Comments on the SACN Draft Carbohydrates and Health report

Submission by
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We declare that both authors have no interests to declare.

Summary

We welcome the extensive analyses and thought that has gone into the SACN report on carbohydrates and much of its original thinking. We consider that the final report can be strengthened by making use of the SACN approach but with new perspectives derived from new data and a reconsideration of some of the assumptions embodied within the report as now set out:

1. The SACN group seem to have essentially adopted the WHO definition of "free sugars" and it is a pity that it did not follow suit when considering the WHO definition of fibre as non - starch polysaccharides. The new SACN choice, much favoured by the food industry, includes lignin and maillard products with no known advantageous biological effects, and will allow the food industry to add a range of ingredients of dubious biological value. These issues have been well considered by other major international groups including WHO and it is regrettable that SACN has suggested this new approach.

2. We note that the SACN evidence only allows data from limited studies to be appraised if they were published after 1990. New data are not intrinsically better than older work and this partial view may explain the discrepancies between some of the systematic analyses conducted by SACN and others.

3. New evidence has emerged on the role of free sugars in inducing blood pressure and other cardiovascular risk factors when body weight is stable. Detailed earlier evidence also suggests that in weight stable states, less dietary fat and more carbohydrates reduce blood pressure perhaps in part because of carbohydrates' greater content of blood pressure reducing minerals.

4. The energy gap calculations by SACN relate predominantly to the extra energy stored during weight gain over a decade in England when the requirement is not just to arrest the continuing exacerbation of the epidemic of overweight and obesity, but to reverse it. The appropriate calculations, using SACN methodology, show that substantially larger reductions in energy intake than the proposed 100kcal reduction are needed.

5 Novel analyses of the robust quantitative relationship between free sugar intakes and dental caries suggest that in England minimum intakes of free sugars are needed and that 5% free sugars should be the absolutely upper limit for individuals with population averages of 2-3% or less. Minimum sugar intakes are needed because dental hygiene and the use of fluoride toothpastes are not the answer to the problem of caries, even if sugar intakes are lowered to 5%.

6 The increase in fibre intakes proposed may not be possible without a marked concomitant reduction in dietary fat if further weight gain is to be avoided when advocating the consumption of higher fibre rich foods. The practicality of the proposed increased fibre intake should be illustrated and the risks of further inappropriate weight gain highlighted.

Introduction.

We welcome and congratulate the SACN on this much needed report. The extent and depth of the analysis of both weight relationships and dental caries sugar are very timely and the use of the systematic reviews gives greater general credibility to the proposals made.

This report clearly has involved a major effort with a sequence of systematic reviews over several years followed by an attempt to take account of more recent findings just before the publication of the draft report. The expert group is to be commended on the effort that this must have involved.

Limitations of the general approach

Perhaps the most remarkable feature of the SACN report is the decision to assess only papers published since 1990 (see Annex 2 section A2.1). The reason given was that the earlier literature had been considered by a previous COMA committee. This means that the supposedly careful and all inclusive systematic reviews excluded a host of potentially meticulous earlier studies which, when combined with the later trials and cohort studies, would have given a far more robust set of results than either COMA could have done at the time or that SACN could now do with its use of the more limited recent evidence. A systematic review is meant to be a collation of all the published evidence on a subject not just what can conveniently be derived from recent studies. This handicap becomes clear when considering the results obtained with some of the systematic reviewed topics.

Limitations of cohort and trial data alone. SACN more conventionally and in keeping with the current WHO process, only uses cohort studies and controlled trials for their analysis and interpretation. This is a pity because tightly controlled metabolic and clinical studies often provide not only mechanistic insights but also allow one to understand better the severe limitations of cohort studies and the need to discern the significance of the precise setting and criteria used in controlled trials.

Cohort analyses have major errors in dietary analysis which are either neglected or purportedly managed by various statistical devices. The SACN authors acknowledge this in their report but one subsequently has little feel for how this altered their judgements. The precise outcomes chosen may also have substantial errors in their determination and cohort studies usually do not often amplify our understanding of the mechanisms involved. This was shown when statistical analyses of very famous and still widely quoted cohort studies showed that the combination of dietary and outcome errors allowed the investigators to produce highly statistically significant but spurious positive or negative relationships. The frequent suggestion that different groups within a cohort provide evidence of what would happen if a dietary change were made also neglects the associated known and unknown differences between the subgroups. Yet at times many authors and sometimes the SACN experts seem to assess these cohort data as almost equivalent to a clinical trial.

A negative finding in a cohort study does not negate a dietary factor's importance. Thus the lack of an association between saturated fat intake and coronary heart disease in cohort studies does not remotely mean that saturated fat is not the primary driver of the coronary heart disease epidemic. The problem arises because the individual genetic and epigenetic determined responses to the specific dietary component i.e. saturated fats are revealed by the very large differences in the blood cholesterol response to a standard saturated fat intake change - a clear finding from many hundreds of tightly controlled metabolic and clinical studies. Mechanistically from animal experiments, including primate studies, as well as clinical and clinical genetic analyses one can discern a critical mechanistic factor such as the blood lipoprotein cholesterol concentration which may not be included as a critical component in a simple cohort study of fat intakes as such and heart disease. So by neglecting the broad array of science one can come to very limited or even erroneous conclusions. This is not a quirky view but one set out clearly by NICE[1] in its analyses of how major public health issues cannot be considered in the same way as the analyses of treatment with a drug or the value of a medical device. One is no less a scientist for dissecting the value of a study and integrating different dimensions of evidence.

