sweetened beverages. The literature search (**Appendix 2**) yielded 16,910 search results after the removal of duplicates (see **Figure 2**). Dual-screening resulted in the exclusion of 14,523 titles, 2,054 abstracts, and 281 full-text articles. Reasons for full-text exclusion are in **Appendix 3**. Five additional articles were identified from the manual search. The body of evidence included 48 articles on sugar-sweetened beverages:

- Infants and young children: 0 articles
- Children and adolescents: 5 articles<sup>1-5</sup>
- Adults and older adults: 43 articles<sup>6-48</sup>

**Figure 2. Literature search and screen flowchart**



## Infants and young children

### Description of the evidence

No articles examined the relationship between sugar-sweetened beverage consumption in infants and young children up to age 24 months and risk of type 2 diabetes.

### Conclusion statement and grade

The Committee was not able to develop a conclusion statement to answer the question “What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?” based on the lack of evidence in infants and young children up to age 24 months (**Table 5**).

**Table 5. Conclusion statement and grade for sugar-sweetened beverage consumption in infants and young children and risk of type 2 diabetes**

<b>Conclusion Statement</b>	<b>A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by infants and young children up to age 24 months and risk of type 2 diabetes because there is no evidence available.</b>
<b>Grade</b>	Grade Not Assignable
<b>Body of Evidence</b>	0 articles
<b>Rationale</b>	There were no eligible articles examining sugar-sweetened beverage consumption by infants and young children and risk of type 2 diabetes.

## Children and adolescents

### Description of the evidence

Five articles examined the relationship between sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes (**Table 8**).<sup>1-5</sup> All included articles were from prospective cohort studies.

### Population

Studies were conducted in populations from the following countries and cohorts:

- Australia: 1 article from the Western Australian Pregnancy Cohort (Raine)<sup>1</sup>
- Germany: 2 articles from the Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study<sup>4,5</sup>
- Iran: 1 article from the Tehran Lipid and Glucose Study<sup>2</sup>
- United States: 1 article from the Exploring Perinatal Outcomes among CHildren (EPOCH) Study<sup>3</sup>

The articles had a mean baseline age ranging from approximately 9.5 to 14 years. Sample sizes ranged from N=226 to N=1124, with 49 to 68% female enrollment. Four articles did not report data on race and/or ethnicity<sup>1,2,4,5</sup>; the EPOCH study comprised of 48% non-Hispanic White, 8% non-Hispanic Black, 5% non-Hispanic Other, and 39% Hispanic participants.<sup>3</sup> Four articles reported data on parental education levels<sup>1,2,4,5</sup>; the EPOCH study<sup>3</sup> did not report data on socioeconomic position. All studies included participants across BMI weight status categories.

### Intervention/exposure and comparator

The intervention or exposure for this systematic review question was sugar-sweetened beverages. Eligible comparators were consumption of different amounts of sugar-sweetened beverages (including no consumption and versions diluted with water), water, or low- and no-calorie sweetened beverages. Studies that compared sugar-sweetened beverages to low- and no-calorie sweetened beverages were included in this review and not the review on low- and no-calorie sweetened beverages.\* All included articles examined different amounts of sugar-sweetened beverages as a categorical variable (i.e., tertiles, quartiles). Most studies examined sugar-sweetened beverage intake at baseline only; only Ambrosini et al<sup>1</sup> measured intake at multiple time points. Three studies measured sugar-sweetened beverage intake using a food frequency questionnaire.<sup>1-3</sup> In the DONALD study, sugar-sweetened beverage intake was measured using two 3-day weighed food records.<sup>4,5</sup> Mirmiran et al<sup>2</sup> examined sugar-sweetened carbonated soft drinks but did not differentiate drinks containing artificial non-caloric sweeteners from drinks containing caloric sugar; the study also examined “combined fruit juice drinks”, which combined sugar-sweetened synthetic juice drinks and 100% fruit juice drinks. Cohen et al<sup>3</sup> included sweetened tea or coffee in their measure of sugar-sweetened beverages.

### Outcome

The following outcomes were reported:

- Fasting blood glucose: 3 articles<sup>1-3</sup>
- Fasting insulin: 3 articles<sup>1,3,4</sup>
- Glucose tolerance/insulin resistance: 4 articles<sup>1,3-5</sup> from 3 studies

None of the included articles examined hemoglobin A1C, prediabetes, or type 2 diabetes. All studies collected fasting blood samples using standard methods. Two studies<sup>1,2</sup> examined change in outcomes over a 3-year follow-up, Cohen et al<sup>3</sup> examined change in outcomes over a 6-year follow-up, and both articles<sup>4,5</sup> from the DONALD study collected fasting blood samples only in adulthood.

### Synthesis of the evidence

Synthesis in children and adolescents focused on fasting blood glucose, fasting insulin, and glucose tolerance/insulin resistance due to a lack of data on hemoglobin A1C, prediabetes, and type 2 diabetes.

### Fasting blood glucose

Out of 3 articles, all reported no association between sugar-sweetened beverage consumption and fasting blood glucose; all but 1 study<sup>1</sup> controlled for total energy intake. Two studies found similar changes in fasting glucose between those with the highest categorical intake of sugar-sweetened beverage compared to those with the lowest intake.<sup>1,3</sup> In Mirmiran et al<sup>2</sup>, children and adolescents in the highest quartile of sugar-sweetened beverage consumption (median intake 142.2 mL/day) had similar odds of incident high fasting plasma glucose ( $\geq 100$  mg/dL according to American Diabetes Association recommendations) at follow-up approximately 3.6 years later compared to those in the lowest quartile with median sugar-sweetened beverage intake of 9.3 mL/d (OR = 1.95, 95% CI=0.73-5.22;  $p=0.108$  for trend).

### Fasting insulin

Out of 3 articles, all reported no association between sugar-sweetened beverage consumption and fasting insulin; all but 1 study<sup>1</sup> controlled for total energy intake. All studies found similar changes in fasting insulin, but with varying direction, between the highest and lowest categorical intake of sugar-sweetened beverages.<sup>1,3,4</sup>

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\* Giovannucci E, Taylor CA, Deierlein AL, et al. *Low- and No-Calorie Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review*. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review; 2025. <https://doi.org/10.52570/NESR.DGAC2025.SR15>

## Glucose tolerance/insulin resistance

Out of 4 articles, all reported no association between sugar-sweetened beverage consumption and glucose tolerance/insulin resistance; all but 1 study<sup>1</sup> controlled for total energy intake. Two studies found similar changes in homeostatic model assessment for insulin resistance (HOMA-IR), but with varying direction and magnitude, between those with the highest categorical intake of sugar-sweetened beverage compared to those with the lowest intake.<sup>1,3</sup> In the DONALD study, children and adolescents in the highest tertile of sugar-sweetened beverage intake (32-33 grams per day) had similar mean values for HOMA-IR and homeostasis model assessment of insulin sensitivity (the reciprocal of HOMA-IR) during adulthood.<sup>4,5</sup>

## Conclusion statement and grade

The Committee was not able to develop a conclusion statement to answer the question “What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?” based on their review of evidence in children and adolescents (**Table 6**).

**Table 6. Conclusion statement and grade for sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes**

<b>Conclusion Statement</b>	<b>A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by children and adolescents and risk of type 2 diabetes because of substantial concerns with directness in the body of evidence.</b>
<b>Grade</b>	Grade Not Assignable
<b>Body of Evidence</b>	5 articles: all prospective cohort studies
<b>Rationale</b>	<ul style="list-style-type: none"> <li>Substantial concerns with directness, particularly with no studies examining the primary outcome of interest</li> <li>Some concerns with risk of bias for potential confounding (due to not accounting for key confounders)</li> </ul>

The body of evidence underlying the conclusion statement includes 5 articles from prospective cohort studies. All studies examined the relationship between different levels of sugar-sweetened beverage consumption and fasting blood glucose, fasting insulin, and glucose tolerance/insulin resistance. Sugar-sweetened beverage intake was assessed as a categorical measure with 3-day weighed food records or food frequency questionnaire. Most outcome measures were examined continuously. There was no evidence that sugar-sweetened beverage consumption was associated with risk of type 2 diabetes. Studies found that increasing sugar-sweetened beverage intake resulted in similar changes in fasting blood glucose, fasting insulin, and measures of glucose tolerance/insulin resistance (specifically HOMA-IR) in children and adolescents. None of the studies specifically examined the endpoint outcome of incidence of type 2 diabetes.

Most studies had sample sizes between 200 and 600 participants. Studies had numerous risks of bias across domains (**Table 9**). Several articles were at higher risk of bias due to confounding for not accounting for multiple key confounders (such as race and/or ethnicity, physical activity, and family history of diabetes). Most studies were at higher risk of bias due to exposure misclassification for not accounting for change in sugar-sweetened beverage intake over time. There were also concerns due to missing data and selection of the reported results, as none of the studies had a pre-determined analysis plan. Additionally, there is risk of publication bias because a search of gray literature was not conducted. However, publication bias was not a serious concern for this body of evidence because studies reported mostly nonsignificant findings. There was a lack of data on race and/or ethnicity; only 1 study from the United States reported data on race and/or ethnicity. Furthermore, children under 9 years of age and adolescents over 14 years were under-represented in this body of evidence.

## Adults and older adults

### Description of the evidence

Forty-three articles (34 studies) examined the relationship between sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes. Six articles<sup>8,14,15,21,27,44</sup> were from randomized controlled trials (RCT) and 37 articles were from 28 observational studies (1 nested case control<sup>39</sup> and 36 prospective cohort studies<sup>6,7,9-13,16-20,22-26,28-38,40-43,45-48</sup>). Evidence for sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes is summarized in **Table 10** (RCT) and **Table 11** (observational). Risk of bias assessments for each article are detailed in **Table 12** (RCT) and **Table 13** (observational).

### Population

Among trials, mean baseline age ranged from 23 to 42 years. One trial<sup>21</sup> enrolled only females and another trial<sup>44</sup> was 84% female; all other trials had even distribution of male and female enrollment. Analytic sample sizes of study groups among the trials ranged from N=27 to N=313.

Among observational studies, mean baseline age ranged from 25 to 69 years. Three studies<sup>19,23,30</sup> were conducted in participants with a mean age  $\geq 60$  years and 1 study<sup>20</sup> provided data separately for adults  $\geq 60$  years. Six studies (9 articles<sup>6,18,23,34,35,38,39,41,43</sup>) enrolled only females and 2 studies<sup>12,40</sup> enrolled only males. Sample sizes ranged from N=93 to N=198,636. Fourteen articles had analytic sample sizes less than 10,000 participants.<sup>11,19,20,22,26,28-30,37,39,40,45-47</sup>

Multiple articles were from the same cohort study (such as EPIC, the European Prospective Investigation into Cancer and Nutrition) and were included when the article reported unique data. Studies were conducted in populations from the following countries:

- Australia: 1 article from an RCT<sup>27</sup>
- Brazil: 1 article from ELSA-Brasil<sup>9</sup> and 1 article from Cohort of Universities of Minas Gerais (CUME)<sup>47</sup>
- Denmark: 1 article from an RCT<sup>15</sup>
- Finland: 1 article from the Finnish Mobile Clinic Health Examination<sup>29</sup>
- France: 1 article from EPIC-E3N (Etude Epidémiologique auprès de femmes de la Mutuelle Générale de l'Education Nationale) cohort<sup>18</sup> and 1 article from the French NutriNet-Santé cohort<sup>42</sup>
- Germany: 1 article from the EPIC-Potsdam study<sup>48</sup>
- Iran: 1 article from the Tehran Lipid and Glucose Study<sup>26</sup>
- Japan: 1 article from the Japan Public Health Center-based prospective study<sup>17</sup>, 1 article from an unnamed cohort study<sup>40</sup>, and 1 article from the Mihama Diabetes Prevention Study<sup>45</sup>
- Mexico: 1 article from an RCT,<sup>21</sup> 1 article from the Mexican Teachers' Cohort,<sup>43</sup> and 1 article from the Health Workers Cohort Study<sup>46</sup>
- Singapore: 1 article from the Singapore Chinese Health study<sup>32</sup>
- South Korea: 1 article from Korean Genome and Epidemiology Study (KoGES)<sup>11</sup>
- Sweden: 3 articles from the Malmö Diet and Cancer study<sup>16,33,37</sup>
- Switzerland: 1 article from an RCT<sup>8</sup>
- United Kingdom: 1 article from the EPIC-Norfolk cohort<sup>31</sup> and 1 article from the Whitehall-II cohort<sup>28</sup>
- United States: 1 article from an RCT<sup>14</sup>, 1 article from the Choose Healthy Options Consciously Everyday (CHOICE) trial,<sup>44</sup> and 16 articles from 10 cohorts<sup>6,7,10,12,13,19,20,22,23,30,35,36,38,39,41</sup>
  - The cohorts included: Atherosclerosis Risk in Communities Study (ARIC)<sup>36</sup>; Black Women's Health Study<sup>34</sup>; Coronary Artery Risk Development in Young Adults Study (CARDIA)<sup>22</sup>; Health Professionals Follow-Up Study (HPFS)<sup>7,10,12,13</sup>; Multi-Ethnic Study of Atherosclerosis (MESA)<sup>30</sup>; NHANES-1 Epidemiologic Follow Up Study (NHEFS)<sup>20</sup>; Northern Manhattan Study<sup>19</sup>; Nurses' Health Study (NHS)<sup>6,7,10,13,38,39</sup> and NHS-II<sup>10,13,35,38,41</sup>; Women's Health Initiative<sup>23</sup>
- Multiple European countries: 2 articles from the EPIC-InterAct study<sup>24,25</sup>

### *Race and/or ethnicity*

Among trials, 3 trials did not report data on race and/or ethnicity.<sup>8,15,27</sup> Of the trials from the United States, 1 trial<sup>14</sup> consisted of 51% White, 20% Black, 13% Asian, 17% Multiple/Other, and 12% Hispanic participants and another trial<sup>44</sup> was 40% White, 54% Black, and 6% Other. Among observational studies, 7 studies (13 articles<sup>6,7,10,12,13,23,28,31,35,36,38,39,41</sup>) were in predominantly white/Caucasian populations (>75%). Four studies from the United States had more diverse racial and/or ethnic representation: 100% African American<sup>34</sup>; 50% Black and 50% White<sup>22</sup>; 53% Hispanic, 22% Black, 23% White, 2% Other<sup>19</sup>; 43% White, 23% African American, 21% Hispanic, 12% Chinese<sup>30</sup>. Twelve articles did not report data on race and/or ethnicity.<sup>16,18,20,24-26,29,33,37,42,47,48</sup>

### *Socioeconomic position*

Among trials, 3 trials<sup>8,15,27</sup> did not report data on socioeconomic position. In 2 trials from the U.S., over half of the participants had a bachelor's degree or higher.<sup>14,44</sup> Among observational studies, 4 studies (10 articles<sup>6,7,10,12,13,35,38,39,41,47</sup>) enrolled primarily well-educated samples; twenty studies (23 articles<sup>9,11,16-18,20,22-26,28,30-34,36,37,40,42,43,46</sup>) were diverse in terms of participant education and/or income levels and 4 studies<sup>19,29,45,48</sup> did not report data on socioeconomic position.

### *Health status*

Among trials, 4 trials<sup>8,15,21,44</sup> had inclusion criteria for BMI  $\geq 25$  kg/m<sup>2</sup> and 2 trials<sup>14,27</sup> included participants across BMI weight status categories. One trial<sup>15</sup> included participants that were nonsmokers and 3 trials<sup>8,14,27</sup> did not report data on smoking. No trials reported data on family history of diabetes. Among observational studies, participant mean BMI at baseline ranged from 23 to 28 kg/m<sup>2</sup>. Most articles (25 out of 37) reported a mean BMI  $\geq 25$  kg/m<sup>2</sup>. One study did not report baseline anthropometry.<sup>48</sup> Most articles (29 out of 37) reported current smoking in 9% to 30% of participants; 2 articles<sup>29,40</sup> had >30% of current smokers, 1 article<sup>16</sup> included 62% current/former smokers, 1 article<sup>23</sup> reported current smoking in 4% of female participants, and 4 articles<sup>18,45,47,48</sup> did not report data on smoking. Participants with family history of diabetes ranged from 7% to 53%; thirteen articles<sup>11,16,19,28,30,32,33,37,39,42,45,47,48</sup> did not report data on family history of diabetes.

### Intervention/exposure and comparator

The intervention or exposure for this systematic review question was sugar-sweetened beverages. Eligible comparators were consumption of different amounts of sugar-sweetened beverages (including no consumption and versions diluted with water), water, or low- and no-calorie sweetened beverages. Studies that compared sugar-sweetened beverages to low- and no-calorie sweetened beverages were included in this review and not the review on low- and no-calorie sweetened beverages.\*

Among trials, all but 1 study<sup>21</sup> compared sugar-sweetened beverage consumption to low- and no-calorie sweetened beverage consumption. Five trials also compared sugar-sweetened beverage consumption to water consumption.<sup>14,15,21,27,44</sup> The interventions and comparators in the trials are detailed below:

- Campos et al<sup>8</sup>: a 12-week intervention in which participants drank 2 or more 660-mL servings a day (habitual intake) of carbonated soft drinks and sugar-sweetened tea compared to a group that drank artificially sweetened beverages to replace 2 or more 660-mL servings a day (habitual intake) of sugar-sweetened beverages; both groups were provided with drinks weekly.
- Ebbeling et al<sup>14</sup>: a 52-week intervention in which the intervention group was instructed to drink sugar-sweetened beverages at the same number of servings per day ( $\geq 12$  fluid ounces) as their usual consumption compared to a group that was instructed to drink artificially-sweetened/diet beverages at the same number of servings per day as their usual consumption of sugar-sweetened beverages and

\* Giovannucci E, Taylor CA, Deierlein AL, et al. *Low- and No-Calorie Sweetened Beverages and Risk of Type 2 Diabetes: A Systematic Review*. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review; 2025. <https://doi.org/10.52570/NESR.DGAC2025.SR15>

compared to a group that was instructed to drink water (but not artificially- or sugar-sweetened beverages); all groups had beverages delivered to their home.

- Engel et al<sup>15</sup>: a 26-week intervention in which the intervention group drank sucrose-sweetened regular cola compared to a group that drank aspartame-sweetened diet cola and compared to a group that drank still mineral water; all groups consumed 1 liter per day of the beverage.
- Hernandez-Cordero et al<sup>21</sup>: a 9-month (39-week) intervention in which participants with habitually high sugar-sweetened beverage intake ( $\geq 250$  kilocalories per day) were randomized to 1 of 2 groups: water/education or education only. In the water/education group, participants received 2-3 liters per day of bottled water plus monthly face-to-face nutrition counseling, which targeted strategies to increase water intake, reduce sugar-sweetened beverage intake, and substitute water for sugar-sweetened beverages. In the education only group, participants consumed their habitual sugar-sweetened beverage intake plus monthly face-to-face nutrition counseling.
- Kendig et al<sup>27</sup>: a 12-week intervention in which the intervention group drank commercially-available sugar-sweetened beverages compared to a group that drank diet drinks sweetened with aspartame, acesulfame-K, and sucralose, and compared to the control group who drank water; all groups had beverages delivered to their home and consumed 4.5 liters per week as twelve 375-mL cans.
- Tate et al<sup>44</sup>: a 26-week weight loss intervention in which participants with high habitual sugar-sweetened beverage intake ( $\geq 280$  kilocalories per day) were assigned to 1 of 3 groups: attention control (maintain sugar-sweetened beverage intake), diet beverages, or water. The “attention control” group participated in monthly group sessions, weigh-ins, and weekly monitoring, and received general weight-loss information; participants in the “attention control” group were not encouraged to change beverage intake and were not provided with beverages. The diet beverage group was encouraged to replace  $\geq 2$  servings a day ( $\geq 200$  kilocalories) of caloric beverages with diet beverages (provided in four 355-500 mL single-servings each day), whereas the water group was encouraged to replace caloric beverages with bottled still and non-sweetened sparkling water.

Among observational studies, 18 articles<sup>6,7,9-12,19,20,22-25,31,35,38,42,43,48</sup> examined continuous beverage intake (e.g., servings per day); whereas, 16 articles<sup>13,16-18,26,28-30,32-34,36,37,40,41,46</sup> only compared intake across categories (e.g., tertiles, quintiles) and 3 articles<sup>39,45,47</sup> examined dichotomous intake. Twenty-eight articles examined sugar-sweetened beverage intake using a food frequency questionnaire<sup>6,7,9-13,16,17,19,20,23,26,28,30,32-39,41,43,46-48</sup>; 5 articles<sup>18,22,29,40,45</sup> used a diet history questionnaire, 1 article<sup>31</sup> used a 7-day food diary, and 1 article<sup>42</sup> used three 24-hour diet records. Two articles from the EPIC-InterAct study used either a dietary questionnaire or country-specific food frequency questionnaire to assess usual intake during the previous year.<sup>24,25</sup> Twenty-six articles examined beverage intake at baseline only<sup>9,11,16,18-20,23-26,28-34,36,37,40,42,43,45-48</sup> and 11 articles<sup>6,7,10,12,13,17,22,35,38,39,41</sup> (5 studies) examined change in beverage intake over time.

## Outcome

The following outcomes were reported:

- Fasting blood glucose: 5 trials<sup>8,14,15,21,44</sup>
- Fasting insulin: 3 trials<sup>8,14,15</sup>
- Glucose tolerance/insulin resistance: 4 trials<sup>8,14,15,27</sup>
- Hemoglobin A1C: 1 trial<sup>21</sup>
- Type 2 diabetes: 37 articles<sup>6,7,9-13,16-20,22-26,28-43,45-48</sup> from 28 observational studies

Among trials, outcome measures included risk factors for the development of type 2 diabetes (i.e., fasting blood glucose, fasting insulin, measures of glucose tolerance/insulin resistance, and hemoglobin A1C). All trials collected fasted blood samples and/or measured oral glucose tolerance using standard methods at baseline and follow-up. For intervention duration, 1 trial<sup>14</sup> was 52 weeks, 1 trial<sup>21</sup> was 9 months (39 weeks), 2 trials<sup>15,44</sup> were 6 months, and 2 trials<sup>8,27</sup> were 12 weeks. No included trials examined incidence of impaired fasting blood glucose, prediabetes, or type 2 diabetes.

Among observational studies, 19 of 37 articles used self-report of type 2 diabetes diagnosis, but all verified the accuracy of diagnosis.<sup>6,7,10,12,13,17-19,23,31,32,34-36,38,39,41,43,47</sup> Nine articles collected fasting blood samples to confirm diagnosis.<sup>9,11,22,26,28,30,40,45,46</sup> and 4 articles<sup>16,29,33,37</sup> used national registries. Five articles used a combination of self-report, linkage to registries, and hospital/mortality data.<sup>20,24,25,42,48</sup> Most studies reported type 2 diabetes incidence using standard criteria or definitions (such as the National Diabetes Data Group, American Diabetes Association, or World Health Organization for biomarkers) and/or treatment with hypoglycemic medication (insulin or oral hypoglycemic agent). Average follow-up duration ranged from 2 to 25 years.

## Synthesis of the evidence

Synthesis in adults and older adults focused on incidence of type 2 diabetes in observational studies, since there were few studies on fasting blood glucose, fasting insulin, glucose tolerance/insulin resistance, and hemoglobin A1C, and no studies on prediabetes. The results for each reported outcome are detailed below:

### Fasting blood glucose

Out of 5 trials, most reported null effect of sugar-sweetened beverage consumption on fasting blood glucose; 2 trials<sup>8,15</sup> adjusted for total energy intake. Campos et al<sup>8</sup> compared habitual intake of sugar-sweetened beverages to replacement with artificially-sweetened beverages, and both groups maintained baseline fasting plasma glucose levels after 12 weeks (mean values ranging from 5.1 to 5.4 mmol/L). In Ebbeling et al<sup>14</sup>, sugar-sweetened beverage intake increased fasting glucose by  $1.8 \pm 0.8$  mg/dL over 12 months, but this increase was similar to water intake and there was no statistically significant difference compared to artificially-sweetened beverage intake. Engel et al<sup>15</sup> found similar changes in fasting glucose over 6 months between groups that consumed sugar-sweetened beverages, water, or artificially-sweetened non-caloric soft drinks. Hernandez-Cordero et al<sup>21</sup> found similar changes in fasting glucose at 3, 6, and 9 months between the group that continued habitual intake of sugar-sweetened beverages (education only) and the water/education group. In Tate et al<sup>44</sup>, the group that replaced caloric beverages with water showed significant improvements in fasting glucose of  $-3.21$  mg/dL (95% CI:  $-3.89, -2.53$ ) compared with the attention control group that continued habitual intake of sugar-sweetened beverages ( $0.59$  mg/dL; 95% CI:  $0.35, 0.83$ ); the group that replaced caloric beverages with diet beverages decreased fasting glucose by  $-1.92$  mg/dL (95% CI:  $-2.38, -1.46$ ), but this was not significantly different from the attention control group.

### Fasting insulin

Out of 3 trials, all reported null effect of sugar-sweetened beverage consumption on fasting insulin compared to intake of water or low- and no-calorie sweetened beverages; 2 trials<sup>8,15</sup> adjusted for total energy intake. When comparing sugar-sweetened beverages to low- and no-calorie sweetened beverages, sugar-sweetened beverage intake tended to increase fasting insulin whereas low- and no-calorie sweetened beverage intake tended to decrease fasting insulin; however, these changes were not statistically significant. Two trials found similar changes in fasting insulin when comparing sugar-sweetened beverage intake to water.<sup>14,15</sup>

### Glucose tolerance/insulin resistance

Out of 4 trials, all reported null effect of sugar-sweetened beverage consumption on glucose tolerance/insulin resistance; 2 trials<sup>8,15</sup> adjusted for total energy intake. Campos et al<sup>8</sup> found similar changes in HOMA-IR over 12 weeks between participants who drank carbonated soft drinks and sugar-sweetened tea compared to those who drank artificially-sweetened beverages. Engel et al<sup>15</sup> found similar changes in measures of insulin sensitivity assessed by a 120-minute oral glucose tolerance test between intake of sucrose-sweetened regular cola, aspartame-sweetened cola, and water. In Ebbeling et al<sup>14</sup>, sugar-sweetened consumption decreased insulin sensitivity by  $-7.5 \pm 6.1$  percent after 12 months but this was not significant; there was no significant difference in insulin sensitivity between intake of sugar-sweetened beverages, water, or artificially-sweetened beverages. Kendig et al<sup>27</sup> found similar changes in a 60-minute oral glucose tolerance test over 12 weeks between participants that drank sugar-sweetened beverages, artificially-sweetened beverages, and water.



## Hemoglobin A1C

In Hernandez-Cordero et al<sup>21</sup>, those that continued habitual intake of sugar-sweetened beverages (education only) increased HbA1C over 9 months (0.02±0.03) whereas the group that was given water decreased HbA1C (-0.03±0.03); however, this difference was not statistically significant.

## Type 2 diabetes

Studies evaluating the association between sugar-sweetened beverage intake and incidence of type 2 diabetes reported different effect measures (hazard ratio, risk ratio, and odds ratio). Overall, the effect measures indicated an elevated risk of type 2 diabetes at 2 to 25 years of follow-up. Out of 37 articles, 26 articles (20 observational studies) reported an association between higher amounts of sugar-sweetened beverages consumed and higher risk of type 2 diabetes.<sup>6,7,9-13,16-20,22-26,29,31,32,35,38,41-43,47</sup> All but 2 studies<sup>9,11</sup> accounted for anthropometry at baseline, and all but 4 studies<sup>9,20,35,43</sup> adjusted for total energy intake. Most articles used a food frequency questionnaire to assess sugar-sweetened beverage consumption; 3 articles<sup>18,22,29</sup> used a diet history questionnaire, 1 article<sup>31</sup> used a 7-day food diary, and 1 article<sup>42</sup> used three 24-hour diet records. Most articles examined beverage intake at baseline only; 10 articles<sup>6,7,10,12,13,17,22,35,38,41</sup> (5 studies) examined change in beverage intake over time.

Eleven articles (10 observational studies) reported no association between sugar-sweetened beverage consumption and incidence of type 2 diabetes.<sup>28,30,33,34,36,37,39,40,45,46,48</sup> Of these articles, 2 studies<sup>34,46</sup> reported an association between higher amounts of sugar-sweetened beverages consumed and higher incidence of type 2 diabetes in models that did not account for anthropometry at baseline, but these findings were attenuated when adjusted for BMI. All but 2 studies<sup>37,45</sup> accounted for anthropometry at baseline, and all but 2 studies<sup>28,45</sup> adjusted for total energy intake. Most articles used a food frequency questionnaire to assess sugar-sweetened beverage consumption; 2 articles<sup>40,45</sup> used a diet history questionnaire. Most articles beverage intake at baseline only; 2 articles<sup>34,39</sup> examined change in beverage intake over time.

## Conclusion statement and grade

The Committee developed a conclusion statement to answer the question “What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?” based on their review of evidence in adults and older adults (**Table 7**).

**Table 7. Conclusion statement and grade for sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes**

<b>Conclusion Statement</b>	<b>Sugar-sweetened beverage consumption by adults and older adults may be associated with higher risk of type 2 diabetes. This conclusion statement is based on evidence graded as moderate.</b>
<b>Grade</b>	Moderate
<b>Body of Evidence</b>	43 articles: 6 RCT, 1 nested case control, 36 from prospective cohort studies
<b>Consistency</b>	Minimal variation in the direction and significance of findings
<b>Precision</b>	Strengths demonstrated by large sample sizes with adequate number of cases and narrow variance
<b>Risk of bias</b>	Some concerns due to potential for confounding and missing data
<b>Directness</b>	Few concerns with directness across studies
<b>Generalizability</b>	Some concerns with low diversity in reported race and/or ethnicity and socioeconomic position

## Assessment of the evidence

The body of evidence underlying the conclusion statement includes 6 randomized controlled trials and 37 observational studies. The strength of the evidence was graded based on an assessment of 5 grading elements, as described below. While the literature search was comprehensive, a search of the gray literature was not done, which may increase the possibility of publication bias. However, publication bias was not a serious concern for this body of evidence because several small and large studies were included, and some studies reported null findings.

### *Consistency*

The direction of findings was generally consistent. Most observational studies reported an association between higher sugar-sweetened beverage consumption and higher incidence of type 2 diabetes. All trials compared sugar-sweetened beverage consumption to water or low- and no-calorie sweetened beverages. While most trials found null effects, the studies suggested that sugar-sweetened beverage intake could be related to unfavorable changes in fasting blood glucose, fasting insulin, glucose tolerance/insulin resistance, and hemoglobin A1C.

### *Precision*

Most trials had concerns with sufficient power (i.e., the study did not report a power calculation or was based on reported power calculations that were not sufficiently powered). Most observational studies demonstrated statistically significant effects that came from a wide range of sample sizes (N=2,019 up to N=198,636) and demonstrated minimal variance (e.g., narrow confidence intervals).

### *Risk of bias*

Most trials had some concerns with bias from the randomization process and lacked a data analysis plan in the trial registry. Six out of 37 articles of observational studies accounted for all key confounders. Several observational studies did not account for race and/or ethnicity, socioeconomic position, and/or family history of diabetes. Some articles were at higher risk of exposure mismeasurement for not accounting for change in beverage intake that may occur during follow-up. There were also concerns due to missing data (large study cohorts with high attrition or exclusion) and selection of the reported results, as none of the observational studies had a pre-determined analysis plan.

### *Directness*

Most studies were designed to directly examine the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes. Three trials<sup>8,15,21</sup> examined cardiometabolic risk factors as the primary outcome, whereas the main objective of the other 3 trials was to examine change in weight. All of the observational studies were designed to directly answer the research question.

### *Generalizability*

The review included studies from 16 countries with similar HDI classification as the United States. The body of evidence has applicable interventions/exposures and outcomes relative to the U.S. population, but there were concerns with low diversity in reported race and/or ethnicity and socioeconomic position. A third of the studies (12 out of 34) were conducted in the United States, and of these, only 7 were diverse in racial and/or ethnic representation and socioeconomic position. The body of evidence included many participants with overweight or obesity, or other risk factors for cardiometabolic disease.

## Summary of conclusion statements and grades

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The Committee answered the systematic review question, “What is the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes?”, with the following conclusion statements.\* The grades reflect the strength of the evidence underlying the conclusion statements.

### Infants and young children

A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by infants and young children up to age 24 months and risk of type 2 diabetes because there is no evidence available. (Grade: Grade Not Assignable)

### Children and adolescents

A conclusion statement cannot be drawn about the relationship between sugar-sweetened beverage consumption by children and adolescents and risk of type 2 diabetes because of substantial concerns with directness in the body of evidence. (Grade: Grade Not Assignable)

### Adults and older adults

Sugar-sweetened beverage consumption by adults and older adults may be associated with higher risk of type 2 diabetes. This conclusion statement is based on evidence graded as moderate. (Grade: Moderate)

## Research recommendations

The Committee identified the following research recommendations that describe the research, data, and methodological advances that are needed to strengthen the body of evidence on sugar-sweetened beverage consumption and risk of type 2 diabetes.

1. Examine sugar-sweetened beverage intake in childhood (from birth to adolescence) in relation to changes in risk of type 2 diabetes across a range of ages and life stages.
2. Conduct well-controlled, randomized interventions, particularly in the United States with diverse populations, to examine the effect of sugar-sweetened beverage consumption and risk of type 2 diabetes.
3. Include diverse populations with varying race and/or ethnicity and socioeconomic position.
4. Control for confounding factors, such as family history of diabetes, that may impact the relationship between sugar-sweetened beverage consumption and risk of type 2 diabetes.
5. Provide repeated measures of beverage intake using validated dietary assessment tools and methods throughout the course of follow-up.
6. Use clear, specific definitions for sugar-sweetened beverages, particularly to differentiate from low- and no-calorie sweetened beverages.

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\* A conclusion statement is carefully constructed, based on the evidence reviewed, to answer the systematic review question. A conclusion statement does not draw implications and should not be interpreted as dietary guidance.

