Models that Explain the Cause of Obesity

Sir,

Two models of obesity exist that describe its causation.[1] The popular theory is based on increased consumption of calories coupled with inadequate energy expenditure, also can be termed as "calorie-based model of obesity" (Cal-O). The less popular but older theory for obesity causation is based on consumption of a diet with increased amounts of high-glycemic index food leading to increased insulin secretion which on a short term depletes nutrients promoting excess food intake and on a long term promotes obesity due to its anabolic actions. This can be termed as carbohydrate-based model of obesity (Carb-O).[2] According to Cal-O, obesity drives insulin resistance leading to hyperinsulinemia, whereas in Carb-O, hyperinsulinemia is thought to be the cause for obesity. The acceptance of Carb-O is on the rise over the past decade due to evidence showing obesity reversal associated with reduced consumption of high-glycemic index carbohydrates.[3] Recently, two studies[4,5] examined Carb-O as a cause of obesity utilizing bidirectional Mendelian randomization analysis,[6] which is a tool used in genetic epidemiology for studying causation. The objective of both these studies was to study whether genetic variants associated with hyperinsulinemia can cause obesity according to Carb-O. Richmond *et al*.[4] utilized fasting plasma insulin levels which usually reflect insulin resistance, while Astley *et al*.[5] utilized glucose-stimulated insulin secretion (plasma insulin levels at 30 min following a standard oral glucose tolerance test) which reflects the beta-cell capacity to respond to dietary carbohydrates as measures of hyperinsulinemia. Body mass index was chosen as the measure for obesity in both these studies. The data were derived from two meta-analyses[7,8] that included studies which had Caucasians as the study population. Richmond *et al*.[4] found obesity as causal for fasting insulin but not vice versa. However, Astley *et al*.[5] found glucose-stimulated insulin secretion as causal for obesity and not vice versa. It could be conceived that Carb-O could be operative in those with high insulin secretory capacity combined with increased consumption of carbohydrates, and Cal-O might be operative in those with decreased insulin secretory capacity such as in Asians who also demonstrate increased insulin resistance than Caucasians.[9] In these populations, obesity could then be the result of excess calorie intake (mostly high-fat diet) leading to insulin resistance and hyperinsulinemia. More such studies are essential to be conducted in other ethnic populations to prove these observations. It is also crucial to understand the differential immune responses in individuals of different ethnicity, which could mediate the processes which promote the obesity epidemic.[10] It should be borne in mind however that although the origin and causative factors may be different in various ethnic groups, once imbalance occurs, then a vicious cycle ensues in all these populations. Another crucial implication of this knowledge is that there are no universal recommendations for healthy diets that can be prescribed uniformly for all individuals to prevent or treat obesity. Rather what is required is a more personalized approach taking into account the responses to dietary carbohydrates and fats in different individuals.

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