The much vaunted controlled trial can also mislead unless one has a real insight into some of the potential drawbacks arising from the choice of subject, the magnitude of the change in diet

envisaged, the nature of the exchange of macronutrients when dealing with sugar or fibre or total carbohydrate intakes. The degree to which the diet is actually controlled as distinct from being prescribed and the duration of the study are also often crucial.

Some of these drawbacks are clearly acknowledged in the report and SACN's expert group is to be congratulated on pointing out the many disadvantages of both cohort and trial studies in sections 1.8 to 1.11.but all these caveats do not seem to come in to play and affect the process of evaluation.

If one considers how these issues apply to the SACN report then the importance of the individual range in responsiveness to defined dietary changes is not mentioned as a fundamental issue because this understanding can only really come from tightly controlled metabolic studies.. Section 1.8 simply ascribes the problems incorrectly to only dietary assessment errors.

Definitions of carbohydrates.

The SACN report correctly mentions the 2003 WHO 916 expert report as using the term "free sugars" but then having rejected the rather cumbersome UK term of " non- milk extrinsic sugars" the SACN report suddenly also now defines the component as "free sugars" but then indicates that it differs from that used by WHO in 2003. In practice the only addition seems to be the inclusion of syrups (paragraph 11.7). This is a helpful emphasis as it would also be accepted as appropriate by WHO.

Definition of fibre. The SACN report does set out the Codex committee's criteria whereby the committee indicates that resistant starch should not be included in the definition of dietary fibre (Section 2.29) but then does not follow the logic of relying on non - starch polysaccharide as the key component and plumps for the new AOAC methods as appropriate. Given the notorious influence that the food industry has on Codex deliberations it is not surprising that Codex opted for this AOAC approach because the industries are interested in magnifying the fibre value by choosing a method (with the newer AOAC techniques) which maximises the carbohydrate components involved. The AOAC method also attempts to measure resistant starch but in practice often does not assess the amount actually consumed. If rice, for example, starts to cool before eating or if reheated - a very frequent occurrence in many societies - then retrogradation of starch begins immediately so the AOAC values will actually underestimate the amount of resistant starch actually present in the meal as eaten. If the AOAC analyses are undertaken by the industry itself for labelling their processed foods then they can maximise the fibre value by arranging for their foods to cool completely before starting the analysis. The AOAC method also provides, for example, a figure for "fibre" which in practice in some products like cornflakes largely reflects the fact that maillard products (with no assumed metabolic effects) are included in the term "fibre".

The discussion in the UN report on dietary fibre by the UK and world experts (Cummings and Stephen - who do not seem to have been consulted for their expertise) also shows how the SACN choice of the AOAC method of definition is conditioned it would seem by the finding that oligosaccharides as well as resistant starch can promote faecal bulking. Yet most of the world literature relates to the older AOAC methods which do not include these components- see Section 11.5.The SACN expert group may not know that the choice of the AOAC by US groups, usually closely linked to the food industry, came after decades of nutritional analyses in the US where almost all their dietary studies (on which a number of analyses in the SACN report depend) did not measure carbohydrates at all. They just inferred its quantity from crude analytical measures of the residual dry weight of a food once the protein (by nitrogen analyses only), the fat and ash content of the dried matter had been quantified.

The SACN expert group specify that their definition of fibre is of “carbohydrates that are naturally integrated components of foods and that are neither digested nor absorbed in the small intestine and has a degree of polymerisation of three or more monomeric units, plus lignin". They do not specify why they include lignin and maillard products when they have no proven health benefit, and the inclusion of carbohydrates with a DP of 3 or more is made despite the physiology of the handling of these 3-10 carbohydrates being totally different from those of non-starch polysaccharides.

Effects of total carbohydrate and selective components on blood pressure.

It is intriguing to read the analyses of the effects of carbohydrate on blood pressure where the disadvantage of having a mechanical approach to the systematic reviews of cohort trials again becomes evident (Section 5.23 and 5.24). The problem of assessing accurately the total carbohydrate intake is not mentioned but at the end of 5.24 and 5.25 the expert group notes that the controlled trial comparisons are almost all made with weight reducing diets. Weight reduction has been recognised for many years for its effects in lowering blood pressure. So these comparisons are only of value in specifying that blood pressure will fall with weight loss and the composition of the diet during weight loss does not seem to be so important.

At no stage is there mention of the classic DASH trials[2] because SACN, for the reasons already specified, seems to assume incorrectly that only recent studies are of high quality and value. In the DASH trials the investigators meticulously ensured that their subjects did not change weight when their fat intakes were reduced from 37.5 % to 25.7% with a total carbohydrate intake increase from 49.2 to 56.5% (when switching from their vegetable rich diet to the combination lower fat, vegetable rich combination diet albeit with an increase in protein intake from 15.1% to 17.9%). This was accompanied by a consistent fall in blood pressure of 2.7 mmHg systolic and 0.8 mm Hg diastolic in non hypertensives and falls of 2.6mmHg systolic and 2.8mmHg diastolic

blood pressure in those individuals with high blood pressure. These changes are substantial from a public health point of view. The hypotensive effects may have related to the increase in calcium absorption as a result of increasing the proportion of low fat dairy products in the combined test diet but there was a notable failure to increase urinary calcium excretion and the authors concluded that the overall study showed the beneficial impact on blood pressure of a higher vegetable and fruit diet with a lower fat (and saturated fat) , higher carbohydrate diet as well as a lower salt intake (measured in another phase of the DASH trials).

