SUGAR INTAKE AND HEALTH OUTCOMES

A Rapid Evidence Review

Physical Activity, Nutrition and Obesity Research Group (PANORG)

Prepared by Sinead Boylan and Seema Mihrshahi
Suggested Citation:
Boylan S., Mihrshahi S. Sugar Intake and Health Outcomes: A Rapid Evidence Review. Prepared for the Centre for Population Health, NSW Ministry of Health. Sydney; Physical Activity Nutrition Obesity Research Group, August 2015

Acknowledgments
The contributions of the following in reviewing earlier drafts are gratefully acknowledged:

Bill Bellew        PANORG
Megan Cobcroft    NSW Ministry of Health
Tim Gill          Boden Institute
## CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.0</td>
<td>EXECUTIVE SUMMARY</td>
<td>4</td>
</tr>
<tr>
<td>1.1</td>
<td>Background</td>
<td>4</td>
</tr>
<tr>
<td>1.2</td>
<td>Methods</td>
<td>4</td>
</tr>
<tr>
<td>1.3</td>
<td>Findings</td>
<td>4</td>
</tr>
<tr>
<td>1.4</td>
<td>Limitations of this evidence review</td>
<td>5</td>
</tr>
<tr>
<td>1.5</td>
<td>Conclusions</td>
<td>5</td>
</tr>
<tr>
<td>2.0</td>
<td>BACKGROUND AND INTRODUCTION</td>
<td>7</td>
</tr>
<tr>
<td>3.0</td>
<td>AIM</td>
<td>8</td>
</tr>
<tr>
<td>4.0</td>
<td>METHODS</td>
<td>9</td>
</tr>
<tr>
<td>4.1</td>
<td>Major sources of evidence</td>
<td>9</td>
</tr>
<tr>
<td>4.2</td>
<td>Additional sources of evidence</td>
<td>9</td>
</tr>
<tr>
<td>4.3</td>
<td>Appraisal of the three main reviews</td>
<td>11</td>
</tr>
<tr>
<td>4.4</td>
<td>Appraisal of additional evidence</td>
<td>14</td>
</tr>
<tr>
<td>5.0</td>
<td>ADDRESSING THE 6 REVIEW QUESTIONS</td>
<td>20</td>
</tr>
<tr>
<td>5.1</td>
<td>Evidence from three specified major reviews</td>
<td>20</td>
</tr>
<tr>
<td>5.2</td>
<td>Summary of recent additional evidence reviewed</td>
<td>29</td>
</tr>
<tr>
<td>6.0</td>
<td>LIMITATIONS OF AVAILABLE EVIDENCE FOR POLICY DEVELOPMENT</td>
<td>29</td>
</tr>
<tr>
<td>6.1</td>
<td>Definitions of sugar/form of sugar used as the exposure variable</td>
<td>29</td>
</tr>
<tr>
<td>6.2</td>
<td>Study design</td>
<td>30</td>
</tr>
<tr>
<td>6.3</td>
<td>Other challenges</td>
<td>30</td>
</tr>
<tr>
<td>7.0</td>
<td>CONCLUSIONS</td>
<td>33</td>
</tr>
<tr>
<td>7.1</td>
<td>Overall summary of evidence</td>
<td>33</td>
</tr>
<tr>
<td>7.2</td>
<td>Policy implications</td>
<td>35</td>
</tr>
</tbody>
</table>

APPENDICES .......................................................... 36
REFERENCES ............................................................. 38
1.0 EXECUTIVE SUMMARY

1.1 Background
The recently released WHO international guidelines recommend that adults and children restrict sugars to less than 10% of total energy intake or further, to less than 5% of intake, for additional health benefits. The purpose of this rapid review is to provide the NSW Ministry of Health with the most recent evidence to guide development of a position on sugar and health. The findings presented will help to determine if and how the NSW Government should include limiting of sugar consumption in healthy food provision guidelines and recommendations at a population level.

1.2 Methods
We undertook a rapid review of the evidence to investigate the links between sugars and specific health outcomes including weight gain, overweight and obesity, cardiovascular disease, metabolic disease including Type 2 diabetes, cognition and dental caries. We used three recent evidence reviews to inform our response to several questions posed by the NSW Ministry of Health [1-3]. We also conducted a systematic search of the literature for recent (since 2009) systematic reviews and rated them for quality.

1.3 Findings
There is good evidence to suggest that dietary sugars may lead to overconsumption of energy, however more solid evidence is needed to determine whether added sugar per se has a negative impact on health. There is convincing evidence that consumption of sugar sweetened beverages (SSBs) in particular is detrimental to health. SSBs which are high in sugars and have no nutrient value are associated with increased risk of weight gain in both adults and children. Evidence from recent high and moderate quality systematic reviews support these findings with significant positive relationships found between SSB intake and adiposity [4, 5]. In addition, several well-conducted randomized controlled trials (RCTs) have shown statistically significant changes in adiposity as a result of corresponding changes in SSB intake, particularly in children [4, 5]. Adults who consume less sugars have lower body weight and increasing the amount of sugars in the diet is associated with a comparable weight increase [2] however, with the exception of SSBs, it is not clear if this relationship is independent of energy intake.

There is strong and convincing evidence that high and frequent consumption of added sugars including SSBs, particularly for infants and young children, is associated with dental caries [1]. The WHO sugar guideline is based on the fact that there are higher rates of dental caries when the intake of free sugars is above 10% of total energy intake compared with an intake of free sugars below 10% of total energy intake [2].

There is accumulating evidence for a relationship between SSBs and blood pressure/hypertension, risk of developing CVD, risk of diabetes and metabolic disease [6, 7]. It is important to note that most of this evidence is from observational studies.

One recent systematic review of RCT evidence that showed that fructose consumption was positively associated with metabolic disorders including increased fasting blood sugar, elevated triglycerides and elevated systolic blood pressure with lower high density lipoprotein (HDL) cholesterol [8]. There is insufficient evidence on the relationship between sugar and cognitive decline[9].

Examining the links between sugar and health is not without its challenges e.g. different definitions of sugar that are used, methodological limitations with study designs, short duration of follow-up,
small sample sizes making it difficult to combine the results of studies. The findings from this rapid review also highlight the lack of studies examining the levels at which adverse effects may occur, with most of the evidence pointing towards adverse effects and consumption of SSBs once or more per day. There have been several reports showing evidence of conflict of interest/ funding bias [10, 11]. These challenges must be addressed going forward to allow researchers to continue to build the evidence for the relationship between this highly topical and widely consumed ingredient and health.

1.4 Limitations of this evidence review
This is a rapid rather than a full systematic review. The goal in a rapid review is to provide a concise summary of evidence that answers specific policy questions presented in a policy-friendly format. This methodology yields a rapid review of existing research and evidence tailored to an agency’s individual needs. Every effort has been made to ensure that a thorough search was undertaken consistent with the agreed scope of work. However the review does not purport to be an exhaustive and comprehensive review of all published research.

1.5 Conclusions
The strongest evidence is for the effect of SSBs and bodyweight, weight gain; and for added sugars and dental caries - health outcomes which are significant for public health in Australia. Any recommendation concerning dietary sugar intake should follow the Australian Dietary Guidelines to “limit intake of foods and drinks containing added sugar such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks”. It is likely that with the growing interest among the population and evidence accumulating on the association between sugar and health, the area will increase in salience for those who have the responsibility to protect the health of their populations. Limitations in the reviewed research preclude more specific guidance on evidence based policy directions or positions at this time.
Glossary

BMI Body Mass Index

CVD Cardiovascular disease

HDL High Low density lipoprotein

LDL Low density lipoprotein

MetS Metabolic Syndrome

SSBs Sugar Sweetened Beverages

RCTs Randomised controlled trials

T2D Type 2 diabetes

VLDL Low density lipoprotein
2.0 BACKGROUND AND INTRODUCTION

Sources of sugar intake

Sugars are carbohydrates and cover a range of mono- and di-saccharides. When sugars occur naturally in foods such as fruit, vegetables and dairy products, they are referred to as intrinsic sugars. However, the major source of sugar in the Australian diet is sucrose from sugar cane that is added to foods and is termed extrinsic sugar. Sucrose is widely used in processed foods and drinks as a sweetener and also plays a role as a flavour and preservative. Fructose and/or high fructose corn syrups are commonly used as a sweetener in the US. These are not commonly used in the Australian food supply [1].

Recent national data on the intake of total sugars for children and adults are available from the Australian Health Survey (2011/2012). Total sugars contributed to 20% of energy and the food groups contributing the greatest amounts of sugars were: fruit products and dishes (15.6%), soft drinks, and flavoured mineral waters (9.7%), dairy milk (8.1%), fruit and vegetable juices, and drinks (7.5%), sugar, honey and syrups (6.5%) and cakes, muffins, scones and cake-type desserts (5.8%). Data on added sugar intake from the Australian Health Survey is currently not available. While sugars provide a readily absorbed source of energy, it has been suggested that added sugars can increase the energy content of the diet while diluting its nutrient density [1].