**Table 8. Evidence examining the relationship between sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes<sup>a</sup>**

Study and Participant Characteristics	Intervention or Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Ambrosini, 2013<sup>1</sup></b>  <b>PCS, Western Australian Pregnancy Cohort (Raine) Study, Australia</b>                      Analytic N=1124 (glucose); 1083 (insulin, HOMA-IR)</p> <p><b>Study objective:</b> To test the hypothesis that higher SSB intakes are associated with increases in cardiometabolic risk factors between 14 and 17y of age</p> <p><b>Participant characteristics at baseline:</b> adolescents</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 14 (0.2)y</li> <li>• Female: 49%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 35% low maternal education (<math>\leq 10</math>y)</li> <li>• Anthropometry: BMI <math>\sim 21</math> (4) kg/m<sup>2</sup>; 22% with overweight or obesity</li> <li>• Physical activity: <math>\sim 111</math> (30) watts on bicycle ergometer working capacity test</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: <math>\sim 300</math> g/d SSB; 89% were SSB consumers</li> </ul> <p><b>Excluded from study or analysis:</b> participants who reported not fasting before venipuncture</p>	<p><b>Exposure:</b> Carbonated soft drinks (excluding artificially sweetened or diet beverages), cordials or squash (fruit drink concentrate), and fruit juice drinks (excluding 100% juice)</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 cup (250mL or 8.45oz) or 261g</li> </ul> <p><b>Comparator:</b> categorical intake (tertiles of svg/d)</p> <ul style="list-style-type: none"> <li>• Tertile 1: 0 to 0.5 svg/d (0-130 g/d)</li> <li>• Tertile 2: <math>&gt;0.5</math> to 1.3 svg/d (130-329 g/d)</li> <li>• Tertile 3: <math>&gt;1.3</math> svg/d (331-2876 g/d)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake over previous year; completed by parents at 14y and adolescents at 17y</li> <li>• Baseline (14y), 3y follow-up (17y)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and HOMA-IR was calculated</li> <li>• Baseline (14y), 3y follow-up (17y)</li> </ul>	<p><b><u>Change in fasting serum glucose (mmol/L) between 14-17y with each tertile increase in SSB intake, <math>\beta</math> (95% CI)</u></b></p> <p><b>Girls</b>                      Tertile 1: REF                      Tertile 2: 0.1 (-1.3, 1.5), p=0.89                      Tertile 3: -1.2 (-3.0, 0.5), p=0.17                      P-trend=0.22</p> <p><b>Boys</b>                      Tertile 1: REF                      Tertile 2: -0.5 (-2.0, 1.0), p=0.50                      Tertile 3: -0.5 (-2.1, 1.1), p=0.55                      P-trend=0.55</p> <p><b><u>Change in fasting serum insulin (mIU/L) between 14-17y with each tertile increase in SSB intake, <math>\beta</math> (95% CI)</u></b></p> <p><b>Girls</b>                      Tertile 1: REF                      Tertile 2: 1.1 (-6.6, 8.8), P=0.79                      Tertile 3: -4.5 (-13.8, 4.9), P=0.35                      P-trend=0.42</p> <p><b>Boys</b>                      Tertile 1: REF                      Tertile 2: 0.2 (-8.2, 8.6), P=0.97                      Tertile 3: -1.4 (-10.3, 7.4), P=0.75                      P-trend=0.74</p> <p><b><u>Change in HOMA-IR between 14-17y with each tertile increase in SSB intake, <math>\beta</math> (95% CI)</u></b></p> <p><b>Girls</b>                      Tertile 1: REF                      Tertile 2: -1.4 (-17.6, 14.7), P=.86                      Tertile 3: -18.1 (-37.7, 1.5), P=0.07                      P-trend=0.42</p> <p><b>Boys</b>                      Tertile 1: REF                      Tertile 2: -3.5 (-20.6, 13.6), P=0.69                      Tertile 3: -7.8 (-25.8, 10.2), P=0.39                      P-trend=0.40</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, socioeconomic position (maternal education, family income), anthropometry, physical activity</li> <li>• Other: pubertal stage, dietary misreporting, 'Healthy' and 'Western' dietary pattern scores</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, family history of diabetes</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      National Heart Foundation of Australia and Beyond Blue Cardiovascular Disease and Depression Strategic Research Program; Australian National Health and Medical Research Council; UK Medical Research Council</p>

Study and Participant Characteristics	Intervention or Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Cohen, 2022<sup>3</sup></b>  <b>PCS, EPOCH (Exploring Perinatal Outcomes among Children Study), U.S.</b>                      Analytic N=597</p> <p><b>Study objective:</b> To assess intermediary metabolic alterations that link sugar-sweetened beverage intake to cardiometabolic risk factors in youth</p> <p><b>Participant characteristics at baseline:</b> children</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): Mean ~10.4 (1.5)y</li> <li>• Female: 50%</li> <li>• Race and/or ethnicity: 48% Non-Hispanic White; 8% Non-Hispanic Black; 6% Non-Hispanic Other; 39% Hispanic</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMIz 0.19-0.37 (NS difference between quartiles)</li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: 17% in utero GDM exposure</li> <li>• Smoking: NR</li> <li>• TEI: ~1775 kcal/d</li> <li>• Beverage intake at baseline: SSB intake for quartiles ranged from 0.11 (0.09) to 1.86 (0.91) svg/d</li> </ul> <p><b>Excluded from study or analysis:</b> missing data</p>	<p><b>Exposure:</b> SSB, including sodas, fruit drinks (i.e., Sunny Delight, Hawaiian Punch, etc.), sports drinks (i.e., Gatorade), and sweetened tea or coffee</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake ( quartiles of svg/d)                      Median energy-adjusted intakes (svg/d) in each quartile as continuous variable</p> <ul style="list-style-type: none"> <li>• Quartile 1: 0-0.25</li> <li>• Quartile 2: 0.26-0.54</li> <li>• Quartile 3: 0.55-1.00</li> <li>• Quartile 4: 1.01-5.12</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing intake during the past week</li> <li>• Baseline (~10y)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and HOMA-IR was calculated</li> <li>• Baseline (~10y), ~6y follow-up</li> </ul>	<p><b><u>Change in fasting glucose (mg/dL) through adolescence by quartile of SSB intake during childhood</u></b>, <math>\beta</math> (95% CI)</p> <p>Quartile 1: REF                      Quartile 2: -0.7 (-4.1, 2.7)                      Quartile 3: -0.7 (-4.2, 2.7)                      Quartile 4: -1.4 (-4.9, 2.2)                      P-trend=0.488</p> <p><b><u>Change in fasting insulin (<math>\mu</math>IU/mL) through adolescence by quartile of SSB intake during childhood</u></b>, <math>\beta</math> (95% CI)</p> <p>Quartile 1: REF                      Quartile 2: -1.7 (-3.5, 0.1)                      Quartile 3: -1.1 (-2.9, 0.7)                      Quartile 4: -1.3 (-3.1, 0.6)                      P-trend=0.426</p> <p><b><u>Change in HOMA-IR through adolescence by quartile of SSB intake during childhood</u></b>, <math>\beta</math> (95% CI)</p> <p>Quartile 1: REF                      Quartile 2: -0.7 (-1.3, -0.1)                      Quartile 3: -0.4 (-1.0, 0.2)                      Quartile 4: -0.5 (-1.1, 0.1)                      P-trend=0.330</p> <p>Analyses conducted for entire sample since no significant effect modification by sex (P-interaction&gt;0.10)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes, using residual method</li> <li>• Key confounders: sex, age, race and/or ethnicity</li> <li>• Other: none</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position, anthropometry, physical activity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIDDK</p>

Study and Participant Characteristics	Intervention or Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Della Corte, 2020<sup>4</sup></b>  <b>PCS, DONALD (Dortmund Nutritional and Anthropometric Longitudinally Designed Study), Germany</b>                      Analytic N=254</p> <p><b>Study objective:</b> To examine the prospective relevance of dietary sugar intake (based on dietary data as well as urinary excretion data) in adolescent years for insulin sensitivity and biomarkers of inflammation in young adulthood.</p> <p><b>Participant characteristics at baseline:</b> children and adolescents</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 12y, 9-15y (females); 13y, 10-16y (males)</li> <li>• Female: 51%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 57% paternal education <math>\leq</math>12y (female), 65% (male)</li> <li>• Anthropometry: 22% with overweight; BMI 17.8 kg/m<sup>2</sup> (female), 18.8 kg/m<sup>2</sup> (male)</li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 32% smoking in household</li> <li>• TEI: 1,697 kcal (female); 2,151 kcal (male)</li> <li>• Beverage intake at baseline: % of total sugar from SSB: 4.5 (3.9) in males, 3.9 (3.7) in females</li> </ul> <p><b>Excluded from study or analysis:</b> fasting glucose concentrations <math>&lt;</math>2.5 mmol/L threshold for calculating HOMA2-%S; non-singleton, born preterm, or abnormal birthweight; missing data</p>	<p><b>Exposure:</b> Total sugars from SSB (sweetened fruit juice drinks and nectars, soft drinks/soda, sweetened teas and water, instant beverages except dairy drinks, sweetened sports drinks) calculated as a percentage of TEI</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (tertiles, g/d)</p> <ul style="list-style-type: none"> <li>• Tertile 1 (g/d): 9.3 (6.3, 12.0) in females; 8.6 (5.8, 12.5) in males</li> <li>• Tertile 2 (g/d): 19.9 (17.0, 22.8) in females; 20.2 (18.6, 22.6) in males</li> <li>• Tertile 3 (g/d): 33.4 (29.3, 24.3) in females; 32.3 (28.1, 38.3) in males</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• At least 2 (range 2-7, mean=6) 3d weighed food records</li> <li>• Baseline (9-15y for females, 10-16y for males)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and HOMA2-%S (the reciprocal of HOMA2-IR) was calculated.</li> <li>• Adulthood (median 9y follow-up, 18-36y)</li> </ul>	<p><b>Fasting plasma insulin (pmol/L) in adulthood, Mean (95% CI)</b></p> <p><b>Females</b></p> <p>Tertile 1: 72.7 (63.7, 81.8)                      Tertile 2: 80.2 (71.7, 88.7)                      Tertile 3: 72.9 (63.5, 82.3)                      P-trend=0.66</p> <p><b>Males</b></p> <p>Tertile 1: 70.8 (59.1, 82.5)                      Tertile 2: 78.2 (66.9, 89.4)                      Tertile 3: 69.2 (58.1, 80.4)                      P-trend=0.85</p> <p><b>HOMA2-%S in adulthood, Mean (95% CI)</b></p> <p><b>Females</b></p> <p>Tertile 1: 84.6 (76.9, 92.2)                      Tertile 2: 75.9 (68.7, 83.1)                      Tertile 3: 81.2 (73.2, 89.2)                      P-trend=0.70</p> <p><b>Males</b></p> <p>Tertile 1: 87.0 (76.3, 97.6)                      Tertile 2: 87.0 (76.3, 97.6)                      Tertile 3: 86.2 (76.1, 96.3)                      P-trend=0.79</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (paternal education), anthropometry</li> <li>• Other: birth weight, gestational weight gain, smoking in the household, parental overweight, adult percent body fat</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, physical activity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Ministry of Science and Research of North Rhine-Westphalia Germany; German Federal Ministry of Health; Ministry of Culture and Science of the State North Rhine-Westphalia; German Federal Ministry of Education and Research</p>

Study and Participant Characteristics	Intervention or Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Goletzke, 2013<sup>5</sup></b>  <b>PCS, DONALD (Dortmund Nutritional and Anthropometric Longitudinally Designed Study), Germany</b>                      Analytic N=226</p> <p><b>Study objective:</b> To examine whether the amount or the quality (dietary glycemic index, glycemic load, and added sugar, fiber, and whole-grain intake) of carbohydrates during puberty is associated with risk markers of T2D in younger adulthood</p> <p><b>Participant characteristics at baseline:</b> children and adolescents</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~9.5y; 9-14y (female); 10-15y (male)</li> <li>• Female: 54%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 47% maternal education ≥12y; 51% maternal occupation</li> <li>• Anthropometry: 15% with overweight</li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 34% smoking in household</li> <li>• TEI: ~1870 kcal/d</li> <li>• Beverage intake at baseline: added sugar from drinks as a % of energy by tertile of dietary glycemic index, T1: 3.2%; T2: 4.0%; T3: 6.1%</li> </ul> <p><b>Excluded from study or analysis:</b> non-singleton, born preterm, or abnormal birthweight (&lt;2500 g); missing data; consistent underreporting of energy intake</p>	<p><b>Exposure:</b> Added sugar from drinks calculated as a percentage of energy intake</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (tertiles)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Average of at least two 3d weighed food records (mean=5 records/participant)</li> <li>• Baseline (9-15y for females, 10-16y for males)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and used to calculate HOMA-IR</li> <li>• Adulthood (mean 22.7y, range 18-36y)</li> </ul>	<p><b>HOMA-IR in adulthood</b>, Mean (95% CI)                      Tertile 1: 2.60 (2.36, 2.86)                      Tertile 2: 2.43 (2.22, 2.65)                      Tertile 3: 2.40 (2.18, 2.63)                      P-trend=0.8</p> <p>Data pooled for analysis since there were no differences by sex (P-interaction&gt;0.02)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes, using residual method</li> <li>• Key confounders: sex, age, socioeconomic position (maternal education), anthropometry</li> <li>• Other: early life factors (firstborn), nutritional factors (carbohydrate, glycemic index, glycemic load, fiber, protein), waist circumference in younger adulthood</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, physical activity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• High missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      German Federal Ministry of Food, Agriculture, and Consumer Protection through the Federal Office for Agriculture and Food; Wereld Kanker Onderzoek Fonds; Ministry of Science and Research of North Rhine-Westphalia Germany</p>

Study and Participant Characteristics	Intervention or Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Mirmiran, 2015<sup>2</sup></b>  <b>PCS, Tehran Lipid and Glucose Study, Iran</b>                      Analytic N=476</p> <p><b>Study objective:</b> To evaluate the association between SSB consumption with incident metabolic syndrome and its components 3.6 years later among children and adolescents in Iran</p> <p><b>Participant characteristics at baseline:</b> children and adolescents</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 13.6 (3.7)y; 6-18y</li> <li>• Female: 68%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 35% parental education level &lt;12y</li> <li>• Anthropometry: BMI ~19 (4) kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• TEI: 2116-3317 kcal/d (SSB quartile p-trend&lt;0.001)</li> <li>• Beverage intake at baseline:                             <ul style="list-style-type: none"> <li>○ SSB: 98 ml/d in boys, 70 ml/d in girls</li> <li>○ Sugar sweetened carbonated soft drinks: 38.5 (75.0) g/d</li> <li>○ Combined fruit juice drinks: 32.3 (60.1) g/d</li> </ul> </li> </ul> <p><b>Excluded from study or analysis:</b> high fasting plasma glucose at baseline; missing data; energy intake to energy requirement ratios beyond ±3SD range</p>	<p><b>Exposure:</b> SSB, including sugar-sweetened carbonated soft drinks ("did not differentiate between artificial non-caloric sweeteners and those containing caloric sugar e.g. fructose or sucrose") and combined fruit juice drinks (both 100% fruit juice and sugar sweetened synthetic juice drinks that are not 100% juice)</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 cup (250mL)</li> </ul> <p><b>Comparator:</b> categorical intake (quartiles)                      Median intakes (mL/d) in each quartile as continuous variable (SSB; Sugar-sweetened carbonated soft drink; Combined fruit juice drinks)</p> <ul style="list-style-type: none"> <li>• Quartile 1: 9.3; 1.1; 1.3</li> <li>• Quartile 2: 32.0; 9.3; 8.3</li> <li>• Quartile 3: 58.6; 33.1; 20.4</li> <li>• Quartile 4: 142.2; 100.0; 67.1</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake over previous year (parent-assisted if needed)</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected; high fasting plasma glucose defined as ≥100 mg/dL based on ADA recommendations or drug treatment in participants &gt;18y after follow-up</li> <li>• Baseline, ~3.6y follow-up</li> </ul>	<p><b><u>Incident high fasting plasma glucose after 3.6y follow-up by baseline intake</u></b>, OR (95% CI)  <b>SSB</b>                      Quartile 1: REF                      Quartile 2: 1.21 (0.48, 3.21)                      Quartile 3: 1.87 (0.75, 4.68)                      Quartile 4: 1.95 (0.73, 5.22)                      P-trend=0.108</p> <p><b>Sugar sweetened carbonated soft drinks</b>                      Quartile 1: REF                      Quartile 2: 0.55 (0.20, 1.54)                      Quartile 3: 1.93 (0.83, 4.50)                      Quartile 4: 1.12 (0.40, 3.12)                      P-trend=0.251</p> <p><b>Combined fruit juice drinks</b>                      Quartile 1: REF                      Quartile 2: 1.13 (0.44, 2.91)                      Quartile 3: 0.64 (0.21, 1.92)                      Quartile 4: 1.19 (0.44, 3.21)                      P-trend: 0.947</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes</li> <li>• Other: dietary fiber, tea and coffee, red and processed meat, fruit, vegetables</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position</li> <li>• Exposure subject to measurement error and only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Research Institute for Endocrine Sciences at the Shahid Beheshti University of Medical Sciences</p>



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<sup>a</sup> Abbreviations: ADA: American Diabetes Association; ASB: artificial sweetened beverage(s); BMI: body mass index; BMIz: body mass index z-score; CI: confidence interval; d: day(s); dL: deciliter(s); FFQ: food frequency questionnaire; g: gram(s); HbA1c: hemoglobin A1C; HOMA-IR: homeostatic model assessment for insulin resistance; hr: hour; IAUC: incremental area under the curve; kcal: kilocalorie(s); kg: kilogram(s); L: liter(s); LNCSB: low- and no-calorie sweetened beverage(s); m: meter(s); mg: milligram(s); min: minute(s); mIU: milli international unit(s); mL: milliliter(s); N/A: not applicable; NIDDK: National Institute of Diabetes and Digestive and Kidney Diseases; nmol: nanomole(s); NR: not reported; NS: not significant; OGTT: oral glucose tolerance test; OR: odds ratio; PCS: prospective cohort study; REF: reference group; SSB: sugar-sweetened beverage(s); SD: standard deviation; SSB: sugar-sweetened beverage(s); svg: serving(s); T2D: type 2 diabetes; TEI: total energy intake;  $\mu$ IU: micro-international unit(s);  $\mu$ mol: micromole(s); wk: week(s); y: year(s)

**Table 9. Risk of bias for observational studies examining sugar-sweetened beverage consumption in children and adolescents and risk of type 2 diabetes<sup>a</sup>**

Article	Confounding	Exposure measurement	Selection of participants	Post-exposure interventions	Missing data	Outcome measurement	Selection of reported result	Overall risk of bias
Ambrosini, 2013 <sup>1</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Cohen, 2022 <sup>3</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Della Corte, 2020 <sup>4</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Goletzke, 2013 <sup>5</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Mirmiran, 2015 <sup>2</sup>	HIGH	HIGH	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH

<sup>a</sup> Possible ratings of low, some concerns, high, very high, no information, or not applicable were determined using the "Risk of Bias in Non-randomized Studies of Exposures (ROBINS-E)" tool (Higgins JPT, Morgan RL, Rooney AA, et al. A tool to assess risk of bias in non-randomized follow-up studies of exposure effects (ROBINS-E). *Environment International* 2024 (published online Mar 24). doi: [10.1016/j.envint.2024.108602](https://doi.org/10.1016/j.envint.2024.108602).)

**Table 10. Intervention studies examining the relationship between sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes<sup>a</sup>**

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Campos, 2015<sup>8</sup></b>  <b>RCT-Parallel, Switzerland</b>                      Baseline N=31, Analytic N=27 (Attrition: 13%)</p> <p><b>Study objective:</b> To test the hypothesis that substituting artificially-sweetened beverages for SSB decreases intrahepatocellular lipid concentrations in subjects with overweight and high SSB consumption.</p> <p><b>Participant characteristics at baseline:</b> adults with overweight that habitually consume SSB</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 18-40y</li> <li>• Female: 48%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI ~31 kg/m<sup>2</sup>; 52% with obesity</li> <li>• Physical activity: ~9000 steps/d (sig diff between groups)</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2300 kcal/d</li> <li>• Beverage intake at baseline: All drank ≥44 oz/d of SSB</li> </ul> <p><b>Excluded from study or analysis:</b> BMI &lt;25 kg/m<sup>2</sup>; consume fewer than two 22oz SSB a day</p>	<p><b>Intervention:</b> SSB (n=13): Habitual intake of 2 or more 22oz servings of carbonated soft drinks and sugar-sweetened tea; every week participants were given new batch of SSB</p> <ul style="list-style-type: none"> <li>• Serving Size: 660 mL (22oz)</li> </ul> <p><b>Comparator:</b> LNCSB (n=14): replace habitual intake of SSB (2 or more 22oz servings of carbonated soft drinks and sugar-sweetened tea) with artificially sweetened beverages; every week participants were given new batch of ASBs</p> <p>Duration: 12wk                      Compliance: &gt;90% for both groups based on returned packages</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and used to calculate HOMA-IR</li> <li>• Baseline (wk4), 12wk follow-up (wk16)</li> </ul>	<p><b>Fasting plasma glucose (mmol/L),</b> Mean (SEM)                      Within group: Baseline, 12wk                      LNCSB: 5.1 (0.1), 5.1 (0.1)                      SSB: 5.4 (0.1), 5.4 (0.1)  <b>Between groups: Change over 12wk</b>                      P=NS</p> <p><b>Fasting insulin (uU/mL),</b> Mean (SEM)                      Within group: Baseline, 12wk                      LNCSB: 16.9 (2.1), 15.0 (2.2)                      SSB: 15.3 (2.2), 15.6 (2.0)  <b>Between groups: Change over 12wk</b>                      P=NS</p> <p><b>HOMA-IR,</b> Mean (SEM)                      Within group: Baseline, 12wk                      LNCSB: 4.1 (0.6), 3.6 (0.6)                      SSB: 3.7 (0.6), 4.0 (0.5)  <b>Between groups: Change over 12wk</b>                      P=NS</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes (NS between-group differences at baseline or during study)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Methods for randomization and concealment NR</li> <li>• Differences in attrition</li> <li>• No power calculation</li> <li>• Trial registry did not include data analysis plan</li> </ul> <p><b>Funding:</b>                      Swiss National Foundation for Science; Fondation Raymond Berger pour la recherche sur le diabete et les maladies metaboliques (Lausanne, Switzerland)</p>

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Ebbeling, 2020<sup>14</sup></b>  <b>RCT-Parallel, U.S.</b>                      Baseline N=203, Analytic N=186 (Attrition: 8%)</p> <p><b>Study objective:</b> To compare effects of consuming SSB, artificially-sweetened beverages, and unsweetened beverages in adults who habitually consumed SSB</p> <p><b>Participant characteristics at baseline:</b> adults that habitually consume SSB</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~27y; 18-40y</li> <li>• Female: 40%</li> <li>• Race and/or ethnicity: 51% White; 20% Black; 13% Asian; 17% Multiple/other/unknown; 12% Hispanic</li> <li>• Socioeconomic position: 53% with bachelors degree or higher; 31% with some college, vocational school, or associate's degree; 27% with household income ≥\$60k/yr</li> <li>• Anthropometry: BMI ~26 (5) kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2100 (650) kcal/d</li> <li>• Beverage intake at baseline: ~1.5 (1.3) svg/d (12-oz svg)</li> </ul> <p><b>Excluded from study or analysis:</b> BMI &lt;18.5 or &gt;40 kg/m<sup>2</sup>; SSB consumption less than one 12-oz svg/d; FBG ≥110 mg/dL, physician diagnosis of a major medical or psychiatric illness; chronic use of any medication that could affect study outcomes; smoking &gt;10 cigarettes/d; pregnancy (preceding 12mo) or plans to become pregnant during study period; lactation (preceding 3mo); change in hormonal contraceptives (preceding 3mo)</p>	<p><b>Intervention:</b> SSB (n=60): participants instructed to drink SSB delivered to home and to not drink LNCSB</p> <ul style="list-style-type: none"> <li>• Serving Size: 12oz; participants were instructed to drink delivered beverages at the same rate (number of svg/d) as usual SSB consumption</li> </ul> <p><b>Comparator:</b> Water (n=66): spring water, purified water, and unsweetened sparking water with or without flavoring; participants instructed to drink water delivered to home and to not drink SSB or LNCSB</p> <p>LNCSB (n=60): artificially-sweetened/diet beverages; participants instructed to drink LNCSB delivered to home and to not drink SSB</p> <p>Duration: 52wk                      Compliance: Biweekly check-in telephone calls to review beverage consumption; NS difference between groups in number of beverage deliveries or check-in calls completed</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and insulin sensitivity (as a % of value for a normal reference population) was evaluated by HOMA using glucose and insulin data</li> <li>• Baseline, 52wk follow-up</li> </ul>	<p><b>Change in fasting glucose (mg/dL) over 12mo.</b> Mean (SE)                      Water: 1.8 (0.7), P=0.01                      LNCSB: 0.9 (0.8), P=0.24                      SSB: 1.8 (0.8), P=0.02  <b>Between groups:</b> P=0.63</p> <p><b>Change in fasting insulin (µIU/L) over 12mo.</b> Mean (SE)                      Water: -0.0 (6.4), P=0.99                      LNCSB: -5.4 (6.3), P=0.39                      SSB: 7.5 (7.1), P=0.26  <b>Between groups:</b> P=0.37</p> <p><b>Change in insulin sensitivity % over 12mo.</b> Mean (SE)                      Water: -0.6 (6.3), P=0.93                      LNCSB: 4.9 (6.9), P=0.46                      SSB: -7.5 (6.1), P=0.22  <b>Between groups:</b> P=0.38</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Methods for randomization and concealment NR</li> <li>• Not sufficiently powered</li> <li>• Trial registry did not include data analysis plan</li> </ul> <p><b>Funding:</b>                      NHLBI; NIDDK; National Center for Research Resources; Harvard Catalyst Clinical and Translational Science Center; New Balance Foundation</p>

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Engel, 2018<sup>15</sup></b>  <b>RCT-Parallel, Denmark</b>                      Baseline N=73, Analytic N=58 (Attrition: 21%)</p> <p><b>Study objective:</b> To investigate the long-term effects of semi-skimmed milk on insulin sensitivity and further to compare milk with sugar-sweetened soft drinks and non-caloric soft drinks</p> <p><b>Participant characteristics at baseline:</b> adults with overweight and obesity</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~39y; 20-50y</li> <li>• Female: 64%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI ~32 kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: All nonsmokers</li> <li>• Alcohol intake: ~8 g/d (NS difference between groups)</li> <li>• TEI: ~2450 kcal/d</li> <li>• Beverage intake at baseline: Mean SSB intake: 184 mL/d</li> </ul> <p><b>Excluded from study or analysis:</b> &lt;20 or &gt;50y; BMI &lt;26 or &gt;40 kg/m<sup>2</sup>; diabetes; blood pressure &gt;160/100 mmHg; medication affecting either blood lipids, blood glucose or body weight; smoking; pregnancy or breastfeeding; allergies to milk or suffering from phenylketonuria; excessive physical activity (&gt;10 hr/wk)</p>	<p><b>Intervention:</b> Sugar-sweetened soft drinks (SSSD, n=14): sucrose-sweetened regular cola (Coca Cola)</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 L/d</li> </ul> <p><b>Comparator:</b> Water (n=16): still mineral water (Aqua d'or; 1 L/d)                      Artificially-sweetened non-caloric soft drinks (NCSD, n=15): aspartame-sweetened diet cola (Coca Colas)</p> <p>Duration: 26wk                      Compliance: Empty bottles or cartons every 3-4wk; 7-d dietary records at baseline, 3mo, 6mo; data NR</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and 120-min OGTT was conducted to assess various measures of insulin sensitivity (Matsuda Index, fasting, and AUC glucose, insulin and homeostasis model assessment values).</li> <li>• Baseline, 6mo follow-up</li> </ul>	<p><b>Fasting glucose (mmol/L),</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 5.26 (0.54), 5.35 (0.15)                      NCSD: 5.52 (0.47), 5.49 (0.15)                      SSSD: 5.48 (0.52), 5.62 (0.18)  <b>Between groups, change over 6mo:</b> P=NS</p> <p><b>Fasting insulin (pmol/L),</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 68.09 (54.67), 93.19 (24.94)                      NCSD: 75.35 (30.64), 74.73 (10.13)                      SSSD: 55.32 (22.88), 61.10 (6.77)  <b>Between groups, change over 6mo:</b> P=NS</p> <p><b>AUC OGTT glucose (mmol/L),</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 829 (204), 812 (62)                      NCSD: 864 (214), 876 (61)                      SSSD: 889 (228), 883 (60)  <b>Between groups, change over 6mo:</b> P=NS</p> <p><b>AUC OGTT insulin (pmol/L),</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 25,784 (10,735), 17,786 (2726)                      NCSD: 32,396 (12,305), 22,766 (3090)                      SSSD: 24,834 (8808), 24,364 (4889)  <b>Between groups, change over 6mo:</b> P=NS</p> <p><b>Matsuda Index,</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 7.98 (4.07), 8.38 (1.27)                      NCSD: 6.01 (3.55), 6.14 (0.84)                      SSSD: 8.08 (4.56), 6.58 (0.74)  <b>Between groups, change over 6mo:</b> P=NS</p> <p><b>HOMA-IR,</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 1.29 (1.04), 1.71 (0.44)                      NCSD: 1.44 (0.58), 1.42 (0.19)                      SSSD: 1.06 (0.44), 1.17 (0.13)  <b>Between groups, change over 6mo:</b> P=NS</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes (NS difference between groups)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Methods for randomization and concealment NR</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No power calculation</li> <li>• Trial registry did not include data analysis plan</li> </ul> <p><b>Funding:</b>                      Danish Council for Strategic Research; The Food Study Group/Danish Ministry of Food, Agriculture and Fisheries; Novo Nordic Foundation; Clinical Institute at Aarhus University, Denmark; Danish Dairy Company, Arla Foods</p> <p><b>HOMA-IR AUC,</b> Mean (SE)                      Within group: Baseline, 6mo                      Water: 3.08 (3.26), 3.08 (0.82)                      NCSD: 2.58 (1.50), 2.58 (0.39)                      SSSD: 2.12 (0.89), 2.12 (0.25)  <b>Between groups, change over 6mo:</b> P=NS</p>

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Hernandez-Cordero, 2014<sup>21</sup></b>  <b>RCT-Parallel, Mexico</b>                      Baseline N=240, Analytic N=240 (Attrition: 0%)</p> <p><b>Study objective:</b> To determine if replacing SSBs with water affects plasma triglycerides TGs (primary outcome), weight, and other cardiometabolic factors</p> <p><b>Participant characteristics at baseline:</b> adult women with overweight and obesity</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 33 (6.7)y; 18-45y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: 100% Hispanic</li> <li>• Socioeconomic position: 45% completed middle and high school; Socioeconomic level index (composite score including age, years of education, and housing condition): WEP: 0.11(1.34), EP: -0.14(1.33), P&gt;0.05</li> <li>• Anthropometry: BMI 31.2 (3.7) kg/m<sup>2</sup>; 46% with overweight; 54% with obesity</li> <li>• Physical activity: 1.455 (0.004) METs/d</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 31% current</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2035 kcal/d</li> <li>• Beverage intake at baseline: All with SSB intake ≥250 cal/d (inclusion criteria) ~1,111 mL/d, ~408 kcal/d, ~21% of TEI</li> </ul> <p><b>Excluded from study or analysis:</b> &lt;18 or &gt;45y; BMI &lt;25 or ≥39 kg/m<sup>2</sup>; SSB intake &lt;250 kcal/d; &gt;5% weight loss in past 6mo; current weight-reducing diet; pregnancy in past 6mo; medical condition that affects metabolic function; history of MI or heart surgery; medication affecting metabolism; recent psychiatric hospitalization; muscle-increasing regime or anabolic use; excessive alcohol (≥21 drinks/wk)</p>	<p><b>Intervention:</b> Participants with habitually high SSB intake (≥250 cal/d) were randomized to 1 of 2 groups: water+education (WEP) or education only (EP).</p> <p>SSB (EP, n=120): habitual SSB intake ≥250 cal/d plus monthly face-to-face nutrition counseling with a dietitian and a psychologist where they identified a healthy diet goal for the next month</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> Water (WEP, n=120): received bottled water (2-3 L/d) with monthly face-to-face nutrition counseling with a dietitian and a psychologist, including individualized and group meetings targeted to the rationale and strategies to increase water intake, reduce SSB intake, and substitute water for SSBs</p> <p>Duration: 9mo (39wk)                      Compliance: Attendance (Mean (SD)): WEP: 7.3(2.4) sessions                      EP: 6.4(2.4) sessions                      P=0.01                      SSB decreased in both groups but more in WEP</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and the proportion of HbA1C was determined by an immunocolorimetric method in whole blood</li> <li>• Baseline, 3, 6, and 9mo follow-up for fasting blood glucose; baseline and 9mo for HbA1C</li> </ul>	<p><b>Fasting plasma glucose (mg/dL), Mean (SE)</b>                      Within group: Baseline, 3mo, 6mo, 9mo                      Water (WEP): 90.2 (0.4), 90.0 (0.3), 90.5 (0.3), 90.7 (0.4)                      SSB (EP): 90.2 (0.3), 90.3 (0.3), 90.5 (0.4), 91.1 (0.3)</p> <p><b>Between groups: Change over 3mo</b>                      Water (WEP): 0.04 (1.5)                      SSB (EP): 0.2 (1.4)                      P=0.90</p> <p><b>Change over 6mo</b>                      Water (WEP): 0.60 (1.90)                      SSB (EP): 0.90 (2.20)                      P=0.90</p> <p><b>Change over 9mo</b>                      Water (WEP): 1.20 (1.70)                      SSB (EP): 1.70 (2.80)                      P=0.90</p> <p><b>HbA1C (%), Mean (SE)</b>                      Within group: Baseline, 9mo                      Water (WEP): 5.80 (0.01), 5.82 (0.01)                      SSB (EP): 5.80 (0.01), 5.80 (0.01)                      P(treatment)=0.20; P(time)=0.30</p> <p><b>Between groups: Change over 9mo</b>                      Water (WEP): -0.03 (0.03)                      SSB (EP): 0.02 (0.03)                      P=0.30</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• The EP group was not required to maintain SSB intake</li> </ul> <p><b>Funding:</b>                      Danone Research Center</p>

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Kendig, 2023<sup>27</sup></b>  <b>RCT-Parallel, Australia</b>                      Baseline N=118, Analytic N=80 (Attrition: 32%)</p> <p><b>Study objective:</b> To assess the effects of a 12-week intervention in which young healthy adults who regularly consumed SSBs were instructed to replace SSB intake with artificially-sweetened beverages, water, or to continue SSB intake.</p> <p><b>Participant characteristics at baseline:</b> young adults that habitually consume SSB</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 22.9 (3.9)y; 18-35y</li> <li>• Female: 40%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI 23.2 (3.6) kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: All drank &gt;2 L/wk of SSB</li> </ul> <p><b>Excluded from study or analysis:</b> &lt;18 or &gt;35y; BMI &lt;17.5 or &gt;30 kg/m<sup>2</sup>; regularly consumed less than 2 L/wk of SSB; participants with average weekly SSB intake &gt;1.5 times their group's IQR</p>	<p><b>Intervention:</b> SSB (n=27): commercially available</p> <ul style="list-style-type: none"> <li>• Serving Size: 4.5 L/wk (as 12 x 375mL cans and ≤3 cans/d)</li> </ul> <p><b>Comparator:</b> Water (n=25): 4.5 L/wk as 12 x 375mL cans</p> <p>Artificially-sweetened beverages (n=28): 4.5 L/wk as 12 x 375mL cans and ≤3 cans/d</p> <p>Duration: 12wk                      Compliance: questionnaire on weekly drink adherence and weekly SSB intake</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected and 60-min OGTT was conducted.</li> <li>• Baseline (test 1), 12wk (test 3)</li> </ul>	<p><b>Change in OGTT blood glucose (mM) over 12wk</b>                      Water vs Artificially-sweetened beverages vs SSB: data NR (figure only), P=NS</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Methods for randomization and concealment NR</li> <li>• Concerns with deviations from intended intervention</li> <li>• High attrition rate with no information on non-completers</li> <li>• Not sufficiently powered</li> <li>• Trial registry did not include data analysis plan</li> </ul> <p><b>Funding:</b>                      Australian Research Council                      Discovery Project</p>