This classic trial should therefore have alerted the SACN expert group to recognise that carbohydrate rich diets are usually also rich in potassium (a blood pressure lowering mineral) and other minerals whereas fats are devoid of these minerals. So total carbohydrate does not have a neutral effect on blood pressure if one assesses subjects in energy balance.

Carbohydrate intake in relation to dietary energy density and weight gain.

The issue of dietary energy density has not really been considered by the SACN experts because they are locked in to cohort or trial data where until recently there were few estimates of dietary energy density. The same problem has applied to the recent WHO analyses of the role of fat and sugar on weight gain where they were forced by the WHO demand for a grading system based on cohort and trial data only to consider not energy density but the separate roles of fats and carbohydrates. If, however, the experts had broadened their perspective they would have realised why the WHO 916 Expert Technical Consultation had identified energy density as one of the key factors leading to weight gain. Dietary density is increased by a greater fat content as well as a greater sugar and refined starch content of the diet and by a lower proportion of fibre rich carbohydrates with their original water retaining structure as well as vegetables and fruit. This feature therefore also relates to the observed potential effect of fibre rich diets in reducing the propensity to weight gain. Thus the dietary proportion of carbohydrate and its specific components and structure in relation to weight gain would have allowed the SACN expert group as well as WHO to set the whole topic in a much more coherent way.

Despite these drawbacks the SACN compilation of the different trials of sugar intake and its relationship to weight gain (Figure 1 on page 202) is very commendable in allowing the reader to gain a perspective on the impact of sugar intakes. Certainly there seems to be no threshold in the effects of sugar intakes on weight gain so this alone would justify specifying as low a sugar intake as possible when the disease burden induced by excess weight in England is so enormous.

Weight reductions needed. The SACN calculations suggest the need for a mere 100kcal average intake reduction to not only prevent weight gain but to induce a slight fall in body weight. This figure is based primarily on the assessment of the excess energy stored per day of 20-40

year old adults between 1999 and 2009, as set out in a very simple set of calculations made by the SACN Calorie Reduction Expert Group. However, one needs to consider the assumptions of both the SACN carbohydrate report and the Calorie Reduction Expert Group and realise that the 100kcalorie reduction is only slightly greater than the estimated daily energy storage of young English adults gaining weight over a 10 year period. The calculations of the Calorie Expert Group make no allowance for the well-recognised increase in routine maintenance energy requirements on weight gain and make absolutely no allowance for adjusting energy intake to cope with the pre-existing major public health problem of overweight and obesity in England. So if one takes the data of the Health Survey for English males and females above 16 years, their average weights (when actually measured rather than self- reporting) were 83.9kg for men and 71kg for women. This means their respective average BMIs were 27.3 for men and 27.1 for women.

It is obvious that to reduce the average BMI to 25.0 simply means that 50% of the population would still be overweight (BMI 25-29.9) or obese (BMI 30+). That is why one of us concluded for the WHO major Millennium analysis of the global burden of disease that the ideal average BMI should be 21 to minimise the proportion of those who were either underweight or overweight[3]. This would mean an average reduction in body weight to 64.5kg in men and 55kg in women. This is, of course, unrealistic in the near future but that is the counterfactual analysis which is still used by WHO albeit the latest non WHO global burden analysis from Seattle and Harvard allows an optimum BMI of 21-23. Even if one takes a BMI of 23 or the SACN Committee's Dietary Reference Energy Report[4] with the use of a BMI of 22.5 this would still mean a BMI reduction of over 4 units. This corresponds to an average desirable body weight reduction of 10kg -15kg. If one then takes the SACN values for dietary energy requirements with the Henry equations, seemingly favoured for their estimates of basal metabolic rate, and the physical activity levels of 1.62 from the $D_2\ 0^{18}$ values quoted by the SACN report on dietary reference values for energy, then reducing the body weights of men and women to the so-called SACN desirable levels of BMI 22.5 would require an average reduction of about 275kcals/d in 30-60 year old men and 150kcal/d in women. This does not include the 15 kcal - 40 kcal reduction to avoid current weight gain as calculated by the Calorie Reduction Expert Group. So the really desirable kcalorie reduction in an average adult in England amounts to about 300kcal/d for men and about 175kcal/d for women. The reductions needed for the majority of overweight and obesity individuals will be even greater e.g. 300 kcal -600kcal/d for men and perhaps 200kcal- 400kcal/d for women.