Sugar intake guidelines

Many countries offer guidance to consumers on appropriate sugar intakes for health – either quantitatively or qualitatively. Guidance is justified based on concerns about the possible adverse impact of sugar on health. The World Health Organization’s (WHO) sugar guideline, issued in March 2015, recommends that adults and children restrict free sugars to less than 10% of total daily energy intake, which is the equivalent of around 12.5 teaspoons of sugar for adults, and suggests a further reduction to below 5% of total daily energy intake for additional benefits [2]. The Australian Dietary Guidelines (ADG) recommend to “Limit intake of foods containing saturated fat, added salt, added sugars and alcohol” and specifically “Limit intake of foods and drinks containing added sugars such as confectionary, sugar –sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks”[1]. The evidence used to support the development of these guidelines seems to point to limiting the intake of soft drinks rather than added sugar in foods more broadly (with the exception of sugary foods in relation to dental caries).

The NSW context

The NSW Ministry of Health wishes to develop a position on sugar and health that will allow the them to determine if and how the NSW Government should include limiting of sugar consumption in healthy food provision guidelines and recommendations at a population level. In particular, there is need to identify if there is sufficient evidence to support a recommendation to limit sugar intake from core, nutrient-dense foods. The NSW Ministry of Health will not be in a position to set guidelines based on ‘added’ or ‘free’ sugars as these are not required to be labelled by law – only total sugar and carbohydrate content is labelled.
The broader strategic context for this work is the NSW Healthy Eating and Active Living (HEAL) Strategy [12] - a five year whole of government plan, linked to achievement of a number of goals within the state plan NSW2021. The plan has four strategic directions: environments to support healthy eating and active living, state-wide support programs, advice as a part of routine service delivery and education and information to enable informed healthy choices. The six objectives within the HEAL Strategy to achieve these improvements in nutrition and physical activity levels are to:

1. Reduce intake of energy dense nutrient poor food and drinks
2. Increase consumption of fruit and vegetables
3. Increase intake of water in preference to sugar-sweetened drinks
4. Increase incidental, moderate and vigorous physical activity
5. Reduce time in sedentary behaviours
6. Increase community awareness of healthy eating and physical activity as protective factors against chronic disease

Report format
This report is split into several sections. Section 3 describes the aim of this report. Section 4 describes the sources of evidence and the appraisal thereof. Section 5 addresses the question of whether sugar has an adverse impact on health, in what form, at what level and on which health outcomes. This section includes responses mapped to the questions posed by the NSW Ministry of the health and a summary of recent evidence. Section 6 describes the challenges in considering the evidence. Section 7 concludes with an overall summary of the evidence and the policy implications following interpretation of the current evidence.

3.0 AIM

The aim of this rapid review is to identify if there is sufficient evidence for an adverse effect of sugar consumption on a range of health outcomes. The purpose is to inform the development by the NSW Ministry of Health of a position on sugar and health, determining if and how sugar should be included in healthy food provision guidelines and recommendations at a population level. In particular, if there is need to consider a recommendation to limiting sugar intake from core, nutrient-dense foods for the general population.
4.0 METHODS

4.1 Major sources of evidence

We used three major sources (as recommended by the MOH) as the foundation for this rapid review:

1. The NHMRC Australian Dietary Guidelines, published in 2013 [1] and we also used chapters 14 and 15 from the Evidence Report (ref no: N55b) which related to Sugars and Beverages (pages 511-550).
3. Sugar and the Heart: Evidence Update Commissioned by the NZ Heart Foundation, published in 2013 (with an addendum in 2014) [3].

4.2 Additional sources of evidence

The most comprehensive of the reviews (NHMRC) drew on literature from 2002 to 2009, therefore it was decided that an additional search for reviews published since 2009 to present (1st July 2015) would be conducted. We searched the Pubmed database for reviews and systematic reviews published in the English language from December 2009 until present (July 2015). The search terms are presented in Table 1. The following terms were excluded from the search: Alcohol, alcohol, artificial, artificial-sweetened, calorie-sweetened, high-fructose, tax$, intervention$, strategy$, policy$, substitut$. Our search resulted in 280 references which were assessed for inclusion using the titles only. Figure 1 presents the flowchart of study search and selection. Our search identified 34 articles for review, of which two were deemed of high quality (sugar and body weight) and six of moderate quality (2 sugar and body weight; 2 sugar and metabolic syndrome; and 2 sugar and CVD).

Table 1: Search terms used to generate recent evidence for this rapid review

<table>
<thead>
<tr>
<th>Topic</th>
<th>Title term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary exposure</td>
<td>Sugar$, sugar$, sugar-sweetened, sweetened, sugar-containing, soft drink$, energy drink$, beverage$, sucrose, glucose, fructose, galactose, lactose, maltose, dietary carbohydrate$, refined carbohydrate$, monosaccharide$, disaccharide$, polysaccharide$, starch$</td>
</tr>
<tr>
<td>General health</td>
<td>Health, health</td>
</tr>
<tr>
<td>Body weight</td>
<td>Body weight, weight, overweight, obesity, obese, body mass index, waist size, anthropometry, body composition, skinfold thickness</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>Cardiovascular disease, heart disease, cardio-metabolic, cerebrovascular disease, triglyceride$, blood pressure, cholesterol, lipids</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>Metabolic syndrome, syndrome x, insulin resistance, dyslipidaemia, type 2 diabetes, diabetic$</td>
</tr>
<tr>
<td>Dental health</td>
<td>Dental health, dental caries, oral health</td>
</tr>
<tr>
<td>Behaviour</td>
<td>Cognitive, behaviour$, learning</td>
</tr>
</tbody>
</table>
Figure 1: Search strategy used to generate additional evidence for this rapid review

280 citations identified from PubMed

234 excluded:
207 not relevant (e.g. glucose homeostasis, disease prevalence, consensus statements)
14 intervention/strategy/policy
7 Other study type e.g. clinical
6 Other carbohydrate e.g. NSP, starch

46 potentially relevant articles

12 excluded:
5 duplicates
4 review quality assessments
2 review protocol papers
1 effects of energy drinks (contain other compounds other than sugar which may affect health)

34 articles reviewed for quality

Health: 12 articles
- 0 +++
- 0 ++
- 12 +

Body weight: 7 articles
- 2 +++
- 2 ++
- 3 +

Metabolic syndrome: 8 articles
- 0 +++
- 2 ++
- 6 +

CVD: 6 articles
- 0 +
- 2 +
- 4 +

Behaviour: 1 article
- 0 +++
- 0 ++
- 1 +

Dental: 0 articles

+++High quality; ++ Moderate quality; + Low quality
4.3 Appraisal of the three main reviews

Table 2 shows the characteristics of the major sources used for this rapid review including the dates of included studies, type of studies, population (children/adults), and the outcomes studied.

4.3.1 NHMRC Evidence review [1]

The primary aim of this evidence review was to undertake a series of systematic reviews of the national and international literature from the year 2002 on the food-diet-health-disease inter-relationship in order to inform the update of the Australian Dietary Guidelines.

On the whole this was a very comprehensive review and the systematic and umbrella reviews included have resulted in body of evidence statements, graded depending on the strength, consistency, potential impact, generalisability and applicability of the evidence base. In addition, the development of guidelines process involved consultation with experts, modelling and public consultation after the guidelines were developed.

Limitations to the NHMRC review

The NHMRC stressed that the reviews should not be considered complete reviews of the relevant literature and that there were limitations including that the search timeframe (2002-2009) and that some of the most important literature was published before 2002 and was not considered in these reviews. Therefore evidence grades for these may be lower than would be anticipated with a time unlimited literature review (e.g. for sugar and dental caries, where the diet disease relationship was well established prior to 2002). Another limitation was that the methods of assessment of evidence (where RCTs are regarded the highest level of evidence) may not be applicable to diet-health relationships and that the inclusion of umbrella reviews may have missed important individual studies. In addition, the quantification of dietary exposures was not possible due to limitations in the dietary methodologies used in studies or the level of detail reported. For example, in many cohort studies quantiles of exposure are reported in relation to disease outcomes, but absolute intakes are not reported for the highest and lowest levels being compared.

4.3.2 WHO Sugars Intake for adults and Children

This document outlines the evidence for the WHO recommendation to reduce consumption of free sugars throughout the life-course based on prevention of dental caries and the prevention and control of unhealthy weight gain. It strongly recommends that both adults and children do not consume more than 10% of total energy as free sugars and conditionally recommends a further reduction to less than 5% of total energy consumed as free sugars. The recommendations are based on two systematic reviews of studies that examined the association between excess energy intake from sugars and body weight and intake of sugar and dental caries.

Limitations to the WHO Sugars Intake for adults and Children

This review, although it is comprehensive and includes data from RCTs and cohort studies (with respect to weight gain) is not focused on other cardiovascular outcomes because they regard the measures aimed at reducing overweight and obesity are likely to also reduce the complications.
associated with all outcomes including CVD and Type 2 diabetes. For this reason the review focuses on bodyweight and dental caries only.

The main limitations are those inherent to the primary research on which they are based, mainly inadequacy of dietary intake data, and variation in the nature and quality of the dietary intervention.

4.3.3 New Zealand Heart Foundation Sugar and the heart report

This report was intended as an “evidence snapshot” of current research on sugar and heart health to update the Heart Foundation of New Zealand’s position on sugar.

Limitations to the Sugar and the Heart Review

As an umbrella review this drew on evidence from reviews and meta-analyses and therefore there were strong methodological limitations. These included bias relating to study design (cross-sectional and cohort studies were included) and that many of the published studies of high quality had small sample sizes and short duration of follow up. The review also noted that results of studies varied according to funding source with industry funded studies being more likely to have more favourable conclusions than non-industry funded studies.