Study and Participant Characteristics	Intervention, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Tate, 2012<sup>44</sup></b>  <b>RCT-Parallel, CHOICE (Choose Healthy Options Consciously Everyday), U.S.</b>                      Baseline N=318, Analytic N=313 (Attrition: 2%)</p> <p><b>Study objective:</b> To compare the replacement of caloric beverages with water or diet beverages as a method of weight loss over 6mo in adults and attention controls</p> <p><b>Participant characteristics at baseline:</b> adults with overweight and obesity</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 42 (10.7)y; 18-65y</li> <li>• Female: 84%</li> <li>• Race and/or ethnicity: 40% White; 54% Black; 6% Other</li> <li>• Socioeconomic position: 52% college graduate or beyond; 39% some college; 9% high school or less; 54% married or living with partner</li> <li>• Anthropometry: BMI 36.3 (5.9) kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 8% current; 25% former</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2170 kcal/d</li> <li>• Beverage intake at baseline: All consumed ≥280 kcal/d of caloric beverages; ~100-200 g/d</li> </ul> <p><b>Excluded from study or analysis:</b> &lt;18 or 65y; BMI &lt;25 or &gt;49.9 kg/m<sup>2</sup>; consumed &lt;280 kcal/d of caloric beverages; recent weight loss of &gt;5%; participation in other weight-loss or physical-activity research; lactation; recent or planned pregnancy; thyroid medication use; diabetes mellitus treated with oral medication or insulin; cancer in prior 5y; history of myocardial infarction or heart surgery; current psychiatric treatment; psychiatric hospitalization in past year; alcohol dependence; plans to move or unable to attend monthly group meetings; inadequate means to transport beverages; self-report of heart problems, frequent chest pains, or faintness or dizziness</p>	<p><b>Intervention:</b> Participants with high habitual SSB intake (≥280kcal/d) assigned to 1 of 3 groups: attention control (maintain SSB intake), water, or diet beverages (LNCSB).</p> <p>“Attention Control” (AC, n=105): equal treatment contact time and attention involving monthly group sessions and weigh-ins, weekly monitoring, general weight-loss information (e.g., product labels, portion control, physical activity). Participants were not provided with beverages, given weight-loss or physical activity goals, or encouraged to change beverage intake.</p> <ul style="list-style-type: none"> <li>• Serving Size: 355-500 mL (12-16oz)</li> </ul> <p><b>Comparator:</b> Diet beverages (n=105): encouraged to replace ≥2 svg/d (≥200 kcal) of caloric beverages with diet beverages (any combination of noncaloric sweetened beverages: carbonated, noncarbonated, noncaffeinated, and caffeinated beverages), which were provided at monthly group meetings</p> <p>Water (n=108): encouraged to replace ≥2 svg/d (≥200 kcal) of caloric beverages with water (any combination of bottled still and non-sweetened sparkling water), which were provided at monthly group meetings</p> <p>Duration: 26wk                      Compliance: Monthly group counseling to promote adherence; DB and Water groups attended significantly more group sessions than AC group. Two unannounced 24-h dietary recalls were administered via telephone (1 weekday and 1 weekend day) at baseline, 3mo, and 6mo.</p> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Fasting blood samples were collected</li> <li>• Baseline, 3mo and 6mo follow-up</li> </ul>	<p><b>Fasting blood glucose (mg/dL), Mean (95% CI)</b>                      Within group: Baseline, 3mo, 6mo                      LNCSB: 91.7 (89.3, 94.1); 90.2 (87.7, 92.7); 89.7 (87.6, 91.9), P=0.0978                      Water: 93.1 (88.9, 97.2); 90.1 (85.7, 94.6); 89.9 (87.4, 92.3), P=0.0027                      Attention Control: 88.6 (86.6, 90.6); 87.5 (85.1, 89.9); 89.2 (87.2, 91.1), P=0.0058</p> <p><b>Between groups: Change over 6mo</b>                      Water: -3.21 (-3.89, -2.53)                      LNCSB: -1.92 (-2.38, -1.46)                      Attention Control: 0.59 (0.35, 0.83)                      Water vs Attention Control: P=0.019                      LNCSB vs Attention Control: P=0.1471</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Amount of carbonated and/or caffeinated versions of beverages was not taken into account</li> <li>• Trial registry did not include data analysis plan</li> </ul> <p><b>Funding:</b>                      Nestlé Waters USA</p>



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<sup>a</sup> Abbreviations: ASB: artificial sweetened beverage(s); BMI: body mass index; d: day(s); dL: deciliter; HbA1c: hemoglobin A1C; HOMA-IR: homeostatic model assessment for insulin resistance; hr: hour; IAUC: incremental area under the curve; IQR: interquartile range; kcal: kilocalorie(s); kg: kilogram(s); L: liter(s); LNCSB: low- and no-calorie sweetened beverage(s); m: meter(s); mg: milligram(s); min: minute(s); mL: milliliter(s); mM: millimole(s); NA: not applicable; NIDDK: National Institute of Diabetes and Digestive and Kidney Diseases; nmol: nanomole; NR: not reported; NS: not significant; OGTT: oral glucose tolerance test; oz: ounce(s); RCT: randomized controlled trial; REF: reference group; SD: standard deviation; SE: standard error; SEM: standard error of the mean; SSB: sugar-sweetened beverage(s); svg: serving(s); TEI: total energy intake;  $\mu$ mol: micromole; wk: week(s); y: year(s)

**Table 11. Observational studies examining the relationship between sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes<sup>a</sup>**

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Bazzano, 2008<sup>6</sup></b>  <b>PCS, NHS (Nurses' Health Study), U.S.</b>                      Analytic N=71,346</p> <p><b>Study objective:</b> To examine the association between fruit, vegetable, and fruit juice intake and development of T2D</p> <p><b>Participant characteristics at baseline:</b>                      women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 50 (7)y; 38-63y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: All nurses</li> <li>• Anthropometry: BMI 23 (7) kg/m<sup>2</sup></li> <li>• Physical activity: 2 (2) hr/wk</li> <li>• Family history of diabetes: 25%</li> <li>• Smoking: 24% current</li> <li>• Alcohol intake: 7 (11) g/d</li> <li>• TEI: 1457-2061 kcal/d</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; loss to follow-up; energy intake &lt;600 or &gt;3500 kcal/d</p>	<p><b>Exposure:</b> 3 separate beverage categories: Sugar-sweetened cola; "Other carbonated beverages" (details NR, likely combined sugar- and artificially-sweetened beverages); Fruit punch</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• 1984, 1986, 1990, 1994, 1998 (cumulative average of median values from all available questionnaires up to the start of each 2y follow-up period)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• 18y</li> </ul>	<p><b><u>T2D by cumulative average of median intake.</u></b>                      HR (95% CI)  <b>Per 1 svg/d increase:</b>  <b>Sugar-sweetened cola:</b> 1.08 (1.04, 1.12)  <b>Carbonated beverages:</b> 1.04 (1.00, 1.09)  <b>Fruit punch:</b> 1.10 (1.06, 1.15)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: postmenopausal hormone use; consumption of whole grains, nuts, processed meats, coffee, and potatoes</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position</li> <li>• Exposure not well defined</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Bhupathiraju, 2013<sup>7</sup></b>  <b>PCS, NHS (Nurses' Health Study) and HPFS (Health Professionals Follow-up Study), U.S.</b>                      Analytic N=74,749 (NHS); 39,059 (HPFS)</p> <p><b>Study objective:</b> To prospectively examine the association of caffeinated compared with caffeine-free beverages, including coffee, tea, SSB, and carbonated artificially sweetened beverages, with T2D risk</p> <p><b>Participant characteristics at baseline:</b> nurses and health professionals</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~50y, 38-63y (NHS); ~53y, 40-75y (HPFS)</li> <li>• Female: 100% (NHS), 0% (HPFS)</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: Mainly educated health professionals</li> <li>• Anthropometry: BMI ~25 kg/m<sup>2</sup> in both NHS and HPFS</li> <li>• Physical activity: METs/wk, ~14 (NHS); ~21 (HPFS)</li> <li>• Family history of diabetes: 28% NHS; 20% HPFS</li> <li>• Smoking: current, 25% NHS; 9% HPFS</li> <li>• Alcohol intake: ~7 g/d in NHS; ~11 g/d in HPFS</li> <li>• TEI: ~1800 kcal/d (NHS); ~2000 kcal/d (HPFS)</li> <li>• Beverage intake at baseline: ~0.3 svg/d SSB in both NHS and HPFS</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; women who left ≥10 items blank on FFQ or energy intakes &lt;500 or &gt;3500 kcal/d; men who left ≥70 items blank on the FFQ or energy intake &lt;800 or &gt;4200 kcal/d</p>	<p><b>Exposure:</b> SSB (colas and carbonated soft drinks, examined separately as caffeinated and caffeine-free)</p> <ul style="list-style-type: none"> <li>• Serving Size: ~355 mL ("one standard glass, can, or bottle")</li> </ul> <p><b>Comparator:</b> continuous intake (per 1 svg/d) categorical intake (&lt;1/mo, 1-4/mo, 2-6/wk, ≥1/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline (1984 in NHS, 1986 in HPFS), and every 4y for 22-24y follow-up (until 2008 in NHS and 2008 in HPFS) - cumulative average of intake</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• Up to 2008, maximum 22y (HPFS) and 24y (NHS)</li> </ul>	<p><b>T2D by cumulative average intake</b>, RR (95% CI)</p> <p><b>NHS</b>  <b>Caffeinated SSB</b>                      &lt;1/mo (ref)                      1-4/mo: 1.08 (1.01, 1.15)                      2-6/wk: 1.19 (1.09, 1.28)                      ≥1/d: 1.29 (1.14, 1.47)                      P-trend&lt;0.0001  <b>Per-serving increment:</b> 1.13 (1.06, 1.20)</p> <p><b>Caffeine-free SSB</b>                      &lt;1/mo (ref)                      1-4/mo: 1.01 (0.95, 1.07)                      2-6/wk: 1.02 (0.95, 1.11)                      ≥1/d: 1.20 (1.01, 1.42)                      P-trend=0.05  <b>Per-serving increment:</b> 1.11 (1.00, 1.23)</p> <p><b>HPFS</b>  <b>Caffeinated SSB</b>                      &lt;1/mo (ref)                      1-4/mo: 1.05 (0.95, 1.16)                      2-6/wk: 1.19 (1.06, 1.34)                      ≥1/d: 1.33 (1.10, 1.60)                      P-trend&lt;0.001  <b>Per-serving increment:</b> 1.16 (1.06, 1.27)</p> <p><b>Caffeine-free SSB</b>                      &lt;1/mo (ref)                      1-4/mo: 1.02 (0.93, 1.13)                      2-6/wk: 1.14 (1.01, 1.29)                      ≥1/d: 1.37 (1.08, 1.74)                      P-trend=0.002  <b>Per-serving increment:</b> 1.23 (1.06, 1.43)</p> <p>No significant interaction between beverage intake and BMI, physical activity, or smoking (data not shown).</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: postmenopausal hormone use (NHS), alternative HEI, and consumption of other beverages other than the main exposure, depending on model, presence of hypertension, hypercholesterolemia, adherence to low-calorie diet</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Canhada, 2023<sup>9</sup></b>  <b>PCS, ELSA-Brasil, Brazil</b>                      Analytic N=10,202</p> <p><b>Study objective:</b> To investigate the association of ultra-processed food (UPF) consumption and specific subgroups with incident T2D in Brazilian adults.</p> <p><b>Participant characteristics at baseline:</b>                      Brazilian adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): Median 50y (IQR: 44-57); 35-74y</li> <li>• Female: 57%</li> <li>• Race and/or ethnicity: 55% White; 15% Black; 2% Asian; 1% Indigenous; 27% Brown</li> <li>• Socioeconomic position: 58% college/university degree, 33% secondary education, 9% elementary education or less; Median 1452 Brazilian reais (IQR: 726-2282)</li> <li>• Anthropometry: BMI 25.9 (23.4-28.9) kg/m<sup>2</sup></li> <li>• Physical activity: Median: 264 METs/wk (IRQ: 0-960)</li> <li>• Family history of diabetes: 36%</li> <li>• Smoking: 12% current; 28% former; 60% never</li> <li>• Alcohol intake: Median 0 g/wk (IQR: 0-65.6)</li> <li>• TEI: Median 2450 kcal/d (IQR: 1946-3120)</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data or loss to follow-up; implausible food intake; bariatric surgery</p>	<p><b>Exposure:</b> Sweetened beverages as subgroup of UPF (diet soda, soda, industrialized juice with sugar, industrialized juice without sugar, industrialized juice with sweetener, artificial juice with sugar, artificial juice without sugar, artificial juice with sweetener)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (per 50 g/d and SD/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report of physician-diagnosed T2D or current use of diabetes medication, or biomarkers (FPG &gt;126 mg/dl, 2hr post-load glucose &gt;200 mg/dL, or HbA1C 6.5%)</li> <li>• 8.2 (0.7)y</li> </ul>	<p><b>T2D by baseline intake</b>, RR (95% CI)                      By 50 g/d increase: 1.03 (1.02, 1.04)                      By SD (230 mL/d) increase: 1.14 (1.10, 1.18)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (income; school achievement), physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: none</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: anthropometry</li> <li>• Exposure subject to measurement error (combined SSB and LNCSB) and only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Brazilian Ministry of Health; Ministry of Science, Technology, and Innovation; National Council for Scientific and Technological Development</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Chen, 2023<sup>10</sup></b>  <b>PCS, NHS (Nurses' Health Study), NHS-II, HPFS (Health Professionals Follow-Up Study), U.S.</b>                      Analytic N=198,636 (71,871 in NHS; 87,918 in NHS-II; 38,847 in HPFS)</p> <p><b>Study objective:</b> To examine the relationship between ultra-processed food intake and T2D risk among 3 large U.S. cohorts</p> <p><b>Participant characteristics at baseline:</b> nurses and health professionals</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~50y, 38-63y (NHS); ~36y, 27-44y (NHS-II); ~52y, 40-75y (HPFS)</li> <li>• Female: 80%</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: Mainly educated health professionals</li> <li>• Anthropometry: BMI ~26 kg/m<sup>2</sup></li> <li>• Physical activity: MET-hr/wk, ~26 (NHS); ~18 (NHS-II); ~34 (HPFS)</li> <li>• Family history of diabetes: 27% NHS; 33% NHS-II; 14% HPFS</li> <li>• Smoking: current, 10% NHS; 9% NHS-II; 6% HPFS</li> <li>• Alcohol intake: ~5.5 g/d (NHS); ~3.5 g/d (NHS-II); ~10.5 g/d (HPFS)</li> <li>• TEI: ~1800 kcal/d (NHS and NHS-II); ~2000 kcal/d (HPFS)</li> <li>• Beverage intake at baseline: 0.3 (0.5) svg/d of SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; loss to follow-up; energy intake &lt;500 or &gt;3500 kcal/d for women, &lt;800 or &gt;4200 kcal/d for men</p>	<p><b>Exposure:</b> SSB (7-up; Coke or Pepsi with caffeine &amp; sugar; Coke or Pepsi without caffeine but with sugar; Hawaiian punch with sugar; Other carbonated beverage; Dairy coffee drinks)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (per 1 svg/d increase)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline (1984 in NHS, 1986 in HPFS, 1991 in NHS-II), and every 4y until 2016 in NHS and HPFS and until 2017 in NHS-II</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• Up to 2016 in NHS and HPFS, and up to 2017 in NHS-II</li> </ul>	<p><b>T2D by cumulative average intake</b>, HR (95% CI)                      Per 1 svg/d increase: 1.15 (1.12, 1.17)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (neighborhood income), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: physical examination; menopausal status (NHS-II); postmenopausal hormone use (NHS and NHS-II); oral contraceptive use (NHS-II); history of hypercholesterolemia and hypertension; other ultra-processed food subgroups</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Cho, 2023<sup>11</sup></b>  <b>PCS, KoGES (Korean Genome and Epidemiology Study), South Korea</b>                      Analytic N=7,438</p> <p><b>Study objective:</b> To examine the associations of ultra-processed food intake (combined, as well as individual ultra-processed food items) with the risk of T2D</p> <p><b>Participant characteristics at baseline:</b>                      Korean middle-aged adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 51.7y; 40-69y</li> <li>• Female: 47%</li> <li>• Race and/or ethnicity: All Asian</li> <li>• Socioeconomic position: 14% college or higher, 31% high school, 54% lower than high school; 33% lowest household income (compared to mid to low, mid to high, and highest); 48% physical labor occupation, 28% homemakers</li> <li>• Anthropometry: 41% with overweight</li> <li>• Physical activity: 66% high, 2% low, 31% none</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 25% current; 15% former; 59% never</li> <li>• Alcohol intake: 52% nondrinker, 30% low intake, 16% high intake</li> <li>• TEI: 1723-2109 kcal/d</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data or loss to follow-up; energy intake &lt;800 or &gt;4000 kcal/d for men, &lt;500 or &gt;3500 kcal/d for women</p>	<p><b>Exposure:</b> SSB, including carbonated sugar-sweetened beverages (coke, sprite) and other sugar-sweetened beverages (rice punch, yuja citron tea, etc.)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (per 1% increase in weight ratio)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report; confirmed through FBG and HbA1C measured at health examinations</li> <li>• 12.9y; Median: 15y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Per 1% increase in weight ratio                      Total SSB: 1.01 (0.99, 1.03)                      Carbonated SSB: 1.02 (1.00, 1.04)                      Other SSB: 0.99 (0.95, 1.04)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (educational attainment; household income; occupation; marital status), physical activity, smoking, alcohol intake</li> <li>• Other: history of coronary artery disease, stroke, hypertension, or dyslipidemia; ultra-processed food items; survey district (Ansan, Ansung)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: anthropometry, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      National Research Foundation of Korea</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>de Koning, 2011<sup>12</sup></b>  <b>PCS, HPFS (Health Professionals Follow-up Study), U.S.</b>                      Analytic N=41,109</p> <p><b>Study objective:</b> To examine the associations of SSB and ASB with incident T2D</p> <p><b>Participant characteristics at baseline:</b> male health professionals</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 51y; 40-75y</li> <li>• Female: 0%</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: All health professionals</li> <li>• Anthropometry: BMI ~25.5 (3.2) kg/m<sup>2</sup></li> <li>• Physical activity: ~21 METs/wk</li> <li>• Family history of diabetes: 12%</li> <li>• Smoking: 9% current</li> <li>• Alcohol intake: ~11 g/d</li> <li>• TEI: ~1950 kcal/d</li> <li>• Beverage intake at baseline: 0.36 (0.61) svg/d of total SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; &lt;40 or &gt;75y at recruitment, T1D, cardiovascular disease (heart attack, stroke, angina, coronary artery bypass graft), cancer (except melanoma skin cancer), energy intake &lt;800 or &gt;4299 kcal/d</p>	<p><b>Exposure:</b> SSB (caffeinated colas, caffeine-free colas, other carbonated sugar-sweetened beverages, and noncarbonated sugar-sweetened beverages such as fruit punches, lemonades, or other fruit drinks )</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 svg = "one standard glass, can, or bottle"</li> </ul> <p><b>Comparator:</b> continuous intake (per 1 svg/d) categorical intake (quartiles)</p> <ul style="list-style-type: none"> <li>• Quartile 1: never</li> <li>• Quartile 2: 2/mo</li> <li>• Quartile 3: 1-4/wk</li> <li>• Quartile 4: 4.5/wk to 7.5/d</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline, and every 4y for 20y</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• 20y</li> </ul>	<p><b>T2D by cumulative average intake of total SSB</b>, HR (95% CI)                      Q1: REF                      Q2: 1.09 (0.97, 1.22)                      Q3: 1.07 (0.95, 1.20)                      Q4: 1.24 (1.09, 1.40)                      P-trend&lt;0.01</p> <p><b>By 1 svg/d increase of total SSB:</b> 1.16 (1.08, 1.25), P&lt;0.01  <b>By 1 svg/d increase of caffeinated and non-caffeinated colas:</b> 1.20 (1.09, 1.32), P&lt;0.01  <b>By 1 svg/d increase of carbonated noncolas:</b> 1.35 (1.08, 1.69), P&lt;0.01  <b>By 1 svg/d increase of fruit punches, lemonades, other noncarbonated fruit drinks:</b> 1.05 (0.89, 1.25), P=0.65</p> <p><b>By baseline intake of total SSB</b>                      Q1: REF                      Q4: 1.19 (1.07, 1.33)                      P-trend&lt;0.01</p> <p>No significant interactions between beverage intake and age, alcohol, physical activity, or family history (data not shown).</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: multivitamin use, high triglycerides, high blood pressure, use of diuretics, previous weight change and low-calorie diet, alternative HEI</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH; Canadian Institutes of Health Research; Canadian Diabetes Association</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Drouin-Chartier, 2019<sup>13</sup></b>  <b>PCS, NHS (Nurses' Health Study), NHS-II, HPFS (Health Professionals Follow-Up Study), U.S.</b>                      Analytic N=192,352 (76,531 in NHS; 81,597 in NHS-II; 34,224 in HPFS)</p> <p><b>Study objective:</b> To evaluate the associations of long-term changes in consumption of SSB and ASB with subsequent risk of T2D</p> <p><b>Participant characteristics at baseline:</b> nurses and health professionals</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~58y, 52-77y (NHS); ~41y, 37-54y (NHS-II); ~57y, 54-89y (HPFS)</li> <li>• Female: 82%</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: Mainly educated health professionals</li> <li>• Anthropometry: BMI ~25 kg/m<sup>2</sup></li> <li>• Physical activity: MET-hr/wk, ~15 (NHS); ~24 (NHS-II); ~20 (HPFS)</li> <li>• Family history of diabetes: 28% NHS; 34% NHS-II; 26% HPFS</li> <li>• Smoking: current, 19% NHS; 11% NHS-II; 8% HPFS</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~1700 kcal/d (NHS and NHS-II); ~2000 kcal/d (HPFS)</li> <li>• Beverage intake at baseline: ~0.4 svg/d (NHS); ~0.7 svg/d (NHS-II); ~0.6 svg/d (HPFS)</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; loss to follow-up; energy intake &lt;500 or &gt;3500 kcal/d for women, &lt;800 or &gt;4200 kcal/d for men</p>	<p><b>Exposure:</b> SSB (carbonated and noncarbonated beverages with sugar, such as soft drink, punch, lemonade, fruit drink, or sugared ice tea)</p> <ul style="list-style-type: none"> <li>• Serving Size: 8oz (converted for analysis from one 12-oz glass, bottle, or can)</li> </ul> <p><b>Comparator:</b> categorical intake (change in beverage intake)</p> <ul style="list-style-type: none"> <li>• Decrease in consumption &gt;0.50 svg/d</li> <li>• Decrease in consumption &gt;0.07-0.50 svg/d</li> <li>• No change or relatively stable consumption (<math>\pm 0.07</math> svg/d)</li> <li>• Increase in consumption &gt;0.07-0.50 svg/d</li> <li>• Increase in consumption &gt;0.50 svg/d</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline (1990 in NHS and HPFS, 1995 in NHS-II), and every 4y for up to 26y follow-up (analyzed 4y change in beverage intake on risk of T2D in the subsequent 4y period)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• Up to 2012 in NHS and HPFS, and up to 2013 in NHS-II</li> </ul>	<p><b>T2D by 4y change in intake</b>, HR (95% CI)  <b>Pooled (all cohorts)</b>                      Decrease &gt;0.50 svg/d: 1.01 (0.94, 1.09)                      Decrease &gt;0.07-0.50 svg/d: 1.02 (0.95, 1.09)                      No change: REF                      Increase &gt;0.07-0.50 svg/d: 1.03 (0.97, 1.09)                      Increase &gt;0.50 svg/d: 1.09 (1.03, 1.17)                      P-trend=0.006</p> <p>Stratified analysis of pooled results also provided by initial AHEI index, obesity, and physical activity – no significant interactions</p> <p><b>NHS</b>                      Decrease &gt;0.50 svg/d: 1.02 (0.92, 1.14)                      Decrease &gt;0.07-0.50 svg/d: 1.03 (0.94, 1.13)                      No change: REF                      Increase &gt;0.07-0.50 svg/d: 1.04 (0.96, 1.12)                      Increase &gt;0.50 svg/d: 1.10 (1.00, 1.20)                      P-trend=0.12</p> <p><b>NHS-II</b>                      Decrease &gt;0.50 svg/d: 0.92 (0.81, 1.06)                      Decrease &gt;0.07-0.50 svg/d: 0.99 (0.87, 1.11)                      No change: REF                      Increase &gt;0.07-0.50 svg/d: 1.04 (0.93, 1.16)                      Increase &gt;0.50 svg/d: 1.13 (1.00, 1.27)                      P-trend=0.001</p> <p><b>HPFS</b>                      Decrease &gt;0.50 svg/d: 1.15 (0.97, 1.37)                      Decrease &gt;0.07-0.50 svg/d: 1.03 (0.90, 1.19)                      No change: REF                      Increase &gt;0.07-0.50 svg/d: 0.99 (0.88, 1.12)                      Increase &gt;0.50 svg/d: 1.03 (0.89, 1.20)                      P-trend=0.29</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: physical examination; menopausal status; postmenopausal hormone use; oral contraceptive use; AHEI score; intake of other beverages</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>



Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Ericson, 2018<sup>16</sup></b>  <b>PCS, Malmö Diet and Cancer, Sweden</b>                      Analytic N=25,069</p> <p><b>Study objective:</b> To examine the interaction between a genetic risk score for T2D and a diet risk score of processed meat, SSB, whole grain, and coffee</p> <p><b>Participant characteristics at baseline:</b> middle-aged and older adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 58 (7)y; 45-74y</li> <li>• Female: 61%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 32% education &gt;10y</li> <li>• Anthropometry: BMI 25 (4) kg/m<sup>2</sup></li> <li>• Physical activity: 20% high leisure time physical activity</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 62% ever</li> <li>• Alcohol intake: 11 (12) g/d</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: 33% zero-consumers of SSB; mean intake ~77 g/d</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; loss to follow-up</p>	<p><b>Exposure:</b> SSB (beverages sweetened with energy-containing sweeteners; mainly sucrose)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (tertiles)</p> <ul style="list-style-type: none"> <li>• Tertile 1: zero-consumers of SSB</li> <li>• Tertile 2: below median of SSB consumers</li> <li>• Tertile 3: above median of SSB consumers</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated, modified diet history (7d food diary, FFQ assessing usual intake during previous year, and 45- or 60-min interview)</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• National registries (90% of cases) or follow-up examinations (10% of cases)</li> <li>• Mean follow-up time: 17 (5.6)y; Range: 0-24y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)</p> <p><b>All</b></p> <p>Tertile 1: REF                      Tertile 2: 1.01 (0.93, 1.10)                      Tertile 3: 1.13 (1.05, 1.22)                      P-trend=0.003</p> <p><b>Women</b></p> <p>Tertile 1: REF                      Tertile 2: 1.06 (0.94, 1.20)                      Tertile 3: 1.12 (1.01, 1.25)                      P-trend=0.03</p> <p><b>Men</b></p> <p>Tertile 1: REF                      Tertile 2: 0.95 (0.84, 1.07)                      Tertile 3: 1.14 (1.01, 1.28)                      P-trend=0.06</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (education), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: diet assessment method version, season of diet collection</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      European Research Council; Swedish Research Council; Swedish Heart and Lung Foundation; Region Skåne; Novo Nordic Foundation; Albert Pålsson Research Foundation</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Eshak, 2013<sup>17</sup></b>  <b>PCS, Japan Public Health Center-based prospective study , Japan</b>                      Analytic N=27,585</p> <p><b>Study objective:</b> To examine whether increased intake of soft drink and juices contribute to T2D</p> <p><b>Participant characteristics at baseline:</b>                      Japanese adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~49y; 40-59y</li> <li>• Female: 56%</li> <li>• Race and/or ethnicity: All Asian</li> <li>• Socioeconomic position: 50% &gt;high school education</li> <li>• Anthropometry: BMI 23 (3) kg/m<sup>2</sup></li> <li>• Physical activity: 15% practice sports ≥3 times/wk</li> <li>• Family history of diabetes: 8%</li> <li>• Smoking: 25% current (mean ~52% in men, ~4% in women)</li> <li>• Alcohol intake: ~53 g/d in men, ~29 g/d in women</li> <li>• TEI: ~2200 kcal/d in men, ~1500 kcal/d in women</li> <li>• Beverage intake at baseline: 59% rarely drink SSB; 24% drink SSB ≤2 times/wk; 10% drink SSB 3-4 times/wk; 6% SSB almost every day</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cardiovascular disease, cancer, kidney disease, chronic liver disease; missing data; energy intake &lt;500 or &gt;3500 kcal/d</p>	<p><b>Exposure:</b> Soft drinks (cola, flavored juices, and non-100% fruit juices)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (rarely, ≤2 times/wk, 3-4 times/wk, almost every day)</p> <p>Also, change in intake from baseline to 5y:</p> <ul style="list-style-type: none"> <li>• Decreased intake at 5y</li> <li>• Consistently no intake</li> <li>• Consistently moderate intake (1-4 times/wk)</li> <li>• Increased intake at 5y</li> <li>• Consistently high intake (almost every day)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline, 5y follow-up</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• "Has a doctor ever told you that you have any of the following diseases? Diabetes (yes/no)" at baseline, 5y and 10y follow-up</li> <li>• 5y and 10y follow-up</li> </ul>	<p><b>T2D by baseline intake, OR (95% CI)</b></p> <p><b>5y incidence</b></p> <p><b>Men (n=13,854)</b>                      Rarely: REF                      ≤2 times/wk: 0.98 (0.76, 1.27)                      3-4 times/wk: 0.79 (0.55, 1.13)                      Almost every day: 0.98 (0.64, 1.50)                      P-trend=0.73</p> <p><b>Women (n=17,007)</b>                      Rarely: REF                      ≤2 times/wk: 1.20 (0.86, 1.67)                      3-4 times/wk: 1.44 (0.90, 2.31)                      Almost every day: 2.10 (1.23, 3.59)                      P-trend=0.004</p> <p><b>10y incidence</b></p> <p><b>Men (n=12,137)</b>                      Rarely: REF                      ≤2 times/wk: 0.86 (0.68, 1.08)                      3-4 times/wk: 0.83 (0.61, 1.12)                      Almost every day: 0.98 (0.68, 1.42)                      P-trend=0.80</p> <p><b>Women (n=15,448)</b>                      Rarely: REF                      ≤2 times/wk: 1.15 (0.88, 1.51)                      3-4 times/wk: 1.17 (0.78, 1.76)                      Almost every day: 1.79 (1.11, 2.89)                      P-trend=0.01</p> <p><b>T2D by change in SSB intake over 5y, OR (95% CI)</b></p> <p><b>Men</b>                      Decreased intake at 5y: 0.92 (0.69, 1.24)                      Consistently no intake: REF                      Consistently moderate intake: 0.77 (0.55, 1.08)                      Increased intake at 5y: 0.90 (0.39, 1.18)                      Consistently high intake: 0.93 (0.64, 1.36)</p> <p><b>Women</b>                      Decreased intake at 5y: 0.87 (0.56, 1.36)                      Consistently no intake: REF                      Consistently moderate intake: 1.17 (0.73, 1.87)                      Increased intake at 5y: 1.33 (1.01, 1.76)                      Consistently high intake: 1.76 (1.11, 2.87)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (education; occupation), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: history of hypertension, consumption of coffee and green tea, magnesium, calcium, vitamin D, rice, total dietary fiber</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• No evidence whether the result was biased due to missing data</li> <li>• Outcome was self-reported</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Ministry of Health, Labor, and Welfare of Japan</p> <p>Also provided results stratified by age (P=0.001), sports activity (P=0.06), education (P=0.004), occupation (P=0.003), BMI (P=0.007), and menopausal status (P=0.03).</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Fagherazzi, 2013<sup>18</sup></b>  <b>PCS, EPIC-E3N (Etude Epidémiologique auprès de femmes de la Mutuelle Générale de l'Éducation Nationale), France</b>                      Analytic N=66,118</p> <p><b>Study objective:</b> To evaluate the association between self-reported SSB, ASB, and 100% fruit juice consumption and T2D risk over 14y of follow-up</p> <p><b>Participant characteristics at baseline:</b>                      French women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 52.6 (6.6)y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 86% graduate/postgraduate education; 14% undergraduate education</li> <li>• Anthropometry: 16% with overweight, 3% with obesity</li> <li>• Physical activity: 54.8 (30.2) MET-hr/wk</li> <li>• Family history of diabetes: 10%</li> <li>• Smoking: NR</li> <li>• Alcohol intake: 11.1 (14.1) g/d</li> <li>• TEI: 2170.5 (574.5) kcal/d</li> <li>• Beverage intake at baseline: 328.3 (485.8) mL/wk of SSB; 81% non-consumers</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; extreme energy intake (lowest and highest 1% of cohort)</p>	<p><b>Exposure:</b> SSB (excluding ASB and 100% fruit juice)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (nonconsumers, &lt;86 mL/wk, 86-164 mL/wk, 165-359 mL/wk, &gt;359 mL/wk)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated diet history questionnaire assessing usual intake during previous year.</li> <li>• Baseline (1993)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report in ≥1 of 8 questionnaires (validated with drug reimbursement claims or by self-reported questionnaire of biomarker concentrations)</li> <li>• Every 2-3y (mean follow-up: 14y (1993-2007))</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Nonconsumers (n=53,538): REF                      &lt;86 mL/wk (n=4482): 1.28 (1.06, 1.55)                      86-164 mL/wk (n=2699): 1.12 (0.86, 1.45)                      165-359 mL/wk (n=2700): 1.22 (0.94, 1.57)                      &gt;359 mL/wk (n=2699): 1.30 (1.02, 1.66)                      P-trend=0.0206</p> <p><b>Stratified by BMI categories:</b>                      Nonconsumers (REF) vs &gt;359 mL/wk                      BMI &lt;25 kg/m<sup>2</sup> (n=53,695): 1.61 (1.12, 2.32)                      BMI between 25-30 (n=10,290): 1.22 (0.79, 1.88)                      BMI &gt;30 (n=2133): 0.94 (0.56, 1.57)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (years of education), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: hypertension, hypercholesterolemia, hormone replacement therapy, antidiabetic drugs, omega-3 fatty acid intake, carbohydrate intake, coffee, fruit and vegetables, processed meat intake, dietary pattern (Western or Mediterranean)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity</li> <li>• Exposure only assessed at baseline</li> <li>• High attrition rate/missing data with no evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Institut National du Cancer; Mutuelle Générale de l'Éducation Nationale; Institut de Cancérologie Gustave Roussy; Institut National de la Santé et de la Recherche Médicale; European Union InterAct project</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Gardener, 2018<sup>19</sup></b>  <b>PCS, NOMAS (Northern Manhattan Study), U.S.</b>                      Analytic N=2,019</p> <p><b>Study objective:</b> To examine the relation between diet soda and regular soda consumption with the risk of incident diabetes in a longitudinal multiethnic population-based cohort</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 69 (10y); ≥40y</li> <li>• Female: 64%</li> <li>• Race and/or ethnicity: 23% White; 22% Black; 2% Other; 53% Hispanic</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI 28 (5) kg/m<sup>2</sup></li> <li>• Physical activity: 10% moderate-heavy physical activity</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 17% current; 35% former; 48% never</li> <li>• Alcohol intake: 36% moderate alcohol use</li> <li>• TEI: 1569 (654) kcal/d</li> <li>• Beverage intake at baseline: 14% drink regular soda ≥1/d; 45% drink regular soda &lt;1/mo</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; stroke; &lt;40y; resided in Northern Manhattan &lt;3mo; no telephone in household; missing data; energy intake &lt;500 or &gt;4000 kcal/d</p>	<p><b>Exposure:</b> Sugar-sweetened soda (regular, not diet)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (number of sodas per day); categorical intake (&lt;1/mo, 1/mo to 6/wk, daily (≥1/d))</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report of diagnosis with diabetes or high blood sugar, or use of insulin or oral hypoglycemic medications; confirmed by medical record review.</li> <li>• 11 (5)y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)</p> <p><b>By number of regular sodas per day</b></p> <p>Adjusted for BMI: 1.15 (1.02, 1.30)</p> <p>Adjusted for waist-to-hip ratio: 1.16 (1.02, 1.31)</p> <p>Among those with BMI &lt;25: 1.55 (1.10, 2.19)</p> <p>Among those with BMI ≥25: 1.09 (0.95, 1.26)</p> <p>By number of regular and diet sodas per day</p> <p>Adjusted for BMI: 1.14 (1.03, 1.27)</p> <p>Adjusted for waist-to-hip ratio: 1.18 (1.06, 1.32)</p> <p><b>By categories of regular soda consumption</b></p> <p><b>Adjusted for BMI</b></p> <p>&lt;1/mo: REF</p> <p>1/mo to 6/wk: 1.10 (0.86, 1.40)</p> <p>Daily: 1.13 (0.81, 1.61)</p> <p><b>Adjusted for waist-to-hip ratio</b></p> <p>&lt;1/mo: REF</p> <p>1/mo to 6/wk: 1.06 (0.82, 1.35)</p> <p>Daily: 1.11 (0.78, 1.57)</p> <p><b>Among those with BMI &lt;25 kg/m<sup>2</sup></b></p> <p>&lt;1/mo: REF</p> <p>1/mo to 6/wk: 0.74 (0.37, 1.45)</p> <p>Daily: 1.09 (0.45, 2.61)</p> <p><b>Among those with BMI ≥25 kg/m<sup>2</sup></b></p> <p>&lt;1/mo: REF</p> <p>1/mo to 6/wk: 1.10 (0.84, 1.43)</p> <p>Daily: 1.09 (0.75, 1.61)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: Mediterranean diet, hypertension, hypercholesterolemia</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• High attrition rate/missing data with no evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH/NINDS</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Greenberg, 2005<sup>20</sup></b>  <b>PCS, NHEFS (NHANES-1 Epidemiologic Follow Up Study), U.S.</b>                      Analytic N=7,006</p> <p><b>Study objective:</b> To assess the effect of weight change on the relationship between coffee and tea consumption and diabetes risk.</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~47 (0.1)y (adults ≤60y); ~72 (0.2)y (adults &gt;60y); 32-88y</li> <li>• Female: 61%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: ~34 (out of 18 educational levels between 10 and 45); ~\$9000 per-capita income</li> <li>• Anthropometry: BMI ~26 (0.1) kg/m<sup>2</sup></li> <li>• Physical activity: ~3.9 (out of 5 physical activity levels)</li> <li>• Family history of diabetes: 7%</li> <li>• Smoking: 30% current</li> <li>• Alcohol intake: 39% ≥1 drink/d</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: 75% overall drink cola sodas; ≤60y: ~84%; &gt;60y: ~58%</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data</p>	<p><b>Exposure:</b> Cola sodas (caffeinated)</p> <ul style="list-style-type: none"> <li>• Serving Size: 12oz</li> </ul> <p><b>Comparator:</b> continuous intake (per two 12oz svg/d increase)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• 3 FFQ questions on number of cups consumed, time period in which they were consumed (d, wk, mo, y), and whether beverage was consumed seasonally (assuming a 6mo season)</li> <li>• Baseline (1982-1984)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported or documented death from diabetes</li> <li>• 8.4y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      All participants (32-88y): 1.21 (1.01, 1.45), P&lt;0.05                      ≤60y: 1.11 (0.87, 1.40), P=NS                      &gt;60y: 1.38 (1.03, 1.84), P&lt;0.05</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (per-capita income, educational level), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: American style diet (quintiles)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: family history of diabetes</li> <li>• Exposure subject to measurement error and only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NR</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Hirahatake, 2019<sup>22</sup></b>  <b>PCS, CARDIA (Coronary Artery Risk Development in Young Adults Study), U.S.</b>                      Analytic N=4,719</p> <p><b>Study objective:</b> To examine the association between ASB, SSB, and total sweetened beverage consumption and T2D risk in young adults.</p> <p><b>Participant characteristics at baseline:</b>                      Black and White adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 25 (3)y; 18-30y</li> <li>• Female: 55%</li> <li>• Race and/or ethnicity: 50% White; 50% Black</li> <li>• Socioeconomic position: ~15y education attained through follow-up</li> <li>• Anthropometry: BMI 24 (5) kg/m<sup>2</sup></li> <li>• Physical activity: ~200 min/wk of moderate-intensity activity</li> <li>• Family history of diabetes: 16%</li> <li>• Smoking: 29% current; 13% former; 58% never</li> <li>• Alcohol intake: ~12 mL/d</li> <li>• TEI: ~2700 kcal/d</li> <li>• Beverage intake at baseline: 45% drink SSB ≥1/d; mean intake: 1.38 (1.7) svg/d</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; loss to follow-up; energy intake &lt;600 kcal or &gt;6000 kcal/d for women, &lt;800 or &gt;8000 kcal/d for men</p>	<p><b>Exposure:</b> SSB (sugar-sweetened soft drinks and fruit drinks)</p> <ul style="list-style-type: none"> <li>• Serving Size: 8oz</li> </ul> <p><b>Comparator:</b> continuous intake (svg/d); categorical intake (none to &lt;1 svg/wk, 1 to &lt;4 svg/wk, 4 to &lt;7/wk, 1-2 svg/d, &gt;2 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated diet history questionnaire on diet during previous month. A cumulative average intake value was calculated for each participant based on dietary data collected at different time points.</li> <li>• Baseline, 7y and 20y follow-up</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Biomarkers</li> <li>• 25.3 (8.3)y (years 0, 7, 10, 15, 20, 25, 30)</li> </ul>	<p><b>T2D by cumulative average SSB, HR (95% CI)</b></p> <p>None to ≤1/wk: REF                      1 to ≤4/wk: 0.98 (0.73, 1.31)                      4 to ≤7/wk: 0.97 (0.71, 1.32)                      1-2/d: 0.96 (0.71, 1.29)                      ≥2/d: 1.27 (0.93, 1.74)</p> <p>Quintile 1: REF                      Quintile 2: 0.98 (0.73, 1.31)                      Quintile 3: 0.97 (0.71, 1.32)                      Quintile 4: 0.96 (0.71, 1.29)                      Quintile 5: 1.27 (0.93, 1.74)</p> <p>Per svg/d: 1.06 (1.01, 1.10), P=0.009</p> <p>Results were also consistent in the subset of population with data on family history of diabetes (data NR)</p> <p><b>Stratified analyses:</b>                      Men: 1.10 (1.03, 1.18)                      Women: 1.02 (0.97, 1.08)                      P-interaction (sex)=0.66</p> <p>Black: 1.05 (1.00, 1.10)                      White: 1.10 (1.02, 1.19)                      P-interaction (race)=0.014</p> <p>BMI ≤24.9 kg/m<sup>2</sup>: 1.08 (1.00, 1.17)                      BMI 25.0-29.9 kg/m<sup>2</sup>: 1.09 (1.02, 1.17)                      BMI ≥30 kg/m<sup>2</sup>: 1.04 (0.98, 1.11)                      P-interaction (BMI)=0.06</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (time-updated measures of education), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: CARDIA center, other beverage intake, dieting behavior, average alternate Mediterranean diet score</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NHLBI</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Huang, 2017<sup>23</sup></b>  <b>PCS, Women's Health Initiative, U.S.</b>                      Analytic N=64,850</p> <p><b>Study objective:</b> To evaluate the associations of ASB and SSB consumption with the risk of developing T2D and the potential benefit of replacing SSBs with ASBs or water.</p> <p><b>Participant characteristics at baseline:</b> postmenopausal women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~63y; 50-79y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: 87% Non-Hispanic White; 6% African American; 4% Other; 3% Hispanic/Latino</li> <li>• Socioeconomic position: 28% ≤high school, 27% some college or associate degree, 45% college or higher; 11% with annual family income &lt;\$20k, 38% with \$20k-&lt;\$50k, and 45% with ≥\$50k (6% missing income data); 2% no health insurance</li> <li>• Anthropometry: BMI ~27 (5) kg/m<sup>2</sup>; 36% with overweight, 24% with obesity</li> <li>• Physical activity: 19% &lt;1.8 MET hr/wk, 55% 1.8-20 MET hr/wk, 26% &gt;20 MET hr/wk</li> <li>• Family history of diabetes: 30%</li> <li>• Smoking: 4% current; 44% former; 52% never</li> <li>• Alcohol intake: ~5.7 g/d</li> <li>• TEI: ~2300 kcal/d</li> <li>• Beverage intake at baseline: SSB ~0.5 svg/d (31% &lt;1 svg/wk)</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; BMI &lt;18.5 kg/m<sup>2</sup>, energy intake &lt;600 or &gt;5000 kcal/d</p>	<p><b>Exposure:</b> SSB (regular/non-diet soda and soft drinks, fruit juice, and fruit drinks); fruit juice included "orange juice and grapefruit juice", and "other fruit juices such as apple and grape"; fruit drinks included "Tang, Kool-Aid, Hi-C, and other fruit drinks"</p> <ul style="list-style-type: none"> <li>• Serving Size: 355mL (one 12oz can)</li> </ul> <p><b>Comparator:</b> continuous intake (every 12oz increase); categorical intake (&lt;1 svg/wk, 1 to &lt;7 svg/wk, 1 to &lt;2 svg/d, ≥2 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ on intake during the past 3 months.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report: answered yes when asked if "a doctor prescribed for the first time any of the following pills or treatments: pills for diabetes or insulin shots for diabetes"; validated by medical record review and laboratory data</li> <li>• 8.4y (annually)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Per 12oz increase in SSB: 1.13 (1.07, 1.20)</p> <p><b>Total SSB</b>                      &lt;1 svg/wk: REF                      1 to &lt;7 svg/wk: 1.05 (0.98, 1.12)                      1 to &lt;2 svg/d: 1.09 (0.97, 1.23)                      ≥2 svg/d: 1.43 (1.17, 1.75)                      P-trend=0.0004</p> <p><b>Regular soda</b>                      &lt;1 svg/wk: REF                      1 svg/wk to &lt;1 svg/d: 1.12 (1.02, 1.22)                      ≥1 svg/d: 1.12 (0.97, 1.29)</p> <p><b>Fruit drinks</b>                      &lt;1 svg/wk: REF                      1 svg/wk to &lt;1 svg/d: 0.99 (0.85, 1.15)                      ≥1 svg/d: 1.33 (0.89, 1.98)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (family income; education; insurance status), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: systolic blood pressure, antihypertensive use, antihyperlipidemic use, hormone replacement therapy use, other beverage consumption, glycemic load and index, AHEI, cardiovascular history, hysterectomy history, sitting time</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Exposure only assessed at baseline</li> <li>• High attrition rate/missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NHLBI</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Imamura, 2019<sup>24</sup></b>  <b>PCS, EPIC-InterAct, 8 European countries (France, Italy, Spain, UK, The Netherlands, Germany, Sweden, and Denmark)</b>                      Analytic N=27,662 (subcohort = 16,103)</p> <p><b>Study objective:</b> To estimate the risk of T2D when consumption of SSB was replaced with consumption of fruit juice, milk, coffee, or tea.</p> <p><b>Participant characteristics at baseline:</b>                      adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~54y</li> <li>• Female: 58%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 18% &gt;high school education; 50% currently employed</li> <li>• Anthropometry: BMI ~28 kg/m<sup>2</sup></li> <li>• Physical activity: 42% ≥moderately active</li> <li>• Family history of diabetes: 30%</li> <li>• Smoking: 28% current</li> <li>• Alcohol intake: ~210 mL/d</li> <li>• TEI: ~2150 (715) kcal/d</li> <li>• Beverage intake at baseline: 55 (105) g/d of SSB; 6% consume ≥250 g/day of SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data</p>	<p><b>Exposure:</b> SSB (carbonated/soft/isotonic drinks, diluted syrups, sweetened or unspecified, containing &gt;2g carbohydrates per 100g, and sweetened milk beverages); artificially-sweetened beverages were not separated out in some centers, and sweetened milk beverages were assessed in limited regions</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (per 250 g/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ or dietary questionnaire (country-specific) assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Combination of self-report, linkage to registries, hospital/mortality data</li> <li>• Median 6y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Per 250 g/d of SSB: 1.18 (1.08, 1.28)                      Not including sweetened milk beverages: 1.16 (1.07, 1.27)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (education, marital status), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: research center, hormone replacement therapy, menopausal status, history of oral contraceptive use, hypertension, dyslipidemia, coronary heart disease, stroke, dietary supplement use, dietary consumption (vegetables, fruit, nuts, cheese, yogurt, red meats, processed meats, fish, confectionary, cereals, other beverages)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity</li> <li>• Exposure subject to measurement error and only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      EU FP6 Programme</p>



Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Jahromi, 2023<sup>26</sup></b>  <b>PCS, Tehran Lipid and Glucose Study, Iran</b>                      Analytic N=2,081</p> <p><b>Study objective:</b> To assess the association of dietary diabetes risk reduction scores (DDRRS) with T2D risk in Iranian adults</p> <p><b>Participant characteristics at baseline:</b>                      Iranian adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 50.4 (8.2)y; ≥40y</li> <li>• Female: 53%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 19% academic education; 84% employed</li> <li>• Anthropometry: BMI 28.3 (4.3) kg/m<sup>2</sup></li> <li>• Physical activity: ~67 MET hr/wk</li> <li>• Family history of diabetes: 10%</li> <li>• Smoking: 12% current</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2300 kcal/d</li> <li>• Beverage intake at baseline: Mean: ~1.2 svg/wk of SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; &lt;40y; missing data; loss to follow-up; energy intakes &lt;800 or &gt;4200 kcal/d or those on specific diets; history of cancer or CVD; pregnant or lactating</p>	<p><b>Exposure:</b> Sugar-sweetened beverages</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (tertiles in svg/wk)</p> <ul style="list-style-type: none"> <li>• Tertile 1: assigned a score of 4 points in DDRRS (highest intake)</li> <li>• Tertile 3: assigned a score of 1 point in DDRRS</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year. Participants were categorized into quartiles based on their intake ranking. For SSB, participants in the lowest quartile were assigned a score of 4 points and those in the highest quartile were assigned 1 point.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Biomarkers (FPG ≥126 mg/dL or 2h post-75g glucose load ≥200 mg/dL) or medication</li> <li>• 6y</li> </ul>	<p><b>T2D by baseline intake</b>, OR (95% CI)                      Tertile 1 (highest intake): REF                      Tertile 2: 0.74 (0.53, 1.03)                      Tertile 3: 0.49 (0.32, 0.76)                      P-trend=0.002</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (educational level; occupation status; marital status), anthropometry, physical activity, smoking</li> <li>• Other: baseline FPG</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, family history of diabetes, alcohol intake</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Shahid Beheshti University of Medical Sciences</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>McNaughton, 2008<sup>28</sup></b>  <b>PCS, Whitehall-II, UK</b>                      Analytic N=6,699</p> <p><b>Study objective:</b> To identify a dietary pattern associated with insulin resistance and investigate whether this pattern was prospectively associated with T2D</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 49.5 (0.1)y; 39-63y</li> <li>• Female: 30%</li> <li>• Race and/or ethnicity: 90% Caucasian</li> <li>• Socioeconomic position: 16% low employment grade (out of 6 levels)</li> <li>• Anthropometry: BMI 25.3 (0.0) kg/m<sup>2</sup></li> <li>• Physical activity: vigorous activity 1.1 (0.0) times/wk</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 14% current</li> <li>• Alcohol intake: 12.1 (0.2) g/d</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: ~58 g/d SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; loss to follow-up</p>	<p><b>Exposure:</b> SSB dietary patterns factor identified from RRR</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (based on quartiles of overall dietary pattern score)                      SSB intake (g/d), Mean (SE):</p> <ul style="list-style-type: none"> <li>• Quartile 1: 37.3 (1.6)</li> <li>• Quartile 2: 49.7 (1.8)</li> <li>• Quartile 3: 65.5 (2.2)</li> <li>• Quartile 4: 112.8 (4.1)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline (1993)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report based on doctor's diagnosis or diabetic medication; confirmed with 2hr OGTT</li> <li>• 11.6y (Phases 5 (1996-1997) and 7 (2004))</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      By quartiles of SSB intake: Data not shown                      P-trend=NS</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (employment grade), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: blood pressure, energy misreporting (ratio of total energy intake to energy expenditure, 1 or &lt;1)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: family history of diabetes</li> <li>• Exposure subject to measurement error and only assessed at baseline</li> <li>• High attrition rate/missing data with no evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      UK Medical Research Council; British Heart Foundation; Health and Safety Executive (UK); Department of Health; NHLBI; NIA; Agency for Health Care Policy Research; MacArthur Foundation Research Network on Socio-economic Status and Health</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Montonen, 2007<sup>29</sup></b>  <b>PCS, Finnish Mobile Clinic Health Examination, Finland</b>                      Analytic N=2360 (soft drinks); 4304 (sweetened berry juice)</p> <p><b>Study objective:</b> To examine the association between intakes of different sugars and the incidence of T2D in a large nationwide cohort of Finns</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~53y; 40-69y</li> <li>• Female: 47%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI ~27 kg/m<sup>2</sup></li> <li>• Physical activity: 58% physically active</li> <li>• Family history of diabetes: 20%</li> <li>• Smoking: 33% current</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~2500 kcal/d</li> <li>• Beverage intake at baseline: ~66 g/d soft drinks; ~23 g/d of sweetened berry juice</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; &lt;40 or &gt;69y; energy intake &lt;800 or &gt;6000 kcal/d</p>	<p><b>Exposure:</b> Soft drinks; Sweetened berry juice</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (quartiles)                      Median intake for quartiles 1, 2, 3, 4</p> <ul style="list-style-type: none"> <li>• Soft drinks: 0, 1, 13, 143 g/d</li> <li>• Sweetened berry juice: 0, 7.5, 21, 51 g/d</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Diet history interview assessing usual diet during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• National registries</li> <li>• 12y</li> </ul>	<p><b>T2D by baseline intake</b>, RR (95% CI)</p> <p><b>Soft drink intake</b></p> <p>Quartile 1 (n=741): REF                      Quartile 2 (n=458): 0.85 (0.42, 1.73)                      Quartile 3 (n=573): 0.80 (0.43, 1.49)                      Quartile 4 (n=588): 1.60 (0.93, 2.76)                      P-trend=0.01</p> <p><b>Sweetened berry juice intake</b></p> <p>Quartile 1 (n=1665): REF                      Quartile 2 (n=726): 0.68 (0.41, 1.14)                      Quartile 3 (n=802): 0.95 (0.60, 1.49)                      Quartile 4 (n=1091): 1.56 (1.08, 2.26)                      P-trend=0.006</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes, smoking</li> <li>• Other: geographic area, prudent dietary score, conservative pattern score, serum cholesterol, blood pressure; history of infarction, angina pectoris, and cardiac failure</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position, alcohol intake</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NR</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Nettleton, 2009<sup>30</sup></b>  <b>PCS, MESA (Multi-Ethnic Study of Atherosclerosis), U.S.</b>                      Analytic N=5,011</p> <p><b>Study objective:</b> To determine associations between diet soda consumption and risk of incident metabolic syndrome, its components, and T2D</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~61y; 45-84y</li> <li>• Female: 53%</li> <li>• Race and/or ethnicity: 43% White; 23% African American; 12% Chinese; 21% Hispanic</li> <li>• Socioeconomic position: 85% high school degree</li> <li>• Anthropometry: BMI 27.9 kg/m<sup>2</sup></li> <li>• Physical activity: ~2400 MET-min/wk</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 14% current</li> <li>• Alcohol intake: NR</li> <li>• TEI: ~1700 kcal/d</li> <li>• Beverage intake at baseline: 14% drink SSB ≥1 svg/d; 45% never drink SSB</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; insufficient or implausible dietary information</p>	<p><b>Exposure:</b> Sugar-sweetened soda (“regular soft drinks, soda, sweetened mineral water (not diet), nonalcoholic beer”)</p> <ul style="list-style-type: none"> <li>• Serving Size: small, medium, or large (weighted as intake frequency x 0.5, x 1.0, and x 1.5 for small, medium, and large, respectively)</li> </ul> <p><b>Comparator:</b> categorical intake (Rare/never, &gt;Rare/never but &lt;1 svg/wk, ≥1 svg/wk to &lt;1 svg/d, ≥1 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report, fasting glucose ≥126 mg/dl at any exam, or use of antidiabetes medication</li> <li>• 5.8y (baseline at 2000-2002 and 3 follow-up exams conducted from 2002-2003, 2004-2005, and 2005-2007)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Rare/never: REF                      &gt;Rare/never but &lt;1 svg/wk: Data NR                      ≥1 svg/wk to &lt;1 svg/d: Data NR                      ≥1 svg/d: Data NR                      P-trend=NS</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (education), anthropometry, physical activity, smoking</li> <li>• Other: dietary supplement use</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: family history of diabetes, alcohol intake</li> <li>• Exposure only assessed at baseline</li> <li>• High attrition rate/missing data with no evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NHLBI</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>O'Connor, 2016<sup>31</sup></b>  <b>PCS, EPIC-Norfolk, UK</b>                      Analytic N=24,653</p> <p><b>Study objective:</b> To evaluate the association of types of SSB, ASB, and fruit juice with incident T2D</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 58.7 (9.3)y; 40-79</li> <li>• Female: 55%</li> <li>• Race and/or ethnicity: Primarily White European</li> <li>• Socioeconomic position: 13% university degree; 44% professional or managerial social class</li> <li>• Anthropometry: BMI 26.3 (3.9) kg/m<sup>2</sup>; 45% with overweight, 15% with obesity</li> <li>• Physical activity: 18% active (based on 4-level index)</li> <li>• Family history of diabetes: 13%</li> <li>• Smoking: 12% current</li> <li>• Alcohol intake: 3.5 (1-10) units/wk</li> <li>• TEI: 1950 (508) kcal/d</li> <li>• Beverage intake at baseline: 30% SSB consumers, 83 (41-176) g/d</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes or unconfirmed status; missing data on diet or covariates; extreme energy intake (top and bottom 1%)</p>	<p><b>Exposure:</b> Sugar-sweetened soft drinks (soft drinks, sports/energy drinks, and squashes sweetened with sugar, and juice-based beverages)</p> <ul style="list-style-type: none"> <li>• Serving Size: 336 g (12oz)</li> </ul> <p><b>Comparator:</b> continuous intake (per 336 g svg); categorical intake (non-consumers and consumers in tertiles)</p> <p>Tertiles (range and median intake, g/d)</p> <ul style="list-style-type: none"> <li>• Tertile 1: &gt;0-49 (35)</li> <li>• Tertile 2: 50-139 (83)</li> <li>• Tertile 3: 140-3172 (234)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• 7d food diary</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report; verified using data internal and external to study through record linkage</li> <li>• 10.8y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)</p> <p>Non-consumers: REF</p> <p>Tertile 1: 0.97 (0.80, 1.18)</p> <p>Tertile 2: 0.98 (0.80, 1.19)</p> <p>Tertile 3: 1.13 (0.94, 1.36)</p> <p>P-trend=0.306</p> <p><b>Per svg (336 g):</b> 1.14 (1.01, 1.32)</p> <p><b>Subgroup analyses:</b></p> <p>P-interaction (age)≥0.483</p> <p>P-interaction (sex)≥0.090</p> <p>P-interaction (BMI)≥0.424</p> <p>P-interaction (waist)≥0.182</p> <p>P-interaction (physical activity)≥0.099</p> <p>P-interaction (smoking)≥0.274</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (occupational social class; educational level), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: season (winter/summer), intake of other sweet beverages, fiber intake, saturated fat intake</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity</li> <li>• Exposure only assessed at baseline</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Medical Research Council UK;                      Cancer Research UK</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Odegaard, 2010<sup>32</sup></b>  <b>PCS, Singapore Chinese Health study, Singapore</b>                      Analytic N=43,580</p> <p><b>Study objective:</b> To investigate the nature of the association between consumption of soft drinks and juices and the risk of incident T2D</p> <p><b>Participant characteristics at baseline:</b>                      Chinese Singaporean adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 54.8 (7.5)y; 45-74y</li> <li>• Female: 57%</li> <li>• Race and/or ethnicity: All Chinese</li> <li>• Socioeconomic position: 34% secondary education</li> <li>• Anthropometry: BMI ~23 kg/m<sup>2</sup></li> <li>• Physical activity: ~50 min/wk moderate activity</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 27% ever</li> <li>• Alcohol intake: ~1 drink/wk</li> <li>• TEI: ~1600 kcal/d</li> <li>• Beverage intake at baseline: 74% never; 11% ≥2x/wk</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes or unclear status; cancer, heart disease, stroke; energy intakes &lt;600 or &gt;5000 kcal/d; loss to follow-up; not Hokkien or Cantonese dialect groups</p>	<p><b>Exposure:</b> Soft drinks, such as Coca Cola and 7UP</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 glass = 237 mL (~1 cup)</li> </ul> <p><b>Comparator:</b> categorical intake (almost never; 1-3x/mo; 1x/wk; ≥2x/wk)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report of physician-diagnosed diabetes; validated with records</li> <li>• 5.7y</li> </ul>	<p><b>T2D by baseline intake</b>, RR (95% CI)                      Almost never (n=32,060): REF                      1-3x/mo (n=4514): 1.11 (0.97, 1.26)                      1x/wk (n=2389): 0.98 (0.81, 1.29)                      ≥2x/wk (n=4617): 1.34 (1.17, 1.52)                      P-trend&lt;0.0001</p> <p>NS effect modification by sex, age, or BMI</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (educational level), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: year of interview, saturated fat, dietary fiber, dairy, juice, coffee</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Olsson, 2021<sup>33</sup></b>  <b>PCS, Malmö Diet and Cancer, Sweden</b>                      Analytic N=26,622</p> <p><b>Study objective:</b> To analyze associations between intakes of 6 types of carbohydrates and thirteen carbohydrate-rich foods with incident T2D</p> <p><b>Participant characteristics at baseline:</b> middle-aged and older adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~58y</li> <li>• Female: 61%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 14% university degree</li> <li>• Anthropometry: BMI ~26 kg/m<sup>2</sup></li> <li>• Physical activity: 10% with &lt;7.5 METhr/wk of leisure-time physical activity</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 28% current</li> <li>• Alcohol intake: 6% zero consumers</li> <li>• TEI: ~2645 kcal/d in men, ~2020 kcal/d in women</li> <li>• Beverage intake at baseline: SSB ~96 g/d in men, ~71 g/d in women</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; limited Swedish proficiency and mental disability</p>	<p><b>Exposure:</b> SSB (all carbonated and non-carbonated beverages; except juices, dairy products, and alcohol beverages)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (non-consumers vs tertiles)</p> <ul style="list-style-type: none"> <li>• Non-consumers (n=12,066)</li> <li>• Tertile 1 (n=5103): 0.3-47.1 g/d</li> <li>• Tertile 2 (n=4596): 47.3-142.8 g/d</li> <li>• Tertile 3 (n=4857): 142.9-3000.0 g/d</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated, modified diet history (7d food diary, FFQ assessing usual intake during previous year, and 60-min interview)</li> <li>• Baseline</li> </ul> <p>Outcome assessment methods and timing:</p> <ul style="list-style-type: none"> <li>• National registries; rescreening of cohort</li> <li>• 18.4 (6.4)y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)  <b>By g/d of SSB:</b>  <b>All Participants</b>                      Non-consumers: REF                      Tertile 1: 1.02 (0.94, 1.12)                      Tertile 2: 1.05 (0.96, 1.15)                      Tertile 3: 1.05 (0.96, 1.14)                      P-trend=0.23                      P-interaction (sex)=0.35</p> <p><b>Men</b>                      Non-consumers: REF                      Tertile 1: 0.97 (0.86, 1.11)                      Tertile 2: 1.00 (0.88, 1.14)                      Tertile 3: 1.00 (0.89, 1.13)                      P-trend=0.92</p> <p><b>Women</b>                      Non-consumers: REF                      Tertile 1: 1.04 (0.92, 1.17)                      Tertile 2: 1.09 (0.97, 1.23)                      Tertile 3: 1.06 (0.94, 1.20)                      P-trend=0.20</p> <p><b>By g/1000 kcal of SSB:</b>  <b>All Participants</b>                      Non-consumers: REF                      Tertile 1: 1.02 (0.93, 1.11)                      Tertile 2: 1.06 (0.97, 1.16)                      Tertile 3: 1.04 (0.95, 1.13)                      P-trend=0.24                      P-interaction (sex)=0.59</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (education), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: diet method version, season of diet collection, coffee, meat, whole grains</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Swedish Research Council; Swedish Heart-Lung Foundation; Albert Pålsson Foundation; Swedish Foundation for Strategic Research</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Palmer, 2008<sup>34</sup></b>  <b>PCS, Black Women's Health Study, U.S.</b>                      Analytic N=43,960</p> <p><b>Study objective:</b> To examine the association between consumption of sugar-sweetened beverages, weight gain, and incidence of T2D in African American women</p> <p><b>Participant characteristics at baseline:</b>                      African American women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~38 (10)y; 21-69y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: 100% African American</li> <li>• Socioeconomic position: 15% education ≤12y</li> <li>• Anthropometry: BMI ~28 (7) kg/m<sup>2</sup></li> <li>• Physical activity: 50% with ≥1 hr/wk</li> <li>• Family history of diabetes: 34%</li> <li>• Smoking: 16% current</li> <li>• Alcohol intake: ~1.4 (4) drinks/wk</li> <li>• TEI: ~1700 (650) kcal/d</li> <li>• Beverage intake at baseline: 17% drank ≥1/d sugar-sweetened soft drink; 32% drank ≥1/d sweetened fruit drink</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; GDM, myocardial infarction, stroke, or cancer; pregnant; &lt;30y at the end of follow-up; missing data; energy intake &lt;500 or &gt;3800 kcal/d</p>	<p><b>Exposure:</b> Sugar-sweetened soft drinks ("regular soft drinks, not diet soda"); sweetened fruit drinks ("fortified fruit drinks, Kool-Aid, and fruit juices other than orange or grapefruit juice")</p> <ul style="list-style-type: none"> <li>• Serving Size: Soft drinks: 12 oz (336 g)                      Fruit drinks: 6 oz (168 g)</li> </ul> <p><b>Comparator:</b> categorical intake (&lt;1 drink/mo, 1-7 drinks/mo, 2-6 drinks/wk, 1 drink/d, ≥2 drinks/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ</li> <li>• Baseline (1995), 6y follow-up (2001) - only baseline used in analyses</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report via biennial questionnaire</li> <li>• 10y</li> </ul>	<p><b>T2D by baseline intake, HR (95% CI)</b></p> <p><b>Sugar-sweetened soft drinks</b>                      &lt;1 drink/mo: REF                      1-7 drinks/mo: 0.89 (0.80, 0.99)                      2-6 drinks/wk: 1.00 (0.89, 1.12)                      1 drink/d: 1.11 (0.96, 1.28)                      ≥2 drinks/d: 1.24 (1.06, 1.45); 1.05 (0.90, 1.23) when adjusting for BMI, 1.04 when adjusting for TEI                      P-trend=0.002</p> <p><b>Sweetened fruit drinks</b>                      &lt;1 drink/mo: REF                      1-7 drinks/mo: 1.08 (0.96, 1.22)                      2-6 drinks/wk: 1.08 (0.96, 1.21)                      1 drink/d: 1.17 (1.02, 1.33)                      ≥2 drinks/d: 1.31 (1.13, 1.52); 1.33 (1.15, 1.54) when adjusting for BMI, 1.32 when adjusting for TEI                      P-trend=0.001</p> <p>Also provided analyses stratified by age (&lt;40y and ≥40y), BMI (&lt;25, 25-29, and ≥30 kg/m<sup>2</sup>), and family history of diabetes - weak positive association between highest category of sweetened fruit drink consumption and risk of T2D across subgroups for each factor except BMI &lt;25 kg/m<sup>2</sup></p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (years of education), anthropometry, physical activity, family history of diabetes, smoking</li> <li>• Other: diet (red meat, processed meat, cereal fiber, other beverages), glycemic index, questionnaire cycle</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: alcohol intake</li> <li>• Analyses based on exposure collected only at baseline</li> <li>• High attrition rate/missing data with no evidence whether the result was biased due to missing data</li> <li>• Outcome was self-reported</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NCI; NIDDK</p>



Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Pan, 2012<sup>35</sup></b>  <b>PCS, NHS-II (Nurses' Health Study II), U.S.</b>                      Analytic N=82,902</p> <p><b>Study objective:</b> To evaluate the relation of plain-water intake and the substitution of plain water for SSB and fruit juice with incident T2D in U.S. women</p> <p><b>Participant characteristics at baseline:</b>                      women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 36.0 (4.7)y; 26-45y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: Primarily European ancestry</li> <li>• Socioeconomic position: All nurses</li> <li>• Anthropometry: BMI ~24 (5) kg/m<sup>2</sup></li> <li>• Physical activity: ~19 METs/wk</li> <li>• Family history of diabetes: 33%</li> <li>• Smoking: 12% current</li> <li>• Alcohol intake: ~3 g/d</li> <li>• TEI: ~1800 kcal/d</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; implausible total energy intake (&lt;500 or &gt;3500 kcal/d)</p>	<p><b>Exposure:</b> SSB (Coke, Pepsi, or other cola with sugar; other carbonated beverages with sugar; and Hawaiian Punch, lemonade, or other noncarbonated fruit drinks)</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 cup = 240mL</li> </ul> <p><b>Comparator:</b> continuous intake (svg/d) categorical intake (≤1 cup/wk, 2-4 cups/wk, 5-7 cups/wk, 2-3 cups/d, ≥4 cups/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• 1991, 1995, 1999, 2003 (cumulative average intake)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• Up to 2009</li> </ul>	<p><b>T2D by cumulative average intake</b>, RR (95% CI)</p> <p>≤1 cup/wk: REF</p> <p>2-4 cups/wk: 1.10 (0.99, 1.22)</p> <p>5-7 cups/wk: 1.22 (1.10, 1.36)</p> <p>2-3 cups/d: 1.34 (1.19, 1.51)</p> <p>≥4 cups/d: 1.29 (1.07, 1.54)</p> <p>P-trend&lt;0.001</p> <p><b>Per 1 svg/d:</b> 1.09 (1.05, 1.14)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, race and/or ethnicity, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: menopausal status and hormone use, oral contraceptive use, AHEI</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Paynter, 2006<sup>36</sup></b>  <b>PCS, ARIC (Atherosclerosis Risk in Communities Study), U.S.</b>                      Analytic N=12,204</p> <p><b>Study objective:</b> To examine the association between coffee and sweetened beverage consumption in a US community-based cohort of middle-aged Black and White men and women</p> <p><b>Participant characteristics at baseline:</b>                      Black and White adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 54y; 45-64y</li> <li>• Female: 56%</li> <li>• Race and/or ethnicity: 78% White; 22% Black</li> <li>• Socioeconomic position: 20% less than high school education</li> <li>• Anthropometry: BMI 27.2 kg/m<sup>2</sup>; 24% with obesity</li> <li>• Physical activity: ~2.4 leisure activity index (out of 5, with 1 indicating least active)</li> <li>• Family history of diabetes: 24% Women; 21% Men</li> <li>• Smoking: Women: 24% current, 53% never; Men: 26% current, 29% never</li> <li>• Alcohol intake: 59% consumers</li> <li>• TEI: ~1750 kcal/d (men), ~1500 kcal/d (women)</li> <li>• Beverage intake at baseline: 15% drink ≥2 cups/d</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; reported ethnicity other than Black or White; missing data; loss to follow-up</p>	<p><b>Exposure:</b> Sweetened beverages (fruit punch, non-diet soda, and orange or grapefruit juice)</p> <ul style="list-style-type: none"> <li>• Serving Size: 1 cup = 0.24 L</li> </ul> <p><b>Comparator:</b> categorical intake (&lt;1, 1, 1.1-1.9, ≥2 cups/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Combination of self-report of physician-diagnosed diabetes, diabetes treatment, and a fasting or nonfasting blood glucose test</li> <li>• Maximum 9y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)</p> <p><b>Men</b></p> <p>&lt;1 cup/d: REF                      1 cup/d: 1.03 (0.79, 1.34)                      1.1-1.9 cup/d: 0.95 (0.79, 1.15)                      ≥2.0 cup/d: 1.03 (0.82, 1.28)                      P-trend=0.94</p> <p><b>Women</b></p> <p>&lt;1 cup/d: REF                      1 cup/d: 1.13 (0.91, 1.42)                      1.1-1.9 cup/d: 1.10 (0.91, 1.33)                      ≥2.0 cup/d: 1.01 (0.79, 1.29)                      P-trend=0.58</p> <p>Stratification by age, BMI, or physical activity did not affect results, nor did removing juice from definition of sweetened beverages (data NR)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (education), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: dietary fiber, hypertension</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Exposure only assessed at baseline</li> <li>• High attrition rate/missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NHLBI</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Ramne, 2020<sup>37</sup></b>  <b>PCS, Malmö Diet and Cancer-Cardiovascular Cohort (MDC-CC), Sweden</b>                      Analytic N=4,382</p> <p><b>Study objective:</b> To examine how added sugar and SSB intake associate with 136 measured plasma proteins and C-reactive protein, and examine if the identified added sugar- and SSB-associated proteins associate with T2D incidence.</p> <p><b>Participant characteristics at baseline:</b>                      middle-aged and older adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 57y; 45-68y</li> <li>• Female: 62%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 12% university degree</li> <li>• Anthropometry: BMI 25.5 kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 26% current</li> <li>• Alcohol intake: 16% highest quintile of consumers</li> <li>• TEI: ~2300 kcal/d</li> <li>• Beverage intake at baseline: 46% reported no SSB intake on 7d food record; median contribution of SSB to added sugar intake: &lt;1%</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes or cardiovascular disease; missing data</p>	<p><b>Exposure:</b> SSB as a percentage of energy intake (which was estimated assuming SSB have a mean sugar content of 10g per 100g and the sugar content in soft drinks varies between 10-13g per 100g and cordial/squash contains ~8g per 100g)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (0%, &gt;0-2%, &gt;2-3%, &gt;3-5%, &gt;5% of energy intake)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated, modified diet history (7d food diary, FFQ assessing usual intake during previous year, and 60-min interview)</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• National registries</li> <li>• NR</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      By SSB intake as a % of energy intake</p> <p>0% (n=2039): REF</p> <p>&gt;0-2% (n=1471): 1.10 (0.93, 1.29)</p> <p>&gt;2-3% (n=310): 1.06 (0.79, 1.42)</p> <p>&gt;3-5% (n=307): 1.05 (0.78, 1.40)</p> <p>&gt;5% (n=255): 1.19 (0.88, 1.60)</p> <p>HR-trend: 1.03 (0.97, 1.10)</p> <p>P-trend=0.28</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (education), physical activity, smoking, alcohol intake</li> <li>• Other: season, screening date</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, anthropometry, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No information on start of follow-up</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Swedish Research Council;                      Heart and Lung Foundation;                      Albert Pahlsson Foundation</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Rhee, 2015<sup>38</sup></b>  <b>PCS, NHS (Nurses' Health Study) and NHS-II, U.S.</b>                      Analytic N=162,416</p> <p><b>Study objective:</b> To evaluate racial and ethnic differences in the association between a dietary diabetes risk reduction score and incidence of type 2 diabetes in U.S. white and minority women.</p> <p><b>Participant characteristics at baseline:</b> women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 40 (7)y; 34-59y (NHS-I); 27-44y (NHS-II)</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: 96% White (including non-Hispanic White); 1.4% Black; 1.2% Asian; 1.3% Hispanic</li> <li>• Socioeconomic position: All nurses</li> <li>• Anthropometry: BMI kg/m<sup>2</sup>, White: 24.4 (5), Black: 26.5 (6), Asian: 22.6 (4), Hispanic: 24.8 (5)</li> <li>• Physical activity: MET-hr/wk, White: 17 (24), Black: 16 (25), Asian: 17 (27), Hispanic: 20 (31)</li> <li>• Family history of diabetes: 31% White, 45% Black, 40% Asian, 43% Hispanic</li> <li>• Smoking: current: 19% White, 18% Black, 8% Asian, 11% Hispanic</li> <li>• Alcohol intake: 4.6 (8.4) g/d in White women (1.6-3.6 g/d in other racial and ethnic groups, P&lt;0.05 compared to White)</li> <li>• TEI: ~1700 kcal/d</li> <li>• Beverage intake at baseline: SSB 0.39 (0.75) svg/d in White women (0.45-0.68 svg/d in other racial/ethnic groups, p&lt;0.05 compared to White)</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; loss to follow-up; energy intake &lt;500 or &gt;3500 kcal/d</p>	<p><b>Exposure:</b> SSB (Coke, Pepsi, or other cola with sugar; other carbonated beverages with sugar; and Hawaiian Punch, lemonade, or other noncarbonated fruit drinks)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (increase of 1 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline (1980 in NHS, 1991 in NHS-II) and every 4y for up to 18y (NHS II) or 28y (NHS)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• Maximum 18y (up to 2009 in NHS-II) and 28y (up to 2008 in NHS)</li> </ul>	<p><b>T2D by cumulative average intake over follow up.</b> HR (95% CI)  <b>Per 1 svg/d increase in SSB</b></p> <p><b>Non-Hispanic White women (n=156,030)</b>                      NHS: 1.42 (1.28, 1.57)                      NHS-II: 1.15 (1.07, 1.25)                      Pooled: 1.25 (1.17, 1.33)</p> <p><b>Minority (Asian, Hispanic, and Black) women (n=6386)</b>                      NHS: 1.51 (1.01, 2.27)                      NHS-II: 1.01 (0.73, 1.24)                      Pooled: 1.16 (1.08, 1.25)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, anthropometry (BMI in sensitivity analysis; association remained positive and significant, data NR), physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: postmenopausal status and menopausal hormone use, oral contraceptive use (NHS-II), modified dietary diabetes risk reduction score</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Romaguera, 2013<sup>25</sup></b>  <b>PCS, EPIC-InterAct, 5 European countries (France, UK, The Netherlands, Germany, and Denmark)</b>                      Analytic N=26,328 (subcohort = 15,374)</p> <p><b>Study objective:</b> To evaluate the association of consumption of sweet beverages (juices and nectars, sugar-sweetened soft drinks and artificially sweetened soft drinks) with T2D incidence in European adults.</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~52 (9)y</li> <li>• Female: 62%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 21% university education</li> <li>• Anthropometry: BMI ~26 (4) kg/m<sup>2</sup></li> <li>• Physical activity: 20% highest activity level (out of 4 levels)</li> <li>• Family history of diabetes: ~19% (NR in Italy, Spain, Germany, and UK)</li> <li>• Smoking: 26% current</li> <li>• Alcohol intake: ~2 g/d</li> <li>• TEI: ~2220 (645) kcal/d</li> <li>• Beverage intake at baseline: 8% ≥1 glass/d of soft drinks, 24% &gt;1-6 glasses/wk</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; missing data; lowest and highest 1% for ratio of total energy intake to energy requirement; Italy, Spain, and Umeå (Sweden) where information on type of soft drink consumption was not collected</p>	<p><b>Exposure:</b> Sugar-sweetened soft drinks</p> <ul style="list-style-type: none"> <li>• Serving Size: 336 g (12oz) in continuous analyses; 1 glass svg = 250 g (~8.8oz) in categorical analyses</li> </ul> <p><b>Comparator:</b> continuous intake (per 336 g svg); categorical intake (&lt;1 glass/mo, 1-4 glasses/mo, &gt;1-6 glasses/wk, ≥1 glass/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ or dietary questionnaire (country-specific) assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Combination of self-report, linkage to registries, hospital/mortality data</li> <li>• ~16y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      By baseline sugar-sweetened soft drink intake                      &lt;1 glass/mo: REF                      1-4 glasses/mo: 1.19 (0.91, 1.56)                      &gt;1-6 glasses/wk: 1.07 (0.94, 1.21)                      ≥1 glass/d: 1.29 (1.02, 1.63)                      P-trend=0.013</p> <p><b>Per svg (336 g):</b> 1.18 (1.06, 1.32)</p> <p><b>Subgroup analyses:</b>                      &lt;55y: 1.20 (0.94, 1.52)                      ≥55y: 1.37 (1.07, 1.76)                      P-interaction=0.75</p> <p>Men: 1.19 (1.03, 1.39)                      Women: 1.29 (1.10, 1.51)                      P-interaction=0.063</p> <p>BMI &lt;25 kg/m<sup>2</sup>: 1.30 (1.03, 1.66)                      BMI 25-29.9 kg/m<sup>2</sup>: 1.17 (0.96, 1.42)                      BMI ≥30 kg/m<sup>2</sup>: 1.28 (1.02, 1.61)                      P-interaction=0.22</p> <p>Low physical activity: 1.25 (1.04, 1.49)                      High physical activity: 1.24 (1.07, 1.43)                      P-interaction=0.73</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (educational level), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: other sweet beverages</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      EU FP6 Programme</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Rose, 2023<sup>39</sup></b>  <b>NCC, NHS (Nurses' Health Study), U.S.</b>                      Analytic N=2,814</p> <p><b>Study objective:</b> To elucidate potential dietary determinants of T2D risk by defining a model that describes habitual beverage consumption profiles in relation to identified networks of circulating plasma biomarkers.</p> <p><b>Participant characteristics at baseline:</b>                      women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 56 (7)y; 30-55y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: Primarily Caucasian</li> <li>• Socioeconomic position: All nurses</li> <li>• Anthropometry: BMI 28 (6) kg/m<sup>2</sup></li> <li>• Physical activity: ~8 MET hr/wk</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 12% current; 41% former; 48% never</li> <li>• Alcohol intake: 8% beer; 19% liquor; 9% red wine; 21% white wine</li> <li>• TEI: ~1800 (450) kcal/d</li> <li>• Beverage intake at baseline: Cola: 4.2 (3.8) svg/wk; Caffeine-free cola: 3.0 (2.5) svg/wk; SSB: 3.1 (3.0) svg/wk</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data</p>	<p><b>Exposure:</b> SSB factor (representing higher intake of caffeinated and caffeine-free cola and other sugar-containing carbonated drinks relative to other beverage types) identified from EFA</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> dichotomous intake (low vs high factor score group)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• 1984, 1986, 1990 (cumulative average intake)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> </ul>	<p><b>T2D by cumulative average intake</b>, OR (95% CI)                      Low factor score: REF                      High factor score: 1.16 (0.88, 1.52)                      P=0.417</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, alcohol factor</li> <li>• Other: other beverage factors (fruit juice, SSB/LNCSB, caffeine-free), fasting status at blood draw, case-control status, menopausal status, postmenopausal hormone use, aspirin and multivitamin use, modified AHEI score</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position, family history of diabetes, smoking</li> <li>• Exposure subject to measurement error</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Sakurai, 2014<sup>40</sup></b>  <b>PCS, Japan</b>                      Analytic N=2,037</p> <p><b>Study objective:</b> To investigate the association between SSB and diet soda consumption and the incidence of T2D in Japanese men.</p> <p><b>Participant characteristics at baseline:</b> lean middle-aged Japanese men</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 46.2y; 35-55y</li> <li>• Female: 0%</li> <li>• Race and/or ethnicity: All Japanese</li> <li>• Socioeconomic position: All employed at factory</li> <li>• Anthropometry: BMI 23.4 kg/m<sup>2</sup></li> <li>• Physical activity: 28% habitual exercise</li> <li>• Family history of diabetes: 13%</li> <li>• Smoking: 53% current; 15% former; 32% never</li> <li>• Alcohol intake: 15% never, 11% occasional, 32% &lt;20g/d, 42% ≥20g/d</li> <li>• TEI: 1998-2627 kcal/d</li> <li>• Beverage intake at baseline: 12% consumed SSB ≥1 svg/d; median daily intake (range): 0.2 (0.0-9.6) svg</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes or FPG ≥126 mg/dL or HbA1C ≥6.5%; energy intake &lt;500 or &gt;5000 kcal/d; missing data on SSB; loss to follow-up</p>	<p><b>Exposure:</b> SSB ("regular soft drinks, sugar-sweetened soda, and sports drinks, excluding 100% fruit juice and vegetable juice")</p> <ul style="list-style-type: none"> <li>• Serving Size: 237mL or 8oz</li> </ul> <p><b>Comparator:</b> categorical intake (rare/never, more often than rare/never but &lt;1 svg/wk, ≥1 svg/wk but &lt;1 svg/d, ≥1 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Validated diet history questionnaire on intake during previous month</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Biomarkers (FPG ≥126 mg/dL and/or HbA1C ≥6.5%)</li> <li>• 5.5 (1.8)y (annually)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Rare/never: REF                      &gt;Rare/never but &lt;1 svg/wk: 0.97 (0.57, 1.64)                      ≥1 svg/wk to &lt;1 svg/d: 1.11 (0.74, 1.66)                      ≥1 svg/d: 1.34 (0.72, 2.36)                      P-trend=0.424</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity (Japanese), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: hypertension, dyslipidemia, diet treatment for chronic disease, total fiber intake, other beverage consumption (diet soda, fruit juice, vegetable juice, coffee)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Ministry of Health, Labor, and Welfare, Health and Labor Sciences; Ministry of Education, Culture, Sports, Science and Technology of Japan for Scientific Research; Japan Arteriosclerosis Prevention Fund</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Schulze, 2004<sup>41</sup></b>  <b>PCS, NHS-II (Nurses' Health Study II), U.S.</b>                      Analytic N=91,249</p> <p><b>Study objective:</b> To examine the association between consumption of SSB and weight change and risk of T2D in women.</p> <p><b>Participant characteristics at baseline:</b>                      women</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~36 (4.7)y; 24-44y</li> <li>• Female: 100%</li> <li>• Race and/or ethnicity: Primarily European ancestry</li> <li>• Socioeconomic position: All nurses</li> <li>• Anthropometry: BMI ~24.4 kg/m<sup>2</sup></li> <li>• Physical activity: ~19 METs/wk</li> <li>• Family history of diabetes: ~16% in a first-degree relative</li> <li>• Smoking: 12% current</li> <li>• Alcohol intake: ~3 g/d</li> <li>• TEI: ~1800 kcal/d</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or cardiovascular disease; missing data; implausible total energy intake (&lt;500 or &gt;3500 kcal/d)</p>	<p><b>Exposure:</b> Sugar-sweetened soft drinks (sugar-sweetened cola and fruit punch); sugar-sweetened cola included "Coke, Pepsi, or other cola with sugar", "caffeine-free Coke, Pepsi, or other cola with sugar", and "other carbonated beverages with sugar"</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> categorical intake (&lt;1/mo, 1-4/mo, 2-6/wk, ≥1/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• 1991, 1995</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-reported via biennial questionnaire; verified with supplementary questionnaire specific to T2D</li> <li>• 8y</li> </ul>	<p><b>T2D by average intake, RR (95% CI)</b>  <b>All sugar-sweetened soft drinks</b>                      &lt;1/mo (REF)                      1-4/mo: 1.06 (0.87, 1.28)                      2-6/wk: 1.49 (1.16, 1.91)                      ≥1/d: 1.83 (1.42, 2.36)                      P-trend&lt;0.001</p> <p><b>When further controlling for BMI</b>                      &lt;1/mo (REF)                      ≥1/d: 1.39 (1.07, 1.76)                      P-trend=0.01</p> <p><b>When further controlling for TEI</b>                      &lt;1/mo (REF)                      ≥1/d: 1.32 (1.01, 1.73)                      P-trend=0.04</p> <p>Also provided results for sugar-sweetened soft drinks stratified by: Obesity status, physical activity level, family history of diabetes, cereal fiber intake, trans-fat intake, and ratio of polyunsaturated to saturated fat (no stratifications showed significant interactions)</p> <p><b>Sugar-sweetened cola</b>                      &lt;1/mo (ref)                      1-4/mo: 0.99 (0.80, 1.23)                      2-6/wk: 1.56 (1.21, 2.02)                      ≥1/d: 1.87 (1.43, 2.45)                      P-trend&lt;0.001</p> <p><b>Fruit punch</b>                      &lt;1/mo (REF)                      1-4/mo: 0.90 (0.68, 1.18)                      2-6/wk: 1.15 (0.79, 1.66)                      ≥1/d: 2.00 (1.33, 3.03)                      P-trend=0.001</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: postmenopausal hormone use, oral contraceptive use, intake of cereal fiber, magnesium, trans-fat, ratio of polyunsaturated to saturated fat, and other sugar-sweetened beverage consumption (other than the main exposure, depending on model); Results were similar when adjusting for caffeine, red meat, french fries, processed meat, sweets, snacks, vegetables, and fruit</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      NIH; European Association for the Study of Diabetes/American Diabetes Association; German Academic Exchange Service (DAAD)</p>



Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Stroup, 2020<sup>42</sup></b>  <b>PCS, French NutriNet-Santé cohort, France</b>                      Analytic N=104,707</p> <p><b>Study objective:</b> To assess the associations between consumption of ultra-processed foods and T2D</p> <p><b>Participant characteristics at baseline:</b>                      French adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 42.7 (14.5)y; ≥18y</li> <li>• Female: 79%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: 59% with education ≥2y after high school</li> <li>• Anthropometry: 20% with overweight, 8% with obesity</li> <li>• Physical activity: 28% high, 37% moderate, 21% low</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: 17% current; 33% former; 50% never</li> <li>• Alcohol intake: 3.91 (5.53) g/1000 kcal/d</li> <li>• TEI: 1847.14 (450.86) kcal/d without alcohol</li> <li>• Beverage intake at baseline: Sugary drinks, 24.94 (53.64) g/1000 kcal/d</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; &lt;18y; missing or invalid dietary data; energy underreporters</p>	<p><b>Exposure:</b> Ultra-processed beverages, including sugary drinks (e.g., regular sodas, sugary fruit-based beverages, industrial chocolate powder beverages, energy drinks, flavoured waters) and artificially sweetened beverages (e.g., diet sodas, artificially sweetened ice teas)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> continuous intake (% consumed)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Three nonconsecutive 24hr diet records every 6mo</li> <li>• Baseline (average of first 2y)</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report based on doctor's diagnosis or diabetic medication; confirmed with national health insurance database or biomarkers (FBG &gt;1.26 g/L)</li> <li>• Median 6.0y (IQR: 2.8-8.4y)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      By % consumed (in absolute increments of 10%): 1.13 (1.07, 1.19), P&lt;0.0001</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (educational level), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: number of 24h dietary records, diet quality index, consumption amount of ultra-processed beverages</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity</li> <li>• Exposure subject to measurement error (combined SSB and LNCSB) and only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Ministère de la Santé; Santé Publique France; Institut National de la Santé et de la Recherche Médicale (INSERM); Institut National de la Recherche Agronomique (INRA); Conservatoire National des Arts et Métiers (CNAM); Université Paris 13</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Stern, 2019<sup>43</sup></b>  <b>PCS, Mexican Teachers' Cohort, Mexico</b>                      Analytic N=72,667</p> <p><b>Study objective:</b> To estimate the association between sugar-sweetened soda consumption and incident diabetes</p> <p><b>Participant characteristics at baseline:</b>                      Mexican women</p> <ul style="list-style-type: none"> <li>Age (mean and/or range): 42.1 (7.2)y; ≥25y</li> <li>Female: 100%</li> <li>Race and/or ethnicity: All Hispanic</li> <li>Socioeconomic position: Median of highest tertile: 6 household assets (IQR: 6-7), including household access to car, telephone, cell phone, microwave, vacuum, computer, and internet; All are public school teachers</li> <li>Anthropometry: BMI 27.2 (4.5) kg/m<sup>2</sup>; 38% with overweight, 21% with obesity</li> <li>Physical activity: Median of highest tertile: 54.50 MET hr/wk (IQR 41.5-125.5)</li> <li>Family history of diabetes: 47%</li> <li>Smoking: 9% current; 12% former; 76% never; 3% missing</li> <li>Alcohol intake: NR</li> <li>TEI: NR</li> <li>Beverage intake at baseline: Median 1.17 svg/d (IQR: 0.47-4.00)</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; cancer or heart disease; missing data; energy intake &lt;500 or &gt;3500 kcal/d</p>	<p><b>Exposure:</b> Sugar-sweetened soda (cola-flavored soda and flavored soda)</p> <ul style="list-style-type: none"> <li>Serving Size: 355 mL</li> </ul> <p><b>Comparator:</b> continuous intake (svg/d) categorical intake (quintiles; ≤1, &gt;1-4, ≥5 svg/wk)</p> <p>Quintiles; Median (IQR):</p> <ul style="list-style-type: none"> <li>Quintile 1 (n=10,680): 0.23 (0.00-0.23)</li> <li>Quintile 2 (n=13,912): 0.47 (0.47-0.47)</li> <li>Quintile 3 (n=18,478): 1.17 (0.82-1.17)</li> <li>Quintile 4 (n=11,404): 3.00 (2.00-3.23)</li> <li>Quintile 5 (n=18,193): 6.00 (5.50-8.00)</li> </ul> <p>Pre-specified categories; Median (IQR):</p> <ul style="list-style-type: none"> <li>Low, ≤1 svg/wk (n=30,109): 0.47 (0.23-0.58)</li> <li>Moderate, &gt;1 to 2-4 svg/wk (n=29,543): 2.00 (1.17-3.23)</li> <li>High, ≥5 svg/wk (n=13,015): 7.23 (6.00-10.00)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>FFQ assessing usual intake during previous year.</li> <li>Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>Self-report of medical diagnosis of diabetes, use of medical treatment, or date of diabetes diagnosis; validated in subsample using supplementary questionnaire</li> <li>Median 2.16y (IQR: 0.75-4.50y)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)</p> <p>Quintile 1: REF                      Quintile 5: 1.14 (1.01, 1.28)                      P-trend=0.028</p> <p>≤1 svg/wk: REF                      &gt;1 to 2-4 svg/wk: 1.07 (0.99, 1.16)                      ≥5 svg/wk: 1.13 (1.02, 1.25)                      P-trend=0.026</p> <p>Per 1 svg/d : 1.13 (1.04, 1.23)</p> <p>Nonlinear relation per 1 svg/d using restricted cubic splines (unadjusted for BMI):                      Data NR (figure only), P=0.0491</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>TEI: no</li> <li>Key confounders: sex, age, race and/or ethnicity, socioeconomic position (number of assets as measure of SES), anthropometry, physical activity, family history of diabetes, smoking</li> <li>Other: region of residence in Mexico (North, Center, Mexico City/Metropolitan area, South); food and beverage groups (fruit, vegetables, red meat, processed meat, whole grains, juice, and diet soda)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>Did not account for key confounders: alcohol intake</li> <li>Exposure only assessed at baseline</li> <li>High attrition rate/missing data</li> <li>No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      AstraZeneca; Bloomberg Philanthropies; American Institute for Cancer Research; Consejo Nacional de Ciencia y Tecnología</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Teshima, 2015<sup>45</sup></b>  <b>PCS, Mihama Diabetes Prevention Study, Japan</b>                      Analytic N=93</p> <p><b>Study objective:</b> To evaluate the effects of SSB intake on the development of T2D in subjects with impaired glucose tolerance</p> <p><b>Participant characteristics at baseline:</b> participants with IGT</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): ~55y; 40-69y</li> <li>• Female: 68%</li> <li>• Race and/or ethnicity: All Asian</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: BMI 24 (3) kg/m<sup>2</sup></li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: NR</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; normal glucose tolerance; &lt;40 or &gt;69y; severe hepatic or renal disease; history of gastrectomy</p>	<p><b>Exposure:</b> SSB (including canned coffee, carbonated drinks, and juices); no differentiation between fructose, glucose, or sucrose as the source of sweetness</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> dichotomous intake (everyday/occasional intake vs no intake)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• 19-item questionnaire</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Annual OGTT</li> <li>• 3.6 (0.2)y</li> </ul>	<p><b>T2D by baseline intake</b>, OR (95% CI)                      No intake: REF                      Everyday/occasional intake: 1.03 (0.331, 3.187)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: no</li> <li>• Key confounders: sex, age, race and/or ethnicity</li> <li>• Other: duration of observation</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: socioeconomic position, anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Exposure subject to measurement error and analyses based on exposure collected only at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      None declared</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Torres-Ibarra, 2020<sup>46</sup></b>  <b>PCS, Health Workers Cohort Study, Mexico</b>                      Analytic N=1,445</p> <p><b>Study objective:</b> To estimate the risk of T2D due to soft drinks consumption in a cohort of Mexicans.</p> <p><b>Participant characteristics at baseline:</b>                      Mexican adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 44.3 (12.5)y; ≥19y</li> <li>• Female: 76%</li> <li>• Race and/or ethnicity: All Hispanic</li> <li>• Socioeconomic position: 49% ≥college; 23% high school, 26% middle school or less, 2% missing; All employed at health and academic institutions</li> <li>• Anthropometry: BMI 26.2 (4.1) kg/m<sup>2</sup>; 17% obesity</li> <li>• Physical activity: 38% active (≥150 min/wk); 1.5 hr/wk of leisure-time physical activity</li> <li>• Family history of diabetes: 53%</li> <li>• Smoking: 17% current; 24% former; 55% never; 4% missing</li> <li>• Alcohol intake: 33% drink &gt;2.4 g/d</li> <li>• TEI: ~2150 kcal/d</li> <li>• Beverage intake at baseline: Median 0.2 svg/d (IQR: 0.10-0.57); 22% consumed ≥5 svg/wk</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; &lt;19y; missing data; energy intake &lt;500 or &gt;6400 kcal/d; heart disease or cancer (except skin or melanoma) at baseline; pregnant</p>	<p><b>Exposure:</b> Soft drinks (cola soft drinks and flavored carbonated soft drinks)</p> <ul style="list-style-type: none"> <li>• Serving Size: 355 mL</li> </ul> <p><b>Comparator:</b> categorical intake (&lt;1 time/wk, 1-4 times/wk, ≥5 times/wk)</p> <ul style="list-style-type: none"> <li>• &lt;1 svg/wk (n=361)</li> <li>• 1-4 svg/wk (n=770)</li> <li>• ≥5 svg/wk (n=314)</li> </ul> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report of physician-diagnosed T2D, fasting glucose &gt;126 mg/dl, or hypoglycemic medication at any examination</li> <li>• Median 6.7y (IQR: 6.2-7.1y)</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      P-trend=0.040 for multivariate adjusted model</p> <p><b>Further adjusted for baseline BMI</b>                      &lt;1 svg/wk: REF                      1-4 svg/wk: 1.0 (0.6, 1.7)                      ≥5 svg/wk: 1.5 (0.8, 2.8)                      P-trend=0.094</p> <p><b>Further adjusted for abdominal obesity</b>                      &lt;1 svg/wk: REF                      1-4 svg/wk: 1.1 (0.6, 1.9)                      ≥5 svg/wk: 1.6 (0.8, 3.0)                      P-trend=0.083</p> <p><b>Stratified by family history of diabetes:</b>                      P-interaction=0.4285</p> <p><b>No family history of diabetes</b>                      &lt;1 svg/wk (n=147): REF                      1-4 svg/wk (n=303): 0.67 (0.25, 1.79)                      ≥5 svg/wk (n=132): 0.66 (0.20, 2.16)                      P-trend=0.674</p> <p><b>Family history of diabetes</b>                      &lt;1 svg/wk (n=189): REF                      1-4 svg/wk (n=402): 1.49 (0.73, 3.07)                      ≥5 svg/wk (n=166): 2.3 (1.04, 5.17)                      P-trend=0.037</p> <p>Data also provided for complete case analysis (N=600), unadjusted for baseline anthropometry</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, race and/or ethnicity, socioeconomic position (levels of education), anthropometry, physical activity, family history of diabetes, smoking, alcohol intake</li> <li>• Other: hypertension (separate model not including BMI or abdominal obesity)</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Analyses based on exposure collected only at baseline</li> <li>• Unclear how subsample was selected</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Consejo Nacional de Ciencia y Tecnología; Bloomberg Philanthropies</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>Viana Dias, 2023<sup>47</sup></b>  <b>PCS, CUME (Cohort of Universities of Minas Gerais), Brazil</b>                      Analytic N=2,480</p> <p><b>Study objective:</b> To evaluate the association between the energy consumption of SSB adjusted for daily energy intake and the incidence of T2D</p> <p><b>Participant characteristics at baseline:</b>                      Brazilian adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 27% ≤30y, 61% 31-49y, 12% ≥50y</li> <li>• Female: 66%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: All graduated from Brazilian public universities; 54% live with partner</li> <li>• Anthropometry: 58% with overweight</li> <li>• Physical activity: 68% scheduled physical exercise</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: 2239 kcal/d</li> <li>• Beverage intake at baseline: Median 47.7 kcal/d (~118 mL/d of soft drink, ~100 mL/d of fruit juice box)</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; energy intake &lt;600 or &gt;6000 kcal/d; pregnant; missing data on physical activity; foreigner or residing outside Brazil</p>	<p><b>Exposure:</b> SSB, including sugar-sweetened soft drinks and industrialized sugar-sweetened fruit juice (canned/box/powder)</p> <ul style="list-style-type: none"> <li>• Serving Size: NR</li> </ul> <p><b>Comparator:</b> dichotomous intake (based on median: ≤47 vs &gt;47 kcal/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report; blood glucose values were validated with a sample of participants</li> <li>• 2-4y</li> </ul>	<p><b>T2D by baseline intake</b>, OR (95% CI)                      SSB ≤47.7 kcal/d: REF                      SSB &gt;47.7 kcal/d: 1.63 (1.00, 2.66)                      P=0.049</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, anthropometry, physical activity</li> <li>• Other: marital status</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, socioeconomic position, family history of diabetes, smoking, alcohol intake</li> <li>• Analyses based on exposure collected only at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      None declared</p>

Study and Participant Characteristics	Exposure, Comparator, and Outcome(s)	Results	Methodological Considerations
<p><b>von Ruesten, 2013<sup>48</sup></b>  <b>PCS, EPIC-Potsdam, Germany</b>                      Analytic N=23,531</p> <p><b>Study objective:</b> To give a comprehensive overview on health-related foods in relation to major chronic diseases based on 8 years of follow-up</p> <p><b>Participant characteristics at baseline:</b> adults</p> <ul style="list-style-type: none"> <li>• Age (mean and/or range): 35-65y</li> <li>• Female: 61%</li> <li>• Race and/or ethnicity: NR</li> <li>• Socioeconomic position: NR</li> <li>• Anthropometry: NR</li> <li>• Physical activity: NR</li> <li>• Family history of diabetes: NR</li> <li>• Smoking: NR</li> <li>• Alcohol intake: NR</li> <li>• TEI: NR</li> <li>• Beverage intake at baseline: median ~3.5 g/d of high-energy soft drinks</li> </ul> <p><b>Excluded from study or analysis:</b> baseline diabetes; CVD or cancer; missing data; energy intake &gt;800 or &gt;6000 kcal/d</p>	<p><b>Exposure:</b> High-energy soft drinks (cola, lemonade, non-alcoholic beer/malt beer)</p> <ul style="list-style-type: none"> <li>• Serving Size: 200 g</li> </ul> <p><b>Comparator:</b> continuous intake (per 1 svg/d)</p> <p><b>Assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• FFQ assessing usual intake during previous year.</li> <li>• Baseline</li> </ul> <p><b>Outcome assessment methods and timing:</b></p> <ul style="list-style-type: none"> <li>• Self-report or registries; medically verified with physician inquiry</li> <li>• 8y</li> </ul>	<p><b>T2D by baseline intake</b>, HR (95% CI)                      Fully-adjusted model: 1.04 (0.96, 1.13)                      Further adjusted for other food groups: 1.04 (0.95, 1.15)</p>	<p><b>Model adjustments:</b></p> <ul style="list-style-type: none"> <li>• TEI: yes</li> <li>• Key confounders: sex, age, socioeconomic position (education), anthropometry, physical activity, smoking, alcohol intake</li> <li>• Other: prevalent hypertension at baseline, history of high blood lipid levels at baseline, vitamin supplementation, non-consumption of the respective food group, 45 food groups</li> </ul> <p><b>Limitations:</b></p> <ul style="list-style-type: none"> <li>• Did not account for key confounders: race and/or ethnicity, family history of diabetes</li> <li>• Exposure only assessed at baseline</li> <li>• No evidence whether the result was biased due to missing data</li> <li>• No preregistered data analysis plan</li> </ul> <p><b>Funding:</b>                      Federal Ministry of Science (Germany); European Community; German Cancer Aid</p>

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<sup>a</sup> Abbreviations: ADA: American Diabetes Association; ASB: artificial sweetened beverage(s); BMI: body mass index; CI: confidence interval; CVD: cardiovascular disease; d: day(s); dL: deciliter; EFA: exploratory factor analysis; EPIC: European Prospective Investigation into Cancer and Nutrition; FFQ: food frequency questionnaire; g: gram(s); HbA1c: hemoglobin A1C; hr: hour; HR: hazard ratio; IAUC: incremental area under the curve; IGT: impaired glucose tolerance; kcal: kilocalorie(s); kg: kilogram(s); L: liter(s); LNCSB: low- and no-calorie sweetened beverage(s); m: meter(s); mg: milligram(s); min: minute(s); mL: milliliter(s); N/A: not applicable; NCC: nested case control study; NIDDK: National Institute of Diabetes and Digestive and Kidney Diseases; NIH: National Institutes of Health; nmol: nanomole; NR: not reported; NS: not significant; OGTT: oral glucose tolerance test; OR: odds ratio; oz: ounce(s); PCS: prospective cohort study; REF: reference group; RR: risk ratio; SD: standard deviation; SE: standard error; SSB: sugar-sweetened beverage(s); svg: serving(s); T2D: type 2 diabetes; TEI: total energy intake;  $\mu$ mol: micromole(s); wk: week(s); y: year(s)

**Table 12. Risk of bias for randomized controlled trials examining sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes<sup>a</sup>**

Article	Randomization	Deviations from intended interventions (per-protocol)	Missing outcome data	Outcome measurement	Selection of reported result	Overall risk of bias
Campos, 2015 <sup>8</sup>	SOME CONCERNS	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Ebbeling, 2020 <sup>14</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	SOME CONCERNS
Engel, 2018 <sup>15</sup>	SOME CONCERNS	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Hernandez-Cordero, 2014 <sup>21</sup>	LOW	LOW	LOW	LOW	LOW	LOW
Kendig, 2023 <sup>27</sup>	SOME CONCERNS	HIGH	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Tate, 2012 <sup>44</sup>	LOW	LOW	LOW	LOW	SOME CONCERNS	SOME CONCERNS

<sup>a</sup> Possible ratings of low, some concerns, or high determined using the "Cochrane Risk-of-bias 2.0" (RoB 2.0) (August 2019 version)" (Sterne JAC, Savović J, Page MJ, et al. RoB 2: a revised tool for assessing risk of bias in randomised trials. *BMJ* 2019; **366**: l4898. doi:10.1136/bmj.l4898)



**Table 13. Risk of bias for observational studies examining sugar-sweetened beverage consumption in adults and older adults and risk of type 2 diabetes<sup>a</sup>**

Article	Confounding	Exposure measurement	Selection of participants	Post-exposure interventions	Missing data	Outcome measurement	Selection of reported result	Overall risk of bias
Bazzano, 2008 <sup>6</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Bhupathiraju, 2013 <sup>7</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Canhada, 2023 <sup>9</sup>	HIGH	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Chen, 2023 <sup>10</sup>	LOW*	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Cho, 2023 <sup>11</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
de Koning, 2011 <sup>12</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Drouin-Chartier, 2019 <sup>13</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Ericson, 2018 <sup>16</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Eshak, 2013 <sup>17</sup>	LOW*	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Fagherazzi, 2013 <sup>18</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH
Gardener, 2018 <sup>19</sup>	HIGH	SOME CONCERNS	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH

Article	Confounding	Exposure measurement	Selection of participants	Post-exposure interventions	Missing data	Outcome measurement	Selection of reported result	Overall risk of bias
Greenberg, 2005 <sup>20</sup>	SOME CONCERNS	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Hirahatake, 2019 <sup>22</sup>	LOW*	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Huang, 2017 <sup>23</sup>	LOW*	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Imamura, 2019 <sup>24</sup>	SOME CONCERNS	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Jahromi, 2023 <sup>26</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
McNaughton, 2008 <sup>28</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH
Montonen, 2007 <sup>29</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Nettleton, 2009 <sup>30</sup>	HIGH	SOME CONCERNS	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH
O'Connor, 2016 <sup>31</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	LOW	LOW	SOME CONCERNS	SOME CONCERNS
Odegaard, 2010 <sup>32</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Olsson, 2021 <sup>33</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH

Article	Confounding	Exposure measurement	Selection of participants	Post-exposure interventions	Missing data	Outcome measurement	Selection of reported result	Overall risk of bias
Palmer, 2008 <sup>34</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	HIGH	LOW	SOME CONCERNS	HIGH
Pan, 2012 <sup>35</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Paynter, 2006 <sup>36</sup>	LOW*	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Ramne, 2020 <sup>37</sup>	HIGH	SOME CONCERNS	SOME CONCERNS	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Rhee, 2015 <sup>38</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Romaguera, 2013 <sup>25</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Rose, 2023 <sup>39</sup>	HIGH	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Sakurai, 2014 <sup>40</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Schulze, 2004 <sup>41</sup>	SOME CONCERNS	LOW	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Srour, 2020 <sup>42</sup>	SOME CONCERNS	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Stern, 2019 <sup>43</sup>	SOME CONCERNS	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS

Article	Confounding	Exposure measurement	Selection of participants	Post-exposure interventions	Missing data	Outcome measurement	Selection of reported result	Overall risk of bias
Teshima, 2015 <sup>45</sup>	HIGH	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
Torres-Ibarra, 2020 <sup>46</sup>	LOW*	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	SOME CONCERNS
Viana Dias, 2023 <sup>47</sup>	HIGH	HIGH	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH
von Ruesten, 2013 <sup>48</sup>	HIGH	SOME CONCERNS	LOW	LOW	SOME CONCERNS	LOW	SOME CONCERNS	HIGH

<sup>a</sup> Possible ratings of low, some concerns, high, very high, no information, or not applicable were determined using the "Risk of Bias in Non-randomized Studies of Exposures (ROBINS-E)" tool (Higgins JPT, Morgan RL, Rooney AA, et al. A tool to assess risk of bias in non-randomized follow-up studies of exposure effects (ROBINS-E). *Environment International* 2024 (published online Mar 24). doi: [10.1016/j.envint.2024.108602](https://doi.org/10.1016/j.envint.2024.108602).) \*Low risk of bias except for concerns about uncontrolled confounding.

