EDITORIAL

What is Causing the Obesity Epidemic?

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Over the last few decades the incidence and prevalence of obesity has become epidemic throughout the world. Why have we seen the swift emergence of this disease? To understand the complexity of the problem, it's necessary to appreciate the concept of "energy balance". ¹

The amount of energy entering the body (as carbohydrate, fat or protein) is used to supply all tissues and organs with metabolizable fuel, or is stored in the form of fat in adipose cells, glycogen in the liver, or protein, or, lastly, is excreted as unabsorbed energy.

Thus, simply put, energy intake is equal to energy expenditure, which is in essence the First Law of Thermodynamics. Intake being all the food we eat, and expenditure being the metabolism necessary for life, the physical activity we perform, along with a relatively miniscule amount of unabsorbed energy. Whereas we have no control over our resting metabolism or the energy necessary to digest food, we can in large part influence the energy expended from physical activity.

Thus, for weight gain to occur, total energy intake must exceed total energy expenditure. One might presume this would happen every year, to all of us (not just in the first two decades of life), since in a typical year we ingest around 800,000 kcal which represents the potential to gain an enormous amount of weight every year. But, in fact, weight gain for most adults is a very slow process, if at all, and amounts to very few pounds/kilograms that accrue irregularly.

The reason we are all not morbidly obese is because there is a complex system of energy balance regulation, by which energy intake and expenditure are tightly, impressively, regulated. There is an array of hormones secreted by the brain and peripheral tissues that affect when we feel hunger or satiety; and there are environmental signals that trigger brain-derived cognitive signals that determine how we perceive a future or current eating experience e.g, the enjoyment of eating, the taste of foods.²

If we reduce food intake or increase physical activity we will be in negative energy balance and we will lose weight. However, as we've learned from innumerable studies³ weight regain occurs, usually back to where we began. The return to the initial weight results from a host of compensatory adaptive mechanisms. The converse is also operative. That is, if we acutely increase food intake or decrease physical activity weight gain will occur but, subsequently, innate driving forces will return us to our previous weight.² Thus, the body appears to defend its current body weight. Yet, obviously, in virtually all populations, ave rage body weight has increased in the last few decades. What has happened?

One explanation is that we have chronically reduced physical activity, opting instead for more time in front of a television or computer and less time walking or doing other exercise. Unfortunately, while that hypothesis is intuitively appealing, there are no randomized controlled trials supporting the belief that increased exercise itself, to any extent, can prevent weight gain, and we've lacked the necessary precision to measure exercise patterns over many years in any population.

Another appealing hypothesis is that we're eating too much over long periods of time, and although we are superb at regulating energy balance (intake vs. output) the regulatory system gets slightly overwhelmed. In that regard, it appears that we have, indeed, been consuming more calories in recent decades, and that the increased energy intake is more than enough to explain population weight gain. On the other hand, measures of food intake are crude, and there is only indirect evidence that we can permanently 'overwhelm' our energy regulatory system.

Alternatively, there are many plausible environmental factors that, apart from increasing intake or decreasing expenditure per se, could be adversely affecting the regulatory system itself. That is, the discrepancy between total energy intake and expenditure would still be the final pathway resulting in obesity, but this hypothesis posits that consumption or exposure to a specific agent disrupts normal regulatory mechanisms in some manner, leading to excess food consumption or a decline in energy expenditure.

There is evidence that endocrine disrupters, an altered intrauterine environment, increased use of weight promoting pharmacologic agents (e.g. psychotropic drugs), insufficient sleep, smoking cessation, an altered microbiome, and various foods themselves may adversely impact energy regulation thereby causing weight gain.⁵⁻⁷

With regard to the possibility that a specific food(s) can cause obesity, much attention has recently focused on sugar, or more specifically its fructose moiety especially in high fructose corn syrup, as the entity that somehow alters the body's ability to maintain energy balance. In addition to the possibility that sugar or fructose disrupts energy balance, some investigators believe that sugar may also adversely affect other aspects of cell metabolism thereby giving rise to additional disease states. This has led to the belief that excess sugar consumption is a major cause, if not the cause, of the obesity epidemic and the most important contributing factor to other common diseases.^{8,9} On the other hand, the totality of the evidence from studies in humans suggests that it is unlikely that sugar itself has any significant effect on weight, ¹⁰⁻¹³ and appears to be even less weight promoting than a variety of other foods.⁷

The confluence of many of the factors mentioned above, along with a few others, seems to me to be the most likely culprit. First, over the last few decades, the per capita cost of food has declined, particularly for foods that are high in calories and are highly palatable (e.g. snacks, desserts, high-calorie beverages). 14 Second, with

the decline in the cost of food, the proportion of meals taken outside of the home has increased, and 'value' in restaurant dining means large portions of foods high in calories. ¹⁵ Third, the food industry, including restaurants, have developed countless products that are highly palatable and very visually appealing, making us tempted to eat more and more often.

This adverse dietary pattern (often called a "Western diet") may predispose one to obesity^{7,16,17} and if accompanied by adverse socio-economic factors (e.g. stressful life-style, job or income issues)¹⁸ and decreased physical activity, may all conspire to override energy balance regulation.

What does the future hold? One could argue that everyone who is genetically predisposed to becoming overweight or obese will, given our environment, achieve that state. Thus, we should expect to see the obesity epidemic plateau. Indeed, that appears to be happening in the U.S. where fully two-thirds of the adult population is overweight or obese (a third each). Although it's certainly possible to lose excess weight, virtually every study has shown that weight regain occurs for the vast majority of subjects, and no dietary pattern clearly stands out as being superior.¹⁹

There have been few controlled studies investigating whether weightgain can be prevented. While study results have shown mixed results,^{20,21} even those heralded as successful showed only a reduced rate in the incidence of obesity-not true prevention.²² Community-based interventions have been disappointing.^{23,24}

Although it's certainly possible to employ taxation and subsidies to significantly alter the pattern of foods consumed, that approach has many political, economic and social drawbacks, and seems unlikely to happen in any meaningful way. Increased taxation of sugar-sweetened beverages, for example, may well lead to a decline in their consumption, but given the wide-array of other high-caloric foods and our overall obesigenic environment, the likelihood of that approach actually preventing obesity to a meaningful degree seems remote.

Perhaps we just have to make the best with the situation we're in, and wait for the development of safe, effective and inexpensive pharmacotherapy or at least a much better understanding of the pathophysiology of obesity.²⁵ Eventually, both should happen. In the meantime, there are a number of dietary patterns that have been shown to be related to a reduced risk of major chronic diseases and mortality.²⁶⁻²⁸ Health professionals and government can certainly advise everyone on what is a healthy diet and to exercise more. Perhaps then we can resign ourselves to a body size less than ideal, but a lifestyle that can possibly slow down our demise.

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