These relatively simple calculations using SACN's own approach means that the SACN carbohydrate report is presenting an extremely modest suggested change in dietary energy intake and in the composition of the diet. Clearly the desirable reduction inferred from previous SACN reports could not be achieved even by eliminating all sugar in the diet. Nevertheless the calculations do suggest the importance of minimizing sugar intakes on a population basis. So a

proposed 5% average sugar intake can now be seen to be an undesirably high intake from an energy balance point of view as well as for the avoidance of caries (see below). The implications of the current excess energy intake also need to be taken into account when considering the SACN's proposal for quite a substantial increase in fibre intake and the suggestion that the 50% value for total dietary carbohydrates is still appropriate (see below) .

When attempting to assess the magnitude of dietary changes based on food intake surveys it is also important to recognise that the current estimates and particularly those which depend on a dietary history may underestimate intake not only in absolute kcalorie terms but specifically because of the selective underestimation of dietary energy intake in those who are overweight and obese. Overweight and obese subjects also tend to underreport selectively their sugar intakes and probably their total fat intakes too.

Sugar and blood pressure.

The SACN report also closed its analyses before the new systematic review of the impact of sugar on blood lipids and blood pressure conducted by the WHO group[5]. This group took great care to consider all trials of whatever age of publication and specifically only included those trials not involving weight loss. The WHO group also considered the impact of any trials funded by the sugar industry - a feature and issue not mentioned by the SACN expert group These new analyses should be included in the final draft of the SACN report as they suggest the need to re-emphasise the value of even lower sugar intakes. Sugars are, of course, also devoid of blood pressure lowering minerals, so the intriguing finding of the greater effect on blood pressure of sugar intakes the longer the trial may relate to the change in mineral content or, as the authors suggest, to the fructose stimulation of uric acid production. Uric acid has effects which include reducing nitrous oxide induced vasodilation and stimulating the renin angiotensin axis.

Sugar and lipids

The same WHO analysis[5] of the impact of sugar intakes on blood pressure also showed - using all the data available - that sugars do have a deleterious impact on lipid levels which are predictive of increased cardiovascular disease even if the changes are modest compared with the effects of saturated fats etc.

Sugar and dental caries

Our comments centre mainly on the SACN recommendation that the intake of free sugars should not exceed 10% of total energy for individuals and 5% for populations in both adults and children.

We consider that SACN should use the new information on dental caries to modify and strengthen these recommendations.

Many helpful points were set out in the SACN report and they are to be congratulated on this. The recommendation that "The dietary reference value for free sugars should be set at a population average of around 5% of dietary energy for age-groups from 2.0 years upwards. This is based on the need to limit free sugars to no more than 10% of total energy intake at an individual level, which is likely to lead to a population average free sugars intake of around 5% of total energy." The recommendations for Dietary Reference Values for sugars outlined in Paragraph 11.13 are clearly based mainly on energy imbalance but a more appropriate interpretation of epidemiological data relating to the disease process of dental caries would have changed the emphasis of the report and led to a more stringent recommendation for free sugar intakes.

Caries does not develop unless dietary free sugars are available. Sugars have been known for several decades to be the unique causative factors leading to caries: when sugar intakes in a region, or country are negligible caries is practically non-existent even in octogenarians who have been exposed to the dietary sugar intakes for many decades. For example, people of all ages on diets low in sugars, such as in Nigeria in the 1960s, had negligible dental caries (98% of all ages were completely free of caries) despite having poor hygiene and many variables considered to be confounders of dental problems in general. This was a detailed population study by one of us[6] but others have found similar populations who were essentially caries free if their sugar intakes are negligible. Some of these studies might be dismissed if one conjures up the idea that there are other factors involved in caries development or its inhibition. However, no such factors have been found, and quantitatively the findings are robust whether one considers detailed population prevalence, cohort studies or so called ecological studies. Thus, the usual great caution associated with ecological analyses is appropriate when one has several confounding casual or major potential modifying factors but there are none in relation to dental caries. The fundamental link between the presence of free sugars and caries, doubtless understood by SACN, cannot be dismissed by epidemiologists if these analysts of population data do not take account of the intrinsic biology of dental caries development. Therefore the sugar/dental caries intrinsic link allows the use of so-called ecological evidence as a fundamental part of the epidemiological analysis. Analyses are intrinsically incomplete and likely to be unsound if they rely only on trial evidence or cohort studies.

The remarkable tissue damage induced by free sugars. The SACN Report might also have highlighted that if any foodstuff commonly consumed were to cause ulceration of tissues in the gastro-intestinal tract, it would be banned. Yet it is extraordinary that the intake of sugars, that causes cavitation and destruction of the hardest tissue in the human body i.e. the dental enamel

(see Figure 1), is not considered a risk factor that needs the strongest controls to ensure that intakes are minimized.

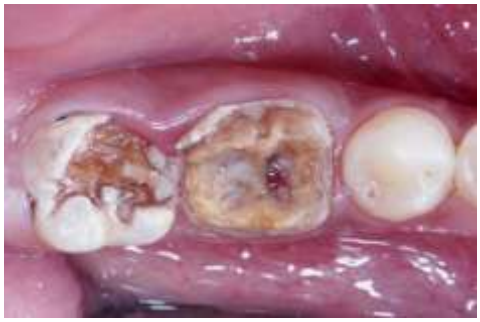

Figure 1. Dental caries in children related to sugars consumption. Dental decay has destroyed most of the molar teeth and the disease has extended into the dental pulp

The burden of disease from sugar induced caries. Caries affects 3.9 billion people worldwide. That indicates that untreated caries is the most prevalent of all 291 conditions assessed in the recent Global Burden of Disease study[7] "Worldwide, oral disease is the fourth most expensive disease to treat; dental caries affects most adults and 60-90% of schoolchildren, leading to millions of lost school days each year, and remains one of the most common chronic diseases; ..."[8]. Indeed in England's children about a fifth of their teeth are carious or have been treated and by the time adults are in their 7th decade, over 70% of all teeth have been affected and are filled, carious or missing[9]. This is despite all the efforts to prevent dental caries and the delaying effects with the very modest inhibitory value of the use of fluoride toothpastes.