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# Appendix 1: Abbreviations

**Table A 1: List of abbreviations**

<b>Abbreviation</b>	<b>Full name</b>
BMI	Body Mass Index
CNPP	Center for Nutrition Policy and Promotion
HbA1C	Hemoglobin A1C
HDI	Human Development Index
HHS	United States Department of Health and Human Services
HOMA-IR	Homeostatic Model Assessment for Insulin Resistance
NESR	Nutrition Evidence Systematic Review
NGAD	Nutrition Guidance and Analysis Division
NIH	National Institutes of Health
RCT	Randomized Controlled Trial
SSB	Sugar-Sweetened Beverage(s)
USDA	United States Department of Agriculture

## Appendix 2: Literature search strategy

This search was first run on July 5, 2022, and then periodically run using NESR’s continuous evidence monitoring methods until January 9, 2024.\*

Database: PubMed

**Provider: U.S. National Library of Medicine**

**Date(s) Searched:** July 5, 2022 (initial search); July 5, 2022 – January 9, 2024 (continuous evidence monitoring)

**Dates Covered:** January 1, 2000 – January 9, 2024

**Table A 2. Search for PubMed**

Search #	Concept	String
#1	Beverages	"Beverages"[Mesh:NoExp] OR "Sugar Sweetened Beverages"[MeSH] OR "Artificially Sweetened Beverages"[Mesh] OR diet drink*[tiab] OR sweetening agent*[tiab] OR artificially sweet*[tiab] OR beverage[tiab] OR beverages[tiab] OR sports drink*[tiab] OR fortified drink*[tiab] OR sweetened drink*[tiab] OR sweet drink*[tiab] OR sugary drink*[tiab] OR dairy drink*[tiab] OR chocolate drink*[tiab] OR smoothie*[tiab] OR carbonated drink*[tiab] OR soft drink*[tiab] OR soda[tiab] OR sodas[tiab] OR caffeinated drink*[tiab] OR "Drinking Water"[Mesh] OR drinking water[tiab] OR bottled water[tiab] OR "Carbonated Beverages"[Mesh] OR carbonated water[tiab] OR sparkling water[tiab] OR flavored water[tiab] OR flavoured water[tiab] OR flavoured drink[tiab] OR flavored drink*[tiab] OR "Energy Drinks"[Mesh] OR energy drink*[tiab] OR "Fruit and Vegetable Juices"[Mesh] OR juice[tiab] OR juices[tiab] OR fruit drink*[tiab] OR fizzy drink*[tiab] OR "Coffee"[Mesh] OR coffee[tiab] OR "Tea"[Mesh] OR tea[tiab] OR "Milk"[Mesh:NoExp] OR milk[tiab] OR "Soy Milk"[Mesh] OR soymilk[tiab] OR "Buttermilk"[Mesh] OR buttermilk[tiab] OR liquid[tiab] OR liquids[tiab]
#2	Type two diabetes	"Diabetes Mellitus"[Mesh:NoExp] OR "Diabetes Mellitus, Type 2"[Mesh] OR "type 2 diabet*" [tiab] OR "T2D"[tiab] OR "adult onset diabetes"[tiab] OR "Prediabetic State"[Mesh] OR "prediabet*" [tiab] OR "pre diabet*" [tiab] OR "Insulin Resistance"[Mesh] OR "insulin resistance"[tiab] OR "insulin resistant"[tiab] OR "glucose intolerance"[tiab] OR "glucose intolerant"[tiab] OR "glucose tolerance"[tiab] OR "glucose tolerant"[tiab] OR "Glycated Hemoglobin A"[Mesh] OR "hemoglobin A1c"[tiab] OR hba1c[tiab] OR "hba 1c"[tiab] OR "haemoglobin A1c"[tiab] OR "Hyperglycemia"[Mesh] OR "hyperglycemia"[tiab] OR hyperglycaemia[tiab] OR "Hypoglycemia"[Mesh] OR "hypoglycemia"[tiab] OR hypoglycaemia[tiab] OR ((impaired[tiab] OR glucose[tiab]) AND fasting[tiab]) OR "blood glucose"[MeSH] OR "blood glucose"[tiab] OR "plasma glucose"[tiab] OR "serum glucose"[tiab] OR "glycemi*" [tiab] OR glycaemi*[tiab] OR "blood sugar"[tiab] OR dysglycemi*[tiab] OR dysglycaemi*[tiab] OR hyperinsulinism[MeSH] OR hyperinsulin*[tiab] OR "Diabetes, Gestational"[Mesh] OR (gestation*[tiab] AND diabet*[tiab]) OR ("Maternal Nutritional Physiological Phenomena"[Mesh] AND diabet*[tiab])

\* USDA Nutrition Evidence Systematic Review Branch. Chapter 10: Continuous Evidence Monitoring. In: *USDA Nutrition Evidence Systematic Review: Methodology Manual*. February 2023. U.S. Department of Agriculture, Food and Nutrition Service, Center for Nutrition Policy and Promotion, Nutrition Evidence Systematic Review. Available at: <https://nesr.usda.gov/methodology-overview>.

<b>#3</b>		#1 AND #2
<b>#4</b>	Limits	<p>#3 NOT ("Animals"[Mesh] NOT ("Animals"[Mesh] AND "Humans"[Mesh])) NOT (editorial[ptyp] OR comment[ptyp] OR commentary[tiab] OR news[ptyp] OR letter[ptyp] OR review[ptyp] OR systematic review[ptyp] OR systematic review[ti] OR meta-analysis[ptyp] OR meta-analysis[ti] OR meta-analyses[ti] OR protocol[ti] OR protocols[ti] OR retracted publication[ptyp] OR retraction of publication[ptyp] OR retraction of publication[tiab] OR retraction notice[ti] OR "retracted publication"[ti] OR "Congress"[Publication Type] OR "Consensus Development Conference"[Publication Type] OR "conference abstract"[tiab] OR "conference proceeding"[tiab] OR "conference paper"[tiab] OR "practice guideline"[ptyp] OR "practice guideline"[ti])</p> <p>Language: English</p> <p>Publication date: from 2000/1/1 - 3000/12/12</p>

Database: Embase

Provider: Elsevier

Date(s) Searched: July 5, 2022 (initial search); July 5, 2022 – January 9, 2024 (continuous evidence monitoring)

Dates Covered: January 1, 2000 – January 9, 2024

Table A 3. Search for Embase

Search #	Concept	String
#1	Beverages	'Beverages'/de OR 'sweetened beverage'/exp OR 'Drinking Water'/exp OR 'Carbonated Beverages'/exp OR 'carbonated water'/exp OR 'Energy Drink'/exp OR 'Fruit and Vegetable Juice'/exp OR 'Coffee'/exp OR 'Tea'/exp OR 'Milk'/de OR 'soybean milk'/exp OR 'Buttermilk'/exp OR 'diet drink*':ab,ti OR 'sweetening agent*':ab,ti OR 'artificially sweet*':ab,ti OR 'beverage':ab,ti OR 'beverages':ab,ti OR 'sports drink*':ab,ti OR 'fortified drink*':ab,ti OR 'sweetened drink*':ab,ti OR 'sweet drink*':ab,ti OR 'sugary drink*':ab,ti OR 'dairy drink*':ab,ti OR 'chocolate drink*':ab,ti OR smoothie*':ab,ti OR 'carbonated drink*':ab,ti OR 'soft drink*':ab,ti OR 'soda':ab,ti OR 'sodas':ab,ti OR 'caffeinated drink*':ab,ti OR 'drinking water':ab,ti OR 'bottled water':ab,ti OR 'carbonated water':ab,ti OR 'sparkling water':ab,ti OR 'flavored water':ab,ti OR 'flavoured water':ab,ti OR 'flavoured drink':ab,ti OR 'flavored drink*':ab,ti OR 'energy drink*':ab,ti OR 'juice':ab,ti OR 'juices':ab,ti OR 'fruit drink*':ab,ti OR 'fizzy drink*':ab,ti OR 'coffee':ab,ti OR 'tea':ab,ti OR 'milk':ab,ti OR 'soymilk':ab,ti OR 'soy milk':ab,ti OR 'buttermilk':ab,ti OR 'liquid':ab,ti OR 'liquids':ab,ti
#2	Type two diabetes	'Diabetes Mellitus'/de OR 'diabetic obesity'/exp OR 'impaired glucose tolerance'/exp OR 'non insulin dependent diabetes mellitus'/exp OR 'insulin resistance'/exp OR 'Hypoglycemia'/exp OR 'glucose blood level'/exp OR 'hyperinsulinism'/exp OR 'pregnancy diabetes mellitus'/exp OR 'type 2 diabet*':ab,ti OR 'T2D':ab,ti OR 'adult onset diabetes':ab,ti OR 'prediabet*':ab,ti OR 'pre diabet*':ab,ti OR 'insulin resistance':ab,ti OR 'insulin resistant':ab,ti OR 'glucose intolerance':ab,ti OR 'glucose intolerant':ab,ti OR 'glucose tolerance':ab,ti OR 'glucose tolerant':ab,ti OR 'hemoglobin A1c':ab,ti OR 'hba1c':ab,ti OR 'hba 1c':ab,ti OR 'haemoglobin A1c':ab,ti OR 'hyperglycemia':ab,ti OR 'hyperglycaemia':ab,ti OR 'hypoglycemia':ab,ti OR 'hypoglycaemia':ab,ti OR (('impaired' OR 'glucose') NEAR/4 'fasting'):ab,ti OR 'blood glucose':ab,ti OR 'plasma glucose':ab,ti OR 'serum glucose':ab,ti OR 'glycemi*':ab,ti OR glycaemi*':ab,ti OR 'blood sugar':ab,ti OR dysglycemi*':ab,ti OR dysglycaemi*':ab,ti OR hyperinsulin*':ab,ti OR ('gestation*' NEAR/4 'diabet*'):ab,ti OR ('maternal nutrition'/exp AND 'diabet*':ab,ti)
#3		#1 AND #2
#4	Limits	#3 AND ([article]/lim OR [article in press]/lim) NOT ([animals]/lim NOT ([animals]/lim AND [humans]/lim)) AND [english]/lim NOT ([conference abstract]/lim OR [conference paper]/lim OR [conference review]/lim OR [editorial]/lim OR [erratum]/lim OR [letter]/lim OR [note]/lim OR 'retraction of publication':ab,ti OR 'retraction notice':ti OR 'retracted publication':ab,ti OR [review]/lim OR [systematic review]/lim OR [meta analysis]/lim OR 'practice guideline':ti) AND [2000-2024]/py

Database: Cochrane Central Register of Controlled Trials (CENTRAL)

**Provider: John Wiley & Sons**

**Date(s) Searched:** July 5, 2022 (initial search); July 5, 2022 – January 9, 2024 (continuous evidence monitoring)

**Dates Covered:** January 1, 2000 – January 9, 2024

**Table A 4. Search for Cochrane CENTRAL**

Search #	Concept	String
#1	Beverages	[mh ^"Beverages"] OR [mh "Sugar Sweetened Beverages"] OR [mh "Artificially Sweetened Beverages"] OR [mh "Drinking Water"] OR [mh "Carbonated Beverages"] OR [mh "Energy Drinks"] OR [mh "Fruit and Vegetable Juices"] OR [mh "Coffee"] OR [mh "Tea"] OR [mh ^"Milk"] OR [mh "Soy Milk"] OR [mh "Buttermilk"] OR ("diet drink*" OR "sweetening agent*" OR "artificially sweet*" OR beverage OR beverages OR "sports drink*" OR "fortified drink*" OR "sweetened drink*" OR "sweet drink*" OR "sugary drink*" OR "dairy drink*" OR "chocolate drink*" OR smoothie* OR "carbonated drink*" OR "soft drink*" OR soda OR sodas OR "caffeinated drink*" OR "drinking water" OR "bottled water" OR "carbonated water" OR "sparkling water" OR "flavored water" OR "flavoured water" OR "flavoured drink" OR "flavored drink*" OR "energy drink*" OR juice OR juices OR "fruit drink*" OR "fizzy drink*" OR coffee OR tea OR milk OR soymilk OR "soy milk" OR buttermilk OR liquid OR liquids):ti,ab,kw
#2	Type two diabetes	[mh ^"Diabetes Mellitus"] OR [mh "Diabetes Mellitus, Type 2"] OR [mh "Prediabetic State"] OR [mh "Insulin Resistance"] OR [mh "Hyperglycemia"] OR [mh "Glycated Hemoglobin A"] OR [mh "Hypoglycemia"] OR [mh "blood glucose"] OR [mh hyperinsulinism] OR [mh "Diabetes, Gestational"] OR ("type 2 diabet*" OR "T2D" OR "adult onset diabetes" OR "prediabet*" OR "pre diabet*" OR "insulin resistance" OR "insulin resistant" OR "glucose intolerance" OR "glucose intolerant" OR "glucose tolerance" OR "glucose tolerant" OR "hemoglobin A1c" OR "hba1c" OR "hba 1c" OR "haemoglobin A1c" OR "hyperglycemia" OR hyperglycaemia OR "hypoglycemia" OR "hypoglycaemia" OR ((impaired OR glucose) AND fasting) OR "blood glucose" OR "plasma glucose" OR "serum glucose" OR "glycemi*" OR glycaemi* OR "blood sugar" OR dysglycemi* OR dysglycaemi* OR hyperinsulin*):ti,ab,kw
#3		#1 AND #2  In Trials, word variations searched  Year First Published: 2000-2024

Database: CINAHL

Provider: EBSCO

Date(s) Searched: July 5, 2022 (initial search); July 5, 2022 – January 9, 2024 (continuous evidence monitoring)

Dates Covered: January 1, 2000 – January 9, 2024

Table A 5. Search for CINAHL

Search #	Concept	String
#1	Beverages	(MH "Beverages") OR MH ("Sweetened Beverages") OR (MH "Water+") OR (MH "Carbonated Beverages") OR (MH "Energy Drinks") OR (MH "Fruit Juices") OR (MH "Coffee") OR (MH "Tea") OR (MH "Milk") OR (MH "Milk Substitutes+") OR TI (diet drink* OR sweetening agent* OR artificially sweet* OR beverage* OR sports drink* OR fortified drink* OR sweetened drink* OR sweet drink* OR sugary drink* OR dairy drink* OR chocolate drink* OR smoothie* OR carbonated drink* OR soft drink* OR soda OR sodas OR caffeinated drink* OR drinking water OR bottled water OR carbonated water OR sparkling water OR flavored water OR flavoured water OR flavoured drink* OR flavored drink* OR energy drink* OR juice OR juices OR fruit drink* OR fizzy drink* OR coffee OR tea OR milk OR soymilk OR buttermilk OR liquid OR liquids) OR AB (diet drink* OR sweetening agent* OR artificially sweet* OR beverage* OR sports drink* OR fortified drink* OR sweetened drink* OR sweet drink* OR sugary drink* OR dairy drink* OR chocolate drink* OR smoothie* OR carbonated drink* OR soft drink* OR soda OR sodas OR caffeinated drink* OR drinking water OR bottled water OR carbonated water OR sparkling water OR flavored water OR flavoured water OR flavoured drink* OR flavored drink* OR energy drink* OR juice OR juices OR fruit drink* OR fizzy drink* OR coffee OR tea OR milk OR soymilk OR buttermilk OR liquid OR liquids)
#2	Type two diabetes	(MH "Diabetes Mellitus") OR (MH "Diabetes Mellitus, Type 2") OR (MH "Diabetes Mellitus, Gestational") OR (MH "Prediabetic State") OR (MH "Insulin Resistance+") OR (MH "Hyperglycemia+") OR (MH "Hemoglobin A, Glycosylated") OR (MH "Hypoglycemia+") OR (MH "blood glucose") OR (MH "hyperinsulinism+") OR (TI "type 2 diabet*" OR "T2D" OR "adult onset diabetes" OR "prediabet*" OR "pre diabet*" OR "insulin resistance" OR "insulin resistant" OR "glucose intolerance" OR "glucose intolerant" OR "glucose tolerance" OR "glucose tolerant" OR "hemoglobin A1c" OR "hba1c" OR "hba 1c" OR "haemoglobin A1c" OR "hyperglycemia" OR hyperglycaemia OR "hypoglycemia" OR "hypoglycaemia" OR ((impaired OR glucose) N4 fasting) OR "blood glucose" OR "plasma glucose" OR "serum glucose" OR "glycemi*" OR glycaemi* OR "blood sugar" OR dysglycemi* OR dysglycaemi* OR hyperinsulin* OR (gestation* N4 diabet*)) OR (AB "type 2 diabet*" OR "T2D" OR "adult onset diabetes" OR "prediabet*" OR "pre diabet*" OR "insulin resistance" OR "insulin resistant" OR "glucose intolerance" OR "glucose intolerant" OR "glucose tolerance" OR "glucose tolerant" OR "hemoglobin A1c" OR "hba1c" OR "hba 1c" OR "haemoglobin A1c" OR "hyperglycemia" OR hyperglycaemia OR "hypoglycemia" OR "hypoglycaemia" OR ((impaired OR glucose) N4 fasting) OR "blood glucose" OR "plasma glucose" OR "serum glucose" OR "glycemi*" OR glycaemi* OR "blood sugar" OR dysglycemi* OR dysglycaemi* OR hyperinsulin* OR (gestation* N4 diabet*)) OR ((MH "Maternal Nutritional Physiology") AND ((TI diabet*) OR (AB diabet*)))

<b>#3</b>		S1 AND S2
<b>#4</b>	Limits	<p>#3 NOT ((MH "Animals+") OR (MH "Animal Studies"))</p> <p>NOT ((MH "Literature Review") OR (MH "Meta Analysis") OR (MH "Systematic Review") OR (MH "News") OR (MH "Retracted Publication") OR (MH "Retraction of Publication))</p> <p>Limiters - English Language, Expanders - Apply equivalent subject, Published Date: 20000101-20240109</p>

## Appendix 3: Excluded articles

The following table lists the articles excluded after full-text screening for this systematic review question. At least one reason for exclusion is provided for each article, though this may not reflect all possible reasons. Information about articles excluded after title and abstract screening is available upon request.

**Table A 6. Articles excluded after full-text screening**

#	Citation	Rationale
1	Aljamal A, Al-Shawabkeh M, Abu-Zaiton A, et al. Effect of Green Coffee and Orlistat on Obese Individuals. <i>International Journal of Pharmacology</i> . 2022. 18:864-868. doi:10.3923/ijp.2022.864.868	Intervention/exposure
2	Alperet DJ, Butler LM, Koh WP, Yuan JM, van Dam RM. Influence of temperate, subtropical, and tropical fruit consumption on risk of type 2 diabetes in an Asian population. <i>Am J Clin Nutr</i> . 2017;105(3):736-745. doi:10.3945/ajcn.116.147090	Intervention/exposure
3	Alvarsson M, Hilding A, Ostenson CG. Factors determining normalization of glucose intolerance in middle-aged Swedish men and women: a 8-10-year follow-up. <i>Diabet Med</i> . 2009;26(4):345-353. doi:10.1111/j.1464-5491.2009.02685.x	Intervention/exposure
4	Angelopoulos TJ, Lowndes J, Rippe J, et al. No change in indices of glucose regulation or insulin resistance after 6 months of daily consumption of sugar sweetened or diet beverages. <i>Endocr Rev</i> . 2016;37(2). doi:10.1210/endo-meetings.2016.DGM.8.SUN-688	Publication status
5	Appelhans BM, Baylin A, Huang MH, et al. Beverage Intake and Metabolic Syndrome Risk Over 14 Years: The Study of Women's Health Across the Nation. <i>J Acad Nutr Diet</i> . 2017;117(4):554-562. doi:10.1016/j.jand.2016.10.011	Outcome
6	Araki R, Fujie K, Yuine N, et al. Olive leaf tea is beneficial for lipid metabolism in adults with prediabetes: an exploratory randomized controlled trial. <i>Nutr Res</i> . 2019;67:60-66. doi:10.1016/j.nutres.2019.05.003	Intervention/exposure
7	Arnberg K, Mølgaard C, Michaelsen KF, Jensen SM, Trolle E, Larnkjær A. Skim milk, whey, and casein increase body weight and whey and casein increase the plasma C-peptide concentration in overweight adolescents. <i>J Nutr</i> . 2012;142(12):2083-2090. doi:10.3945/jn.112.161208	Intervention/exposure
8	Auerbach BJ, Littman AJ, Tinker L, et al. Associations of 100% fruit juice versus whole fruit with hypertension and diabetes risk in postmenopausal women: Results from the Women's Health Initiative. <i>Prev Med</i> . 2017;105:212-218. doi:10.1016/j.ypmed.2017.08.031	Intervention/exposure
9	Azadbakht L, Nurbakhsh S. Effect of soy drink replacement in a weight reducing diet on anthropometric values and blood pressure among overweight and obese female youths. <i>Asia Pac J Clin Nutr</i> . 2011;20(3):383-389.	Outcome
10	Azzini E, Venneria E, Ciarapica D, et al. Effect of Red Orange Juice Consumption on Body Composition and Nutritional Status in Overweight/Obese Female: A Pilot Study. <i>Oxid Med Cell Longev</i> . 2017;2017:1672567. doi:10.1155/2017/1672567	Study design
11	Babio N, Becerra-Tomás N, Martínez-González MÁ, et al. Consumption of Yogurt, Low-Fat Milk, and Other Low-Fat Dairy Products Is Associated with Lower Risk of Metabolic Syndrome Incidence in an Elderly Mediterranean Population. <i>J Nutr</i> . 2015;145(10):2308-2316. doi:10.3945/jn.115.214593	Intervention/exposure
12	Bahorun T, Luximon-Ramma A, Neergheen-Bhujun VS, et al. The effect of black tea on risk factors of cardiovascular disease in a normal population. <i>Prev Med</i> . 2012;54 Suppl:S98-S102. doi:10.1016/j.ypmed.2011.12.009	Intervention/exposure
13	Balk L, Hoekstra T, Twisk J. Relationship between long-term coffee consumption and components of the metabolic syndrome: the Amsterdam Growth and Health Longitudinal Study. <i>Eur J Epidemiol</i> . 2009;24(4):203-209. doi:10.1007/s10654-009-9323-1	Intervention/exposure



#	Citation	Rationale
14	Banini AE, Boyd LC, Allen JC, Allen HG, Sauls DL. Muscadine grape products intake, diet and blood constituents of non-diabetic and type 2 diabetic subjects. <i>Nutrition</i> . 2006;22(11-12):1137-1145. doi:10.1016/j.nut.2006.08.012	Outcome
15	Barr SI, McCarron DA, Heaney RP, et al. Effects of increased consumption of fluid milk on energy and nutrient intake, body weight, and cardiovascular risk factors in healthy older adults. <i>J Am Diet Assoc</i> . 2000;100(7):810-817. doi:10.1016/S0002-8223(00)00236-4	Intervention/exposure
16	Barr SI, McCarron DA, Heaney RP, et al. Effects of increased consumption of fluid milk on energy and nutrient intake, body weight, and cardiovascular risk factors in healthy older adults. <i>J Am Diet Assoc</i> . 2000;100(7):810-817. doi:10.1016/S0002-8223(00)00236-4	Duplicate
17	Barrio-Lopez MT, Martinez-Gonzalez MA, Fernandez-Montero A, Beunza JJ, Zazpe I, Bes-Rastrollo M. Prospective study of changes in sugar-sweetened beverage consumption and the incidence of the metabolic syndrome and its components: the SUN cohort. <i>Br J Nutr</i> . 2013;110(9):1722-1731. doi:10.1017/S0007114513000822	Outcome
18	Basu A, Betts NM, Mulugeta A, Tong C, Newman E, Lyons TJ. Green tea supplementation increases glutathione and plasma antioxidant capacity in adults with the metabolic syndrome. <i>Nutr Res</i> . 2013;33(3):180-187. doi:10.1016/j.nutres.2012.12.010	Intervention/exposure
19	Basu A, Du M, Sanchez K, et al. Green tea minimally affects biomarkers of inflammation in obese subjects with metabolic syndrome. <i>Nutrition</i> . 2011;27(2):206-213. doi:10.1016/j.nut.2010.01.015	Intervention/exposure
20	Basu A, Sanchez K, Leyva MJ, et al. Green tea supplementation affects body weight, lipids, and lipid peroxidation in obese subjects with metabolic syndrome. <i>J Am Coll Nutr</i> . 2010;29(1):31-40. doi:10.1080/07315724.2010.10719814	Intervention/exposure
21	Bellikci-Koyu E, Sarer-Yurekli BP, Akyon Y, et al. Effects of Regular Kefir Consumption on Gut Microbiota in Patients with Metabolic Syndrome: A Parallel-Group, Randomized, Controlled Study. <i>Nutrients</i> . 2019;11(9):2089. doi:10.3390/nu11092089	Intervention/exposure
22	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. <i>Am J Clin Nutr</i> . 2015;102(2):487-496. doi:10.3945/ajcn.114.105049	Intervention/exposure
23	Beydoun MA, Fanelli-Kuczmariski MT, Beydoun HA, et al. Dairy product consumption and its association with metabolic disturbance in a prospective study of urban adults. <i>Br J Nutr</i> . 2018;119(6):706-719. doi:10.1017/S0007114518000028	Intervention/exposure
24	Bhupathiraju SN, Pan A, Manson JE, Willett WC, van Dam RM, Hu FB. Changes in coffee intake and subsequent risk of type 2 diabetes: three large cohorts of US men and women. <i>Diabetologia</i> . 2014;57(7):1346-1354. doi:10.1007/s00125-014-3235-7	Intervention/exposure
25	Bidel S, Silventoinen K, Hu G, Lee DH, Kaprio J, Tuomilehto J. Coffee consumption, serum gamma-glutamyltransferase and risk of type II diabetes. <i>Eur J Clin Nutr</i> . 2008;62(2):178-185. doi:10.1038/sj.ejcn.1602712	Intervention/exposure
26	Boggs DA, Rosenberg L, Ruiz-Narvaez EA, Palmer JR. Coffee, tea, and alcohol intake in relation to risk of type 2 diabetes in African American women. <i>Am J Clin Nutr</i> . 2010;92(4):960-966. doi:10.3945/ajcn.2010.29598	Intervention/exposure
27	Bondonno NP, Davey RJ, Murray K, et al. Associations Between Fruit Intake and Risk of Diabetes in the AusDiab Cohort. <i>J Clin Endocrinol Metab</i> . 2021;106(10):e4097-e4108. doi:10.1210/clinem/dgab335	Intervention/exposure
28	Bonnet F, Tavenard A, Esvan M, et al. Consumption of a Carbonated Beverage with High-Intensity Sweeteners Has No Effect on Insulin Sensitivity and Secretion in Nondiabetic Adults. <i>J Nutr</i> . 2018;148(8):1293-1299. doi:10.1093/jn/nxy100	Intervention/exposure
29	Brouwer-Brolsma EM, van Woudenberg GJ, Oude Elferink SJ, et al. Intake of different types of dairy and its prospective association with risk of type 2 diabetes: The Rotterdam Study. <i>Nutr Metab Cardiovasc Dis</i> . 2016;26(11):987-995. doi:10.1016/j.numecd.2016.08.003	Intervention/exposure

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30	Bruun JM, Maersk M, Belza A, Astrup A, Richelsen B. Consumption of sucrose-sweetened soft drinks increases plasma levels of uric acid in overweight and obese subjects: a 6-month randomised controlled trial. <i>Eur J Clin Nutr.</i> 2015;69(8):949-953. doi:10.1038/ejcn.2015.95	Other (e.g., duplicative data)
31	Bundrick SC, Thearle MS, Venti CA, Krakoff J, Votruba SB. Soda consumption during ad libitum food intake predicts weight change. <i>J Acad Nutr Diet.</i> 2014;114(3):444-449. doi:10.1016/j.jand.2013.09.016	Outcome
32	Buziau AM, Soedamah-Muthu SS, Geleijnse JM, Mishra GD. Total Fermented Dairy Food Intake Is Inversely Associated with Cardiovascular Disease Risk in Women. <i>J Nutr.</i> 2019;149(10):1797-1804. doi:10.1093/jn/nxz128	Intervention/exposure
33	Campos V, Despland C, Brandejsky V, et al. Metabolic Effects of Replacing Sugar-Sweetened Beverages with Artificially-Sweetened Beverages in Overweight Subjects with or without Hepatic Steatosis: A Randomized Control Clinical Trial. <i>Nutrients.</i> 2017;9(3):202. doi:10.3390/nu9030202	Other (e.g., duplicative data)
34	Campos V, Despland C, Kreis R, et al. Metabolic effects of replacing sugar-sweetened by artificially sweetened beverages in overweight subjects with or without hepatic steatosis: a randomized control clinical trial. <i>Obes Facts.</i> 2017;10:190-191.	Publication status
35	Carroll SJ, Niyonsenga T, Coffee NT, Taylor AW, Daniel M. Associations between local descriptive norms for overweight/obesity and insufficient fruit intake, individual-level diet, and 10-year change in body mass index and glycosylated haemoglobin in an Australian cohort. <i>Int J Behav Nutr Phys Act.</i> 2018;15(1):44. doi:10.1186/s12966-018-0675-3	Outcome
36	Cesar T, Fidelix M, Sivieri K, Millenkovic D. Daily consumption of orange juice modulated intestinal microbiota and improved glucose and lipids metabolism in women. <i>Proc Nutr Soc.</i> 2020;79:(OCE2):E634. doi:10.1017/S0029665120005832	Study design
37	Chatterjee S, Roy N, Saha A, et al. Black tea consumption enhance antioxidant status, reduce inflammatory stress vis-a-vis insulin resistance: Hint from a small clinical cohort study on pre-diabetic subjects. <i>Int J Pharm Sci Rev Res.</i> 2014;28:278-283.	Country
38	Chen Y, Feng R, Yang X, et al. Yogurt improves insulin resistance and liver fat in obese women with nonalcoholic fatty liver disease and metabolic syndrome: a randomized controlled trial. <i>Am J Clin Nutr.</i> 2019;109(6):1611-1619. doi:10.1093/ajcn/nqy358	Comparator
39	Cho HJ, Okekunle AP, Yie GE, et al. Association of coffee consumption with type 2 diabetes and glycemic traits: a Mendelian randomization study. <i>Nutr Res Pract.</i> 2023;17(4):789-802. doi:10.4162/nrp.2023.17.4.789	Intervention/exposure
40	Cho Y, Ryu S, Kim R, Shin MJ, Oh H. Ultra-processed Food Intake and Risk of Type 2 Diabetes in Korean Adults. <i>J Nutr.</i> 2024;154(1):243-251. doi:10.1016/j.tjnut.2023.11.021	Duplicate
41	Cordova R, Viallon V, Fontvieille E, et al. Consumption of ultra-processed foods and risk of multimorbidity of cancer and cardiometabolic diseases: a multinational cohort study. <i>Lancet Reg Health Eur.</i> 2023;35:100771. doi:10.1016/j.lanepe.2023.100771	Intervention/exposure
42	Corrêa TAF, Tobaruela EC, Capetini VC, et al. Blood orange juice intake changes specific bacteria of gut microbiota associated with cardiometabolic biomarkers. <i>Front Microbiol.</i> 2023;14:1199383. doi:10.3389/fmicb.2023.1199383	Study design
43	Corrêa TAF, Rogero MM, Mioto BM, et al. Paper-filtered coffee increases cholesterol and inflammation biomarkers independent of roasting degree: a clinical trial. <i>Nutrition.</i> 2013;29:977-81. doi:10.1016/j.nut.2013.01.003	Comparator
44	Creighton S, Jay M. Are non-nutritive sweetened beverages comparable to water in weight loss trials?. <i>J Clin Outcomes Manag.</i> 2014;21:490-492.	Study design
45	Daily JW, Liu M, Park S. High genetic risk scores of SLIT3, PLEKHA5 and PPP2R2C variants increased insulin resistance and interacted with coffee and caffeine consumption in middle-aged adults. <i>Nutr Metab Cardiovasc Dis.</i> 2019;29(1):79-89. doi:10.1016/j.numecd.2018.09.009	Study design

