Obesity and Public Health

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OBESITY is a growing global problem with serious health consequences. The obesity epidemic cannot be explained by genetics or individual behaviour alone. Neither are medical intervention or dietary education sufficient to stop the spread of obesity. To strike the root, we must recognize that energy-dense dietary choices and sedentary life choices are growing ever more accessible in comparison to their healthier alternatives, both here and worldwide. There is now intense interest in the so-called obesogenic society. The nascent literature on obesogenicity has already identified several economic factors contributing to the distribution and spread of obesity. These findings reinforce the need to create social environments that make healthy diet and fitness accessible to everyone. Public health policy must play a greater role in combating obesity.

The obesity epidemic and its costs

Obesity has emerged as one of the most visible and fastest-growing medical problems in today's world. People everywhere are accumulating excess body fat and facing ever-greater risk of weight-related illness. Once found in the wealthiest parts of a mostly

malnourished world, obesity grew more common in tandem with food security and lifestyle change, soaring after 1980[1] and heading worldwide.

The World Health Organization (WHO) defines overweight as having a body mass index (BMI) over 25 kg/m^2 and obesity as having BMI over 30 kg/m^2. The WHO estimates that, in 2005, 1.6 billion adults, a third of the adult world population, were overweight, of which at least 400 million, almost 9% of the adult population, were obese; and they expect as many as 2.3 billion overweight and 700 million obese adults by 2015. In many parts of the world, the prevalence of obesity has more than tripled since 1980.[2] Child overweight and obesity are also reaching epidemic levels: 10 percent worldwide, over 20% in Europe, and 30% in North America.[3]

The obesity epidemic is most pronounced in the United States, where two in three adults and 23 million children are overweight or obese.[4] In Canada, the prevalence of obesity and overweight increased between 1981 and 1996 in men, women, and children. The fastest trends were seen in children: overweight tripled in boys and doubled in girls, while obesity rose five-fold to 10% in boys and 9% in girls.[5]

Obesity is a dangerous condition that reduces the length and quality of life. Adiposity is a risk factor in many deadly and debilitating conditions, including heart disease,

stroke, hypertension, osteoarthritis, some cancers, and type II diabetes,[1,2,6] which occurs almost exclusively in the overweight. At one point, the US Centers for Disease Control (CDC) claimed that as a risk factor obesity was second only to tobacco, contributing to 400 000 premature US deaths each year.[1] In response to academic criticism, the CDC revised the estimate downward in 2005 to over 100 000 premature deaths annually, still enough to warrant alarm about an American obesity epidemic.[7]

Annual US medical costs and productivity losses due to obesity reached US$99 billion in 1998[6] and rose to $139 billion (2003 dollars) by 2003, including 5-7% of US medical expenditures.[1] Canadian health care costs attributable to obesity in 1997 were estimated to be conservatively 2.4% or as high as 4.6% of total expenditures.[8] Naturally, the ill health associated with obesity has spurred medical efforts to reverse weight gain in individual procedures that range from nutrition counselling to invasive bariatric surgery, which account for some of the ballooning medical costs. Drugs can, at best, stabilize weight, not reliably reduce it.[3] Invasive procedures, e.g. gastric bypass, are effective but hazardous. They are considered a last resort, unsuited for mass use.[3,9] Interventions at the individual level have not effectively slowed the rise of obesity or its comorbidities.[6]

The biomedical study of obesity

The immediate medical cause of obesity is widely understood: adipose tissue, or body fat, a medium for energy storage, is accumulated when the dietary energy intake exceeds the expenditure on physical activity and rest metabolism. Weight gain is a result of more energy consumed than exerted.

