Prevalence trends tell us what did not precipitate the US obesity epidemic

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Hypotheses abound concerning the origins of the global obesity epidemic. Genetic predisposition, maternal obesity, excessive maternal weight gain, diabetes, tobacco use during pregnancy, and prenatal exposure to obesogens or endocrine disruptors have all been implicated.[1,2] An international survey[3] of more than 300 policymakers reported that more than 90% believed personal motivation was a strong or very strong influence on the rise of obesity. We describe how simple descriptive statistics can explain a great deal about what factors may, and may not, have caused the rapid rise in prevalence of obesity in the USA, where this phenomenon was first apparent.

Since 1960, the National Health Examination Study and National Health and Examination Surveys (NHANES) have been done on a regular basis, by the US Centers of Disease Control, among representative samples of the general population (non-institutionalised population).[4] Height and weight are measured in a standardised fashion, and are used to calculate body mass index. Results are shown for men and women of different ages (figure). Similar patterns to those shown in the figure were seen when trends were graphed separately for non-Hispanic white, non-Hispanic black, and Mexican American men and women.[4]

The increases in the prevalence of obesity began in the late 1970s across the whole US population.[5] The speed and extent of weight gain varied somewhat by age, sex and ethnicity[5] but for all subgroups most people became heavier at about the same time. This simple observation indicates something important about factors that did not precipitate the US obesity epidemic. We believe it is implausible that each age, sex and ethnic group, with massive differences in life experience and attitudes, had a simultaneous decline in willpower related to healthy nutrition or exercise. If intrauterine exposures played a major causative role,[1,2] one would have to hypothesise that there was a time-lag of about 70 years for babies born in 1910, 60 years for those born in 1920, and so on. This is also clearly implausible. Changes in genetic predisposition do not occur over the period of a few years, nor do they affect all age groups simultaneously. Indeed, it is unlikely that any factor with a long induction period had a major role in precipitating the US obesity epidemic. Nor can quick-acting exposures have been important if they only affect certain subgroups of the population, because whole population distributions of bodyweight shifted to the right. Rather, the epidemic must have been caused by factors that led to rapid population-wide changes.

In the search for factors that can plausibly explain the precipitation of this epidemic, the question is not whether the factors in question cause obesity, or even if they affect all population subgroups and have been increasing over time. Instead, the question is, are these factors a cause of obesity that increased substantially in all major US population subgroups in the late 1970s?

One candidate is the change to US farm bills in the 1970s, which led to a rapid increase in food production and thus an increase in food portion sizes;[6] accelerated marketing, availability, and affordability of energy dense foods;[7] and widespread introduction of cheap and potent sweetening agents, such as high-fructose corn syrup, which infiltrated the food system and affected the whole population simultaneously.[8] Although other countries that are experiencing a sharp rise in obesity rates might not have the equivalent of the US farm bill, most have been exposed to similar substantial changes

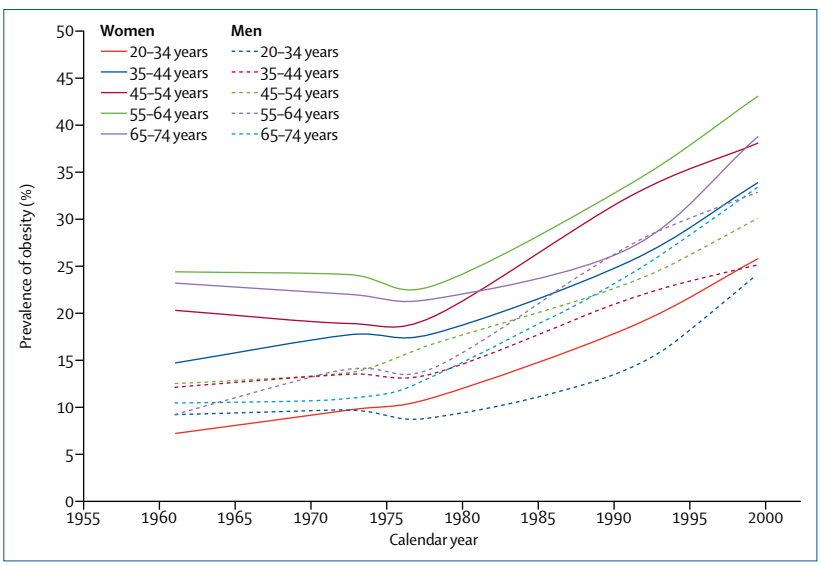

Figure: **Prevalence of obesity, by age and sex**
Data from US Centers for Disease Control and Prevention, National Health and Examination Surveys (1960–2000).[4]

in food supplies, with consequences for dietary patterns, such as increased portion sizes.

By contrast, there have been marked differences between age groups in the slowing of the epidemic—eg, obesity rates have plateaued or reversed in children.[9] These cohort effects suggest a more heterogeneous set of explanations, less ubiquitous than those that applied at the onset of the epidemic.

More than 30 years ago, Geoffrey Rose noted that the determinants of population-level changes in incidence could be missed if one only focused on the causes of individual cases.[9] But the tendency continues to confuse factors that act on individuals with the fundamental, underlying causes of population epidemics. Many factors, such as genetic predisposition, help explain where individuals are placed on the distribution of individual weights. However, they do not explain why the whole distribution of body mass has shifted towards heavier weights so rapidly in many populations. The search for the causes of such epidemics requires consideration of factors that have a mass exposure, are widely distributed, and act with short time-lags. It is not necessarily simple to identify causes of period effects (ie, those that act on the whole population at the same time) and then to intervene. It should also not be forgotten that immediate benefits can accrue from addressing factors with long induction periods—for example, gains due to smoking cessation. But when strong period effects are evident, as they are here, they signal quick potential wins in population health.[10]

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