**ICN2 Draft Framework for Action, Zero Draft**

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**Chapters 1-2:**

These chapters are commendable summaries of the vision, actions and resources necessary to achieve the goals outlined. In particular, the chapters emphasize the need to solve the unacceptably lingering nutrition problems of the 20th Century. All of the public health measures necessary to do so have been known for at least 100 years and all of the recognized essential nutrients have been known for more than 50 years. Likewise, ancillary aids such as antibiotics necessary to treat the common infectious agents associated with the malnourished state have also been available for nearly as long. In practice, the proof-of-principle experiments were completed long ago. Under-nutrition is largely non-existent in Nations that have been able to implement the public health foundations required for clean water and food and that have been able to provide diverse foods in amounts that allow adequate intakes of the essential nutrients in healthy diets. As such, then, the 20th Century’s lingering nutrition problems are not the result of a lingering deficit in nutrition science. They are the consequence of the lingering investment, financing, policy facilitation, and implementation deficits. Chapters 1 and 2 highlight the action elements necessary to turn these issues around. All are critically important, but explicit commitment to the knowledge and evidence-base is the foundation for effective conduct of all of the other recommendations.

Because the fundamental biological causes of under-nutrition are largely understood, final solution to the nutrition problems of the 20Th Century is achievable on a global scale. On the other hand, in Chapter 1, the draft aligns its commitment with the 66th WHA goal of reducing NCDs by 25% by 2025. While certainly commendable, this goal is potentially less tractable. First, on the whole, neither the knowledge nor evidence-base for NCDs are as complete or as convincingly clear and unambiguous as is the information on essential nutrient deficiencies. The pathophysiological bases of NCDs, the nutrition problems of the 21st Century, are not nearly as well understood as the biological causes of under-nutrition. Secondly, proof-of-principle experiments in developed countries have not been nearly as demonstrably effective for prevention of many NCDs, cancers for instance. Third, in NCDs where success has been achieved, it is plausible that these accomplishments were achievable because the overall environmental, societal, public health and economic problems that remain impediments to successful elimination of under-nutrition were corrected first. Fourth, in many NCDs medical/pharmaceutical preventive and therapeutic advances have been responsible for a significant fraction of the successes. The past and future scientific, technological and industrial contributions to medicine get little mention in the draft. Fifth, in large part, behavioral change underlies correction of most modifiable NCD risk factors and science and society have been remarkably poor at changing behaviors in almost any sphere of life. Finally, the large, 25% reduction in NCDs must be accompanied by a corresponding reduction in all-cause mortality. If not little net human benefit results. Thus, for instance, if reduction in cardiovascular deaths is accompanied by an increase in deaths from cancer and other causes (as has been the case in some cardiovascular disease trials), how does one measure success or recommend pubic health policies?

**Chapter 3 (Section 3.1):**

Chapter three provides insightful guidance by recognizing the critical interplay of food systems, supply chains, economic incentives, income growth, and food-system based policies as necessary elements in any overall action plan. Most importantly, the chapter explicitly recognizes that healthy diets and diverse diets are necessary for any nutritional success, either in eliminating under-nutrition or in reducing the risk of NCDs. The absolutely critical word here is “diets” and in whole diets, not individual foods. Despite regular and repeated forays into dietary and/or nutrient fads that have promised long-term beneficial returns, the field of nutrition has demonstrated over and over again that overall health maintenance is a function of an individuals whole diet pattern, not of any specific, individual food or class of foods.

More than fifty years ago, expert nutrition advice was that there are no good foods or bad foods, only good or bad diets. Nonetheless, in the intervening decades, various “bad food” hypotheses were tested repeatedly. Over time, the integrated results of these studies have provided proof that the overall diet pattern is what is critical to maintaining optimal nutritional health, not the presence or absence of specific nutrients in any individual food. Thus, for instance, egg were once vilified as a “bad food” by the American Heart Association because consumption of the cholesterol in eggs would lead to increased serum cholesterol and, consequently, to increased cardiovascular risk. Rather quietly when compared to the highly vocal AHA campaigns to reduce cholesterol intake, in its 2013 Guideline on Lifestyle Management to Reduce Cardiovascular Risk, the AHA now says in a single short sentence that “There is insufficient evidence to determine whether lowering dietary cholesterol reduces Low Density Lipoprotein Cholesterol” (1).