In turn, we are motivated to determine what causes the energy imbalance. In line with the biomedical model of health, there is lasting interest in the genetic predisposition for weight gain. Evolutionary theory predicts the existence of such genes. Humanity has faced frequent starvation. Until recent times, restraint in the face of plenty could mean death later on. Those genetically predisposed to better energy storage when food was available were more likely to survive famine, attract mates, and procreate. Indeed, genetic variety seems to be a significant factor in the individual variation in weight.[10,11] However, it does not contribute to the current rapid rise in mean weight. The epidemic has emerged too quickly to be caused by recent changes in the gene pool.[9,10] If anything, the lack of genetic change contributes to obesity, as our old programming leaves us generally maladapted to structural changes in food availability.[11]

The obesogenic society

With no apparent hereditary change responsible for the changes in individual nutrition and activity which have led to obesity, answers are now being sought in the milieu. Lately there has been an explosion of interest in the social determinants of obesity. A review on neighbourhood influences discovered the majority of the literature was less than four years old.[6] Important results are piling up and drawing an ever-clearer picture of how the social environment promotes weight gain. In 1997, Egger and Swinburn coined the word *obesogenic* to describe environments that stimulate obesity. They predicted obesity's continued spread in an obesogenic society that thwarts medical treatment and educational efforts.[12] The term and its associated ideas have become a major theme of the field. Specific obesity stimuli are called *obesogens.*

It may not be surprising that weight gain has accelerated in a society full of fast food, pre-packaged snack food, automobiles, television, and computer games, especially with homemaking and physical labour on the decline. However, not all of these popularly cited social factors are necessarily significant obesogens. Finkelstein et al. argue[1] that many of them were already ubiquitous before the epidemic arose and are therefore absolved. For example, television broadcast viewership rose faster prior to 1980 than afterward, so the rise in "total screen time" since then must be attributed instead to the emergence of video games, desktop computers, and the home cinema. They also note

that a rise of only 50–100 kilocalories in the average daily energy surplus was enough to cause the whole observed weight gain trend.[1,9] Their argument implies that only those economic trends coincident with the epidemic are likely to be responsible for it.

Dietary energy intake

Food prices may form the most important trend which coincided with the obesity epidemic. As the consumption of food is not perfectly price-inelastic,[13] dietary energy should be seen to increase when food is cheaper. Indeed, this relationship is observed. From 1960 to 1980, when obesity was stable, food prices rose slightly against inflation. From 1980 to 2000, when obesity rose to epidemic numbers, food prices were falling. Perhaps more importantly, the prices of energy-dense food and drink fell faster than other food prices, encouraging them to be used in place of healthier choices.[1,9,11] This imbalance may be the result of technological change[1,10] or price distortion caused by the system of agricultural subsidies.[14]

Dietary intake is more closely related to the amount of food than the number of calories in it. If each serving is more energy-dense, more energy is consumed. Compounding the problem of calories per gram, cheaper food is also causing a parallel trend of "supersized" portions. A number of studies have documented an almost universal

growth of serving sizes since the 1970s both in and out of the home and have associated serving sizes with obesity.[1,14]

Also coincident with the rise of obesity are a number of shifts in how food is marketed, in particular the explosion and aggressive promotion of fast food restaurants and pre-packaged snack food products. Unhealthy fast food and snacks earned the name "junk food" in 1972 thanks to their high fat, carbohydrate, and sodium content, chemical additives, and lack of micronutrients.[15] Junk food is thought to have emerged in response to an increasing demand for food convenience following the shift toward women's labour force participation and away from household production.[1,11] In turn, junk food has played an increasing role in weight gain.

The US fast food industry grew from 30 000 to 220 000 restaurants between 1970 and 2001.[6] Fast food restaurants have also been spreading worldwide, increasing almost five-fold in Asia and seven-fold in Latin America in the 1990s.[3] Fast food diets are higher in calories and fat and are associated with obesity.[6,11] Fast food has had a particularly strong impact on children. Between the late 1970s and the late 1990s, eating out almost doubled for American children and the fraction of dietary energy taken as fast food quintupled to ten percent. Children's consumption of soft drinks also rose.[9]

Snack food has grown ubiquitous in parallel with fast food. Daily meal structure has declined while snacking frequency and the energy density of snacks have increased, especially for children,[1,11,14] trends which are linked with child obesity.[10] By some accounts, the rise in snacking accounted for 76% to 95% of the total increase in energy intake by American adults from the late 1970s to the mid-1990s; and the increase in energy intake is responsible, in turn, for the entire increase in adult obesity.[1]