Dental care as a preventive measure in dental caries

Given that caries is uniquely caused by free sugars, one still has to recognise that for several decades the preventive approach has focused on the potential benefit of exceptional dental care on a routine basis and the use of fluoride in drinking water and in toothpastes. We are now, however, fortunate to have exceptionally good evidence on the potential long term impact of these two preventive approaches. The first is dental care, but this can now be discarded as of major preventive importance because even with superb systematic dental care in an affluent society there is a progressive increase in the prevalence of caries. This was shown in a meticulous longitudinal study where New Zealand children from the age of 5 years were provided with specific advice plus individual dental hygiene by experts every 6 months for 27 years in Dunedin[10,11]. In Dunedin the sugar intakes are similar to those in many affluent countries. In New Zealand different estimates of sugar intakes suggest that children are eating on average 60-70g sucrose daily (about 15% of energy) and adults are consuming about 10% sugar i.e. similar intakes to those suggested for England. So, effective repeated dental care - actually incorporating the use of fluoride enriched toothpastes – did not stop the progressive increase in dental caries

from the age of 5 years up to the age of 32 years in Dunedin. The SACN do not explicitly deal with this issue to demonstrate that the standard dental view of the benefits of routine toothbrushing and even repeated free dental care for children at school cannot prevent the progressive increase in dental caries if so much free sugar is provided in the diet and in soft drinks.

Fluoride is valuable in reducing and delaying sugar induced caries. The second supposed preventive approach is the use of fluoride in toothpaste and in drinking water. We are fortunate to have plenty of evidence that fluoride does prevent the development of some lesions, but delays its onset and reduces the overall burden of caries by about 10%[12]. This explains why there are high numbers of teeth affected by caries in adults despite the low levels of caries in children and adolescents. Fluoride, the main reason given for the caries decline in children, does not appear to increase the resistance of enamel enough to control the demineralizing effects of acids produced from dietary sugar. Fluorides slows the progression and re-mineralizes some of the sub-clinical caries processes. This may be more effective than in previous decades when fluoride toothpastes were not so widely available and used. However, fluorides are delaying the clinical manifestation of caries as a cavity until later in the life course. The caries process continues, however, because the determining factor, sugars, has not been adequately controlled.

We stress the benefits of fluoride, but it is not a substitute for drastically limiting sugars in the diet. Fluoride use still leaves a prevalence of sugar induced caries affecting over 70% of adult populations even when intakes of sugar are relatively low. We regret the SACN Report did not make this point sothat the importance of fluoride could be emphasized. Yet when populations consuming fluoridated water e.g. Australia where 80% are consuming fluoride, or Malaysia (76%), Ireland (73%), United States (72%), and in New Zealand (61%) where there is still a striking increase in caries with age and most of the caries experience is in adults, not in children[10].

Quantitative relationship between intakes of free sugars and dental caries

As WHO reports on Diet, Nutrition and the Prevention of Chronic Diseases (TRS 797, 916)[13,14] indicated, dental caries gives us the unique ability to set quantitative limits for sugar intakes in relation to disease. SACN states that “Where possible, the dose-response relationship between carbohydrate intakes and health outcomes has been considered and used to inform the dietary reference values. (Para 1.10). The specification of a 5% average sugar intake with a 10% maximum is a very commendable way of looking at both the general population health issue and the susceptibilities of individuals. The SACN Committee should be congratulated on this emphasis. However, if one considers this concept with our new understanding of the real lifelong impact of sugar on dental enamel then one can see (Figure 2) that as sugar intakes move up from about 0% to 2-3% the level of dental caries is doubled if the teeth are exposed for only 8 years - not the 70+ years we really need to consider on a population basis.

The SACN report needs now to take into account the old evidence, recently made available in English, of the established quantitative relationships between sugars and dental caries and the more recent evidence that there is a major burden of dental caries in middle aged and older adults who have been exposed to fluoridated drinking water for most of their lives. SACN recognizes that for caries, "… there appears to be continuous benefit as percentage energy from sugars decreases. Therefore it appears that in relation to both improving oral health and reducing the risk of weight gain, sugars should provide no more than 10% of dietary energy." (Para 11.9). However, as we show below, the dose-response relationship reveals that there is no threshold at 10%. Indeed the information on dental caries that we outline below should be used to overcome "the uncertainty around the figure" (para 11.10) and thereby strengthen the arguments for the recommendations on the population average and the individual level[15,16].