Likewise, in the current framework draft (bullet list, Section 3.1), simplified recommendations for reduction in salt, saturated fat and sugar intakes no longer adequately or transparently reflect the complexity of the current state of nutrition science in these areas.

Specifically:

* Intake of saturated fat is less than 10% of total energy intake: This item fails to address the accumulated evidence that the macronutrient replacements for the saturated fats removed from the diet are critical in regard to the overall health consequences, that there are profound differences in the health effects of individual saturated fatty acids, and that the consequent health risks are dependent on the individual fatty acids, not saturated fats as a class, and that effects of reducing saturated fats, per se, on heart disease risk may not be as profound as once suspected, once the effect of the presence of trans-fats in earlier studies is removed (1-24). Recently, published data led Dr. Frank Hu at Harvard to remark that, “The single macronutrient approach is outdated… I think future dietary guidelines will put more and more emphasis on real food rather than giving an absolute upper limit or cutoff point for certain macronutrients.” (25)
* Intake of free sugars is less than 10% of total energy or, preferably, less than 5%: This recommendation is just not supportable from current evidence. There are no direct human experimental data to support a 5% intake level. As far as I can determine, the 10% level is an arbitrary one based on the subjective opinion of a WHO Study Group that met in Geneva in 1989 when, without any systematic evidence-based assessment, the “Group judges that the upper limit of the population nutrient goal for free sugars should be about 10% of energy” (WHO Technical Report Series 797, page 113). Earlier this year, in response to the WHO draft sugars guideline, I submitted formal comments on the lack of evidence basis for this guideline. To my knowledge, this document remains a draft with recommendations that are not yet formally approved. Rather than duplicate in detail here the evidence I sent to the WHO in March, I have attached my earlier comments as an Appendix at the end of the current comments and supporting citations. Since my earlier comments to the WHO on this issue, an additional related meta-analysis has been published (26). This analysis demonstrated statistically significant increments in circulating triglycerides, LDL-Cholesterol and blood pressure as a function of dietary sugars intake. However, the changes in surrogate variables were quite small and their clinical significance is surely arguable without further data, especially hard clinical endpoints (26).
* Intake of Salt is less than 5 g per day: New data question the advisability of severe restrictions in dietary sodium intake based on risk/benefit ratio of salt restrictions beyond modest decrements in intake (27-33). These data continue to show that individuals who consume very high quantities of sodium as salt will have significant, beneficial effects on blood pressure and support the findings of the DASH diet study of hypertensive individuals. However, they question extrapolation of DASH data to populations as a whole since very low sodium intakes are not only associated with little additional benefit but the adverse risk profile increases. In an editorial accompanying the most recent reports in the New England Journal of Medicine, Dr. Susan Oparil discusses the new data and concludes that the articles “highlight the need to collect high-quality evidence on both the risks and benefits of low-sodium diets.” (33) Thus, the current Framework for Action draft needs to reconsider the absolute value chosen for its recommended salt intake.

**Chapter 3 (Section 3.1.1):**

Food Environments: Additionally, as a consequence of the necessary modifications of the Section 3.1 bullet items discussed above, there will be a corresponding need to reword the related bullet items in Section 3.1.1

**Summary:**

The current ICN2 Zero Draft Framework for Action represents a thoughtful document overall and one that provides comprehensive and inclusive recommendations on the whole. However, the draft overstates the level of the today’s evidence when it makes simplified recommendations about individual foods. Several of these specific restrictions are no longer supported convincingly by hard scientific data. Moreover, this negative approach fails to emphasize more positive approaches that focus on healthy dietary patterns as a whole. Not only are these more scientifically sound based on current evidence, healthy whole diet patterns will permit each of the 193 Nations in the U.N. to more readily adapt their individual dietary guidelines to local foods available within these Nations and to food patterns and consumption habits that continue to support the unique social and cultural contexts of the citizens of these countries.

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**APPENDIX**

Comments to Members of the World Health Organization (WHO) Steering Committee for Nutrition Guideline Development and Nutrition Guidance Expert

Advisory Group (NUGAG) Subgroup on Diet and Health.

31 March 2014

I appreciate the opportunity to provide comments to the WHO on its Draft Guideline: Sugars Intake for Adults and Children. Before I do, however, I want to make it clear to the WHO committee that I believe firmly that 1) optimal health is maintained by appropriate meal planning and an active lifestyle, including regular physical activity, 2) that the concept of healthy eating mandates moderating energy consumption to maintain a lean body habitus without becoming overweight or obese, 3) that healthy eating likewise mandates that dietary macronutrients (fat, protein or carbohydrate, including free sugars) be consumed in amounts that do not displace consumption of other beneficial nutrients that may lead to essential nutrient inadequacy/deficiency, and 4) that dental health requires proper attention to oral hygiene, including daily brushing of teeth, fluoridation of water, and regular professional dental care.