4.3.4 Overall conclusion of the main sources

Overall, because the NHMRC and WHO reviews were systematic reviews which used a comprehensive search and involved reviewing a large body of literature with rigorous critical appraisal processes including a consultation with experts, they were regarded as high quality.

The NHMRC review found that high or frequent consumption of added sugars including SSBs, particularly for infants and young children, is associated with dental caries. Consumption of SSBs is associated with increased risk of weight gain in both adults and children. A reduction in sugar consumption prevents increases in measures of bodyweight and/or body fat in adults.

The WHO review, which represents the most recent of the three sources, suggests that adults who consume less sugars have lower body weight and, second, that increasing the amount of sugars in the diet is associated with a comparable weight increase. In addition, research shows that children with the highest intakes of SSBs are more likely to be overweight or obese than children with a low intake of SSBs. In addition, there are higher rates of dental caries when the intake of free sugars is above 10% of total energy intake compared with an intake of free sugars below 10% of total energy intake.
Table 2: Characteristics of the main evidence sources used for this rapid review

<table>
<thead>
<tr>
<th>Review</th>
<th>Type/Dates/ Sources used</th>
<th>Population/Dietary exposure</th>
<th>Outcome</th>
<th>Tool used to assess quality of studies included</th>
<th>Quality assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>A review of the evidence to address targeted questions to inform the revision of the ADGs NHMRC 2011 [1]</td>
<td>Systematic review 2002-2009. Included systematic reviews, meta-analyses and prospective cohort studies.</td>
<td>Adults and children Sugars, added sugars, SSBs, sugar sweetened food, fruit juice</td>
<td>Obesity and weight, dental disease, bone health, cardiovascular disease, type 2 diabetes, cancer</td>
<td>GRADE plus consultation and consensus to develop guidelines</td>
<td>High</td>
</tr>
<tr>
<td>Sugars intake for adults and children WHO 2015 [2]</td>
<td>Systematic review 1959-2014. RCT, observational and ecological studies</td>
<td>Adults and children Free sugars include mono and disaccharides added to foods by cook or consumers and sugars naturally present in honey, syrups, fruit juices and concentrates</td>
<td>Excess weight gain and dental caries are primary outcomes but also included Obesity, CHD, Stroke, Type 2 Diabetes, Cancer, Hypertension, Chronic obstructive pulmonary disease, Eye health, Bone health, Dental health, Mental health, Other</td>
<td>GRADE and technical consultation for consensus on strength of recommendations</td>
<td>High*</td>
</tr>
<tr>
<td>Sugar and the Heart National Heart Foundation (NZ) Jan 2013 [3].</td>
<td>Narrative review 2002-2012 (with Addendum in 2014 including the systematic review by Te Morenga et al)[13] included all study types</td>
<td>Adults and children Sugars defined as mono-or disaccharides and SSBs (soft drinks or drinks with sugar added)</td>
<td>Heart disease only</td>
<td>AMSTAR</td>
<td>Low</td>
</tr>
</tbody>
</table>

*High quality evidence for recommending reducing intake of free sugars to below 10% of total energy
4.4 Appraisal of additional evidence

All recent reviews found using the literature search described in Section 4.2 were assessed for quality and rated as low, medium, high by one of two assessors (SB and SM) using the AMSTAR criteria as a guide. This included grading information about the comprehensiveness of the review including minimum number of electronic sources searched, inclusion and exclusion criteria, data extraction processes, assessment of bias and quality assessment of included studies, methods used to combine the studies, and possible conflicts of interest or funding bias.

Narrative reviews, opinions, commentaries or articles with a possible risk of funding bias/conflict of interest were categorised as low quality.

Systematic reviews that included only observational (cohort, cross sectional studies) or with limited information about the process of reviewing (e.g. number of reviewers, number of articles screened) or that did not assess the quality of studies included were regarded as moderate quality.

Systematic reviews that reported all necessary items (listed above) and where the risk of bias was low were regarded as high quality.

Reviews which were rated as high and moderate were discussed and a summary of these reviews is given in Table 3.
Table 3: Summary of high and moderate quality systematic review evidence relating to sugars/SSBs and body weight, metabolic disease and CVD

<table>
<thead>
<tr>
<th>First author (date)</th>
<th>Aim</th>
<th>Population</th>
<th>Number of included studies/types</th>
<th>Exposure</th>
<th>Outcome</th>
<th>Effect size / Main findings related sugar and body weight</th>
<th>Comments</th>
<th>Rating of Quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malik VS (2013)[4]</td>
<td>To assess the relationship between SSBs and body weight.</td>
<td>Systematic review and meta-analysis. 32 original articles 20 in children (15 cohort; 5 trials) and 12 in adults (7 cohort studies; 5 trials).</td>
<td>SSBs</td>
<td>Body weight, BMI, overweight, obesity, related cardio-metabolic outcomes (i.e., diabetes mellitus, insulin resistance, cardiovascular diseases, hypertension).</td>
<td>Cohort studies: 1 serve/day associated with a 0.06 (95% CI: 0.02, 0.10) and 0.05 (95% CI: 0.03, 0.07)-unit increase in BMI in children and 0.22 kg (95% CI: 0.09, 0.34 kg) and 0.12 kg (95% CI: 0.10, 0.14 kg) weight gain in adults over 1yr. RCTs in children showed reductions in BMI gain when SSBs were reduced [random- and fixed-effects: 20.17 (95% CI: 20.39, 0.05 kg) and 20.12 (95% CI: 20.22, 20.2 kg)], whereas RCTs in adults showed increases in body weight when SSBs were added (random- and fixed-effects: 0.85 kg; 95% CI: 0.50, 1.20 kg). Sensitivity analyses of RCTs in children showed more pronounced benefits in preventing weight gain in SSB substitution trials (compared with school-based educational programs) and among overweight children (compared with normal-weight children).</td>
<td>Details provided on comprehensive search strategy. Effect sizes presented for weight gain presented. Restricted publications to the English language, and did not consider cross-sectional or ecologic studies because they are highly prone to confounding and reverse causation.</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>Woodward-Lopez (2011)[5]</td>
<td>To determine whether SSB intake increases the risk for obesity, and the extent to which it has contributed to recent increases in energy intake and adiposity in the USA</td>
<td>Systematic review. Observational (56 studies), intervention (5 studies among children), experimental (4 studies), meta-analyses (2).</td>
<td>Soda; soft drinks; sweetened beverages; fruit drinks</td>
<td>Obesity, overweight, body weight.</td>
<td>Studies consistently show that higher intake of sweetened beverages is associated with higher energy intake. Energy in liquid form is not well compensated for by reductions in the intake of other sources of energy. <strong>Well-designed observational studies consistently show a significant positive relationship between SSB intake and adiposity.</strong> More importantly, several well-conducted randomized controlled trials have shown statistically significant changes in adiposity as a result of corresponding changes in SSB intake. <em>Levels: sweetened soda v HFCS v no soda supplementation – regular soda consumers gained 0.7kg; sucrose v artificial sweeteners: sucrose group increased by 1.6kg, other group increased by 1kg; 0.8kg unit change in BMI per 335ml of soda.</em></td>
<td>Details provided of search strategy and quality grading. Training studies, weight loss studies and studies of populations with chronic diseases were excluded. Studies had to have been conducted in a developed country. Mostly observational studies. Possible conflict of interest identified in one of the meta-analyses.</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>First author (date)</td>
<td>Aim</td>
<td>Population Number of included studies/ types</td>
<td>Exposure</td>
<td>Outcome</td>
<td>Effect size / Main findings related sugar and body weight</td>
<td>Comments</td>
<td>Rating of Quality</td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
<td>-----</td>
<td>---------------------------------------------</td>
<td>----------</td>
<td>---------</td>
<td>----------------------------------------------------------</td>
<td>----------</td>
<td>------------------</td>
<td></td>
</tr>
<tr>
<td>Trumbo (2014)[14]</td>
<td>To systematically review role of SSBs in obesity risk, taking into account energy balance.</td>
<td>Systematic review. Excluded 17/17 intervention studies and 45/59 observational studies so only 14 observational studies in children, adolescents or adults were used</td>
<td>SSBs (collectively or individually soda only)</td>
<td>BMI ≥30 kg/m² (adults) or BMI at or above 95th percentile (for children)</td>
<td>Inconsistent results for children, adolescents, and adults with some showing effects and some showing no effect of SSBs on obesity or change in BMI. 4 studies in children 1 showing a positive effect on BMI and obesity; 4 studies in adolescents with 2 showing some gender specific or culture specific effects; 6 studies in adults, 4 which show positive effects.</td>
<td>Systematic review however no description of search strategy. Excluded those studies with overweight as an outcome, that did not adjust for total energy intake, inappropriate controls (ie strict criteria). There was a quality rating of studies (high, moderate, or low) but no detailed description of how rated.</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>Perez-Morales (2013) [15]</td>
<td>To conduct a systematic review of prospective studies that examined the association between SSB intake before 6y of age and later weight or BMI status among older children.</td>
<td>Systematic review. 7 prospective studies were analysed. The study population was from 72 to 10,904 children.</td>
<td>SSB, including soft drinks, soda, fruit drinks, sports drinks, sweetened iced tea, and lemonade.</td>
<td>Weight, BMI, and waist circumference status.</td>
<td>3 studies showed a consistent association; 1 study showed a positive trend of SSB consumption and childhood obesity (OR 1.04), another study found that an increase in total sugar intake and sugar from sweets and beverages tended to increase BMI; two studies showed no association. While there is a trend, results are inconsistent. The 2 studies with the higher number of children showed a positive association.</td>
<td>Details of search strategy provided. Selection of articles was restricted to prospective cohort studies in children younger than 6 y of aged. Quality of studies not assessed. No detail on exposure levels at which specific adverse effects may occur/effect sizes.</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>Kelishadi R (2014) [8]</td>
<td>To review current human studies to determine the association of various doses and durations of fructose consumption on metabolic syndrome.</td>
<td>Systematic review of human studies including healthy adults (many were overweight or obese) observational studies and clinical intervention trials</td>
<td>Fructose, and Fructose beverage consumption</td>
<td>Systolic blood pressure (SBP), fasting blood sugar (FBS), high-density lipoprotein</td>
<td>Fructose consumption was positively associated with increased fasting blood sugar (FBS; summary mean difference, 0.307; 95% CI, 0.149–0.465; P = 0.002), elevated triglycerides (TG; 0.275; 95% CI, 0.014–0.408; P = 0.002); and elevated systolic blood pressure (SBP; 0.297; 95% CI, 0.144–0.451; P = 0.002). The corresponding figure was inverse for high-density lipoprotein (HDL) cholesterol (0.267; 95% did not include non-clinical trials, or trials in which fructose was recommended exclusively as sucrose or HFCS because these did not permit isolation of the effect of fructose. Probably needed to</td>
<td></td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>First author (date)</td>
<td>Aim</td>
<td>Population</td>
<td>Number of included studies/types</td>
<td>Exposure</td>
<td>Outcome</td>
<td>Effect size / Main findings related to sugar and body weight</td>
<td>Comments</td>
<td>Rating of Quality</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----</td>
<td>------------</td>
<td>---------------------------------</td>
<td>----------</td>
<td>---------</td>
<td>------------------------------------------------------------</td>
<td>----------</td>
<td>------------------</td>
</tr>
<tr>
<td>Malik VS* (2010)[16]</td>
<td>To investigate the role of SSBs in the development of related chronic metabolic diseases, such as metabolic syndrome and type 2 diabetes</td>
<td>Systematic review of prospective cohort studies from 1966-2010 in adults. 11 studies (three for metabolic syndrome and eight for type 2 diabetes) for inclusion in a random-effects meta-analysis comparing SSB intake in the highest to lowest quartiles in relation to risk of metabolic</td>
<td>Soft drinks, carbonated soft drinks, fruitades, fruit drinks, sports drinks, energy and vitamin water drinks, sweetened iced tea, punch, cordials, squashes, and lemonade</td>
<td>Metabolic syndrome and type 2 diabetes</td>
<td>Individuals in the highest quantile of SSB intake (most often 1-2 servings/day) had a 26% greater risk of developing type 2 diabetes than those in the lowest quantile (none or &lt;1 serving/month) (relative risk [RR] 1.26 [95% CI 1.12-1.41]). Among studies evaluating metabolic syndrome, including 19,431 participants and 5,803 cases, the pooled RR was 1.20 [1.02-1.42] ie 20% increase risk of metabolic disease</td>
<td>Unclear about methods and quality assessment</td>
<td>Moderate</td>
<td></td>
</tr>
</tbody>
</table>