There is no evident threshold for sugars but a log-linear increase with at least a doubling in caries rates when sugar intakes increase from <1kg sugar/caput/yr. (≈0.05%E) to 5–7·5kg sugar/caput/yr. (≈2·7%–4·1%E) if teeth that have been erupted for 7–8 years are considered. The positive correlation between sugars and caries was +0·7 with a log-linear relation at both lower and higher sugar intake levels for all tooth types if 1–8 years of sugar exposure is considered (Figure 2). However, this does not take account of the further increases when sugar intakes are increased throughout life (see below)

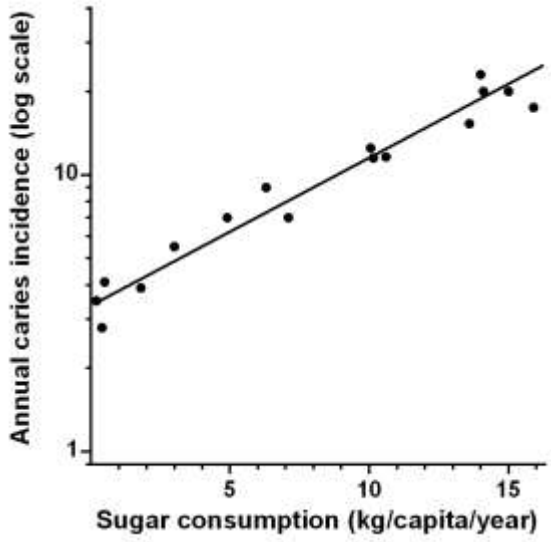

Figure 2. Relationship between annual per capita sugar consumption and annual caries incidence in lower first molars. Data based on a nationally representative sample of 10,553 Japanese children who were monitored yearly from the age of 6 to when they were 11 years old. Data plotted on a log scale. (See Sheiham & James 2014a[15], and attached).

Adult caries should be emphasized in the context of the recommendations relating sugar intakes to dental caries as well as to adult weight gain.

We consider that the recommendation should be reworded to highlight the applicability of the recommendation to adults as well as to children as the report's readers may not realize that in terms of dental caries, even international dental experts still focus incorrectly on the problem in children only. This improper emphasis arises from the traditional approach of those in dental epidemiology who found that school children had rampant caries and it was possible to conduct relatively simple surveys with a high response rate if they assessed schoolchildren of about 12 years of age. This has led to a total distortion of the public health approach to sugar and the crude assumption that fluoride would fix the problem when although it prevents some lesions, its main role appears to be to delay the later stage of caries, namely cavitation.

The citing of adult caries in the SACN report is indeed valuable but the problem is underplayed in the draft release. Adult caries accounts for about 80% of the dental care costs relating to caries compared with only 20% for children up to the age of 18 years. So the traditional focus on 12 year old children probably related to only about 10% of the health burden. If one then considers the adult problem, this means that one has to consider the multiple of the two key factors of importance in the development of dental caries i.e. the magnitude of sugar exposure and the time the teeth have to cope with repeated acid attacks on the enamel. Once this time element is understood with tooth exposure times of 70+ years on average then this means that we need to keep the magnitude of exposure as low as possible throughout the life course.
The evidence that most dental caries occurs in adults is shown in Figure 3[9,17]. Taken together, the data showing lower dental caries rates in children at intakes of sugars equivalent to less than 5% of total energy intake, still reveal a progressive and substantial burden of current or treated dental caries when one considers adults. Thus, to prevent the accumulated burden of a disease that progresses throughout the life-course, suggests that greater benefits in adult life will arise if free sugars intake is far less than 5% of total energy intake. Given the greatest burden of caries occurs in adult life and the majority of treatment costs also occur in adults, we now need to see the general understanding of this problem extend to both academic and practicing dentists.

Most policies, research programmes and surveys on dental caries have focused on children. However, in a study in 26 countries with comparable summary data on dental caries for different World Health Organization (WHO) index ages, **v**ery much higher levels of caries occurred in adults in all 26 countries. For most countries, irrespective of the DMFT levels in 12-year-olds, the percentage difference in levels of DMFT between 12-year-olds and 35-44year-olds was above 500% and the relative difference was 5 or more[17]. Caries levels were also very much higher in

adults than in children in all countries with high percentages of their population consuming fluoridated water[17]. The numbers of fillings needed to treat the extra caries between the age of 12 and 35-44 years is as follows: Finland 20 fillings per person, Germany 13.8, Denmark 15.8, UK 10.4, Czech Republic 14.9. Figure 3 shows the age relationships of dental disease in children and adults of different ages where it is evident that there is a very marked increase in dental caries after the teenage years. Even in countries with fluoridated water supplies, the burden of dental decay progresses throughout adult life[12,15] .

Since fluoride and toothbrushing only modify the magnitude of the relationship between free sugars and dental caries, one needs to take account of the prevailing fluoride content of drinking water and the availability and practice of toothbrushing with fluoride toothpastes in different countries to allow the setting of very clear limits to appropriate free sugar intakes.