The increasing recognition of junk food's role in the continuing rise in obesity has raised concerns and unrest about how pre-packaged food is marketed and promoted.[10] By 2003, US$40 billion were spent annually on advertising food products worldwide, over 500 times the WHO's budget for promoting dietary health. Much of this promotion is for unhealthy, energy-dense foods advertised on children's television programming and through school sponsorships, resulting in parents losing control over their children's diet.[3] In contrast to the limitations on the promotion of tobacco and alcohol in much of the world, controls on energy-dense food exist only in some European countries despite the need to make everyone, especially impressionable children, aware of the health consequences.[10,14]

Physical inactivity and the built environment

Several studies have linked the degree of physical activity with weight gain and distributional inequities in weight. Much of this research involves individual lifestyle and time use; the rest focuses on a group of factors known as the *built environment,* relating to the design and features of neighbourhoods and access to healthy activities.

When it comes to the role of physical inactivity in weight gain, television and computer use are the usual suspects. Time spent watching a screen is postulated to cause weight gain in three ways: first, by displacing time spent in physical activity; second, by being less metabolically demanding than other sedentary behaviours like writing, typing, playing cards, and even reading—indeed, being virtually as sedate as sleep; and third, by stimulating dietary intake through snacking and exposure to snack and fast food adverts.[3,16] A survey of Canadian adults found evidence for all three relationships, of which the metabolic link may have been the strongest. Even among adults with ostensibly similar patterns of physical activity and diet, obesity was still positively associated with the amount of time spent watching television. In light of the habit-forming nature of television in childhood, the authors recommended public policy to reduce television time among children and adults.[16]

In the study of the built environment, a number of neighbourhood factors have been linked with BMI, including land use and access to parks, greenbelt, recreation and fitness facilities. In the case of land use planning, more physical activity and lower BMIs were recorded in neighbourhoods with more mixed land use, where residents could more easily and safely walk from place to place.[6]

One weakness in the research on physical activity is that studies have tended to be cross-sectional. Due to the immaturity of the field, there is less research into how physical activity and the built environment have changed over time.[6] Consequently, their results do not necessarily strengthen the case that physical activity fell in concert with the obesity epidemic. Citing time-series studies on the composition of labour and leisure activity, Finkelstein et al. conjecture little to no decline in energy expenditure since 1980.[1] Consequently, the physical activity inequities that have been found in the built environment must predate the epidemic to some degree.

Socioeconomic status

Adult and child obesity are associated with high socioeconomic status (SES) in poor countries and low SES in rich countries.[4,10,14] One study found the turning point to be US$2500 per capita. In poorer countries, well-educated women were more obese, and in richer countries the relationship was reversed.[17] These results are consistent with a shift

from starchy staple diets in poor, rural populations toward fatty, sugary diets in urban populations.[2] There is some evidence for a link between income inequality and obesity within developed countries.[6,11,18]

The role of public policy

The need for public action to combat obesity is stronger than ever. Despite playing an important role in promoting food security in the midst of massive social change, the modern food industry has also begun to create serious worldwide health problems in the form of adult and child obesity, which degrade health, impose medical costs and productivity losses, and contribute to disability and premature death. Furthermore, although obesity is a medical problem, it has no easy, widely applicable medical solution. Obesity must be prevented, not cured, and the answer lies in making appropriate changes to the social environment. A combination of public policy approaches should be considered.

First, in light of the role junk food has played in creating the obesity epidemic, the marketing of fast food and pre-packaged snack foods must change substantially. The WHO urges governments to reform their agricultural subsidy programs, impose sales taxes on less healthy foods, and limit food and drink adverts targeted at children.[7] The PorGrow project, a dialogue with EU policy stakeholders including food producers,

found support for better nutritional labelling, better school nutrition and nutritional education, and limits on advertising; additional taxes and subsidies were not popular.[19] Another dialogue set out a variety of snack industry responsibilities, e.g. to improve nutrition and nutritional transparency and to refrain from advertising to children.[10]

Nevertheless, the obesity epidemic was not caused by ads and information gaps alone, and individual behaviour has only limited capacity to change so long as the most important cue, the low price of junk food, remains unchanged. Generational trends in food prices have diminished the ability to maintain healthy diet. Real incentives will be necessary to restore healthy diet and halt the spread of obesity.[1] Whether through subsidy reform or new taxes, and while taking care not to threaten the public's food security, junk food must be made more expensive than healthy food.

