

Consultation on the draft report:

Lower carbohydrate diets for adults with type 2 diabetes

Comments Form

| | |
|--|--|
| Organisation | British Association for Nutrition and Lifestyle Medicine |
| Name of commentator and contact details | Dr Susan McGinty |

- Please do not PDF the form.
- Please do not amend the formatting of this form.
- Please do not embed attachments into this form.
- Please list any references in full that you wish the committee to consider.
- Please email this form to: sacndiabetes@phe.gov.uk
- Closing date: 9:30am 30 April 2020

| General comments | Comments |
|---|---|
| | Please insert each new comment in a new row |
| Report and General Terms of Reference and Omissions | The WG terms of reference do not align with the general SACN terms of reference. Omissions relate to vulnerable groups and risks/benefits of nutrients/food components for adults with T2D and background to T2D. |
| Inconsistencies in markers, outcomes and goals | <p>WG Terms of reference are to consider “.. markers and clinical outcomes of T2D including any potential adverse effects” but there is no narrative as to the justification for the omission of diabetic retinopathy, diabetic nephropathy and diabetic neuropathy which are prominent clinical outcomes of T2D as set out in the Background.</p> <p>Diagnosis of T2D is on basis of glycaemic markers. Body weight, which is neither a symptom or marker for T2D and is described in 3.5 as ‘associated with risk of developing T2D’, is set as a primary outcome. 3.10 states ultimate aim of treatment is to reduce and maintain HbA1C concentration at a value below the cut-off for the definition of T2D. A successful dietary intervention is described (4.13) as reduction in T2D symptoms (glycaemia, blood pressure and blood lipids) and reduced need for diabetes medication. Background omits exposition as fat infiltration of pancreas and liver as causal to beta-cell insufficiency. Body weight as a primary outcome skews the review and analysis of evidence.</p> <p>Secondary outcomes (4.2) omit blood pressure which does not align with statement at 4.13 as establishing successful dietary intervention. Secondary outcomes also omit liver steatosis which is an important factor and is in line with PHE(2019) E06b Public Health Outcomes Indicator (under 75 premature mortality from liver disease considered preventable).</p> |
| Clinical Practice Evidence | <p>The introduction (1.2) explains SACN does not normally make recommendations on clinical conditions. The SACN Framework (2020) is designed for population health. Adjustments should be made so that evidence from clinical practice studies is included. Clinicians must meet a patient-centred standard of care which is logical and reasonable, ref <i>House of Lords 1997 Bolitho v City and Hackney Health Authority</i>.</p> <p>Evidence cited at 5.9 should therefore be considered.</p> |

| | |
|---------|---|
| General | The pathologies of T2D, treatment goals and markers and clinical outcomes should be aligned in paragraphs 3 and 4, and reflect the terms of reference in 1.4. |
|---------|---|

Please add extra rows as needed

| Comments by paragraph | Comments |
|-------------------------|--|
| | Please insert each new comment in a new row |
| 2.9 | <p>Processed starches (calorific maltodextrins) should be flagged as having high GI ranking.</p> <p>Ref: Hofman DL, van Buul VJ, Brouns FJ. Nutrition, Health, and Regulatory Aspects of Digestible Maltodextrins. <i>Crit Rev Food Sci Nutr</i>. 2016;56(12):2091–2100. doi:10.1080/10408398.2014.940415</p> |
| 2.11 and 2.14 | Fructose is insufficiently characterised particularly in relation to liver steatosis. See point on para 3.14 below. |
| 3.5 | <p>First sentence should be more precise and read “T2D accounts.... and occurs following <u>beta-cell dedifferentiation</u> which results in reduced insulin secretion and increased insulin resistance”. In addition to ADA 2019a, Cinti 2016 should be cited.</p> <p>Ref: Cinti F, Bouchi R, Kim-Muller JY, et al. Evidence of β-Cell Dedifferentiation in Human Type 2 Diabetes. <i>J Clin Endocrinol Metab</i>. 2016;101(3):1044–1054. doi:10.1210/jc.2015-2860</p> |
| 3.6 | Winkley et al, 2013 also stated “..in multi-ethnic inner-city populations, onset of type 2 diabetes occurred almost 10 years earlier in non-white populations than in white participants, predicating a prolonged morbidity.” This should be noted in the report. |
| 3.11 (plus 3.8 and 4.4) | Paragraph 3.11 should be deleted as it is not consistent with the position taken in 4.4 that blood pressure would not be included as a secondary outcome. Nor is it consistent with statements in 3.8 which also includes renal and eye diseases. PHE (2019) Public Health Outcomes Framework Indicator refers to preventable sight loss -diabetic eye disease (E12c). If 3.11 is maintained it should include treatment |

| | |
|------|--|
| | goals of improved renal function and liver steatosis (E06b PHE Public Health Indicator), which should also then be secondary outcomes in chapter 4. |
| 3.14 | <p>In underweight and normal weight T2D a reduction in energy intake cannot be recommended. This paragraph fails to spotlight specific nutrients/food components which increase risk of liver steatosis. The report fails to consider unique aspects of fructose metabolism in stimulating lipogenesis and inducing insulin resistance, and how it alters the metabolism of glucose by driving more glucose through oxidation pathways.</p> <p>Refs: Softic S, Cohen DE, Kahn CR. Role of Dietary Fructose and Hepatic De Novo Lipogenesis in Fatty Liver Disease. <i>Dig Dis Sci</i>. 2016;61(5):1282–1293. doi:10.1007/s10620-016-4054-0</p> <p>Varma V, Boros LG, Nolen GT, et al. Fructose Alters Intermediary Metabolism of Glucose in Human Adipocytes and Diverts Glucose to Serine Oxidation in the One-Carbon Cycle Energy Producing Pathway. <i>Metabolites</i>. 2015;5(2):364–385. Published 2015 Jun 16. doi:10.3390/metabo5020364</p> |
| 4.1 | Body weight cannot be supported as a primary outcome – and even as a secondary outcome as it is not a clinical outcome of T2D. Body weight (or % weight gain/loss) is not a marker for T2D and not consistent with the aim set out in 3.10. While weight loss may drive improvements in T2D for those who are obese, it is not reconcilable for underweight (Asian Indians) or normal weight T2D patients. Nor is it compatible with the state of metabolically healthy obesity (normal glucose and lipid parameters and absence of hypertension). |
| 4.13 | Markers of a successful intervention need to be consistent – so blood pressure should be excluded if not included as a secondary outcome. |
| 5.9 | Evidence from clinical practice studies should be included if the report is expected to be a guide for clinicians who must meet a patient-centred standard of care. |
| 7.3 | This paragraph should include an explicit statement that practice-based evidence did not meet the inclusion criteria. |
| 7.49 | Is there any evidence for this statement? In this context vegetables includes both low and high starch so the statement is imprecise. |
| 7.64 | If no trials included in the review provided information about the type of carbohydrate consumed, how can any conclusions be made by this WG at all? |

Please add extra rows as needed