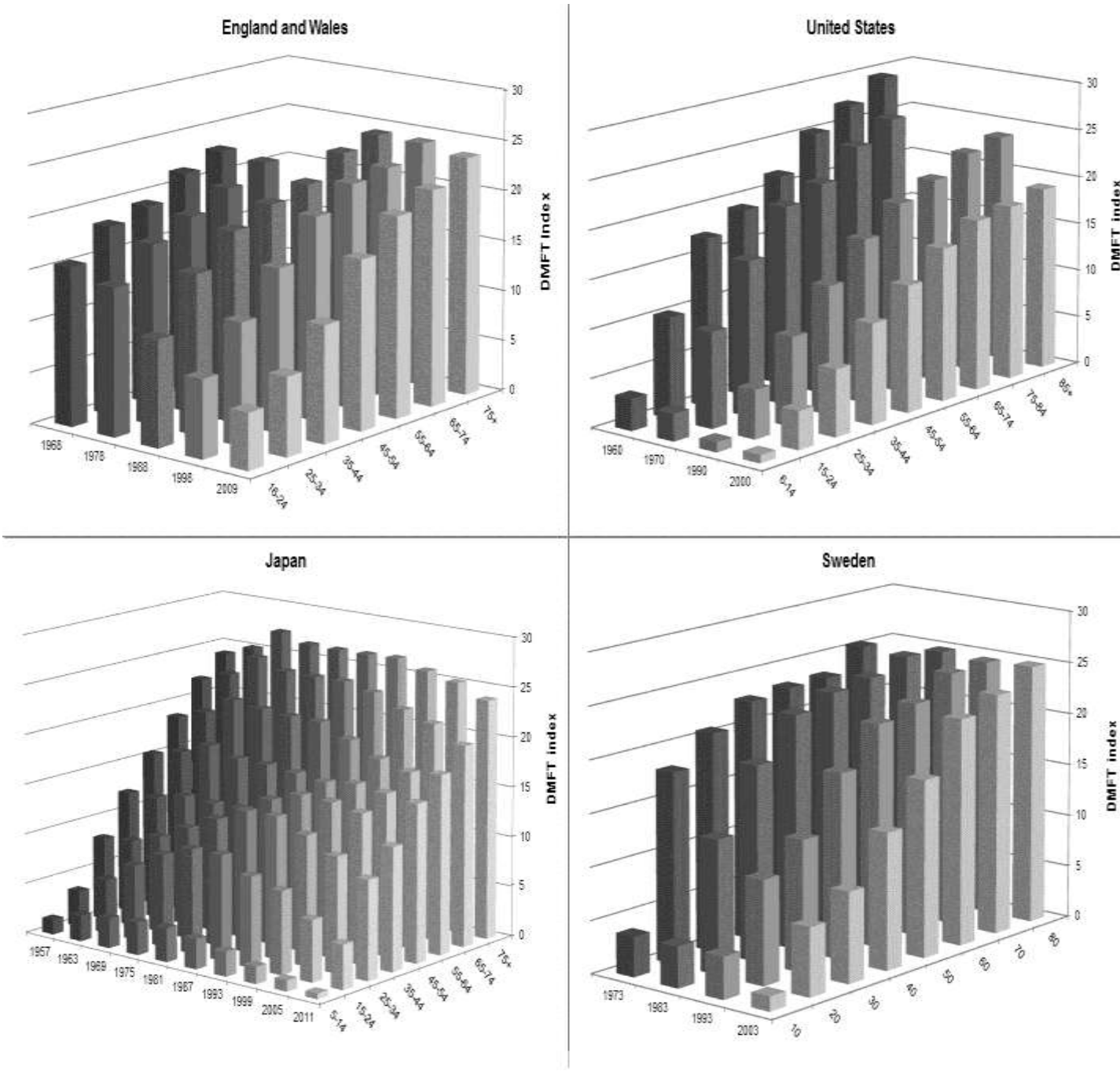

Figure 3. Increase in caries in four countries showing that the largest burden of caries (Decayed, Missing and Filled Teeth = DMFT), is in adults and that caries rates increase as people get older (See Bernabé and Sheiham 2014a[9],and attached).

Figure 3 shows the prevalence by age of the damage done by dental caries by documenting the number of decayed, missing and filled teeth (DMFTs). Note that there has been a marked reduction in the DMFT index of English children aged 5-14 since about 1975. Before that in the immediate post war years when sugar intakes were quite low children were not so severely affected as in the 1970s and 1980s. This is why the focus has been on the value of fluoride toothpastes. However, consider the subsequent age related increases in caries despite the

introduction of fluoride toothpastes and the value of dental care and the easier access to NHS dental practices in the 1970s and 1980s. At this time the caries rates were rocketing throughout early adult life and by the time adults are of pensionable age, fluoride toothpaste has had only a very modest effect indeed.

One can then argue that these effects neglect the fundamental importance of fluoride in the drinking water which we note the SACN report does not advocate in conjunction with its sugar recommendations. If we consider the US data in Fig 3, however, these dental indices were observed in a country where fluoridation of drinking water has been introduced in 42 of the 50 largest cities since the 1960s. In 1960 on average all the teeth of adults were affected by the time they were very old but the potential impact of fluoride in the year 2000 is revealed by the population having on average 17-18 of their 28 teeth affected. So fluoride has helped, but the population is still subject to a huge dental caries burden induced by the high prevailing sugar intakes.

Japan is more like England in that fluoride is not added to water but fluoride toothpastes are available. In 2011 the estimated average sugar intake in Japan was about 5% which is now what SACN is advocating. Yet this still leads to adults having on average over half their teeth affected (See Figure 3). Since in the UK where there is no routine fluoridation of the drinking water for the majority of the UK population, any inhibitory effects on dental caries depend on the use of routine individual dental hygiene techniques with fluoride being available in toothpastes. This is a very limited approach on a population basis now when the availability of dental care and surveillance has decreased and where those in lower socio-economic circumstances are particularly vulnerable.