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46	de Moraes MM, Mediano MFF, de Souza RAG, Moura AS, da Veiga GV, Sichieri R. Discouraging soft drink consumption reduces blood glucose and cholesterol of Brazilian elementary students: Secondary analysis of a randomized controlled trial. <i>Prev Med.</i> 2017;100:223-228. doi:10.1016/j.ypmed.2017.04.035	Intervention/exposure
47	De B, Shrivastav A, Das T, Goswami TK. Physicochemical and nutritional assessment of soy milk and soymilk products and comparative evaluation of their effects on blood gluco-lipid profile. <i>Applied Food Research.</i> 2022;2(2):100146. doi:https://doi.org/10.1016/j.afres.2022.100146	Study design
48	Dhingra R, Sullivan L, Jacques PF, et al. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community [published correction appears in <i>Circulation.</i> 2007 Dec 4;116(23):e557]. <i>Circulation.</i> 2007;116(5):480-488. doi:10.1161/CIRCULATIONAHA.107.689935	Outcome
49	Díaz-López A, Bulló M, Martínez-González MA, et al. Dairy product consumption and risk of type 2 diabetes in an elderly Spanish Mediterranean population at high cardiovascular risk. <i>Eur J Nutr.</i> 2016;55(1):349-360. doi:10.1007/s00394-015-0855-8	Intervention/exposure
50	Ding M, Pan A, Manson JE, et al. Consumption of soy foods and isoflavones and risk of type 2 diabetes: a pooled analysis of three US cohorts. <i>Eur J Clin Nutr.</i> 2016;70(12):1381-1387. doi:10.1038/ejcn.2016.117	Intervention/exposure
51	Dong XX, Wang RR, Liu JY, Ma QH, Pan CW. Habitual tea consumption and 5-year incident metabolic syndrome among older adults: a community-based cohort study. <i>BMC Geriatr.</i> 2021;21(1):728. doi:10.1186/s12877-021-02707-8	Intervention/exposure
52	Doo T, Morimoto Y, Steinbrecher A, Kolonel LN, Maskarinec G. Coffee intake and risk of type 2 diabetes: the Multiethnic Cohort. <i>Public Health Nutr.</i> 2014;17(6):1328-1336. doi:10.1017/S1368980013000487	Intervention/exposure
53	Dourado GK, Cesar TB. Investigation of cytokines, oxidative stress, metabolic, and inflammatory biomarkers after orange juice consumption by normal and overweight subjects. <i>Food Nutr Res.</i> 2015;59:28147. doi:10.3402/fnr.v59.28147	Study design
54	Driessen MT, Koppes LL, Veldhuis L, Samoocha D, Twisk JW. Coffee consumption is not related to the metabolic syndrome at the age of 36 years: the Amsterdam Growth and Health Longitudinal Study. <i>Eur J Clin Nutr.</i> 2009;63(4):536-542. doi:10.1038/ejcn.2008.6	Intervention/exposure
55	Drouin-Chartier JP, Hernández-Alonso P, Guasch-Ferré M, et al. Dairy consumption, plasma metabolites, and risk of type 2 diabetes. <i>Am J Clin Nutr.</i> 2021;114(1):163-174. doi:10.1093/ajcn/nqab047	Intervention/exposure
56	Drouin-Chartier JP, Li Y, Ardisson Korat AV, et al. Changes in dairy product consumption and risk of type 2 diabetes: results from 3 large prospective cohorts of US men and women. <i>Am J Clin Nutr.</i> 2019;110(5):1201-1212. doi:10.1093/ajcn/nqz180	Intervention/exposure
57	Drouin-Chartier JP, Gagnon J, Labonté MÈ, et al. Impact of milk consumption on cardiometabolic risk in postmenopausal women with abdominal obesity. <i>Nutr J.</i> 2015;14:12. doi:10.1186/1475-2891-14-12	Intervention/exposure
58	Duffey KJ, Gordon-Larsen P, Steffen LM, Jacobs DR Jr, Popkin BM. Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. <i>Am J Clin Nutr.</i> 2010;92(4):954-959. doi:10.3945/ajcn.2010.29478	Outcome
59	Ei-Elimat T, Qasem WM, Al-Sawalha NA, et al. A Prospective Non-Randomized Open-Label Comparative Study of The Effects of Matcha Tea on Overweight and Obese Individuals: A Pilot Observational Study. <i>Plant Foods Hum Nutr.</i> 2022;77(3):447-454. doi:10.1007/s11130-022-00998-9	Intervention/exposure
60	Ellis AC, Mehta T, Nagabooshanam VA, Dudenbostel T, Locher JL, Crowe-White KM. Daily 100% watermelon juice consumption and vascular function among postmenopausal women: A randomized controlled trial. <i>Nutr Metab Cardiovasc Dis.</i> 2021;31(10):2959-2968. doi:10.1016/j.numecd.2021.06.022	Comparator

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61	Elwood PC, Pickering JE, Fehily AM. Milk and dairy consumption, diabetes and the metabolic syndrome: the Caerphilly prospective study. <i>J Epidemiol Community Health</i> . 2007;61(8):695-698. doi:10.1136/jech.2006.053157	Intervention/exposure
62	Esfandiar Z, Hosseini-Esfahani F, Mirmiran P, Azizi F. Higher dietary flavonol and isoflavonoid intakes are associated with lower incidence of type 2 diabetes. <i>Int J Vitam Nutr Res</i> . doi:10.1024/0300-9831/a000782	Intervention/exposure
63	Fagherazzi G, Gusto G, Mancini FR, et al. Determinants of 20-year non-progression to Type 2 diabetes in women at very high risk: the E3N cohort study. <i>Diabet Med</i> . 2018;35(12):1716-1721. doi:10.1111/dme.13774	Intervention/exposure
64	Fagherazzi G, Vilier A, Saes Sartorelli D, Lajous M, Balkau B, Clavel-Chapelon F. Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidemiologique aupres des femmes de la Mutuelle Generale de l'Education Nationale-European Prospective Investigation into Cancer and Nutrition cohort. <i>Am J Clin Nutr</i> . 2013;97(3):517-523. doi:10.3945/ajcn.112.050997	Duplicate
65	Faghih S, Hedayati M, Abadi A, Kimiagar M. Comparing the effects of cow's milk, and calcium supplementation on components of the metabolic syndrome in overweight or obese women. <i>Iranian J Endocrinology and Metabolism</i> . 2013;14(5):430-436.	Intervention/exposure
66	Fernandez-Cao JC, Arija V, Aranda N, et al. Heme iron intake and risk of new-onset diabetes in a Mediterranean population at high risk of cardiovascular disease: an observational cohort analysis. <i>BMC Public Health</i> . 2013;13:1042. doi:10.1186/1471-2458-13-1042	Intervention/exposure
67	Ferreira I, Twisk JW, van Mechelen W, Kemper HC, Stehouwer CD. Development of fatness, fitness, and lifestyle from adolescence to the age of 36 years: determinants of the metabolic syndrome in young adults: the amsterdam growth and health longitudinal study. <i>Arch Intern Med</i> . 2005;165(1):42-48. doi:10.1001/archinte.165.1.42	Intervention/exposure
68	Ferreira-Pêgo C, Babio N, Bes-Rastrollo M, et al. Frequent Consumption of Sugar- and Artificially Sweetened Beverages and Natural and Bottled Fruit Juices Is Associated with an Increased Risk of Metabolic Syndrome in a Mediterranean Population at High Cardiovascular Disease Risk. <i>J Nutr</i> . 2016;146(8):1528-1536. doi:10.3945/jn.116.230367	Outcome
69	Fidélis M, Milenkovic D, Sivieri K, Cesar T. Microbiota modulation and effects on metabolic biomarkers by orange juice: a controlled clinical trial. <i>Food Funct</i> . 2020;11(2):1599-1610. doi:10.1039/c9fo02623a	Study design
70	Flieh SM, Miguel-Berges ML, Huybrechts I, et al. Associations between food portion sizes, insulin resistance, VO2 max and metabolic syndrome in European adolescents: The HELENA study. <i>Nutr Metab Cardiovasc Dis</i> . 2022;32(9):2061-2073. doi:10.1016/j.numecd.2022.05.017	Study design
71	Floegel A, Pischon T, Bergmann MM, Teucher B, Kaaks R, Boeing H. Coffee consumption and risk of chronic disease in the European Prospective Investigation into Cancer and Nutrition (EPIC)-Germany study. <i>Am J Clin Nutr</i> . 2012;95(4):901-908. doi:10.3945/ajcn.111.023648	Intervention/exposure
72	Fonollá J, López-Huertas E, Machado FJ, et al. Milk enriched with "healthy fatty acids" improves cardiovascular risk markers and nutritional status in human volunteers. <i>Nutrition</i> . 2009;25(4):408-414. doi:10.1016/j.nut.2008.10.008	Intervention/exposure
73	Fresan U, Gea A, Bes-Rastrollo M, Basterra-Gortari FJ, Carlos S, Martinez-Gonzalez MA. Substitution of water or fresh juice for bottled juice and type 2 diabetes incidence: The SUN cohort study. <i>Nutr Metab Cardiovasc Dis</i> . 2017;27(10):874-880. doi:10.1016/j.numecd.2017.07.010	Intervention/exposure
74	Fuhrman BJ, Smit E, Crespo CJ, Garcia-Palmieri MR. Coffee intake and risk of incident diabetes in Puerto Rican men: results from the Puerto Rico Heart Health Program. <i>Public Health Nutr</i> . 2009;12(6):842-848. doi:10.1017/S1368980008003303	Intervention/exposure
75	Fujioka K, Greenway F, Sheard J, Ying Y. The effects of grapefruit on weight and insulin resistance: relationship to the metabolic syndrome. <i>J Med Food</i> . 2006;9(1):49-54. doi:10.1089/jmf.2006.9.49	Intervention/exposure

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76	Fumeron F, Lamri A, Abi Khalil C, et al. Dairy consumption and the incidence of hyperglycemia and the metabolic syndrome: results from a french prospective study, Data from the Epidemiological Study on the Insulin Resistance Syndrome (DESIR). <i>Diabetes Care</i> . 2011;34(4):813-817. doi:10.2337/dc10-1772	Intervention/exposure
77	Garcia DO, Morrill KE, Aceves B, et al. Feasibility and acceptability of a beverage intervention for Hispanic adults: results from a pilot randomized controlled trial. <i>Public Health Nutr</i> . 2019;22(3):542-552. doi:10.1017/S1368980018003051	Outcome
78	Gilbert JA, Joanisse DR, Chaput JP, et al. Milk supplementation facilitates appetite control in obese women during weight loss: a randomised, single-blind, placebo-controlled trial. <i>Br J Nutr</i> . 2011;105(1):133-143. doi:10.1017/S0007114510003119	Comparator
79	Gonçcalinho GHF, Nascimento JRO, Mito BM, et al. Effects of Coffee on Sirtuin-1, Homocysteine, and Cholesterol of Healthy Adults: Does the Coffee Powder Matter?. <i>J Clin Med</i> . 2022;11(11):2985. doi:10.3390/jcm11112985	Comparator
80	Goto A, Song Y, Chen BH, Manson JE, Buring JE, Liu S. Coffee and caffeine consumption in relation to sex hormone-binding globulin and risk of type 2 diabetes in postmenopausal women. <i>Diabetes</i> . 2011;60(1):269-275. doi:10.2337/db10-1193	Intervention/exposure
81	Green BP, Stevenson EJ, Rumbold PLS. Metabolic, endocrine and appetite-related responses to acute and daily milk snack consumption in healthy, adolescent males. <i>Appetite</i> . 2017;108:93-103. doi:10.1016/j.appet.2016.09.029	Comparator
82	Guess N, Wijesuriya M, Vasantharajah L, et al. The effect of dietary changes on distinct components of the metabolic syndrome in a young Sri Lankan population at high risk of CVD. <i>Br J Nutr</i> . 2016;116(4):719-727. doi:10.1017/S0007114516002476	Intervention/exposure
83	Gul S, Khatoon H, Ahmed N, Rashid H, Mirza AZ. Possible role of grape fruit in controlling hyperglycemia and associated complications: Better glycemic control in healthy subjects through fruits fibers as compared to fruit juices. <i>Bangladesh Journal of Medical Science</i> . 2020;19(3):480-485. doi:10.3329/bjms.v19i3.45866	Outcome
84	Guo H, Zhong R, Liu Y, et al. Effects of bayberry juice on inflammatory and apoptotic markers in young adults with features of non-alcoholic fatty liver disease. <i>Nutrition</i> . 2014;30(2):198-203. doi:10.1016/j.nut.2013.07.023	Comparator
85	Hägele FA, Büsing F, Nas A, et al. High orange juice consumption with or in-between three meals a day differently affects energy balance in healthy subjects. <i>Nutr Diabetes</i> . 2018;8(1):19. doi:10.1038/s41387-018-0031-3	Comparator
86	Ham JY, Shon YH. Natural Magnesium-Enriched Deep-Sea Water Improves Insulin Resistance and the Lipid Profile of Prediabetic Adults: A Randomized, Double-Blinded Crossover Trial. <i>Nutrients</i> . 2020;12(2):515. doi:10.3390/nu12020515	Comparator
87	Hamer M, Witte DR, Mosdøl A, Marmot MG, Brunner EJ. Prospective study of coffee and tea consumption in relation to risk of type 2 diabetes mellitus among men and women: the Whitehall II study. <i>Br J Nutr</i> . 2008;100(5):1046-1053. doi:10.1017/S0007114508944135	Intervention/exposure
88	Harrold JA, Hill S, Radu C, et al. Non-nutritive sweetened beverages versus water after a 52-week weight management programme: a randomised controlled trial. <i>Int J Obes (Lond)</i> . 2024;48(1):83-93. doi:10.1038/s41366-023-01393-3	Duplicate
89	Hayashino Y, Fukuhara S, Okamura T, Tanaka T, Ueshima H; HIPOP-OHP Research Group. High oolong tea consumption predicts future risk of diabetes among Japanese male workers: a prospective cohort study. <i>Diabet Med</i> . 2011;28(7):805-810. doi:10.1111/j.1464-5491.2011.03239.x	Intervention/exposure
90	Hiltunen LA. Are there associations between coffee consumption and glucose tolerance in elderly subjects?. <i>Eur J Clin Nutr</i> . 2006;60(10):1222-1225. doi:10.1038/sj.ejcn.1602441	Intervention/exposure
91	Hinkle SN, Rawal S, Bjerregaard AA, et al. A prospective study of artificially sweetened beverage intake and cardiometabolic health among women at high risk. <i>Am J Clin Nutr</i> . 2019;110(1):221-232. doi:10.1093/ajcn/nqz094	Health status

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92	Hirata A, Ohnaka K, Tashiro N, et al. Effect modification of green tea on the association between rice intake and the risk of diabetes mellitus: a prospective study in Japanese men and women. <i>Asia Pac J Clin Nutr.</i> 2017;26(3):545-555. doi:10.6133/apjcn.042016.04	Intervention/exposure
93	Hirota T, Kawasaki I, Hirota K, et al. P267 - Milk intake accompanied with higher intake of vitamin D might efficiently decrease body fat mass and increase lean mass during moderate weight loss in normal weight young Japanese women. <i>Osteoporosis International.</i> 2010;21:S98. doi:10.1007/s00198-010-1247-9	Publication status
94	Hjellvik V, Tverdal A, Strøm H. Boiled coffee intake and subsequent risk for type 2 diabetes. <i>Epidemiology.</i> 2011;22(3):418-421. doi:10.1097/EDE.0b013e31821083e3	Intervention/exposure
95	Hollands WJ, Armah CN, Doleman JF, Perez-Moral N, Winterbone MS, Kroon PA. 4-Week consumption of anthocyanin-rich blood orange juice does not affect LDL-cholesterol or other biomarkers of CVD risk and glycaemia compared with standard orange juice: a randomised controlled trial. <i>Br J Nutr.</i> 2018;119(4):415-421. doi:10.1017/S0007114517003865	Comparator
96	Hollis JH, Houchins JA, Blumberg JB, Mattes RD. Effects of concord grape juice on appetite, diet, body weight, lipid profile, and antioxidant status of adults. <i>J Am Coll Nutr.</i> 2009;28(5):574-582. doi:10.1080/07315724.2009.10719789	Intervention/exposure
97	Hong X, Xu F, Wang Z, Liang Y, Li J. Dietary patterns and the incidence of hyperglycemia in China. <i>Public Health Nutr.</i> 2016;19(1):131-141. doi:10.1017/S1368980015000774	Intervention/exposure
98	Hosseinpour-Niazi S, Aghayan M, Mirmiran P, Azizi F. Does weight change modify the association between the consumption of sugar-sweetened beverages and 100% fruit juice and the risk of metabolic syndrome?. <i>Clin Nutr.</i> 2021;40(10):5261-5268. doi:10.1016/j.clnu.2021.08.017	Outcome
99	Hou C, Zeng Y, Chen W, et al. Medical conditions associated with coffee consumption: Disease-trajectory and comorbidity network analyses of a prospective cohort study in UK Biobank. <i>Am J Clin Nutr.</i> 2022;116(3):730-740. doi:10.1093/ajcn/nqac148	Outcome
100	Hruby A, Ma J, Rogers G, Meigs JB, Jacques PF. Associations of Dairy Intake with Incident Prediabetes or Diabetes in Middle-Aged Adults Vary by Both Dairy Type and Glycemic Status. <i>J Nutr.</i> 2017;147(9):1764-1775. doi:10.3945/jn.117.253401	Intervention/exposure
101	Hsia DS, Zhang DJ, Beyl RS, Greenway FL, Khoo C. Effect of daily consumption of cranberry beverage on insulin sensitivity and modification of cardiovascular risk factors in adults with obesity: a pilot, randomised, placebo-controlled study. <i>Br J Nutr.</i> 2020;124(6):577-585. doi:10.1017/S0007114520001336	Intervention/exposure
102	Hu G, Jousilahti P, Peltonen M, Bidel S, Tuomilehto J. Joint association of coffee consumption and other factors to the risk of type 2 diabetes: a prospective study in Finland. <i>Int J Obes (Lond).</i> 2006;30(12):1742-1749. doi:10.1038/sj.ijo.0803341	Intervention/exposure
103	Hur YI, Park H, Kang JH, et al. Associations between Sugar Intake from Different Food Sources and Adiposity or Cardio-Metabolic Risk in Childhood and Adolescence: The Korean Child-Adolescent Cohort Study. <i>Nutrients.</i> 2015;8(1):20. doi:10.3390/nu8010020	Intervention/exposure
104	Ibsen DB, Laursen ASD, Lauritzen L, Tjønneland A, Overvad K, Jakobsen MU. Substitutions between dairy product subgroups and risk of type 2 diabetes: the Danish Diet, Cancer and Health cohort. <i>Br J Nutr.</i> 2017;118(11):989-997. doi:10.1017/S0007114517002896	Intervention/exposure
105	Ibsen DB, Overvad K, Laursen ASD, et al. Changes in intake of dairy product subgroups and risk of type 2 diabetes: modelling specified food substitutions in the Danish Diet, Cancer and Health cohort. <i>Eur J Nutr.</i> 2021;60(6):3449-3459. doi:10.1007/s00394-021-02524-0	Intervention/exposure
106	Indrayani, Rahmadi A, Syarifah R, et al. Effect of dates and 7dates on blood glucose levels among adolescent girls: A randomized controlled trial. <i>Pakistan J Medical and Health Sciences.</i> 2021;15:393-398.	Intervention/exposure

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108	Jacobo Cejudo MG, Ochoa-Rosales C, Ahmadizar F, Kavousi M, Geleijnse JM, Voortman T. The healthy beverage index is not associated with insulin resistance, prediabetes and type 2 diabetes risk in the Rotterdam Study. <i>Eur J Nutr.</i> 2023;62(7):3021-3031. doi:10.1007/s00394-023-03209-6	Intervention/exposure
109	Jacobs S, Kröger J, Floegel A, et al. Evaluation of various biomarkers as potential mediators of the association between coffee consumption and incident type 2 diabetes in the EPIC-Potsdam Study. <i>Am J Clin Nutr.</i> 2014;100(3):891-900. doi:10.3945/ajcn.113.080317	Intervention/exposure
110	Jarman M, Mathe N, Ramazani F, et al. Dietary Patterns Prior to Pregnancy and Associations with Pregnancy Complications. <i>Nutrients.</i> 2018;10(7):914. doi:10.3390/nu10070914	Intervention/exposure
111	Jeon J, Jang J, Park K. Effects of Consuming Calcium-Rich Foods on the Incidence of Type 2 Diabetes Mellitus. <i>Nutrients.</i> 2018;11(1):31. doi:10.3390/nu11010031	Intervention/exposure
112	Jin R, Collin L, Vos M, Welsh J. Replacement of sugar-sweetened beverages with water and its impact on insulin sensitivity among overweight adolescents and young adults. <i>FASEB J.</i> 2015;29(S1). doi:10.1096/fasebj.29.1_supplement.584.12	Publication status
113	Jin T, Youn J, Kim AN, et al. Interactions of Habitual Coffee Consumption by Genetic Polymorphisms with the Risk of Prediabetes and Type 2 Diabetes Combined. <i>Nutrients.</i> 2020;12(8):2228. doi:10.3390/nu12082228	Intervention/exposure
114	Johansson I, Esberg A, Nilsson LM, Jansson JH, Wennberg P, Winkvist A. Dairy Product Intake and Cardiometabolic Diseases in Northern Sweden: A 33-Year Prospective Cohort Study. <i>Nutrients.</i> 2019;11(2):284. doi:10.3390/nu11020284	Intervention/exposure
115	Johansson I, Nilsson LM, Esberg A, Jansson JH, Winkvist A. Dairy intake revisited - associations between dairy intake and lifestyle related cardio-metabolic risk factors in a high milk consuming population. <i>Nutr J.</i> 2018;17(1):110. doi:10.1186/s12937-018-0418-y	Intervention/exposure
116	Jung HJ, Han SN, Song S, Paik HY, Baik HW, Joung H. Association between adherence to the Korean Food Guidance System and the risk of metabolic abnormalities in Koreans. <i>Nutr Res Pract.</i> 2011;5(6):560-568. doi:10.4162/nrp.2011.5.6.560	Study design
117	Juturu V, Wilson D, Evans M, Kasper K. Effect of a cranberry beverage on insulinotropic response in moderately hypercholesterolemic overweight/obese adults: a randomized, Double-blind, Placebo-controlled clinical trial. <i>Endocrine Practice.</i> 2011;17:19A.	Publication status
118	Kang Y, Kim J. Soft drink consumption is associated with increased incidence of the metabolic syndrome only in women. <i>Br J Nutr.</i> 2017;117(2):315-324. doi:10.1017/S0007114517000046	Outcome
119	Karimnezhad N, Mahdavi Roshan M, Izaddoust, et al. The simultaneous effects of green coffee and combine exercise training on body composition and glucose homeostasis in obese and overweight women. <i>J Med Plants.</i> 2019;18 (72):215-227. doi:10.29252/JMP.4.72.215	Intervention/exposure
120	Kato M, Noda M, Inoue M, Kadowaki T, Tsugane S; JPHC Study Group. Psychological factors, coffee and risk of diabetes mellitus among middle-aged Japanese: a population-based prospective study in the JPHC study cohort [published correction appears in <i>Endocr J.</i> 2011;58(5):421]. <i>Endocr J.</i> 2009;56(3):459-468. doi:10.1507/endocrj.k09e-003	Intervention/exposure
121	Keshavarz SA, Nourieh Z, Attar MJ, Azadbakht L. Effect of Soymilk Consumption on Waist Circumference and Cardiovascular Risks among Overweight and Obese Female Adults. <i>Int J Prev Med.</i> 2012;3(11):798-805.	Intervention/exposure

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122	Khorraminezhad L, Bilodeau JF, Greffard K, Larose J, Rudkowska I. Impact of Dairy Intake on Plasma F <sub>2</sub> -IsoProstane Profiles in Overweight Subjects with Hyperinsulinemia: A Randomized Crossover Trial. <i>Nutrients</i> . 2021;13(6):2088. doi:10.3390/nu13062088	Intervention/exposure
123	Kim AN, Cho HJ, Youn J, et al. Coffee Consumption, Genetic Polymorphisms, and the Risk of Type 2 Diabetes Mellitus: A Pooled Analysis of Four Prospective Cohort Studies. <i>Int J Environ Res Public Health</i> . 2020;17(15):5379. doi:10.3390/ijerph17155379	Intervention/exposure
124	Kim D, Kim J. Dairy consumption is associated with a lower incidence of the metabolic syndrome in middle-aged and older Korean adults: the Korean Genome and Epidemiology Study (KoGES). <i>Br J Nutr</i> . 2017;117(1):148-160. doi:10.1017/S000711451600444X	Intervention/exposure
125	Kim J. Dairy food consumption is inversely associated with the risk of the metabolic syndrome in Korean adults. <i>J Hum Nutr Diet</i> . 2013;26 Suppl 1:171-179. doi:10.1111/jhn.12098	Study design
126	Kim MJ, Kim JI, Ryu CH, Kang MJ. Effects of Fermented Beverage in Subjects with Metabolic Syndrome. <i>Prev Nutr Food Sci</i> . 2021;26(1):12-20. doi:10.3746/pnf.2021.26.1.12	Intervention/exposure
127	Koloverou E, Panagiotakos DB, Pitsavos C, et al. The evaluation of inflammatory and oxidative stress biomarkers on coffee-diabetes association: results from the 10-year follow-up of the ATTICA Study (2002-2012). <i>Eur J Clin Nutr</i> . 2015;69(11):1220-1225. doi:10.1038/ejcn.2015.98	Intervention/exposure
128	Koloverou E, Panagiotakos DB, Pitsavos C, et al. The long term effect of dietary habits and physical activity on type 2 diabetes incidence: 10-year follow up of the ATTICA study (2002-2012): Diet, physical activity and diabetes. <i>Hell J Atheroscler</i> . 2018;9:5-16.	Intervention/exposure
129	Kummer K, Jensen PN, Kratz M, et al. Full-Fat Dairy Food Intake is Associated with a Lower Risk of Incident Diabetes Among American Indians with Low Total Dairy Food Intake. <i>J Nutr</i> . 2019;149(7):1238-1244. doi:10.1093/jn/nxz058	Intervention/exposure
130	Kwok MK, Leung GM, Schooling CM. Habitual coffee consumption and risk of type 2 diabetes, ischemic heart disease, depression and Alzheimer's disease: a Mendelian randomization study. <i>Sci Rep</i> . 2016;6:36500. doi:10.1038/srep36500	Intervention/exposure
131	Lee HA, Son N, Lee WK, Park H. A Diabetes-Related Dietary Pattern Is Associated with Incident Diabetes in Obese Men in the Korean Genome Epidemiology Study. <i>J Nutr</i> . 2019;149(2):323-329. doi:10.1093/jn/nxy274	Intervention/exposure
132	Lee JH, Oh MK, Lim JT, Kim HG, Lee WJ. Effect of Coffee Consumption on the Progression of Type 2 Diabetes Mellitus among Prediabetic Individuals. <i>Korean J Fam Med</i> . 2016;37(1):7-13. doi:10.4082/kjfm.2016.37.1.7	Intervention/exposure
133	Lee JK, Kim K, Ahn Y, Yang M, Lee JE. Habitual coffee intake, genetic polymorphisms, and type 2 diabetes. <i>Eur J Endocrinol</i> . 2015;172(5):595-601. doi:10.1530/EJE-14-0805	Intervention/exposure
134	Lee KW, Shin D. A Healthy Beverage Consumption Pattern Is Inversely Associated with the Risk of Obesity and Metabolic Abnormalities in Korean Adults. <i>J Med Food</i> . 2018;21(9):935-945. doi:10.1089/jmf.2017.0119	Study design
135	Leermakers ET, Felix JF, Jaddoe VW, Raat H, Franco OH, Kiefte-de Jong JC. Sugar-containing beverage intake at the age of 1 year and cardiometabolic health at the age of 6 years: the Generation R Study. <i>Int J Behav Nutr Phys Act</i> . 2015;12:114. doi:10.1186/s12966-015-0278-1	Outcome
136	Li M, Yang L, Ma M, Liu Y. Improving the metabolism of glucose and lipids in patients with prediabetes by affecting the gut microbiota. <i>Diabetes Metab Res Rev</i> . 2017;33:S1. doi:10.1002/dmrr.2948	Publication status
137	Li X, Zeng J, Chen B, et al. Daily higher tea consumption is associated with a reduced risk of type 2 diabetes: A cohort study and updated systematic review and meta-analysis. <i>Nutr Res</i> . 2023;118:116-127. doi:10.1016/j.nutres.2023.08.002	Intervention/exposure



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138	Li X, Yin J, Zhu Y, et al. Effects of whole milk supplementation on gut microbiota and cardiometabolic biomarkers in subjects with and without lactose malabsorption. <i>Nutrients</i> . 2018;10(10):1403. doi:10.3390/nu10101403	Study design
139	Li Y, Wang DD, Ley SH, et al. Time Trends of Dietary and Lifestyle Factors and Their Potential Impact on Diabetes Burden in China [published correction appears in <i>Diabetes Care</i> . 2018 May;41(5):1116. doi: 10.2337/dc18-er05]. <i>Diabetes Care</i> . 2017;40(12):1685-1694. doi:10.2337/dc17-0571	Study design
140	Lima ACD, Cecatti C, Fidélis MP, et al. Effect of Daily Consumption of Orange Juice on the Levels of Blood Glucose, Lipids, and Gut Microbiota Metabolites: Controlled Clinical Trials. <i>J Med Food</i> . 2019;22(2):202-210. doi:10.1089/jmf.2018.0080	Study design
141	Liu M, Chen QT, Li ZC, Zhang J, Wang PG, He QQ. Association Between Diet Quality and Cardiometabolic Risk Factor Clustering Stratified by Socioeconomic Status Among Chinese Children. <i>J Acad Nutr Diet</i> . 2021;121(10):1975-1983.e2. doi:10.1016/j.jand.2021.03.009	Intervention/exposure
142	Liu S, van der Schouw YT, Soedamah-Muthu SS, Spijkerman AMW, Sluijs I. Intake of dietary saturated fatty acids and risk of type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition-Netherlands cohort: associations by types, sources of fatty acids and substitution by macronutrients. <i>Eur J Nutr</i> . 2019;58(3):1125-1136. doi:10.1007/s00394-018-1630-4	Intervention/exposure
143	Liu X, Xu W, Cai H, et al. Green tea consumption and risk of type 2 diabetes in Chinese adults: the Shanghai Women's Health Study and the Shanghai Men's Health Study. <i>Int J Epidemiol</i> . 2018;47(6):1887-1896. doi:10.1093/ije/dyy173	Country
144	Löfvenborg JE, Andersson T, Carlsson PO, et al. Sweetened beverage intake and risk of latent autoimmune diabetes in adults (LADA) and type 2 diabetes. <i>Eur J Endocrinol</i> . 2016;175(6):605-614. doi:10.1530/EJE-16-0376	Study design
145	Louie JC, Flood VM, Rangan AM, et al. Higher regular fat dairy consumption is associated with lower incidence of metabolic syndrome but not type 2 diabetes. <i>Nutr Metab Cardiovasc Dis</i> . 2013;23(9):816-821. doi:10.1016/j.numecd.2012.08.004	Intervention/exposure
146	Lu J, Wang Z. C-reactive protein partially mediates the inverse effect of coffee consumption on risk of type 2 diabetes: Evidence from two-stage Mendelian randomization analysis. <i>Clin Nutr</i> . 2023;42(9):1747-1748. doi:10.1016/j.clnu.2023.07.024	Intervention/exposure
147	Luo Y, He L, Ma T, et al. Associations between consumption of three types of beverages and risk of cardiometabolic multimorbidity in UK Biobank participants: a prospective cohort study. <i>BMC Med</i> . 2022;20(1):273. doi:10.1186/s12916-022-02456-4	Outcome
148	Ma J, Jacques PF, Meigs JB, et al. Sugar-Sweetened Beverage but Not Diet Soda Consumption Is Positively Associated with Progression of Insulin Resistance and Prediabetes. <i>J Nutr</i> . 2016;146(12):2544-2550. doi:10.3945/jn.116.234047	Outcome
149	Ma Z, Hao M. Longitudinal study of the relationship between coffee consumption and type 2 diabetes in Chinese adult residents: Data from China Health and Nutrition Survey. <i>PLoS One</i> . 2021;16(5):e0251377. doi:10.1371/journal.pone.0251377	Country
150	Malik VS, Sun Q, van Dam RM, et al. Adolescent dairy product consumption and risk of type 2 diabetes in middle-aged women. <i>Am J Clin Nutr</i> . 2011;94(3):854-861. doi:10.3945/ajcn.110.009621	Intervention/exposure
151	Martin KR, Coles KM. Consumption of 100% Tart Cherry Juice Reduces Serum Urate in Overweight and Obese Adults. <i>Curr Dev Nutr</i> . 2019;3(5):nzz011. doi:10.1093/cdn/nzz011	Intervention/exposure
152	Martini D, Rosi A, Tassotti M, et al. Effect of coffee and cocoa-based confectionery containing coffee on markers of cardiometabolic health: results from the pocket-4-life project. <i>Eur J Nutr</i> . 2021;60(3):1453-1463. doi:10.1007/s00394-020-02347-5	Intervention/exposure
153	Maskarinec G, Kristal BS, Wilkens LR, et al. Risk Factors for Type 2 Diabetes in the Multiethnic Cohort. <i>Can J Diabetes</i> . 2023;47(8):627-635.e2. doi:10.1016/j.cjcd.2023.06.004	Intervention/exposure

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154	Maskarinec G, Kristal BS, Wilkens LR, et al. Risk Factors for Type 2 Diabetes in the Multiethnic Cohort. <i>Can J Diabetes</i> . 2023;47(8):627-635.e2. doi:10.1016/j.jcjd.2023.06.004	Duplicate
155	Mena P, Tassotti M, Rosi A, et al. A comprehensive approach to the bioavailability and cardiometabolic effects of the bioactive compounds present in espresso coffee and confectionery-derived coffee. <i>Proc Nutr Soc</i> . 2020;79(OCE2):E123. doi:10.1017/S0029665120000713	Publication status
156	Miranda AM, Steluti J, Fisberg RM, Marchioni DM. Association between Coffee Consumption and Its Polyphenols with Cardiovascular Risk Factors: A Population-Based Study. <i>Nutrients</i> . 2017;9(3):276. doi:10.3390/nu9030276	Study design
157	Mirmiran P, Carlström M, Bahadoran Z, Azizi F. Long-term effects of coffee and caffeine intake on the risk of pre-diabetes and type 2 diabetes: Findings from a population with low coffee consumption. <i>Nutr Metab Cardiovasc Dis</i> . 2018;28(12):1261-1266. doi:10.1016/j.numecd.2018.09.001	Intervention/exposure
158	Moreira TKB, Santos HCD, Mendes FD, Molina MDCB, Mill JG, Faria CP. Examining the Usage Patterns of Non-Nutritive Sweeteners among Non-Diabetic Individuals: Insights from the Longitudinal Study of Adult Health (ELSA-Brasil). <i>Nutrients</i> . 2023;15(22):4785. doi:10.3390/nu15224785	Study design
159	Moreno LA, Bel-Serrat S, Santaliesra-Pasías A, Bueno G. Dairy products, yogurt consumption, and cardiometabolic risk in children and adolescents. <i>Nutr Rev</i> . 2015;73 Suppl 1:8-14. doi:10.1093/nutrit/nuv014	Study design
160	Moslehi N, Shab-Bidar S, Mirmiran P, Sadeghi M, Azizi F. Associations between dairy products consumption and risk of type 2 diabetes: Tehran lipid and glucose study. <i>Int J Food Sci Nutr</i> . 2015;66(6):692-699. doi:10.3109/09637486.2015.1034249	Intervention/exposure
161	Mueller NT, Odegaard AO, Gross MD, et al. Soy intake and risk of type 2 diabetes in Chinese Singaporeans [corrected] [published correction appears in Eur J Nutr. Eur J Nutr. 2012 Dec;51(8):1041]. <i>Eur J Nutr</i> . 2012;51(8):1033-1040. doi:10.1007/s00394-011-0276-2	Intervention/exposure
162	Mukamal KJ, MacDermott K, Vinson JA, Oyama N, Manning WJ, Mittleman MA. A 6-month randomized pilot study of black tea and cardiovascular risk factors. <i>Am Heart J</i> . 2007;154(4):724.e1-724.e7246. doi:10.1016/j.ahj.2007.07.008	Intervention/exposure
163	Muraki I, Imamura F, Manson JE, et al. Fruit consumption and risk of type 2 diabetes: results from three prospective longitudinal cohort studies [published correction appears in BMJ. 2013;347:f6935]. <i>BMJ</i> . 2013;347:f5001. doi:10.1136/bmj.f5001	Intervention/exposure
164	Mursu J, Virtanen JK, Tuomainen TP, Nurmi T, Voutilainen S. Intake of fruit, berries, and vegetables and risk of type 2 diabetes in Finnish men: the Kuopio Ischaemic Heart Disease Risk Factor Study. <i>Am J Clin Nutr</i> . 2014;99(2):328-333. doi:10.3945/ajcn.113.069641	Intervention/exposure
165	Nakamura Y, Watanabe H, Tanaka A, Yasui M, Nishihira J, Murayama N. Effect of increased daily water intake and hydration on health in Japanese adults. <i>Nutrients</i> . 2020;12(4):1191. doi:10.3390/nu12041191	Intervention/exposure
166	Nettleton JA, Harnack LJ, Scrafford CG, Mink PJ, Barraj LM, Jacobs DR Jr. Dietary flavonoids and flavonoid-rich foods are not associated with risk of type 2 diabetes in postmenopausal women. <i>J Nutr</i> . 2006;136(12):3039-3045. doi:10.1093/jn/136.12.3039	Intervention/exposure
167	Nettleton JA, Steffen LM, Ni H, Liu K, Jacobs DR Jr. Dietary patterns and risk of incident type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). <i>Diabetes Care</i> . 2008;31(9):1777-1782. doi:10.2337/dc08-0760	Other (e.g., duplicative data)
168	Ng R, Sutradhar R, Yao Z, Wodchis WP, Rosella LC. Smoking, drinking, diet and physical activity-modifiable lifestyle risk factors and their associations with age to first chronic disease. <i>Int J Epidemiol</i> . 2020;49(1):113-130. doi:10.1093/ije/dyz078	Intervention/exposure
169	Nie J, Yu C, Guo Y, et al. Tea consumption and long-term risk of type 2 diabetes and diabetic complications: a cohort study of 0.5 million Chinese adults. <i>Am J Clin Nutr</i> . 2021;114(1):194-202. doi:10.1093/ajcn/nqab006	Country

