Sugar consumption in New Zealand—with Thornley and McRobbie response

We write in response to Thornley et al’s viewpoint article *The New Zealand sugar (fructose) fountain: time to turn the tide?* published in *The New Zealand Medical Journal* on 19 March 2010. The data quoted on sugar consumption in New Zealand are presented misleadingly and are not correctly referenced to primary sources.

The opening two sentences state: “In 2005 New Zealanders drank and ate, on average, over half a cup (158g) of sucrose (sugar) per day. In contrast, less than 40g a day (about 1½ tablespoons) are recommended by the World Health Organization (WHO) to prevent dental caries, obesity and chronic disease.”

Firstly what is quoted is *total* sugar intake, not *sucrose* intake, as 158g per day. Total sugar intake is not equivalent to sucrose intake, which is only one type of dietary sugar, along with lactose, glucose, fructose and maltose.

Secondly, the WHO recommendation is for *added* sugars, not *total* sugars (which includes all sugars naturally present in foods). The generally accepted definition of *added* sugars is: “Sugars and syrups that are added to foods during processing or preparation. Added sugars do not include naturally occurring sugars such as those that occur in milk and fruits.”

While it’s not the same thing, data on *sucrose* intake are often used as a proxy for *added sugars* intake, since the majority of added sugars come in the form of sucrose in New Zealand. Comparing our *total* sugar (not sucrose) intakes with the WHO recommendation for *added sugar* (thereby inferring a nearly four fold difference), and using the terms sucrose and sugar interchangeably is both inaccurate and confusing. One would expect these terms to describe markedly different amounts, since the former encompasses sugar intake from all sources including fruit, vegetables, milk and honey, as well as added sugars.

In addition, the viewpoint article by Simon Thornley references a key New Zealand paper in this field, that by Parnell et al (Public Health Nutrition, 2007). The findings on beverages as source of sugar for New Zealand children are stated, but the author fails to acknowledge some of the key findings of this research. The paper titled *Exploring the relationship between sugars and obesity,* sought to discover the relationship (if any) between sugar intake and obesity, by analysing data from the 1997 National Nutrition Survey for Adults and the 2002 Children’s Nutrition Survey. The researchers found no relationship between current sucrose intake from beverages (the predominant source of added sugar in children’s diets) and obesity. Total current sugar intake (but not sucrose) was in fact significantly lower among obese children compared to children of a normal weight.

Adults and children with the lowest current intakes of sugar were actually significantly more likely to be overweight or obese. Subsequently, there was no relationship found between current sugar (sucrose) intake and body weight status in the New Zealand population. It is acknowledged that as a cross-sectional study we are
unable to equate these findings to sugar and sucrose intakes over time, however in the
absence of longitudinal data for New Zealand this study is considered to provide
credible and valuable information regarding current intake of sugar and body weight
in both adults and children.

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Thornley and McRobbie respond

Parnell and colleagues draw attention to two items raised in our viewpoint article,¹
questioning our estimates of sugar consumption in New Zealand, and our fidelity in
weighing the evidence linking sugar consumption with obesity.

The first point relates to our estimate of New Zealander’s sugar consumption of
158g/day. This is derived from UN food balance sheet data which assesses the
national “disappearance” of food, calculated from production, minus exports, plus
imports. What is left is then considered to have been consumed, although this estimate
does not account for wastage. Our figure was based on the disappearance of the item
“sugar and sweetener's (Total)”, which does not—as Parnell and colleagues claim—
estimate total sugar intake, both added and intrinsic. This figure estimates the
disappearance of added sugar and sweeteners in the food supply.

We concede that our report was slightly high, and that we should have restricted our
estimate to sugar only, because other sweeteners may be glucose, fructose or a range
of other mono or di saccharides. So our revised estimate, based on this source, is 50.5
kg/capita/year, or 138 g/day (32 teaspoons per day per person). Notwithstanding the
lower figure, this still indicates that as a nation, we consume added sugar at a rate
well above WHO recommendations and higher than indicated from survey data, based
on self report, as in Parnell’s study.

Parnell’s assessment of New Zealander’s sugar intakes are lower than food balance
sheets.² However, what is not commonly reported in nutrition studies is that human
memory of both the quantity and nature of food eaten is fallible, with recall estimates
reporting about 20% less sucrose intake compared to more objective methods.³ This
inaccuracy occurs non-randomly—obese people are more likely to under report
consumption than normal weight people.⁴ Non-response also further underestimates
food intake. Both sources, however, indicate that the majority of New Zealanders eat
quantities of added sugar far in excess of the WHO guideline of 40g/day (10
teaspoons per day).

We are then accused of leaving out crucial analytical results of Parnell’s study, which
suggested that obese children report consuming less sugar than counterparts of
normal weight. The study was not included for specific reasons. First, it employed a
cross-sectional design. Second, it was funded by the sugar industry, and third, it
consisted of a secondary analysis of the data collected for other purposes. All characteristics have been identified in a meta-analysis which reports a positive association between soft drink consumption and obesity, as being less likely to report such a link.5 The weak study design, therefore, lead us not to include Parnell’s analytical conclusions. Our article, instead, summarises the evidence of the adverse effects of sugar from either randomised controlled studies, or meta-analyses (of observational studies). Such designs are considered stronger than cross-sectional studies for assessing causation. The consistency of adverse effects, that we observe, linking added sugar, fructose and sugar-sweetened soft drink intake with obesity, dental decay, hypertension, insulin resistance and raised serum triglycerides remains.

We concur with Parnell that we have modestly overestimated national sugar consumption, but disagree that we have misrepresented research which links sugar intake with adverse health outcomes. In 2009, the American Heart Association reversed its earlier position,6 publishing guidelines that advise severe restriction of added sugar intake.

We consider that the potential consequences of this ubiquitous exposure are too important to narrow our gaze to one cross-sectional study, sponsored by an industry with a lot to lose.

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References