15 studies were included in the meta-analysis that investigated the association of oral fructose on the components of MetS in a healthy population.

Cholesterol (HDL-C), and TG before and after fructose

CI, 0.406 to 0.128; P = 0.001).

Good commentary on doses: Previous research found that fructose intake approximately > 8% to 12% of energy intake (> 50 g/d) is associated with MetS. The researchers found that the amount of fructose intake through fruits and vegetables even in the highest quartile was only 5% of energy (30 g/d). Thus, fructose in industrialized foods is considered the main underlying cause for the increasing risk for MetS.

Significant heterogeneity existed between studies, except for fasting blood sugar studies. So no sig effect on TG and other outcomes.

After excluding studies that led to the highest effect on the heterogeneity test, the association between fructose consumption and TG, SBP, and HDL became non-significant.

Analyse the obese overweight participants separately?

The results did not show any evidence of publication bias. Very small numbers (7 to 74) and short duration (as short as 2 days- 10 weeks) for some trials.

Malik VS* (2010)[16]

To investigate the role of SSBs in the development of related chronic metabolic diseases, such as metabolic syndrome and type 2 diabetes.

Systematic review of prospective cohort studies from 1966-2010 in adults. 11 studies (three for metabolic syndrome and eight for type 2 diabetes) for inclusion in a random-effects meta-analysis comparing SSB intake in the highest to lowest quartiles in relation to risk of metabolic.

Soft drinks, carbonated soft drinks, fruitades, fruit drinks, sports drinks, energy and vitamin water drinks, sweetened iced tea, punch, cordials, squashes, and lemonade.

Metabolic syndrome and type 2 diabetes.

Individuals in the highest quartile of SSB intake (most often 1-2 servings/day) had a 26% greater risk of developing type 2 diabetes than those in the lowest quartile (none or <1 serving/month) (relative risk [RR] 1.26 [95% CI 1.12-1.41]). Among studies evaluating metabolic syndrome, including 19,431 participants and 5,803 cases, the pooled RR was 1.20 [1.02-1.42] ie 20% increase risk of metabolic disease.

Unclear about methods and quality assessment.

No cross-sectional studies included, no short term studies included.

Moderate.
<table>
<thead>
<tr>
<th>First author (date)</th>
<th>Aim</th>
<th>Population Number of included studies/types</th>
<th>Exposure</th>
<th>Outcome</th>
<th>Effect size / Main findings related sugar and body weight</th>
<th>Comments</th>
<th>Rating of Quality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malik AH (2014) [6]</td>
<td>To conduct a systematic review exploring the relation between consumption of SSB and BP</td>
<td>Systematic review in adolescents and adults (excluded children &lt;12) Of 605 studies only 12 studies (409,707 participants) met the inclusion criteria; 6 were cross-sectional studies, and remainder were prospective cohort studies.</td>
<td>SSB</td>
<td>BP</td>
<td>All 12 studies showed positive relation between increased SSB intake and hypertension; however, statistical significance was reported in 10 of these studies. Of the 12 studies, 5 reported an increase in mean BP whereas 7 reported an increase in the incidence of high BP. In conclusion, consumption of SSBs is associated with higher BP, leading to increased incidence of hypertension. Dose: even using conservative estimate, intake of &gt;12 fl oz of SSB per day can increase the risk of having hypertension by at least 6%, and it can increase mean SBP by a minimum of 1.8 mm Hg over 18 months. This can be very significant at a population level.</td>
<td>Comprehensive search, good search terms Excluded studies with small SS (&lt;100 subjects) and those involving subjects aged &lt;12 years. Restriction on SSB consumption should be incorporated in the recommendations of lifestyle modifications for the treatment of hypertension. Interventions to reduce intake of SSBs should be an integral part of public health strategy to reduce the incidence of hypertension.</td>
<td>Moderate</td>
</tr>
<tr>
<td>Huang C (2014) [17]</td>
<td>To summarize the evidence with respect to sugar sweetened beverages (SSBs) consumption and risk of coronary heart disease (CHD) and to recommend field standards for future analysis on this topic.</td>
<td>Systematic review of 4 prospective cohort studies including a total of 7396 CHD cases among 173,753 participants and durations of follow-up ranged from 9.8 to 28 years.</td>
<td>SSB</td>
<td>CHD Myocardial infarction, cerebrovascular disease, peripheral arterial disease and heart failure.</td>
<td>Prospective studies with 7396 CHD cases among 173,753 participants were included in the meta-analysis. The pooled RR (95% CI) for CHD in the highest category of SSBs consumption in comparison with the lowest category of SSBs was 1.17 (1.07-1.28). Stratified analyses indicated a significant association for men but not for women, with pooled RRs (95%CI) of 1.17 (1.05-1.29) and 1.19 (0.94-1.50), respectively. For studies carried out in America, the pooled RR for CHD was 1.18 (1.07-1.30). Additionally, a one-serving per day increase in SSBs consumption was associated with a 16% increased risk of CHD.</td>
<td>Used Newcastle-Ottawa Scale (NOS) for quality assessment Really well written paper – good discussion of mechanisms and also limitations on study type and confounding dominated by 2 large American studies of health professionals and thus might not be</td>
<td>Moderate</td>
</tr>
<tr>
<td>First author (date)</td>
<td>Aim</td>
<td>Population Number of included studies/types</td>
<td>Exposure</td>
<td>Outcome</td>
<td>Effect size / Main findings related sugar and body weight</td>
<td>Comments</td>
<td>Rating of Quality</td>
</tr>
<tr>
<td>--------------------</td>
<td>-----</td>
<td>-----------------------------------------------</td>
<td>----------</td>
<td>---------</td>
<td>--------------------------------------------------------</td>
<td>----------</td>
<td>------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CHD (RR: 1.16, 95%CI: 1.10-1.23). After adjusting for BMI and energy intake the result was attenuated but still significant</td>
<td>generalizable. Publication bias cannot be fully omitted, given the small number of included studies however Egger’s test revealed no evidence of publication bias.</td>
<td></td>
</tr>
</tbody>
</table>

*This reference was included in the NHMRC review*
The following sections represent a mapping of the evidence to the research questions.

