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Variation in genetic susceptibility drives increasing dispersion of population BMI

Dear Editor:

The recent report by Krishna et al. (1) shows unequivocally that the recent secular increase in BMI in the US population is associated with increased dispersion (SD) across the whole population and within subgroups defined by sex, race-ethnicity, and education. They conclude that future research should focus on understanding the causes of this increased dispersion. We submit that, at a fundamental level, the causes are already understood to be a consequence of between-individual variations in genetic susceptibilities to obesogenic environmental factors (2). What is lacking at present is the general recognition of this truth and, importantly, any subsequent discussion of the implications of such varying genetic susceptibilities for clinical, public health, and political responses to high rates of obesity.

The strong genetic contribution to obesity has been well established for many years by studies of familial effects on various measures of obesity, most clearly by the adoption study of Stunkard et al. (3). It seems to us that these findings have been overshadowed by the failure of large-scale genomic studies to identify the responsible sequence variants, which has been misinterpreted to cast doubt on the contribution of genetics (2). It is now apparent that large-scale genomic studies lack power to detect the many rare variants that are almost certainly responsible for the genetic contribution, and possibly full discovery is impractical in the medium term. The strong genetic effect therefore lacks a current genomic explanation, but the effect is still real and measurable and ought to be included as prior knowledge in the design and interpretation of epidemiologic studies of obesity. Had that been the case in Krishna et al. (1) it may well have been possible to estimate useful population variables such as the proportion of genetically susceptible individuals.

The presence of a strong genetic contribution to obesity invalidates the fundamental basis of current practice in preventative approaches to obesity (population-wide strategies). The suitability of those strategies for so-called lifestyle disorders such as obesity has been questioned almost from their inception (4), but neither those fundamental questions nor the recurrent failures of the strategy as applied to obesity appear to have affected the continued promotion of the approach. If epidemiologic and related studies continue to ignore the genetic basis of obesity, they will only serve to promote a false picture of the problem and thereby perpetuate futile population-wide preventative strategies and the dominant public and clinical view of obesity as a personal failing (2).

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Reply to M Kivimäki et al. and AB Jenkins and LV Campbell

Dear Editor:

We read with interest the letters by Kivimäki et al. and Jenkins et al. related to our findings of increasing interindividual inequalities or dispersion in the BMI distribution (1). Both posit that interactions between genetic susceptibility and an obesogenic environment may contribute to increasing dispersion. A recent study showing an interaction between birth cohort and fat mass and obesity–associated (*FTO*) genotype on BMI lends some support to the genetic susceptibility explanation (2). Jenkins et al., however, problematically contend that a "strong genetic contribution to obesity invalidates the fundamental basis of current practice in preventative approaches to obesity (population-wide strategies)." A strong genetic contribution to a disease does not invalidate population approaches to its prevention. For example, consider the very strong genetic basis of lung cancer, and yet a population strategy to eliminate smoking would still eliminate almost all cases of lung cancer (3). It is therefore unhelpful to pin all unexplained variability to a catchall phrase of "genetics" or, for that matter, "epigenetics" (3).

Importantly, the right-skewed BMI distribution may develop for a wide variety of reasons, ranging from basic biology to psychology to social phenomena such as assortative mating (4–6). Adipose tissue likely grows through a multiplicative rather than an additive process, resulting in a log-normal distribution with natural rightward