5% of energy as free sugars is a high value for individuals and is unsuitable as a population goal. The evidence just presented is strong evidence for considering 5%E from free sugars as the maximum levels for the long term exposure of individuals. The average sugar population intake should clearly be as low as possible with even a 2-3 % free sugar intake. Even at that level people will require regular dental care and fluoride toothpastes in an attempt to limit the progressive carious disease in adults. The attached papers, generated from a background document, provide the evidence that there is an absolutely clear log linear relationship between sugar intake exposure over a 5-8 years period and the incidence of dental caries. Indeed a 2-3% sugar intake (equivalent to a 5kg/year intake leads to about a 2-3 fold increase in caries rates in these young children so that if one takes the accumulation of caries over 6-7 decades then this, in a predominantly non-fluoridated water society, induces a heavy burden of disease. So a 5% value (equivalent to <10kg/yr as shown in Figure 2) in a non-fluoridated country leads to almost a 9 fold increase in caries rates and such a high percentage

sugars figure can only be justified in a fluoride water treated country where toothbrushing with fluoride is also routine. This clearly does not apply to England. Therefore, this major public health point needs to be made in the final SACN document.

A 10% sugar value for individuals is near sugar saturation levels for inducing a maximum dental caries burden. Because SACN concentrated so much on clinical trials and cohort studies, the point that 10% sugar value is near sugar saturation levels for maximum dental caries burden seems to have been overlooked. This saturation effect is clear from Figure 2 but also from numerous observational studies. For example, in Low and Middle income countries where fluoridation was not yet applied to water sources, e.g. Bangladesh, Cambodia, China, Ethiopia, Ghana, Laos, Mozambique, Nepal, Nigeria, Laos, Tanzania, Uganda, Vietnam and where annual per capita sugar intakes as low as 10Kg/year (about 5%of dietary energy) was common levels of dental caries in 12 year olds were already high; about 50% of children were affected[18]. Given the expected marked subsequent increase in dental caries to be expected in adults, a 5% free sugars intake for individuals would still lead to a major increase in the burden of dental disease.

Counterfactual analyses by experts in the analysis of disease specific global burdens.

It seems clear that the SACN review group report may not be conversant with the wealth of new analyses which specify an optimum level of a risk factor when analysing the burden of disease. WHO made a major contribution in developing this concept which illuminates the need globally to consider the global diet and not just the Western "norms". We advocate the continued use of this counterfactual approach and therefore the importance of setting optimum levels. The SACN report perhaps should have considered the original WHO 797 report[13] on sugars in more detail because in the WHO 797 report (where one of us was the author of the expert report on dental caries and the other the Chair of the Expert Technical Consultation), we set the lowest limit at 0% free sugars on the grounds that it is a totally unnecessary dietary ingredient. At that stage the counterfactual concept had not emerged but now one should be able to say confidently that the counterfactual level for free sugars intakes is 0% of energy intake. This argument would have immediately strengthened the very generous choice of the high level of 5%E sugar intake provided one has suitably fluoridated water for universal use. Certainly most lower income countries do not have dental facilities and dental caries is a recognized cause of slow growth in children in these environments. This may also be a factor in the poorer growth of disadvantaged children in England.

Beneficial effects of increased fibre intakes

We note the intriguing analyses and graphic displays in Figures 3-6 of the SACN report. The SACN proposes that fibre intakes should be increased to 30g/d on average and evidence is cited

to suggest that this is compatible with current intakes of a proportion of the population. One suspects that these individuals are on a relatively high total carbohydrate intake and some assessment should be made of this because if SACN produces its final report with a proposal for a 30 g fibre intake then this might lead to an increase in energy intake unless it was explicitly made clear the extent to which sugar, refined starches and fat intakes need to be reduced to allow this fibre intake to be achieved without weight gain..

We attach 4 papers which are either published or in press.

1. Bernabé E; Sheiham A. 2014a. Age, Period and Cohort Trends in Caries of Permanent Teeth in Four Developed Countries. Amer J Pub Health 2014;104(7):115-121. Shows the increase in caries with increasing age based on national surveys in four countries.
2. Bernabé E, Sheiham A. 2014b. Extent of Differences in Dental Caries in Permanent Teeth Between Childhood and Adulthood in 26 Countries. International Dental Journal 2014 26th May doi: 10.1111/idj.12113. Shows the percentage and actual increases in caries between the ages of 12 and 35-44 years.
3. Sheiham A, James WPT. 2014a. A reappraisal of the quantitative relationship between sugar intake and dental caries; the need for new criteria for developing goals for sugar intake. BMC Public Heath 2014,14:863 http://www.biomedcentral.com/1471-2458/14/863 (Accepted. To be published on 16th September). This outlines the basis for the dose response relationship between caries in children and sugars being linear.
4. Sheiham A, James WPT. 2014b. A new understanding of the relationship between sugar, dental caries and fluoride use: implications for limits on sugars consumption. Public Health Nutrition 2014 Jun 3:1-9.

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