5.1 Evidence from three specified major reviews

5.1.1 What is the level of evidence for an adverse effect of sugar consumption on health outcomes? (e.g. cardiovascular disease, overweight and obesity, diabetes, dental caries, cognition/behaviour)

| Table 3 presents the reported adverse health outcomes of sugar consumption on health (body weight, CVD and metabolic syndrome/type 2 diabetes) and the level of evidence. For more specific detail, please see the responses to the questions below. |

5.1.2 For which health outcomes is there evidence of an adverse effect? What is the strength of evidence?

5.1.2.1 Heart disease

There remains insufficient and currently inconclusive evidence on the direct influence of sugar on heart disease itself [3]. The NHMRC stated that there is no new evidence that sugars play a causal role in the development or moderation of any cardiovascular risk factors. However there is some suggestive evidence (6 reviews and a meta-analysis) that sugar intake increases triglycerides [3]. There is growing evidence on the potential impact of added sugars on blood pressure in humans, however results remain inconsistent[3].

There is a small body of cohort evidence (n=3) that SSBs are associated with increased risk of heart disease [3]. Specifically, there is some evidence that SSB consumption is associated with higher blood pressure [1]. However the NHMRC reviewed only one RCT and therefore decided that there were not enough studies to form a body of evidence statement.

5.1.2.2 Body weight

There is insufficient evidence that increased dietary sugar intake increases body weight. It is probable that consumption of SSBs is positively associated with body weight, however more RCTs are needed to confirm this.

Dietary sugar (added/ free)

One of the reviews examined a robust systematic review and meta-analyses [13]. This review indicates that an increase in dietary sugar increases body weight and conversely that a reduction in dietary sugar decreases body weight. The effect on body weight from reducing dietary sugars was not evident in children, and the review states that this is likely due to poor compliance. There appears to be good evidence that when substituted iso-energetically (with the same energy value), sugar does not appear to impact on body weight. However, dietary sugar appears to lead to
overconsumption of energy, which is associated with increases in body weight. **The NHF conclude that “evidence strongly suggests that sugar and SSBs can contribute to weight gain, and that reducing their intake reduces weight.”** Caution is warranted however as the NHF was an umbrella review and drew upon a relatively small number of meta-analyses and reviews to reach its conclusion – only one of these sources [13] sources was examined in relationship to dietary sugar and body weight per se, while the others were concerned with the relationship between dietary sugar/SSBs and energy intake [18].

The NHMRC evidence report examined a systematic review, one cohort study and three randomised control trials (RCTs) [19-23]. Overall, if a reduction in sugar intake results in a lower total energy intake then weight gain is controlled. However in order to determine whether there are specific effects of reducing sugar compared with reducing another macronutrient or carbohydrate type then it is essential that total energy intake be tightly controlled to investigate this. The NHMRC admitted difficulty in developing a body of evidence statement as the methods of the examined studies are quite different from one another and are confounded by other factors. They conclude that “**Further evidence of a higher quality is required and analysis of long standing cohorts to determine if habitual consumers of added sugars have higher body weight than those who do not would be helpful to build the evidence base.”**

The other review which assessed the impact of free sugars found that the overall quality of the available evidence for changes in body weight in relation to both increasing and decreasing free sugars intake in adults was considered to be moderate [2]. **Potential biases** were identified in a minority of studies, including publication bias as only a small number of trials were identified. In children, the quality of evidence for an association between a reduction in free sugars intake and reduced body weight was similarly considered to be moderate, whereas the quality of the evidence for an association between an increase in free sugars intake and increased body weight was considered to be low.

**SSBs**

**There is stronger evidence for the impact of SSB on body weight.** As referred to above, the NHF review concluded that reducing their intake reduces weight, however the review has its limitations. The NHMRC concluded that there is a **probable association between sugar-sweetened drinks and weight gain in adults and children.** The review identified differences in study designs with case control and cohort studies not providing good evidence (changes in drinking behaviours of overweight individuals) and there were a limited number of RCTs of large size or duration. Evidence also indicated that industry-funded studies provided results of smaller effect size. RCTs showed more consistency, therefore good quality RCTs of adequate duration are needed to improve the evidence base. Many of the significant associations were found in sub-groups of cohort studies and RCTs so may be more significant for some populations. Specific evidence in children was provided by one meta-analysis that found effect size of SSB intakes on BMI to be medium in children, and 4 cohort studies all providing evidence of an effect with higher BMI associated with intakes of SSB (NHMRC did not cite reference).
There is insufficient consistent evidence available to form an evidence statement about fruit juices and weight gain [1]. Eight studies were reviewed, 7 studies in children (six cohort and one clinical trial) and one cohort study in adults [1]. Overall, 4/8 studies found increased risk of weight gain. Four of seven studies in children found no association whilst two cohorts and one clinical trial found increased risk of weight gain in children. The single cohort study in adults found increased risk of weight gain.

5.1.2.3 Type 2 diabetes / metabolic syndrome

There is insufficient evidence that intake of sugars is related to an increased risk of metabolic syndrome/type 2 diabetes. However, there is suggestive evidence that SSB intake is associated with risk of type 2 diabetes.

Following an examination of 4 reviews, the NHF found that the evidence does not suggest an effect of sugars on risk of metabolic syndrome and type 2 diabetes [24-27]. The NHMRC review stated that there were insufficient studies identified to develop an evidence statement for intake of sugars and type 2 diabetes. One systematic review identified by the NHF review [26] and one cohort study [28] were used to consider sugars and type 2 diabetes. The search strategy and study selection criteria were not adequately described in the systematic review and a meta-analysis was not performed making it difficult to determine clinical impact. The cohort study was well designed and described, however the representativeness is questionable (African-American women; median age of 38 years (range 21 - 69 years); median BMI of 28 kg/m2; and 34% of participants had a family history of diabetes).

However, more recent studies indicate that SSBs may increase the risk of developing type 2 diabetes [29]. After reviewing 2 reviews, the NHF concluded there is a “probable” association between SSB intake and increased risk of type 2 diabetes [16, 25]. Only one study was reviewed by NHRMC examining the relationship between fruit juice consumption and type 2 diabetes (1 serving/day of fruit juice was associated with an increased risk of diabetes) [30].

5.1.2.4 Dental caries

There is suggestive evidence that added sugar and soft drink consumption are associated with increased dental caries, particularly in children.

**Added sugars**

The NHRMC reviewed a systematic review [31] and 4 small cohort studies [32-35]. While the evidence suggests that a frequent intake of foods and beverages high in added sugars increases the risk of dental caries there is insufficient evidence provided in the searched published literature between 2002 and 2009 to determine a dose-response. However, there have been a several key references published prior to 2002 that could be used to guide decision making. The review concluded that high or frequent consumption of added sugars, particularly for infants and young children, is associated with increased risk of dental caries.
Soft drink

The NHMRC reviewed 1 systematic review (4 studies, design not described)[18] and 3 cohort studies [32, 33, 35]. There were no studies in adults; all studies were performed in children, concentrating on the primary dentition. Study subjects were often from low SES demographic groups and exposure to fluoridated water varied. Still, the NHMRC concluded that consumption of soft drink is associated with increased risk of dental caries in children.

Free sugars

An analysis of cohort studies in children suggests a positive association between the level of free sugars intake and dental caries [2]. The overall quality of the available evidence from cohort studies was considered to be moderate, whereas that from the national population studies was considered to be very low.

5.1.3 Is the evidence for specific food and beverage products only or for intake of added, free or total sugars?

Section 5.1.2 (above) presents the relationship between various different dietary exposures of sugar and health outcomes. Two of the reviews examined the relationship between various types of sugar and sources of sugar intake (e.g. total sugar, added sugar, SSBs, fructose, sucrose) [1, 3] and one review focused on the effects of free sugars on health[2]. Much of the evidence related to sugar in the form of SSBs. It is also plausible that SSBs could have a different effect to sugar consumed as part of a food. Sugar may be more detrimental in fluid form due to weaker energy compensation, but evidence is inconsistent [3]. While it is important to consider a whole dietary pattern, SSBs do have a number of features that create an opportunity for public health intervention [3]: “they are an easily defined category; they contribute excess energy to the diet; there is more evidence for SSBs and excess weight than for many other foodstuffs; they provide no nutritional benefit [other than helping meet fluid intakes]; and reducing intakes could have a measurable impact on body weight without any negative dietary consequences”.

Table 3 indicates that there was one study of moderate evidence for a positive association between fructose consumption and metabolic syndrome [8]. Please find below further detail on the effect of fructose and sucrose on various health outcomes as reported in the three major reviews.

5.1.3.1 Fructose

There is insufficient evidence that fructose intake has an adverse effect on the health outcomes studied.
**Cardiovascular disease**

The NHF reviewed one RCT [36] and 2 meta-analyses [37, 38] all of which indicated a relationship between fructose intake and **blood pressure**. However, one of the meta-analyses showed an effect with very high doses (213-219g/day) [37] and the other meta-analyses was found to have included studies of poor quality, short duration and methodological limitations [38].

