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Origins of the obesity pandemic can be analysed

Statistical and biological methods are available to probe why the prevalence of obesity has risen more in some countries than in others, says **John Frank**.

What started the obesity pandemic? We remain unsure. And although we do not need to know the answer to tackle the symptoms, a clearer picture might produce better strategies.

Analytical methods for sorting out the epidemiological evidence on this question now lie within our reach. Economists have used these methods for many years to look at the impact of large natural experiments such as changes in policy. And in the past five years or so, epidemiologists have realized that they can be applied to health outcomes.

These statistical tricks include 'difference in differences' and 'fixed-effect variables' to control for 'unobserved heterogeneity' (confounding factors that were not measured and thus not taken into account). They are not foolproof, but they can be used to test — and often rule out — associations between population-level health changes and previous exposure to possible causes.

Obesity is particularly suited to this kind of epidemiological analysis because there is wide variation by country in when the problem began, and how quickly it developed.

As a Canadian living in Scotland (a severely overweight society), I have my own theory on the origins of obesity. And it could now be tested.

The most revealing 'epidemic curves' of the prevalence of obesity and overweight over time were published by the Organisation for Economic Co-operation and Development, with annual updates to 2012 (see go.nature.com/2bb5ns). They depict survey data on national obesity prevalence in nine developed countries and Mexico from the 1970s onwards. And, to this older epidemiologist's eye, there is a striking trend.

The United States, England, Australia, Mexico and possibly Canada show rapid growth in obesity prevalence to world-leading levels. Switzerland, Italy, Spain, South Korea and (at least initially) France, show slower growth, often with later onset, and certainly lower current levels. It is as if the two sets of countries had more, or less, resistance to the forces driving the pandemic. These are widely identified as some combination of increased calorie intake and unchanged or declining physical-activity levels.

What do the countries in each group have in common in relation to changes in lifestyle between 35 and 15 years ago? The societies that experienced later and slower weight gains have, in my opinion, much stronger cultural attachment to traditional cuisines, now thought to be healthier than most modern foods. (Mexico is obviously the exception; it is also in the economic backyard of the United States, where the pandemic hit first and hardest.) By contrast, the traditional foods of the predominantly English-speaking countries that saw an early and rapid rise in obesity are notoriously bland. When processed food laden with sugar, fat and salt arrived on the shelves, these peoples quickly switched.

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To rigorously test this conjecture, what sorts of data could be analysed? Ironically, commercial sales data may hold more promise than human-health surveys. Self-reporting of diet and physical activity is well known to be unreliable: people forget, or say what they think they should say. Over extended periods, they may alter reports to fit evolving social norms, and different cultures may systematically provide differentially biased responses.

More promising are national time series of sales of those foods currently implicated in the pandemic's origins and spread, such as sugary beverages and fast foods with high caloric density, including French fries. Detailed sales data may be difficult to obtain, especially from big fast-food chains, but proxy measures, such as total sales, should be available. Indeed, publicly traded firms in this market have often boasted to their shareholders of historical sales increases.

Time series of credible data on physical activity at the national level are harder to come by, so proxy measures of sedentary habits, such as total hours of television-watching (and, more recently, 'screen time'), might be the best available.

This quasi-experimental approach could also test the most unusual hypothesis on the pandemic's origins. In 2013, infectious-disease researcher Lee Riley and his colleagues at the University of California, Berkeley, suggested that an increasing cumulative lifetime exposure to antibiotics could be responsible, driven by these powerful chemicals' presence in meat and dairy products in our food chain, and careless overprescribing in medical care.

To test this hypothesis, one could examine time series of sales of the relevant antibiotics, both human and veterinary. However, the lag times here are much less certain: it might take decades of antibiotic exposure, perhaps beginning in childhood or even at birth, to profoundly alter the bowel microbiota and presage weight gain.

A much better data source would be frozen stool samples obtained during national surveys, collected over the relevant time period. These could be analysed for effects (now well described) of prolonged antibiotic exposure on the bowel microbiota, whose nucleic-acid and protein fingerprints should still be detectable.

So there it is: a whole programme of new and potentially important scientific work, for anyone with the nerve — and resources — to execute it. Any takers? ■

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