Second, although there is less support for the idea that physical inactivity has contributed to the rise in obesity, we have seen inequities in socioeconomic status and the built environment that cause poor fitness and contribute to obesity. Many of these inequities predate the epidemic and will persist until something is done about them. Universal access to health care is not a sufficient condition for health equality. If we want to help the disadvantaged to fight off obesity, we must also work toward universal access to healthy food and fitness choices. PorGrow found popular support for

improving availability and access to sports facilities.[19] However, policy must go further than this to address all aspects of community access and land use, especially in poor communities, where obesity is most prevalent.

Above all, more research is needed in order to fully document the obesity epidemic and its costs, the obesogenic society, the built environment, and related public health issues.

References

1. Finkelstein EA, Ruhm CJ, Kosa KM. 2005. "Economic Causes and Consequenses of Obesity." *Annual Review of Public Health* 26: 239–57.

2. World Health Organization. 2006. "Fact sheet N°311: Obesity and overweight." <http://www.who.int/mediacentre/factsheets/fs311/en/index.html> (accessed April 6, 2009).

3. Lobstein T. 2004. "Child obesity: public health meets the global economy." *Consumer Policy Review* 14(1).

4. Hamburg R. March 26, 2009. "Obesity in the U.S." *FDCH Congressional Testimony.*

5. Tremblay MS, Katzmarzyk PT, Willms JD. 2002. "Temporal trends in overweight and obesity in Canada, 1981-1996." *International Journal of Obesity* 26(4): 538–543.

6. Black JL, Macinko J. 2008. "Neighborhoods and obesity." *Nutrition Reviews* 66(1): 2–20.

7. Malvasi MG. 2007. *Obesity.* Broomall, Pa: Mason Crest Publishers.

8. Birmingham CL, Muller JL, Palepu A, Spinelli JJ, Anis AH. 1999. "The cost of obesity in Canada." *Canadian Medical Association Journal* 160(4): 483-8.

9. Ebbeling CB, Pawlak DB, Ludwig DS. 2002. "Childhood obesity: public-health crisis, common sense cure." *The Lancet* 360: 473–82.

10. Astrup A, Bovy MWL, Nackenhorst K, Popova AE. 2006. "Food for thought or thought for food? – A stakeholder dialogue around the role of the snacking industry in addressing the obesity epidemic." *Obesity Reviews* 7: 303–312.

11. Ulijaszek SJ. 2007. "Obesity: a disorder of convenience." *Obesity Reviews* 8 (Suppl. 1): 183–187.

12. Egger G, Swinburn B. 1997. "An 'ecological' approach to the obesity pandemic." *British Medical Journal* 315(7106): 447.

13. USDA Economic Research Service. 2003. "International Food Consumption Patterns." United States Department of Agriculture. <http://www.ers.usda.gov/Data/InternationalFoodDemand/> (accessed April 10, 2009).

14. Rosenkranz RR, Dzewaltowski DA. 2008. "Model of the home food environment pertaining to childhood obesity." *Nutrition Reviews* 66(3): 123–140.

15. Smith AF. 2006. *Encyclopedia of Junk Food and Fast Food.* Greenwood Publishing Group. <http://books.google.com/books?id=mYc4aw7tnekC> (accessed April 12, 2009).

16. Shields S, Tremblay MS. 2008. "Sedentary behavior and obesity." *Statistics Canada Health Reports* 19(2).

17. Monteiro CA, Conde WL, Lu B, Popkin BM. 2004. "Obesity and inequities in health in the developing world." *International Journal of Obesity* 28: 1181–1186.

18. Lobstein T, Millstone E. 2007. "Context for the PorGrow study: Europe's obesity crisis." *Obesity Reviews* 8 (Suppl. 2): 7–16.

19. Millstone E, Lobstein T. 2007. "The PorGrow project: overall cross-national results, comparisons and implications." *Obesity Reviews* 8 (Suppl. 2): 29–36.