I believe as well that the WHO should be commended for its activities in addressing the fundamental environmental contributions to maintenance of healthy body weight and prevention of dental caries. However, I disagree that the present level of scientific evidence supports the WHO’s “strong recommendation” that “intake of free sugars should not exceed 10% of total energy”. In addition, I find that there are insufficient data to support the recommendation for “further reduction to below 5%”. The basis for my opinions is predicated on existing evidence and summarized below.

**Summary of Evidence: Issues of Definition**

The WHO Draft Guidelines rightly comment that “free sugars contribute to the overall energy density of diets.” Further, it is imperative that consumption of free sugars not reduce the caloric intake of foods containing essential dietary nutrients. However, sugars are hydrated in foods and beverages, and the energy density of sugars is relatively low, only approximately 1 kcal/g in foods and considerably less in beverages. Although recognition that overconsumption of sugar sweetened beverages may be an appropriate target in public health efforts to prevent and treat obesity, the proposed WHO focus on sugars ignores the fact that fat ingestion contributes far more to energy intake than do dietary sugars. Moreover, small amounts of fats (for example, 1.5 tablespoons of cooking oil) that would likely be underestimated or unreported, especially when data is retrieved from dietary intake surveys and 24-hour dietary recall, contribute more calories to energy intake than 12 ounces of sugar sweetened beverage.

Furthermore, I question the biological basis of the WHO classification of “total sugars”, which includes fruit juices but excludes sugars present in whole fruit. The latter are excluded because they are “incorporated within the structure of the intact fruit”. However, the sugars in fruits that are not starch polysaccharides are present as free sugars within the pulp vesicles of the fruit. This can be demonstrated readily by squeezing an orange to produce orange juice. This process requires no more than release of the free fruit sugars by mechanical rupturing of the pulp vesicles. It is difficult to understand how one form of these sugars is, in fact, different from the other when it comes to dietary recommendations or to biological consequences of eating the sugars. The latter conceptual difficulty is particularly problematic when enterocytes that absorb the hydrolyzed sugars in the gut have no way to distinguish sugars that came from intact fruit or from fruit juice.

The WHO Draft Guidelines conclude that “because there is no evidence of adverse effects of consumption of intrinsic sugars, recommendations focus on the effect of consumption of free sugars.” I know of no human studies that have tested adverse effects of intrinsic sugars, per se, on human health consequences. There have been many observational studies on fruit and vegetable consumption and various randomized controlled trials (RCTs) that have included fruits and vegetables in their experimental design intervention. However, in each instance it is virtually impossible to dissociate closely-coupled lifestyle and dietary covariates in interpreting outcomes. Thus, in my opinion, whether intrinsic sugars have effects (detrimental or otherwise) that are different from other “free sugars” remains an open question.

**Summary of Evidence: Body Weight and Other Outcomes**

The WHO position on dietary sugars and body weight relies heavily on the systematic review and meta-analyses published by Te Morenga, Mallard and Mann in the British Medical Journal (BMJ. 2012 Jan 15;346:e7492. doi: 10.1136/bmj.e7492). While this article is an excellent summary of the present literature, and while Professor Mann is a member of the WHO NUGAG Subgroup on Diet and Health, I respectfully submit that the WHO interpretation of this review extends beyond the limits of the data in the publication itself. In my opinion, appropriate recognition of the limitations of the data analyzed by Te Morenga et. al. creates uncertainty in the study outcomes, and precludes awarding a “strong recommendation” to a WHO position that limits intake of free sugars to less than 10% of total energy intake.

When added dietary sugars were exchanged isocalorically with other macronutrients, Te Morenga et. al. found no effect on adult body weight in eleven of the twelve RCTs reviewed. In an analysis of five RCTs studying the effect of reduced dietary sugars on “measures of body fatness’ in adults, Te Morenga et. al. reported a statistically significant but quite small effect. However, the authors then state that “three of the five reported data for completers only. However, only two of these studies considered this to be a potential source of bias.” Nonetheless, these two studies represent 40% of subjects in the five RCTs. Moreover, the authors did not consider a completers only analysis as a source of bias, although other independent assessors would potentially disagree citing a lack of intention-to-treat analysis as a critical interpretive flaw. Additionally, Te Morenga et. al. found that exclusion of “three studies that had a high risk of bias for two or more validity criteria, the effect estimate (i.e. on body fatness) was no longer significant although the difference in weight was similar…” In other words, the authors could find no convincing evidence from RCTs that there was an effect of limiting dietary sugars on “measures of body fatness when their analysis was limited to studies that did not have a high risk of bias.