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170	Nordestgaard AT, Thomsen M, Nordestgaard BG. Coffee intake and risk of obesity, metabolic syndrome and type 2 diabetes: a Mendelian randomization study. <i>Int J Epidemiol.</i> 2015;44(2):551-565. doi:10.1093/ije/dyv083	Intervention/exposure
171	Oba S, Nagata C, Nakamura K, et al. Consumption of coffee, green tea, oolong tea, black tea, chocolate snacks and the caffeine content in relation to risk of diabetes in Japanese men and women. <i>Br J Nutr.</i> 2010;103(3):453-459. doi:10.1017/S0007114509991966	Intervention/exposure
172	Ochoa-Rosales C, van der Schaft N, Braun KVE, et al. C-reactive protein partially mediates the inverse association between coffee consumption and risk of type 2 diabetes: The UK Biobank and the Rotterdam study cohorts. <i>Clin Nutr.</i> 2023;42(5):661-669. doi:10.1016/j.clnu.2023.02.024	Intervention/exposure
173	O'Connor LM, Lentjes MA, Luben RN, Khaw KT, Wareham NJ, Forouhi NG. Dietary dairy product intake and incident type 2 diabetes: a prospective study using dietary data from a 7-day food diary. <i>Diabetologia.</i> 2014;57(5):909-917. doi:10.1007/s00125-014-3176-1	Intervention/exposure
174	O'Connor S, Julien P, Weisnagel SJ, Gagnon C, Rudkowska I. Impact of a High Intake of Dairy Product on Insulin Sensitivity in Hyperinsulinemic Adults: A Crossover Randomized Controlled Trial. <i>Curr Dev Nutr.</i> 2019;3(8):nzz083. doi:10.1093/cdn/nzz083	Intervention/exposure
175	Odegaard AO, Pereira MA, Koh WP, Arakawa K, Lee HP, Yu MC. Coffee, tea, and incident type 2 diabetes: the Singapore Chinese Health Study. <i>Am J Clin Nutr.</i> 2008;88(4):979-985. doi:10.1093/ajcn/88.4.979	Intervention/exposure
176	Ohnaka K, Ikeda M, Maki T, et al. Effects of 16-week consumption of caffeinated and decaffeinated instant coffee on glucose metabolism in a randomized controlled trial. <i>J Nutr Metab.</i> 2012;2012:207426. doi:10.1155/2012/207426	Intervention/exposure
177	Ohnaka K, Ikeda M, Maki T, et al. Effects of 16-week consumption of caffeinated and decaffeinated instant coffee on glucose metabolism in a randomized controlled trial. <i>J Nutr Metab.</i> 2012;2012:207426. doi:10.1155/2012/207426	Duplicate
178	Olofsson C, Discacciati A, Åkesson A, Orsini N, Brismar K, Wolk A. Changes in fruit, vegetable and juice consumption after the diagnosis of type 2 diabetes: a prospective study in men. <i>Br J Nutr.</i> 2017;117(5):712-719. doi:10.1017/S0007114516002257	Intervention/exposure
179	Olsen NJ, Andersen LB, Wedderkopp N, Kristensen PL, Heitmann BL. Intake of liquid and solid sucrose in relation to changes in body fatness over 6 years among 8- to 10-year-old children: the European Youth Heart Study. <i>Obes Facts.</i> 2012;5(4):506-512. doi:10.1159/000341631	Intervention/exposure
180	Olsson K, González-Padilla E, Janzi S, et al. Clusters of carbohydrate-rich foods and associations with type 2 diabetes incidence: a prospective cohort study. <i>Nutr J.</i> 2023;22(1):71. doi:10.1186/s12937-023-00906-0	Intervention/exposure
181	Palatini P, Benetti E, Mos L, et al. Association of coffee consumption and CYP1A2 polymorphism with risk of impaired fasting glucose in hypertensive patients. <i>Eur J Epidemiol.</i> 2015;30(3):209-217. doi:10.1007/s10654-015-9990-z	Intervention/exposure
182	Papier K, D'Este C, Bain C, et al. Consumption of sugar-sweetened beverages and type 2 diabetes incidence in Thai adults: results from an 8-year prospective study. <i>Nutr Diabetes.</i> 2017;7(6):e283. doi:10.1038/nutd.2017.27	Country
183	Parnell LD, Noel SE, Bhupathiraju SN, et al. Metabolite patterns link diet, obesity, and type 2 diabetes in a Hispanic population. <i>Metabolomics.</i> 2021;17(10):88. doi:10.1007/s11306-021-01835-x	Intervention/exposure
184	Pereira MA, Jacobs DR Jr, Van Horn L, Slattery ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. <i>JAMA.</i> 2002;287(16):2081-2089. doi:10.1001/jama.287.16.2081	Intervention/exposure
185	Pereira MA, Parker ED, Folsom AR. Coffee consumption and risk of type 2 diabetes mellitus: an 11-year prospective study of 28 812 postmenopausal women. <i>Arch Intern Med.</i> 2006;166(12):1311-1316. doi:10.1001/archinte.166.12.1311	Intervention/exposure

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186	Perelli L, Alcaraz A, Vianna CMM, et al. Health and economic burden of sugar-sweetened beverages consumption in Brazil. <i>Cad Saude Publica</i> . 2023;39(12):e00249422. doi:10.1590/0102-311XEN249422	Study design
187	Platt DE, Ghassibe-Sabbagh M, Salameh P, et al. Caffeine Impact on Metabolic Syndrome Components Is Modulated by a CYP1A2 Variant. <i>Ann Nutr Metab</i> . 2016;68(1):1-11. doi:10.1159/000441481	Study design
188	Ponce O, Benassi R, Cesar T. Orange juice associated with a balanced diet mitigated risk factors of metabolic syndrome: A randomized controlled trial. <i>J Nutr Intermed Metab</i> . 2019;17:100101. doi:10.1016/j.jnim.2019.100101	Intervention/exposure
189	Rezvani R, Cianflone K, McGahan JP, et al. Effects of sugar-sweetened beverages on plasma acylation stimulating protein, leptin and adiponectin: relationships with metabolic outcomes. <i>Obesity (Silver Spring)</i> . 2013;21(12):2471-2480. doi:10.1002/oby.20437	Intervention/exposure
190	Ribeiro C, Dourado G, Cesar T. Orange juice allied to a reduced-calorie diet results in weight loss and ameliorates obesity-related biomarkers: A randomized controlled trial. <i>Nutrition</i> . 2017;38:13-19. doi:10.1016/j.nut.2016.12.020	Intervention/exposure
191	Rivera-Paredes B, Torres-Ibarra L, González-Morales R, et al. Cumulative soft drink consumption is associated with insulin resistance in Mexican adults. <i>Am J Clin Nutr</i> . 2020;112(3):661-668. doi:10.1093/ajcn/nqaa169	Outcome
192	Rosado JL, Garcia OP, Ronquillo D, et al. Intake of milk with added micronutrients increases the effectiveness of an energy-restricted diet to reduce body weight: a randomized controlled clinical trial in Mexican women. <i>J Am Diet Assoc</i> . 2011;111(10):1507-1516. doi:10.1016/j.jada.2011.07.011	Intervention/exposure
193	Rosengren A, Dotevall A, Wilhelmsen L, Thelle D, Johansson S. Coffee and incidence of diabetes in Swedish women: a prospective 18-year follow-up study. <i>J Intern Med</i> . 2004;255(1):89-95. doi:10.1046/j.1365-2796.2003.01260.x	Intervention/exposure
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195	Said MA, van de Vegte YJ, Verweij N, van der Harst P. Associations of Observational and Genetically Determined Caffeine Intake With Coronary Artery Disease and Diabetes Mellitus. <i>J Am Heart Assoc</i> . 2020;9(24):e016808. doi:10.1161/JAHA.120.016808	Intervention/exposure
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197	Samara A, Herbeth B, Ndiaye NC, et al. Dairy product consumption, calcium intakes, and metabolic syndrome-related factors over 5 years in the STANISLAS study. <i>Nutrition</i> . 2013;29(3):519-524. doi:10.1016/j.nut.2012.08.013	Intervention/exposure
198	Sarriá B, Martínez-López S, Sierra-Cinos JL, García-Diz L, Mateos R, Bravo-Clemente L. Regularly consuming a green/roasted coffee blend reduces the risk of metabolic syndrome. <i>Eur J Nutr</i> . 2018;57(1):269-278. doi:10.1007/s00394-016-1316-8	Comparator
199	Sarriá B, Sierra-Cinos JL, García-Diz L, Martínez-López S, Mateos R, Bravo-Clemente L. Green/Roasted Coffee May Reduce Cardiovascular Risk in Hypercholesterolemic Subjects by Decreasing Body Weight, Abdominal Adiposity and Blood Pressure. <i>Foods</i> . 2020;9(9):1191. doi:10.3390/foods9091191	Other (e.g., duplicative data)
200	Sartorelli DS, Fagherazzi G, Balkau B, et al. Differential effects of coffee on the risk of type 2 diabetes according to meal consumption in a French cohort of women: the E3N/EPIC cohort study. <i>Am J Clin Nutr</i> . 2010;91(4):1002-1012. doi:10.3945/ajcn.2009.28741	Intervention/exposure
201	Scheffers FR, Boer JM, Wijga AH, van der Schouw YT, Smit HA, Verschuren WM. Substitution of pure fruit juice for fruit and sugar-sweetened beverages and cardiometabolic risk in European Prospective Investigation into Cancer and Nutrition (EPIC)-NL: a prospective cohort study. <i>Public Health Nutr</i> . 2022;25(6):1504-1514. doi:10.1017/S1368980021000914	Intervention/exposure

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203	Schmidt KA, Cromer G, Burhans MS, et al. The impact of diets rich in low-fat or full-fat dairy on glucose tolerance and its determinants: a randomized controlled trial. <i>Am J Clin Nutr.</i> 2021;113(3):534-547. doi:10.1093/ajcn/nqaa301	Intervention/exposure
204	Schoppen S, Pérez-Granados AM, Carbajal A, et al. A sodium-rich carbonated mineral water reduces cardiovascular risk in postmenopausal women. <i>J Nutr.</i> 2004;134(5):1058-1063. doi:10.1093/jn/134.5.1058	Study design
205	Schulze MB, Hoffmann K, Boeing H, et al. An accurate risk score based on anthropometric, dietary, and lifestyle factors to predict the development of type 2 diabetes. <i>Diabetes Care.</i> 2007;30(3):510-515. doi:10.2337/dc06-2089	Study design
206	Scott C, Au-Yeung F, Strom N, et al. PTFS08-02-23 Comparison of a Daily Stevia Beverage vs. a Sucrose Sweetened Beverage on the Human Gut Microbiome, Cardiometabolic Functions and Anthropometric Measurements. <i>Curr Dev Nutr.</i> 2023;7:S1. doi:10.1016/j.cdnut.2023.101447	Publication status
207	Shenoy SF, Poston WS, Reeves RS, et al. Weight loss in individuals with metabolic syndrome given DASH diet counseling when provided a low sodium vegetable juice: a randomized controlled trial. <i>Nutr J.</i> 2010;9:8. doi:10.1186/1475-2891-9-8	Intervention/exposure
208	Shi L, Brunius C, Johansson I, et al. Plasma metabolite biomarkers of boiled and filtered coffee intake and their association with type 2 diabetes risk. <i>J Intern Med.</i> 2020;287(4):405-421. doi:10.1111/joim.13009	Comparator
209	Shin H, Yoon YS, Lee Y, Kim CI, Oh SW. Dairy product intake is inversely associated with metabolic syndrome in Korean adults: Anseong and Ansan cohort of the Korean Genome and Epidemiology Study. <i>J Korean Med Sci.</i> 2013;28(10):1482-1488. doi:10.3346/jkms.2013.28.10.1482	Intervention/exposure
210	Simão TN, Lozovoy MA, Simão AN, et al. Reduced-energy cranberry juice increases folic acid and adiponectin and reduces homocysteine and oxidative stress in patients with the metabolic syndrome. <i>Br J Nutr.</i> 2013;110(10):1885-1894. doi:10.1017/S0007114513001207	Outcome
211	Siqueira JH, Pereira TSS, Moreira AD, et al. Consumption of sugar-sweetened soft drinks and risk of metabolic syndrome and its components: results of the ELSA-Brasil study (2008-2010 and 2012-2014). <i>J Endocrinol Invest.</i> 2023;46(1):159-171. doi:10.1007/s40618-022-01895-3	Outcome
212	Sluijs I, Forouhi NG, Beulens JW, et al. The amount and type of dairy product intake and incident type 2 diabetes: results from the EPIC-InterAct Study. <i>Am J Clin Nutr.</i> 2012;96(2):382-390. doi:10.3945/ajcn.111.021907	Intervention/exposure
213	Sluijs I, Forouhi NG, Beulens JW, et al. The amount and type of dairy product intake and incident type 2 diabetes: results from the EPIC-InterAct Study. <i>Am J Clin Nutr.</i> 2012;96(2):382-390. doi:10.3945/ajcn.111.021907	Duplicate
214	Slurink IA, Chen L, Magliano DJ, Kupper N, Smeets T, Soedamah-Muthu SS. Dairy Product Consumption and Incident Prediabetes in the Australian Diabetes, Obesity, and Lifestyle Study With 12 Years of Follow-Up. <i>J Nutr.</i> 2023;153(6):1742-1752. doi:10.1016/j.tjnut.2023.03.032	Intervention/exposure
215	Slurink IA, Corpeleijn E, Bakker SJ, et al. Dairy consumption and incident prediabetes: prospective associations and network models in the large population-based Lifelines Study. <i>Am J Clin Nutr.</i> 2023;118(6):1077-1090. doi:10.1016/j.ajcnut.2023.10.002	Intervention/exposure
216	Slurink IA, Corpeleijn E, Bakker SJ, et al. Dairy consumption and incident prediabetes: prospective associations and network models in the large population-based Lifelines Study. <i>Am J Clin Nutr.</i> 2023;118(6):1077-1090. doi:10.1016/j.ajcnut.2023.10.002	Duplicate

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218	Slurink IAL, Voortman T, Ochoa-Rosales C, et al. Dairy Product Consumption in Relation to Incident Prediabetes and Longitudinal Insulin Resistance in the Rotterdam Study. <i>Nutrients.</i> 2022;14(3):415. doi:10.3390/nu14030415	Intervention/exposure
219	Smith B, Wingard DL, Smith TC, Kritz-Silverstein D, Barrett-Connor E. Does coffee consumption reduce the risk of type 2 diabetes in individuals with impaired glucose?. <i>Diabetes Care.</i> 2006;29(11):2385-2390. doi:10.2337/dc06-1084	Intervention/exposure
220	Soedamah-Muthu SS, Masset G, Verberne L, Geleijnse JM, Brunner EJ. Consumption of dairy products and associations with incident diabetes, CHD and mortality in the Whitehall II study. <i>Br J Nutr.</i> 2013;109(4):718-726. doi:10.1017/S0007114512001845	Intervention/exposure
221	Song Y, Manson JE, Buring JE, Sesso HD, Liu S. Associations of dietary flavonoids with risk of type 2 diabetes, and markers of insulin resistance and systemic inflammation in women: a prospective study and cross-sectional analysis. <i>J Am Coll Nutr.</i> 2005;24(5):376-384. doi:10.1080/07315724.2005.10719488	Intervention/exposure
222	Stanhope KL, Schwarz JM, Keim NL, et al. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. <i>J Clin Invest.</i> 2009;119(5):1322-1334. doi:10.1172/JCI37385	Intervention/exposure
223	St-Onge MP, Goree LL, Gower B. High-milk supplementation with healthy diet counseling does not affect weight loss but ameliorates insulin action compared with low-milk supplementation in overweight children. <i>J Nutr.</i> 2009;139(5):933-938. doi:10.3945/jn.108.102079	Comparator
224	Struijk EA, Heraclides A, Witte DR, et al. Dairy product intake in relation to glucose regulation indices and risk of type 2 diabetes. <i>Nutr Metab Cardiovasc Dis.</i> 2013;23(9):822-828. doi:10.1016/j.numecd.2012.05.011	Intervention/exposure
225	Stuber JM, Vissers LET, Verschuren WMM, Boer JMA, van der Schouw YT, Sluijs I. Substitution among milk and yogurt products and the risk of incident type 2 diabetes in the EPIC-NL cohort. <i>J Hum Nutr Diet.</i> 2021;34(1):54-63. doi:10.1111/jhn.12767	Intervention/exposure
226	Swarbrick MM, Stanhope KL, Elliott SS, et al. Consumption of fructose-sweetened beverages for 10 weeks increases postprandial triacylglycerol and apolipoprotein-B concentrations in overweight and obese women. <i>Br J Nutr.</i> 2008;100(5):947-952. doi:10.1017/S0007114508968252	Intervention/exposure
227	Taba N, Valge HK, Metspalu A, et al. Mendelian Randomization Identifies the Potential Causal Impact of Dietary Patterns on Circulating Blood Metabolites. <i>Front Genet.</i> 2021;12:738265. doi:10.3389/fgene.2021.738265	Outcome
228	Talaei M, Pan A, Yuan JM, Koh WP. Dairy intake and risk of type 2 diabetes. <i>Clin Nutr.</i> 2018;37(2):712-718. doi:10.1016/j.clnu.2017.02.022	Intervention/exposure
229	Tan LJ, Jeon HJ, Park S, et al. Association of Coffee Consumption and Its Types According to Addition of Sugar and Creamer with Metabolic Syndrome Incidence in a Korean Population from the Health Examinees (HEXA) Study. <i>Nutrients.</i> 2021;13(3):920. doi:10.3390/nu13030920	Intervention/exposure
230	Tanaka S, Uenishi K, Ishida, et al. A randomized intervention trial of 24-wk dairy consumption on waist circumference, blood pressure, and fasting blood sugar and lipids in Japanese men with metabolic syndrome. <i>J Nutr Sci Vitaminol (Tokyo).</i> 2014. 60:305-12. doi:10.3177/jnsv.60.305 .	Intervention/exposure
231	Tate DF, Turner-McGrievy G, Stevens J, et al. Replacing caloric beverages with water or diet beverages for weight loss in adults: results of a 6-month randomized controlled trial. <i>Obesity (Silver Spring).</i> 2011;19:S68. doi:10.1038/oby.2011.222	Publication status
232	Tehrani HG, Allahdadian M, Zarre F, Ranjbar H, Allahdadian F. Effect of green tea on metabolic and hormonal aspect of polycystic ovarian syndrome in overweight and obese women suffering from polycystic ovarian syndrome: A clinical trial. <i>J Educ Health Promot.</i> 2017;6:36. doi:10.4103/jehp.jehp_67_15	Intervention/exposure

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234	Toxqui L, Vaquero MP. An Intervention with Mineral Water Decreases Cardiometabolic Risk Biomarkers. A Crossover, Randomised, Controlled Trial with Two Mineral Waters in Moderately Hypercholesterolaemic Adults. <i>Nutrients</i> . 2016;8(7):400. doi:10.3390/nu8070400	Comparator
235	Trapp G, Hurworth M, Jacoby P, et al. Energy drink intake and metabolic syndrome: A prospective investigation in young adults. <i>Nutr Metab Cardiovasc Dis</i> . 2020;30(10):1679-1684. doi:10.1016/j.numecd.2020.06.012	Intervention/exposure
236	Trichia E, Koulman A, Stewart ID, et al. Plasma Metabolites Related to the Consumption of Different Types of Dairy Products and Their Association with New-Onset Type 2 Diabetes: Analyses in the Fenland and EPIC-Norfolk Studies, United Kingdom. <i>Mol Nutr Food Res</i> . 2024;68(1):e2300154. doi:10.1002/mnfr.202300154	Intervention/exposure
237	Trichia E, Luben R, Khaw KT, Wareham NJ, Imamura F, Forouhi NG. The associations of longitudinal changes in consumption of total and types of dairy products and markers of metabolic risk and adiposity: findings from the European Investigation into Cancer and Nutrition (EPIC)-Norfolk study, United Kingdom. <i>Am J Clin Nutr</i> . 2020;111(5):1018-1026. doi:10.1093/ajcn/nqz335	Intervention/exposure
238	Tsai KZ, Huang WC, Sui X, Lavie CJ, Lin GM. Moderate or greater daily coffee consumption is associated with lower incidence of metabolic syndrome in Taiwanese militaries: results from the CHIEF cohort study. <i>Front Nutr</i> . 2023;10:1321916. doi:10.3389/fnut.2023.1321916	Intervention/exposure
239	Tsang C, Smail NF, Almoosawi S, Davidson I, Al-Dujaili EA. Intake of polyphenol-rich pomegranate pure juice influences urinary glucocorticoids, blood pressure and homeostasis model assessment of insulin resistance in human volunteers. <i>J Nutr Sci</i> . 2012;1:e9. doi:10.1017/jns.2012.10	Comparator
240	Tsitsimpikou C, Tsarouhas K, Kioukia-Fougia N, et al. Dietary supplementation with tomato-juice in patients with metabolic syndrome: a suggestion to alleviate detrimental clinical factors. <i>Food Chem Toxicol</i> . 2014;74:9-13. doi:10.1016/j.fct.2014.08.014	Intervention/exposure
241	Tuomilehto J, Hu G, Bidel S, Lindström J, Jousilahti P. Coffee consumption and risk of type 2 diabetes mellitus among middle-aged Finnish men and women. <i>JAMA</i> . 2004;291(10):1213-1219. doi:10.1001/jama.291.10.1213	Intervention/exposure
242	van Dam RM, Dekker JM, Nijpels G, et al. Coffee consumption and incidence of impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes: the Hoorn Study. <i>Diabetologia</i> . 2004;47(12):2152-2159. doi:10.1007/s00125-004-1573-6	Intervention/exposure
243	van Dam RM, Feskens EJ. Coffee consumption and risk of type 2 diabetes mellitus. <i>Lancet</i> . 2002;360(9344):1477-1478. doi:10.1016/S0140-6736(02)11436-X	Intervention/exposure
244	van Dam RM, Pasma WJ, Verhoef P. Effects of coffee consumption on fasting blood glucose and insulin concentrations: randomized controlled trials in healthy volunteers. <i>Diabetes Care</i> . 2004;27(12):2990-2992. doi:10.2337/diacare.27.12.2990	Intervention/exposure
245	van Dam RM, Willett WC, Manson JE, Hu FB. Coffee, caffeine, and risk of type 2 diabetes: a prospective cohort study in younger and middle-aged U.S. women. <i>Diabetes Care</i> . 2006;29(2):398-403. doi:10.2337/diacare.29.02.06.dc05-1512	Intervention/exposure
246	van Dieren S, Uiterwaal CS, van der Schouw, et al. Coffee and tea consumption and risk of type 2 diabetes. <i>Diabetologia</i> . 2009;52:2561-9. doi:10.1007/s00125-009-1516-3	Intervention/exposure
247	Van Hulst A, Ybarra M, Mathieu ME, Benedetti A, Paradis G, Henderson M. Determinants of new onset cardiometabolic risk among normal weight children. <i>Int J Obes (Lond)</i> . 2020;44(4):781-789. doi:10.1038/s41366-019-0483-0	Outcome
248	van 't Riet E, Dekker JM, Sun Q, Nijpels G, Hu FB, van Dam RM. Role of adiposity and lifestyle in the relationship between family history of diabetes and 20-year incidence of type 2 diabetes in U.S. women. <i>Diabetes Care</i> . 2010;33(4):763-767. doi:10.2337/dc09-1586	Intervention/exposure

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249	van Woudenberg GJ, Kuijsten A, et al. Tea consumption and incidence of type 2 diabetes in Europe: the EPIC-InterAct case-cohort study. <i>PLoS One</i> . 2012;7(5):e36910. doi:10.1371/journal.pone.0036910	Intervention/exposure
250	Ventura AK, Loken E, Birch LL. Risk profiles for metabolic syndrome in a nonclinical sample of adolescent girls. <i>Pediatrics</i> . 2006;118(6):2434-2442. doi:10.1542/peds.2006-1527	Outcome
251	Vieira Senger AE, Schwanke CH, Gomes I, Valle Gottlieb MG. Effect of green tea ( <i>Camellia sinensis</i> ) consumption on the components of metabolic syndrome in elderly. <i>J Nutr Health Aging</i> . 2012;16(9):738-742. doi:10.1007/s12603-012-0081-5	Intervention/exposure
252	Villegas R, Gao YT, Dai Q, et al. Dietary calcium and magnesium intakes and the risk of type 2 diabetes: the Shanghai Women's Health Study. <i>Am J Clin Nutr</i> . 2009;89(4):1059-1067. doi:10.3945/ajcn.2008.27182	Intervention/exposure
253	Villegas R, Gao YT, Yang G, et al. Legume and soy food intake and the incidence of type 2 diabetes in the Shanghai Women's Health Study. <i>Am J Clin Nutr</i> . 2008;87(1):162-167. doi:10.1093/ajcn/87.1.162	Country
254	Villegas R, Yang G, Gao YT, et al. Dietary patterns are associated with lower incidence of type 2 diabetes in middle-aged women: the Shanghai Women's Health Study. <i>Int J Epidemiol</i> . 2010;39(3):889-899. doi:10.1093/ije/dyq008	Intervention/exposure
255	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. <i>Int J Obes (Lond)</i> . 2021;45(8):1751-1762. doi:10.1038/s41366-021-00841-2	Intervention/exposure
256	Vissers LET, Sluijs I, van der Schouw YT, et al. Dairy Product Intake and Risk of Type 2 Diabetes in EPIC-InterAct: A Mendelian Randomization Study. <i>Diabetes Care</i> . 2019;42(4):568-575. doi:10.2337/dc18-2034	Intervention/exposure
257	Voortman T, Kieft-de Jong JC, Ikram MA, et al. Adherence to the 2015 Dutch dietary guidelines and risk of non-communicable diseases and mortality in the Rotterdam Study. <i>Eur J Epidemiol</i> . 2017;32(11):993-1005. doi:10.1007/s10654-017-0295-2	Intervention/exposure
258	Wang J, Light K, Henderson M, et al. Consumption of added sugars from liquid but not solid sources predicts impaired glucose homeostasis and insulin resistance among youth at risk of obesity. <i>J Nutr</i> . 2014;144(1):81-86. doi:10.3945/jn.113.182519	Intervention/exposure
259	Wang X, Jia J, Huang T. Coffee Types and Type 2 Diabetes Mellitus: Large-Scale Cross-Phenotype Association Study and Mendelian Randomization Analysis. <i>Front Endocrinol (Lausanne)</i> . 2022;13:818831. doi:10.3389/fendo.2022.818831	Intervention/exposure
260	Wedick NM, Brennan AM, Sun Q, Hu FB, Mantzoros CS, van Dam RM. Effects of caffeinated and decaffeinated coffee on biological risk factors for type 2 diabetes: a randomized controlled trial. <i>Nutr J</i> . 2011;10:93. doi:10.1186/1475-2891-10-93	Intervention/exposure
261	Wedick NM, Pan A, Cassidy A, et al. Dietary flavonoid intakes and risk of type 2 diabetes in US men and women. <i>Am J Clin Nutr</i> . 2012;95(4):925-933. doi:10.3945/ajcn.111.028894	Intervention/exposure
262	Wennergren MH, Smedman A, Turpeinen AM, et al. Dairy products and metabolic effects in overweight men and women: results from a 6-mo intervention study. <i>Am J Clin Nutr</i> . 2009;90(4):960-968. doi:10.3945/ajcn.2009.27664	Intervention/exposure
263	Wong THT, Burlutsky G, Gopinath B, Flood VM, Mitchell P, Louie JCY. The longitudinal association between coffee and tea consumption and the risk of metabolic syndrome and its component conditions in an older adult population. <i>J Nutr Sci</i> . 2022;11:e79. doi:10.1017/jns.2022.78	Intervention/exposure
264	Wuni R, Lakshmipriya N, Abirami K, et al. Higher Intake of Dairy Is Associated with Lower Cardiometabolic Risks and Metabolic Syndrome in Asian Indians. <i>Nutrients</i> . 2022;14(18):3699. doi:10.3390/nu14183699	Country
265	Yang H, Kim H, Kim JM, Chung HW, Chang N. Associations of dietary intake and metabolic syndrome risk parameters in Vietnamese female marriage immigrants in South Korea: The KoGES follow-up study. <i>Nutr Res Pract</i> . 2016;10(3):313-320. doi:10.4162/nrp.2016.10.3.313	Intervention/exposure



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266	Yang J, Tobias DK, Li S, et al. Habitual coffee consumption and subsequent risk of type 2 diabetes in individuals with a history of gestational diabetes - a prospective study. <i>Am J Clin Nutr.</i> 2022;116(6):1693-1703. doi:10.1093/ajcn/nqac241	Health status
267	Yang Q, Lin SL, Au Yeung SL, et al. Genetically predicted milk consumption and bone health, ischemic heart disease and type 2 diabetes: a Mendelian randomization study. <i>Eur J Clin Nutr.</i> 2017;71(8):1008-1012. doi:10.1038/ejcn.2017.8	Intervention/exposure
268	Ye S, Chen H, Ren X, et al. Carbonated beverage consumption is associated with lower C-peptide in adolescents. <i>J Pediatr Endocrinol Metab.</i> 2019;32(5):447-454. doi:10.1515/jpem-2018-0286	Study design
269	Yeates AJ, Gilmartin N, O'Kane SM, Pourshahidi LK, Mulhern MS, Strain JJ. The effect of cow's milk consumption on cardiometabolic health in women of childbearing age. <i>Proc Nutr Soc.</i> 2017;76(OCE3):E58. doi:10.1017/S0029665117001318	Publication status
270	Yu S, Wang B, Li G, Guo X, Yang H, Sun Y. Habitual Tea Consumption Increases the Incidence of Metabolic Syndrome in Middle-Aged and Older Individuals. <i>Nutrients.</i> 2023;15(6):1448. doi:10.3390/nu15061448	Intervention/exposure
271	Yuan S, Sun J, Lu Y, et al. Health effects of milk consumption: phenome-wide Mendelian randomization study. <i>BMC Med.</i> 2022;20(1):455. doi:10.1186/s12916-022-02658-w	Intervention/exposure
272	Yuan S, Larsson SC. An atlas on risk factors for type 2 diabetes: a wide-angled Mendelian randomisation study. <i>Diabetologia.</i> 2020;63(11):2359-2371. doi:10.1007/s00125-020-05253-x	Intervention/exposure
273	Yun H, Sun L, Wu Q, et al. Lipidomic Signatures of Dairy Consumption and Associated Changes in Blood Pressure and Other Cardiovascular Risk Factors Among Chinese Adults. <i>Hypertension.</i> 2022;79(8):1617-1628. doi:10.1161/HYPERTENSIONAHA.122.18981	Country
274	Yuzbashian E, Pakseresht M, Vena J, Chan CB. Association of dairy consumption patterns with the incidence of type 2 diabetes: Findings from Alberta's Tomorrow Project. <i>Nutr Metab Cardiovasc Dis.</i> 2022;32(12):2760-2771. doi:10.1016/j.numecd.2022.09.022	Duplicate
275	Yuzbashian E, Asghari G, Mirmiran P, Chan CB, Azizi F. Changes in dairy product consumption and subsequent type 2 diabetes among individuals with prediabetes: Tehran Lipid and Glucose Study. <i>Nutr J.</i> 2021;20(1):88. doi:10.1186/s12937-021-00745-x	Intervention/exposure
276	Yuzbashian E, Nosrati-Oskouie M, Asghari G, Chan CB, Mirmiran P, Azizi F. Associations of dairy intake with risk of incident metabolic syndrome in children and adolescents: Tehran Lipid and Glucose Study. <i>Acta Diabetol.</i> 2021;58(4):447-457. doi:10.1007/s00592-020-01651-0	Intervention/exposure
277	Zair Y, Kasbi-Chadli F, Housez B, et al. Effect of a high bicarbonate mineral water on fasting and postprandial lipemia in moderately hypercholesterolemic subjects: a pilot study. <i>Lipids Health Dis.</i> 2013;12:105. doi:10.1186/1476-511X-12-105	Comparator
278	Zhang S, Meng G, Zhang Q, et al. Dairy intake and risk of type 2 diabetes: results of a large prospective cohort. <i>Food Funct.</i> 2023;14(21):9695-9706. doi:10.1039/d3fo02023a	Intervention/exposure
279	Zhang Y, Bian Z, Lu H, Wang L, Xu J, Wang C. Association between tea consumption and glucose metabolism and insulin secretion in the Shanghai High-risk Diabetic Screen (SHiDS) study. <i>BMJ Open Diabetes Res Care.</i> 2023;11(2):e003266. doi:10.1136/bmjdr-2022-003266	Study design
280	Zhang Y, Lee ET, Cowan LD, Fabsitz RR, Howard BV. Coffee consumption and the incidence of type 2 diabetes in men and women with normal glucose tolerance: the Strong Heart Study. <i>Nutr Metab Cardiovasc Dis.</i> 2011;21(6):418-423. doi:10.1016/j.numecd.2009.10.020	Intervention/exposure
281	Zhang Y, Wang R, Tang X, et al. A Mendelian Randomization Study of the Effect of Tea Intake on Type 2 Diabetes. <i>Front Genet.</i> 2022;13:835917. doi:10.3389/fgene.2022.835917	Intervention/exposure