The NHF also considered a number of reviews and meta-analyses examining the relationship between fructose and lipids [39-41] [42]. In short-term studies, fructose slightly increased triglycerides in comparison to sucrose, glucose, or starch after approximately three hours. However, long-term studies did not show evidence of an increase in triglycerides after ingestion of fructose, provided excess energy was not consumed. Longer term studies tended to be of higher quality. Dolan et al [40] supported the conclusion that fructose does not cause biologically relevant changes in triglycerides (or body weight) at less than 136g/day, in comparison to other sugars. This has been supported by other reviews [41, 42].

The NHMRC reviewed 2 studies which were not about fructose per se (sucrose and SSBs) and CVD risk, therefore there is insufficient evidence of a relationship between fructose and SSBs [43, 44].

**Diabetes**

The NHF reviewed one review of 16 trials among people with type 2 diabetes indicating an isocaloric switch to fructose from carbohydrate raised triglycerides and lowered total cholesterol, with no effect on LDL or HDL, however results were only consistent when follow up was for less than four weeks, crystalline fructose was used, or participants had switched to fructose from starch [45].

### 5.1.3.2 Sucrose

**Cardiovascular disease**

The NHMRC review stated that there were insufficient studies to form a body of evidence for sucrose and CVD risk. Early studies suggested that reducing dietary sucrose could lower elevated **triglyceride** levels, but it is likely that the effects were the result of a reduction in energy intake and body weight [46].

#### 5.1.4 At what level of intake of the specific type of sugar, refined carbohydrate or specific sugary foods do the adverse effects occur?

Table 4 presents the intake levels at which the adverse effects occur. Most of the evidence that is available points towards an adverse effect of SSB consumption at least one serve per day. Please find below further detail regarding levels of intake.
5.1.4.1 Cardiovascular disease

As discussed in Section 5.1.2.1, there remains insufficient and currently inconclusive evidence on which to draw conclusions on the direct influence of sugar on heart disease itself [3]. However, there is a small body of cohort evidence that sugar-sweetened beverages are associated with increased risk of heart disease [3].

Percentage of energy from sugar

The European Food Safety Authority concluded there is some evidence that high intakes (>20% total energy) of sugars may increase triglycerides and cholesterol and adversely impact on glucose and insulin response [47]. Similarly, the American Heart Association advises that a diet with more than 20% of energy from sugars (glucose, fructose, sucrose) is linked with raised fasting triglycerides [48].

SSBs

Results from the Nurses’ Health Study showed that women who drank two or more SSBs a day had a 35% greater relative risk of coronary heart disease compared with infrequent consumers [43]. In the Health Professionals Study, men consuming 355mL of SSB per day had a 19% increased relative risk of CVD, and statistically significant adverse changes in HDL, triglycerides and C-reactive protein, after adjustment for multiple lifestyle factors [49]. The Framingham Heart Study, a prospective cohort study, found drinking one or more SSBs per day (350ml) was associated with a range of risk factors for cardiovascular disease [50].

The PREMIER study found reducing intake of SSBs by one serving a day (355mL) was associated with 1.8mmHg reduction in systolic blood pressure and a 1.1mmHg reduction in diastolic blood pressure over an 18-month period [24]. In the Framingham Heart Study, consumption of more than one SSB per day was associated with 18% greater odds of experiencing higher blood pressure [50]. A cross-sectional analysis of 2696 people from the UK and US in the INTERMAP study found SSB intake related directly to blood pressure. Each additional serving (355mL) of SSB per day was associated with 1.1mmHg higher SBP and 0.4mmHg higher DBP, after adjusting for weight and height [51].

Fructose

The NHF review discussed the levels of fructose intake which are associated with an increased CVD risk. A recent RCT in Spanish men found very high doses (200g) of fructose raised blood pressure and induced features of metabolic syndrome [15]. In short-term studies, ingestion of 30–100 g/day fructose (either in a liquid bolus or in a meal) slightly increased triglycerides in comparison to sucrose, glucose, or starch after approximately three hours. However, long-term studies did not show evidence of an increase in triglycerides after ingestion of up to 133-136 g/day fructose, provided excess energy was not consumed [40].
Other reviews and meta-analyses have come to similar conclusions, and suggest that in the long-term fructose intakes of less than 100g per day do not have any statistically significant effect on fasting triglyceride levels, with a dose-dependent increase over that level [3]. For postprandial triglycerides, dose-dependent increases were seen at intakes over 50g fructose [52] [53]. Statistically significant increases in fasting triglycerides and LDL cholesterol have been associated with fructose intakes of 20-25% total energy or more [54]. An isoenergetic switch to fructose from carbohydrate has been associated with raised triglycerides and lowered total cholesterol, with no effect on LDL or HDL. However, results were only consistent when there was a dose-threshold of >60g day, follow up was for less than four weeks, crystalline fructose was used, or participants had switched to fructose from starch [45].

As previously discussed, it is important to note that the dose, form and study length all impact on results. That is, doses of fructose used in studies are often unrealistically high, in a form not usually consumed (e.g. a bolus of fructose or as crystalline fructose), and acute effects differ from longer-term effects. Energy content of the usual diet is often not controlled, and effects may differ between males and females, or whether a person has diabetes.

5.1.4.2 Body weight
When fructose was added hyper-energetically at high doses (+104 to 250g/day) it did lead to a mean 0.53kg weight gain [38]. Overall, long-term studies have not supported an effect of fructose at intakes up to 100g/day on increased food intake or body weight, when consumed instead of glucose or sucrose [39, 40].

5.1.4.3 Metabolic syndrome / type 2 diabetes
The NHF presented results from a meta-analyses (11 prospective cohort studies) which indicated that consuming 1-2 servings of SSBs/day compared with none to 1 serving/month, was associated with a 20% greater risk of developing metabolic syndrome and a 26% greater risk of developing type 2 diabetes [7]. Looking at individual studies, longer and larger studies tended to show stronger associations. The NHF also presented results from a review (prospective cohort studies and a meta-analysis) showing probable evidence of an association between regular consumption of SSBs (daily basis) and increased risk of type 2 diabetes [55]

One study found an increase of 1 serving/day of fruit juice was associated with an increased risk of diabetes HR1.18 (1.10-1.26). The cohort study was undertaken with 71,346 females from the Nurses’ Health study [30]. There is insufficient evidence to make a statement.

5.1.4.4 Dental caries
Caries are associated with national per capita yearly sucrose consumption, with very few caries in children consuming 10kg/year (about 30g/day) or less. A steep increase may occur from 15kg/year upwards.
WHO evidence suggests **higher rates of dental caries when the level of free sugars intake is more than 10% of total energy intake** compared with it being less than 10% of total energy intake. Furthermore, in three national population studies, lower levels of dental caries development were observed when per capita sugars intake was less than 10 kg/person/year (approximately 5% of total energy intake). Additionally, a positive log-linear dose response relationship between free sugars intake and dental caries was observed across all studies, at free sugars intakes well below 10 kg/person/year (i.e.<5% of total energy intake) [2].

5.1.5 For the health outcomes that have been studied, are the adverse effects seen limited to sugar intake or include refined carbohydrates more broadly?

As is evident from **Sections 5.1.2 and 5.1.3**, the three reviews did not examine the evidence of adverse effects from refined carbohydrates per se. The evidence found points more towards adverse effects from sugar intake, in particular consumption of SSBs.

5.1.6 What evidence is there (within the scope of studies retained in answering review questions 1-5) for the mechanism of action for the adverse effects between the exposure (sugar, refined carbohydrate or specific sugary food) and the specified health outcomes?

A number of mechanistic actions of added sugar on health were identified in the evidence presented by the three major reviews and are outlined below.

5.1.6.1 Form of sugar

It has been suggested that the form in which sugar is consumed could play a role in its biological effect [3]. SSB consumption, in particular, has been a variable of interest as is sugar in consumed in the form of a liquid rather than a solid. While some suggest that the form of sugar, rather than the energy content, is an important consideration, others do not agree [56]. Indeed, evidence does point to a weaker dietary compensation with energy in a liquid compared to solid form [57, 58]; [59] Other reviews found conflicting and insufficient evidence [60] [61].

5.1.6.2 Body weight

The NHF review presented several mechanisms which have been proposed for sugar’s contribution to weight gain: energy density, lack of fibre, high palatability; unique effects of fructose (see below); consumption as a liquid rather than food, thus little satiating effect; and SSB consumption possibly leading to lower thermogenesis and subsequently a positive energy balance [62, 63]. However other studies imply that the effect of dietary sugar on body weight is due to an increased consumption of energy rather than an effect of sugar per se. In studies that exchanged carbohydrates or other macronutrients with dietary sugars of an equal calorie level and where energy intakes were strictly controlled, there was no effect on body weight [13]. The NHMRC review echoes the findings from the NHF review - that compensation for energy from SSBs is less complete than that for energy in solid form, therefore consumption thereof adds to total energy intake [1].