In a systematic review of adult cohort studies, Te Morenga et. al. found one or more statistically positive associations among intake of sugars and a wide assortment of measures of body fatness. However, only six of the 23 associations were statistically significant and these came primarily from one or two studies. The authors were unable to construct a forest plot in which all cohort studies were represented because outcome measures differed among the studies. Of the individual forest plots that could be constructed, less than half showed summary statistical significance among various adiposity measures and sugars consumption.

In children, the data were even less convincing. Te Morenga et. al. found no published isocaloric substitution RCTs. The authors found five RCTs that tested whether reduction of dietary sugars would result in lower body weight. The overall effect estimate for the five studies was not significant. Two more recently reported childhood RCTs that were not included in the Te Morenga analysis involved more subjects and longer follow-up ( Ebbeling et. al., *N Engl J Med* 2012; 367:1407-16 and de Ruyter et. al., *N Engl J Med* 2012; 367:1397-406). However, their results do not materially change the conclusion that reduction of energy intake from sugars does not improve body weight. In the Ebbeling et. al. study the registered primary endpoint of weight loss at 2 years was not significant. In de Ruyter et. al., the drop-out rate was 22-29%, a significant portion of children consuming the artificially sweetened beverage (completers and drop-outs) were aware of the kind of beverage they were consuming, the BMI in the sugar-free intervention group did not go down but only increased less than the control group, the point estimate difference between the control and intervention groups was small (95% CI very close to zero). Importantly, when the completers and drop-outs were combined “the BMI Z-Score increased by 0.06 SD units in the sugar-free group and by 0.12 SD units in the sugar group (P=0.06)”. Overall, then, this study does not provide robust evidence for an effect of reduced sugars intake from sugar-sweetened beverages on body weight.

Te Morenga et. al. identified 21 pediatric cohort studies assessing the relationships of increased sugars intake to measures of adiposity. Of the various associations found, six were significant. However, only three of the summary forest plots showed a statistically significant effect. In fact, four studies reported a negative association.

Malik et. al. (*Am J Clin Nutr* 2013:98: 1084-102) and Kaiser et. al. (*Obesity Reviews* 2013;14:620-33) each reported similar meta-analyses assessing the relationships of sugars intake to BMI or weight. Overall, these two studies analyzed largely the same primary research reports cited by Te Morenga et. al. However, each study had a somewhat different approach to inclusion criteria and the grouping of subjects. While both Malik et. al. and Kaiser et. al. each reported some positive associations, they are plagued by the same issues of small point effect estimates, heterogeneity, publication bias, lack of adjustment for total energy intake or loss of significance when adjustments for energy intake were made. Overall, they too failed to make a convincing case that sugars intake, per se, is associated with body weight.

Te Morenga et. al. concluded that their systematic review and meta-analyses indicated that “the change in body fatness that occurs with modifying intakes seems to be mediated via changes in energy intakes, since isoenergetic exchange of sugars with other carbohydrates was not associated with weight change.” In my opinion, this conclusion is supported by essentially all clinical studies published over the last several decades. All macronutrients, including but not limited to sugars, are problematic when over consumed as a dietary energy source, leading to obesity. Sugars, per se, are not in any way special in this context.

