Main Submission

A 'sweet' take on obesity

Adam Upshaw

University of Western Ontario

Of the myriad paths, upon which we have set out in pursuit of discovering the cause and ultimately, the treatment of obesity, how is it we continually seem to miss the mark? We are always concerned with low fat, non-fat, half fat, less fat etc... and have been concerned with this for decades. However as Melanson et al.[1] noted, the current literature supports only a weak association, let alone a causal relationship, between total dietary fat and saturated fat intake on weight gain. If not fat intake, then total calories consumed and levels of physical inactivity have been of primary focus for managing the current obesity trends. However a recent report in the Journal of the American Medical Association[2] explained the current national efforts at promoting healthful eating, which have historically focused on low fat diets, low total calorie intake and increased daily physical activity, are having little effect on overweight and obesity rates. Conversely, it is likely that the high intakes of sugar and refined carbohydrates (S&RC) may in fact be causing the most havoc with our body particularly when it comes to weight gain.[3] The average daily intake of sugar in North America in 2009 was 162g/day which is an increase of approximately 20% since 1990.[4] Similarly, in the last 40 years, the average daily intake of fructose, which along with glucose from table sugar, has increased by more than 40%[3] while the intake of refined carbohydrate has risen quite dramatically as well.[5] Interestingly, this trend in S&RC consumption mirrors the rise in obesity[5] while during the same period, physical activity levels have increased,[6] dietary saturated fat consumption has not changed and total calorie consumption among both men and women has decreased.[7] All this suggests that there is a poor association between fat consumption, total calorie intake and physical activity with obesity while a stronger association exists for S&RC.

The effects of S&RC, as described by Kahn & Flier,[8] are chronically high insulin levels which in turn promote adipocyte triglyceride stores (fat tissue) by stimulating lipogenesis (formation of fat), inhibiting lipolysis (breakdown of fat) and increasing the uptake of fatty acids into visceral and subcutaneous fat cells. The chronic consumption of the sugar fructose, that occurs mainly through the increased intakes of high fructose corn syrups, can also promote lipogenesis and insulin resistance; which in turn further increases fat synthesis in adipose tissue.[9] The exact mechanisms of fructose induce lipidimia and insulin resistance remain controversial; however, it is generally understood that the chronic consumption of high amounts of fructose overwhelms the liver, the organ capable of metabolizing fructose, which then disrupts glucose metabolism and glucose uptake pathways ultimately leading to insulin resistance.[10] Additionally, fructose reduces the response of adiponectin, a hormone which, when in adequate amounts, increases insulin sensitivity.[11] Although the effects of S&RC are profound, not all people will respond in a similar way; that is, consuming large amounts of these nutrients will not cause all people to become obese. These observations suggest a strong genetic contribution as one's genetic make-up will influence how a body responds to high S&RC intake. It has been suggested, that the likelihood to respond unfavourably to S&RC (i.e., gain fat more quickly) is determined by one's ancestors. Studies by Pettitt et al.[12], Vickers et al.[13] and Samuelsson et al[14] have all shown that excessive consumption of S&RC by human and animal mothers who are obese and insulin resistant as a result of high carbohydrate diet during pregnancy and lactation, resulted in an increased propensity for offspring to fatten more quickly. Additionally, Bayol et al.[15] found that the offspring of mother rats fed a high sugar diet were more likely during the early part of life to indulge in sugary foods. Thus if gestational intake of S&RC increases the susceptibility of offspring to fatten more easily, then we, a population who has steadily increased the consumption of S&RC over the last 50 years,[5] could be

setting our children up for weight problems not just during childhood (childhood obesity), but their entire life.

It is therefore plausible that the ever increasing trend in obesity is an additive effect of the previous generation overindulging in S&RC producing subsequent generations with increased susceptibility who themselves, engage in a high intake S&RC. Given the current trends presented by Flegal et al.,[2] children will likely be entering adulthood heavier than they ever had before. Conventional wisdom would suggest that higher levels of physical activity and a lower intake of 'bad fats' and total calories is the cure for obesity. However given the information presented, perhaps it is time that we, the research community, acknowledge the powerful effects of sugars and refined carbohydrates on weight gain and obesity and re-focus anew.

References

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Adam Upshaw

Adam Upshaw is currently pursuing his PhD in Nutrition and Exercise at University of Western Ontario in London Ontario. His research interests include vegan nutrition and aerobic performance. Adam is a course intructor for fitness aaund nutrition courses at Humber College in Toronto and is passionate about his family, friends, Ironman training and research!