**Body weight & fructose**

The NHF review also discusses the possible mechanism for an adverse effect of fructose on body weight. Fructose may be less satiating than other sugars e.g. compared to glucose it has a lower
glycaemic index (postprandial glycaemia may impact on mechanisms controlling satiety), less suppression of ghrelin and less increase in leptin [42, 64]; fructose is sweeter than sucrose, allowing less to be used in a product for an equivalent sweetness; and it’s metabolism does not require insulin to enter body cells, which means it can be more readily converted to fat (EFSA, 2010; [64, 65]. Despite the possible mechanisms, long-term studies have not supported an effect of fructose at intakes up to 100g/day on increased food intake or body weight, when consumed instead of glucose or sucrose. In addition, six studies have shown that fructose increases energy expenditure compared to glucose and starch [66].

5.1.6.3 Metabolic syndrome / type 2 diabetes
Fructose may be associated with the metabolic syndrome/type 2 diabetes through several mechanisms, including weight gain from increased consumption of SSBs and their high glycaemic load [7]. Some studies also suggest an association between a high intake of SSBs or sugary foods and acute inflammation and oxidative stress [48]. However, the NHF review concluded that further long-term studies are required to confirm this.

5.1.6.4 Dental caries
Fermentable carbohydrates (both sugars and starches) are a substrate for bacteria which increase the acid-producing potential of dental plaque [67]. Dietary sugars other than sucrose (e.g. glucose and lactose) can also induce caries, although these sugars are less cariogenic than sucrose [1]. The more frequently foods containing added sugars are consumed, the greater the risk of caries, since frequent consumption does not allow remineralisation of the teeth [68]. Therefore, advice on sugar intake for preventing dental caries should include frequency of intake as well as the amount [1]. The other factor to consider in dental erosion is the acidity of sweetened drinks (including diet soft drinks) (American Academy of Paediatrics 2004).

5.1.6.5 Heart disease
Fructose may increase the risk of heart disease by increasing triglyceride and VLDL production in the liver [64]; and increasing blood pressure by increasing serum uric acid levels and reducing endothelial nitric oxide [69]. Fructose from fruit is not thought to have an adverse effect due to the level of antioxidants and flavanols present blocking the hypertensive effect and the uric acid lowering effect of ascorbate through increased renal excretion [70].
5.2 Summary of recent additional evidence reviewed

Most of the recent high and moderate quality evidence reviewed was focused on the effects of SSBs on health. It is well known that higher intake of sweetened beverages is associated with higher energy intake. Well-designed observational studies consistently show a significant positive relationship between SSB intake and adiposity [4, 5]. Several well-conducted RCTs have shown statistically significant changes in adiposity as a result of corresponding changes in SSB intake [5]. The effect is especially important in children and RCTs have shown reductions in BMI gain when SSBs intake was reduced [4]. There is accumulating evidence for a relationship between SSB’s and risk of developing CVD (including high blood pressure/hypertension), Type 2 diabetes and metabolic disease [6, 7]. It is important to note that most of this evidence is from observational studies.

There is one systematic review of RCT evidence that showed that fructose consumption was positively associated with increased fasting blood sugar, elevated triglycerides and elevated systolic blood pressure. In addition, it was associated with lower high density lipoprotein (HDL) cholesterol [8]. Based on this current search of the literature, there is insufficient evidence linking sugar consumption and cognitive decline [9].

Recent literature discussing the mechanisms of sugar’s/SSBs impact on health did not add anything to the evidence already presented by the three main reviews.

6.0 LIMITATIONS OF AVAILABLE EVIDENCE FOR POLICY DEVELOPMENT

Assessing the relationships between sugar and health is not without its challenges - the range of definitions of sugar that are used, methodological limitations with the study design, short duration of follow-up or small sample sizes and in some cases evidence of conflict of interest/ funding bias.

6.1 Definitions of sugar/form of sugar used as the exposure variable

Definitions of sugar differ between studies which makes it difficult to combine the results in most cases. Generally the term “sugars” covers all mono- and disaccharides and excludes all other refined carbohydrates. Sucrose (or table sugar) is a disaccharide, made up of two monosaccharides: glucose and fructose. Other common sugars are lactose, found in milk, and galactose. Some studies use fructose (which is the natural sugar which occurs in fruit and some vegetables) as the exposure variable but in the normal diet free fructose is rarely consumed in high doses and high fructose corn syrup (HFCS) is often used in the US food supply but is not commonly used currently in the Australian food supply.

The WHO review uses the term “free sugars” which are classified as all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices. Dietary guidelines generally use the term “added sugars” which are sugars and syrups added to foods during processing or preparation. Many of the studies which are
reviewed for this document use SSBs (drinks with any form of added sugar), such as soft drinks, cordials, sports drinks or energy drinks but again the definition differs between studies and some do/do not include fruit juice as a SSB. Appendix 1 shows the different definitions of sugars which are used in dietary guidelines in Australia and other nations.

In addition, the form in which sugar is consumed could impact on its biological effect. For example, SSBs could have a different metabolic effect to sugar consumed as part of a food, or to naturally occurring sugars from fruit for example. This has been thoughtfully discussed in the NHF review.

6.2 Study design
The majority of research has been from observational studies (cross-sectional and cohort studies), with limited evidence from experimental or RCTs. In cross-sectional studies it is difficult to control for bias including the possibility of confounding, recall bias, and reverse causation. Cohort studies may also have a likelihood of residual confounding and reverse causation. Cross-sectional and cohort studies cannot be used to infer causation. The most robust evidence, and the only type of studies that can show a causal relationship are experimental studies such as RCTs and these are expensive and difficult to conduct.

6.3 Other challenges
In general, many of experimental studies on sugar or SSBs have small sample sizes and short duration of follow up. Challenges are also compounded by the nature of nutritional epidemiology e.g. limitations with dietary assessments used, with some methods more likely to over-estimate intakes for example. Study results may also vary according to funding source and there is some evidence that industry funded intervention studies are more likely to report favourable conclusions or significantly smaller associations than non-industry funded research [18, 71]
### Table 4: Reported adverse effects of sugar on health and levels of exposure reported

<table>
<thead>
<tr>
<th>Health outcome</th>
<th>Exposure</th>
<th>Level of exposure</th>
<th>Level of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease</td>
<td>Sugar [3]</td>
<td>n/e</td>
<td>Inconclusive</td>
</tr>
<tr>
<td></td>
<td>SSBs [3]</td>
<td>≥1 serves/day</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Sucrose [1]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>SSBs [17]</td>
<td>Increase in one serve/day</td>
<td>Insufficient</td>
</tr>
<tr>
<td>Increased triglycerides</td>
<td>Sugar [3]</td>
<td>&gt;20% of total energy</td>
<td>Strong</td>
</tr>
<tr>
<td></td>
<td>SSBs [3]</td>
<td>30-100g; &gt;50g (post-prandial); &gt;60g; 20-25% of total energy</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Fructose [3]</td>
<td>200g</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Fructose [8]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td>Increased blood pressure</td>
<td>Sugar [3]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>SSBs [3]</td>
<td>&gt;1 serve/day</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Fructose [3]</td>
<td>200g</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>SSBs [6]</td>
<td>Intake of &gt;12 floz/day</td>
<td>Strong</td>
</tr>
<tr>
<td></td>
<td>Fructose [8]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td>Increased body weight</td>
<td>Dietary sugar (added and SSB) [3]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Dietary sugar [1]</td>
<td>n/e</td>
<td>Inconclusive</td>
</tr>
<tr>
<td></td>
<td>SSBs [3]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>SSBs [1]</td>
<td>n/e</td>
<td>Strong</td>
</tr>
<tr>
<td></td>
<td>Fruit juice [1]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Free sugars [2]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Free sugars [2]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Fructose [72]</td>
<td>&gt;104-250g/day</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>SSBs [7]</td>
<td>Increment in 1 serve/day</td>
<td>Strong</td>
</tr>
<tr>
<td></td>
<td>SSB [5]</td>
<td>335ml soda/daily</td>
<td>Strong</td>
</tr>
<tr>
<td></td>
<td>SSBs [15]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>SSBs [14]</td>
<td>n/e</td>
<td>Inconclusive</td>
</tr>
<tr>
<td>Metabolic syndrome / type 2 diabetes</td>
<td>SSBs [3, 7]</td>
<td>1-2 serves/day; Daily consumption</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Sugar [1]</td>
<td>n/e</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Fructose [8]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Fruit juice [1]</td>
<td>1 serve/day</td>
<td>Insufficient</td>
</tr>
<tr>
<td>Increased dental caries</td>
<td>Added sugar [1]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Soft drink [1]</td>
<td>n/e</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Sucrose [1]</td>
<td>&gt;15kg/per capita/year</td>
<td>Insufficient</td>
</tr>
<tr>
<td></td>
<td>Free sugars [2]</td>
<td>&gt;10kg/person/yr or &gt;10% total energy</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Free sugars [2]</td>
<td>5-10% of total energy</td>
<td>Insufficient</td>
</tr>
</tbody>
</table>

n/e= Not evident from the review
# Level of evidence

Shading denotes level of evidence e.g. darkest grey = strong level of evidence. **Strong** = there is probable evidence of an association based on at least one high/moderate quality systematic review; **Good** = evidence of an association (however non-significant) based on at least one high/moderate quality systematic review; **Insufficient** = the evidence for an association is currently weak so caution is warranted; **Inconclusive** = some studies that show a positive and some a negative/no association i.e. conflicting results from studies.
7.0 CONCLUSIONS

7.1 Overall summary of evidence

This review indicates that it is likely that added sugar has an adverse effect on health. Some believe that this is because sugars are found in beverages and foods which are inexpensive, palatable and readily available and may be more likely to be purchased by lower-income consumers [14]. Some studies which exchanged carbohydrates or other macronutrients with dietary sugars of an equal calorie level and where energy intakes were tightly controlled, body weight remained unchanged [3]. Therefore while there is good evidence to suggest that dietary sugars may lead to overconsumption of energy, more solid evidence is needed to determine whether added sugar \textit{per se} has a negative impact on health. The strongest evidence indicates a positive association between SSB consumption and body weight. Some argue that this association is due to the form of sugar (i.e. fluid form meaning weaker energy contribution), but evidence is inconsistent [3]. SSBs provide no nutritional benefit to the diet therefore recommendations to reduce intakes are not likely to result in negative dietary consequences.