A recent study by Yang et. al. (*JAMA Intern Med*. 2014 Feb 3. doi: 10.1001/jamainternmed.2013.13563) reported on the effects of added sugars over a median 14.6 year follow-up of the National Health and Nutrition Examination Survey (NHANES) dietary intake data (NHANES 1988-1994, 1999-2004 and 2005-2010). These authors reported that the risk of cardiovascular disease (CVD) mortality increased once added sugars intake exceeded 15% of total daily calories, and further exponentially increased with additional dietary sugars consumption. This overall risk of CVD mortality was significantly associated with sugar sweetened beverage consumption. However, the reported risk of CVD mortality by Yang et. al. was independent of BMI, was not associated with CVD risk up to a sugars intake of 9.3%, was not associated with CVD mortality across the entire range of sugars intake if individuals maintained a BMI <25 kg/m2, and was unrelated to all-cause mortality across the entire level of added dietary sugars, including the highest level of 25% of total calorie intake. Yang et. al. calculated that more than 100 individuals consuming 13.1 to <16.7% of energy intake as added sugars would have to reduce sugar consumption to <9.6% to prevent a single CV death, without a corresponding change in all-cause-mortality. Likewise, more than 250 people consuming 9.6 to <13.1% of energy as added sugars would have to reduce sugar consumption to <9.6% to prevent one cardiovascular death, without a corresponding change in all-cause-mortality. Limitations cited by Yang et. al. in their study include 1) added sugars intake was assessed by first day 24-hour dietary recall at baseline and not updated during the follow-up period, and 2) their finding of CVD mortality with increased sugars intake >15% of calories did not apply to the non-Hispanic black ethnic group, despite the prevalence of heavy sugars consumption (>25%) being reported elsewhere as disproportionately high among blacks.

In my opinion, the totality of these data raise the question of the advisability for a recommendation to reduce free sugars dietary intake to less than 10% of total calories. More human studies with hard clinical outcomes are necessary before the scientific community can confidently claim any net gain from efforts aimed at reducing CVD risks by limiting dietary free sugars with corresponding effects, or lack thereof, on all-cause-mortality. In the interim, the subject at least remains arguable. In summary, I believe that the above critique of published data argues against a “strong recommendation” for the 10% limit on dietary sugars.

**Summary of Evidence: Dental Caries**

My understanding is that dental caries result from the interplay of multiple factors including genetics, nutrition, poor oral hygiene, fluoridation of the water supply, tobacco smoking, and a variety of other variables including inadequate professional dental care. Nonetheless, since sugars intake in the U.S. has increased over the last several decades while the prevalence of dental caries assessed in the NHANES database has declined, one might potentially surmise that the summation of the factors other than dietary sugars intake, per se, play the predominant role.

Although I am not qualified to discuss the basic science of dental caries, I do possess considerable expertise in evaluating the level of scientific evidence necessary to support clinical recommendations. Thus, I would like to comment on the level of evidence proposed for the conditional recommendation to reduce free sugars intake to 5% of energy intake for prevention of dental caries. In this case, too, I find inadequate evidence to support such a recommendation even at the level of a conditional recommendation.

The proposed WHO recommendation relies largely on the systematic review of Moynihan and Kelly, a somewhat puzzling decision since the authors of this review themselves judged the evidence for the <5% energy position “to be of very low quality”. This assessment was echoed in a commentary by the UK NHS National Institute for Health Research Centre for Reviews and Dissemination at the University of York that states, “Given that the authors’ conclusion seems to be based on a small number of cohort studies, the authors’ conclusion may be overstated. The supporting evidence was derived from studies with potentially high risk of bias.” (<http://www.crd.york.ac.uk/crdweb/ShowRecord.asp?LinkFrom=OAI&ID=12013071674#.UzCava1dVg4>).

The full text of the three Japanese papers relied on for the WHO Draft Guidelines recommendation appears to be available only in Japanese. Since only English abstracts are available for two of these articles our own assessment of the evidence is limited, but I believe sufficient to cast serious doubt. Koike and Hiromu evaluated caries in the first molar of more than 10,000 children born between the years of 1924 and 1946 and admitted to school between the years of 1931 and 1953. In the English summary of their work, Koike and Hiromu state that “Accumulated sugar consumption during the maturation period of teeth has only slight influence upon caries incidence.” In similar fashion, Takahashi studied more than 7,800 children born between 1929 and 1951. The English Summary provided at the end of the Takahashi report reads “The interrelative ratio between the amount of total sugar consumption of a child during the maturation period of the tooth (0-5 years old) and the rate of new caries incidence in the first molar during 6-11 years of age, indicates that there is an extremely little co-relation between them.” However, as reported by Moyihan and Kelly, both studies found relationships between “dental cares increment and sugar intakes” and showed relationships of new caries to sugar consumption in the same year or the year before caries assessment.