While there is insufficient evidence to suggest a direct influence of sugar on risk of heart disease, metabolic syndrome or type 2 diabetes, there is growing evidence of a positive association between SSBs and increased risk of these conditions [3, 6, 7, 17]. In addition, one study of moderate quality found an association between fructose consumption and indicators of the metabolic syndrome (increased fasting blood sugar, elevated triglycerides, elevated blood pressure and decreased HDL cholesterol) [8]. While fructose naturally occurs in fruit and vegetables, it contributes very little to daily energy intake [8] therefore it is the fructose present in processed foods which is considered the main underlying cause for the association between fructose and this syndrome. However added fructose is not commonly found in the Australian food supply where sucrose is the major sugar added to food and beverages.

The evidence suggests a positive association between sugar and SSB intake and increased risk of dental caries, particularly in children. From our additional search of the recent literature, there was no additional sufficient evidence found linking sugar intake with dental caries. Indeed the NHMRC do highlight that several key references were published previous to 2002 which could be used to guide decision making. It is also recommended that advice on sugar intake to prevent dental caries should include the frequency of intake as well as the amount [1]. Our search found no reviews of moderate or high quality examining the relationship between sugar intake and behaviour.

It is evident that SSB consumption is a variable of interest and it appears that even one daily serving of SSBs is enough to have a negative impact on health. It has been more difficult to decipher a detrimental level of exposure for other sugars due to the large heterogeneity among study dosages (fructose levels examined in this current review ranged from 30g/day to 100g/day) and some have questioned the doses used in feed trials as they may not be applicable to the human diet [73].

It is also evident from this review that it is biologically plausible that sugar has an adverse impact on health. There has been a lot of discussion regarding the mechanisms for the positive association
between sugar and body weight e.g. form of sugar, energy density, lack of fibre, little satiating effect [3]. There has been less discussion around the mechanistic actions of sugar on increasing risk of other conditions, however it is likely that fructose may have an adverse effect on the metabolic syndrome and type 2 diabetes via a variety of mechanisms including its’ ready conversion to fat [3, 8].

While not within the scope of this current review, three additional important resources were found in the grey literature. The first resource is a website (http://www.sugarscience.org/) developed by The University of California, San Francisco, where soft drinks were recently banned from all campuses. The website includes evidence-based, scientific information about sugar and its impact on health. The team have reviewed more than 8,000 scientific papers published to date and focus in areas where the science is the strongest (diabetes, heart disease and liver disease).

The second resource identified was the Carbohydrates and Health report, a recently released (17 July 2015) report of evidence from the (UK) Scientific Advisory Committee on Nutrition (SACN)[74]. Although the report includes carbohydrates as the exposure it also addresses the effects of individual sugars. SACN concluded that “population average intake of free sugars should not exceed 5% of total dietary energy. This advice, that people’s intake of ‘free sugars’ should be lower than that currently recommended for non-milk extrinsic sugars, is based on SACN’s assessment of evidence on the effect of free sugars on the risk of dental caries and on total energy intake.” [74]. Appendix 2 gives a summary of the major characteristics of this report. It is important to note that case-control, cross sectional and ecological studies were not considered so the number of studies included was fewer than the WHO report and there were very few studies on individual sugars (glucose, fructose or sucrose) which met the inclusion criteria.

Overall, the findings from the SACN review were similar to the reviews described in this current report - higher consumption of sugars and sugars-containing foods and beverages associated with a greater risk of dental caries; prospective cohort studies indicating that greater consumption of SSBs is associated with risk of type 2 diabetes mellitus; RCT evidence indicating that a higher sugars intake increases energy intake; and that the consumption of SSBs results in weight gain and an increase in BMI.

A third piece of evidence which has been recently published online is recent analysis from the Global Burden of Disease study, which has estimated that a total of 8.5 (2.8, 19.2) million disability-adjusted life years (DALYs) were related to SSB intake (4.5% of diabetes-related DALYs) in 2010 [75]. This research suggests that SSBs, which are a single,modifiable component of diet, that can impact preventable death/disability in adults in high, middle, and low-income countries, indicating an urgent need for strong global prevention programs.
7.2 Policy implications

Based on the current evidence it would seem appropriate to adopt the recent WHO recommendation that adults and children do not consume more than 10% of total energy as free sugars. WHO also makes the conditional recommendation of a further reduction to less than 5% of total energy consumed as free sugars. This latter recommendation was deemed conditional as “the desirable effects of adhering to the recommendation probably outweigh the undesirable effects but these tradeoffs could not be clarified” due to the quality of evidence available [76]. It is however recognised that it is difficult to make recommendations based on added or free sugars which are not currently required to be labelled by law in NSW. In addition, while the levels of added sugar intake in Australia is currently unknown, recent data from the Australian Health Survey indicate that total sugars contribute to 20% of dietary energy with the greatest contributors comprising of discretionary as well as core, nutrient-dense foods [77]. Although this current review did not find studies which would support a recommendation to limit sugar intake from core, nutrient-dense foods for the general population, there is sufficient evidence to recommend reducing the energy-dense, nutrient-poor foods that currently provide a significant contribution to total sugar and energy intake in the Australian diet, in particular SSBs but also cakes and sugary spreads and additionally for children, biscuits, ice cream and confectionary as evidence indicates that these products are consumed in large quantities particularly by children, consumption dilutes nutrient density of the diet and a reduction thereof of presents no harm to diet adequacy [1, 78].

Any recommendation concerning dietary sugar intake should therefore follow the Australian Dietary Guidelines to “limit intake of foods and drinks containing added sugar such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks”. [1]. It is likely that with the growing interest among the population and evidence accumulating on the association between sugar and health the area will increase in salience for policy makers. Limitations in the reviewed research preclude more specific guidance on evidence based policy directions or positions at this time.
### APPENDICES

**Appendix 1: Different definitions of sugar used in dietary recommendations**

<table>
<thead>
<tr>
<th>Country/Authority</th>
<th>Term used</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>Added sugar</td>
<td>Added sugars includes sucrose, glucose, fructose and corn syrup. These are separate from naturally occurring sugars found in milk, fruit, vegetables and legumes.</td>
</tr>
<tr>
<td>UK</td>
<td>Non-milk extrinsic sugars</td>
<td>Sugars not contained within the cellular structure of a food and sugars in milk and milk products.</td>
</tr>
<tr>
<td>WHO</td>
<td>Free sugars</td>
<td>Sugars added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups fruit juices and fruit concentrates.</td>
</tr>
<tr>
<td>US</td>
<td>Added sugars</td>
<td>Sugars and syrups that are added to foods during processing and preparation.</td>
</tr>
<tr>
<td>European food Safety Authority</td>
<td>Added sugars</td>
<td>Sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup, iso-glucose) and other isolated sugar preparations used as such or added during food preparation and manufacturing</td>
</tr>
</tbody>
</table>
## Appendix 2: Characteristics of the Carbohydrates and Health Report (SACN)

<table>
<thead>
<tr>
<th>Review</th>
<th>Type/Dates/ Sources used</th>
<th>Population/Dietary exposure</th>
<th>Outcome</th>
<th>Tool used to assess quality of studies included</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrates and Health Report SACN</td>
<td>From 1990-Dec 2009 (update search, not sys review up to June 2012) only included cohort</td>
<td>Adults and children ‘Sugars’ all dietary sugars in cohort studies. For randomised controlled</td>
<td>Colorectal health (Colorectal cancer, IBS, constipation)</td>
<td>Developed their own framework and grading system with technical consultation and public comments</td>
</tr>
<tr>
<td></td>
<td>and RCT evidence. No case control or cross sectional studies included.</td>
<td>trials, ‘sugars’ is used when considering trials where the exposure cannot be clearly attributed to specific sugar(s) types investigated (e.g. sucrose, lactose, glucose or fructose).</td>
<td>Cardio-metabolic health (CVD, insulin resistance, glycaemic response), Obesity, Oral health</td>
<td>Evidence Adequate, Moderate, Limited (see reference for more details)</td>
</tr>
<tr>
<td></td>
<td>67 prospective cohort studies and 41 RCTs investigating relationship between sugars,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>individual sugars, sugars-sweetened foods and beverages and sugar alcohol intake and</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>cardio-metabolic, colorectal and oral health outcomes. Thirty six prospective cohort</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>studies and nine randomised controlled trials were conducted in children and adolescents</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>and are considered separately at the end of the chapter.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
REFERENCES