Nevertheless, the known confounding societal, psychosocial, emotional, economical, oral hygiene, dental care, dietary habits, health care and other biologically impactful changes in Japan during the period of the 1930’s to the 1950s (especially during the war years) were so unimaginably immense as to make any conclusion related to added sugars *per se* simply unjustifiable on a scientific level. Furthermore, the Japanese studies do not appear to have included any information on dietary essential nutrients (i.e., calcium, phosphate, total calorie and protein intakes) or on oral microflora that contribute to dental health and that would surely be required today as covariates in any model to analyze the presumptive effects of dietary sugars intake on dental caries. In summary, I again conclude on the basis of the available published data that there is no credible evidence for reducing dietary sugars intake below 10% of total calories for the prevention of dental caries.

**Summary of Evidence: Potential Harm Regarding a 5% Limit on Free Sugars**

In several places in the text, the WHO Draft Guidelines include the remark that “no harm is associated with reducing the intake of free sugars to less than 5% of total energy, especially when considering the risk of dental caries throughout the life cycle.” While this statement may be true for dental caries and while dental caries are surely a health outcome worth reducing, the apparently single-minded focus on caries neglects the potential for adverse effects elsewhere. While direct adverse effects of restricting free sugars to less than 5% energy may be the case for some biomedical consequences, this hypothesis has not been tested formally in humans, especially at the level of randomized controlled trials.

Moreover, I am not entirely complacent about the potential for at least some adverse effects. For example, in the U.S. thirty to forty years ago, a common cause for toddler admissions to pediatric wards was a clinical condition called ketotic hypoglycemia, a form of substrate-limited hypoglycemia that was the consequence of inadequate carbohydrate supply during prolonged periods between food ingestion, especially overnight. Particularly, but not exclusively, this condition was most manifest in toddlers who were on the small, thin side and in whom risk of development of obesity was nil. Ketotic hypoglycemia has been essentially eliminated from the pediatric population by parental education about provision of adequate dietary carbohydrate, especially in the evening. I remain concerned that recommendations to reduce simple sugars to <5% energy intake may be associated with parental practices that lead to consequences like ketotic hypoglycemia, particularly when these recommendations are applied to infants and children at the lower percentiles of weight where obesity is a highly unlikely consequence of not restricting free sugars. Surely, any recommendations to limit intake of free sugars to less than 5% should consider all the potential consequences of this action, not just the potential for reducing dental caries.

Finally, I believe that there are at least two other potential adverse consequences and neither seems to be addressed by the WHO Draft Guidelines as possible harms. First, it is impossible to foresee any potential adverse “psychosocial and behavioral” effects as a consequence of restriction of one of the most important contributors to the sensory, hedonic enjoyment of foods and, by extension, to enjoyment in the spheres of life in which foods play a part such as family meals, social events, and the like. However, I also believe that there is no easy way to approach studying this question, or that any formal systematic studies have been performed that can provide the evidence to prove or refute the hypothesis. Finally, it is not clear whether the WHO Draft Guidelines has taken into account potential economic harm. Presumably, to meet the <5% energy recommendation, the food industry would have to reformulate a wide variety of products and these costs will undoubtably be passed on to the consumer. Has the WHO performed a formal economic risk/benefit analysis using various theoretical models to assess the potential for economic harm to the population at large?

**Summary Conclusion:**

Since at least the second half of the 20th Century, nutritionists have recommended various single nutrient, “Silver Bullet”, approaches to preventing or treating various health consequences to which nutrition is a known contributor. Not surprisingly, the food industry responded by developing a panoply of products to comply with the recommendations of expert committees. On the whole, none of these has satisfied hypotheses about population outcomes based on such approaches.

Professor Frank Hu, Co-Director of the Program in Obesity Epidemiology and Prevention at the Harvard School of Public Health, said “The single macronutrient approach is outdated.” “I think future dietary guidelines will put more and more emphasis on real food rather than giving an absolute upper limit or cutoff point for certain macronutrients.” (Study Doubts Saturated Fat’s Link to Heart Disease. New York Times, March 18, 2014, page A3). I am not alone is supporting the whole diet, “real food” approach to a healthy nutritional lifestyle in preference to the outdated single macronutrient limitation paradigm recommended in the WHO Draft Guideline: Sugars Intake for Adults and Children. Particularly, at the moment, however, I can find no high quality sufficient scientific evidence to support the WHO Draft Guideline that aims for a target of 5% or less. Far more human clinical research data will be required to feel confident that there is benefit of this action, what the true extent of the benefit (or lack thereof) might be, and whether there are as yet unaccounted-for harm(s) as a consequence.

In today’s world of Evidence-Based decision making, I believe that policies directed at improving public health should be based on a strong base of scientific evidence that explores the full range of potential effects resulting from the policy pronouncement.