

Why causality, and not prediction, should guide obesity prevention policy



The large increase in obesity worldwide is a major public health crisis.¹⁻³ Obesity has been associated with several non-communicable diseases, such as diabetes, cardiovascular diseases, and cancers, and is a major cause of premature death.² According to WHO, at least 2.8 million deaths and more than 35 million (2.3%) global disability-adjusted life-years are linked to overweight or obesity.³ Furthermore, obesity is a major cause of osteoarthritis and chronic disabilities. Owing to the increase of obesity and population ageing, especially in low-income and middle-income countries, the obesity-related burden of disease will rise.¹

In *The Lancet Public Health*, Solja Nyberg and colleagues⁴ analysed data from ten large cohort studies to estimate the extent to which body-mass index (BMI) categories, and obesity in particular, are associated with the number of years free from major non-communicable diseases. Compared with normal weight, the loss of disease-free years in men was 1.8 (95% CI -1.3 to 4.9) for underweight, 1.1 (0.7 to 1.5) for overweight, 3.9 (2.9 to 4.9) for class I obese, and 8.5 (7.1 to 9.8) for class II-III obese; corresponding estimates for women were 0.0 (-1.4 to 1.4) for underweight, 1.1 (0.6 to 1.5) for overweight, 2.7 (1.5 to 3.9) for class I obese, and 7.3 (6.1 to 8.6) for class II-III obese. The association between obesity and loss of disease-free years was observed across all categories of physical activity, smoking, and socioeconomic status. The investigators concluded that these results “lend support to obesity prevention as an important strategy for the reduction of morbidity”.⁴

What are the true policy implications of these findings? A straightforward implication is that preventing obesity will decrease the number of years lived with diseases. This statement implies a causal link between obesity and these diseases (figure). Although this implication seems evident, stating that we can prevent diseases or delay their occurrence if we reduce obesity raises several complex issues.⁵ One major issue is the scarcity of strong evidence on how to prevent obesity. Prevention surely requires a complex, multilevel, environmental, socioeconomic, and life-course approach.⁶ However, despite a large number of studies designed to tackle

the causes of obesity and several health promotion programmes to prevent obesity, we still do not have efficient, evidence-based, well defined, and applicable interventions to prevent obesity.

A second major—and difficult to solve—issue is that the impact of an obesity prevention programme on the burden of disease depends on the method used to prevent weight gain.⁵ If there was a simple and direct causal effect of obesity on the risk of diseases (figure), the number of diseases prevented or delayed for a given reduction in BMI could be easily predicted using, for example, the results by Nyberg and colleagues.⁴ However, causal links between obesity and the risk of disease are not so simple. Obesity results from a mix of factors such as diet or physical activity, embedded in a causal web of environmental and socioeconomic determinants, which have direct and specific effects on the risk of obesity-related diseases (figure). If you target physical activity to prevent high BMI, you may not have the same effect on the burden of disease than if you target diet, even if you have the same effect on BMI.⁷ One can assume that BMI has per se no direct causal effect on the risk of disease, only related causal mechanisms.⁷ In this perspective, high BMI is merely a marker of risk, and as such should not be the primary target of prevention strategies. Such a perspective on

Published Online
August 31, 2018
[http://dx.doi.org/10.1016/S2468-2667\(18\)30158-0](http://dx.doi.org/10.1016/S2468-2667(18)30158-0)
See [Articles](#) page e490

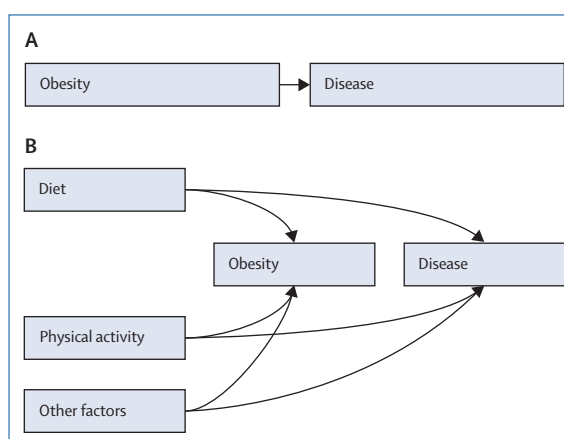


Figure: Simplified causal relationship between obesity and disease
(A) A direct causal effect of obesity on the occurrence of diseases. (B) Diet, physical activity, and other factors have a direct effect on obesity and diseases, but there is no direct effect of obesity on the risk of disease. In both cases, obesity is associated with a loss of disease-free years.

obesity is also key because it helps deal with the fact that optimal BMI might increase with age; evidence suggests that BMI in the overweight or obesity I range, particularly in older adults, is associated with a lower mortality risk compared with normal weight.^{1,8} Hence, policy aiming to prevent overweight or obesity could be, at least in theory, deleterious in this segment of the population.

Causality is necessary to define appropriate prevention policy because it indicates the possibility for intervention.^{5,9} Modifiable causal factors, such as diet or physical activity, should be the explicit targets of prevention programmes. The study by Nyberg and colleagues⁴ is an eloquent and very well done prediction exercise, informing us that people with obesity have a reduced life expectancy free of disease. There is, however, no explicit causal consideration in this study. Although this study offers arguments to conduct further research and prevention activities related to obesity, it does not help to directly inform prevention policy. Research to guide such policy should assess the effect of interventions to increase physical activity or improve diet, or to influence their determinants, on the burden of obesity-related disease; that would be a truly consequential public health prevention research agenda.¹⁰

Arnaud Chiolero

Institute of Primary Health Care (BIHAM), University of Bern, Bern, Switzerland; Institute of Social and Preventive Medicine (IUMSP), University of Lausanne, Lausanne, Switzerland; Department of Epidemiology, Biostatistics, and Occupational Health, McGill University, Montreal, QC, Canada
 achiolero@gmail.com

I declare no competing interests.

Copyright © 2018 The Author(s). Published by Elsevier Ltd. This is an Open Access article under the CC BY 4.0 license.

- 1 Hugues V. The big fat truth. *Nature* 2013; **497**: 428–30.
- 2 NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet* 2017; **390**: 2627–42.
- 3 WHO. Global Health Observatory data. Obesity. Situation and trends. http://www.who.int/gho/ncd/risk_factors/obesity_text/en/ (accessed July 22, 2018).
- 4 Nyberg ST, Batty GD, Pentti J, et al. Obesity and loss of disease-free years owing to major non-communicable diseases: a multicohort study. *Lancet Public Health* 2018; published online Aug 31. [http://dx.doi.org/10.1016/S2468-2667\(18\)30139-7](http://dx.doi.org/10.1016/S2468-2667(18)30139-7).
- 5 Chiolero A, Paccaud F. An obesity epidemic booga booga? *Eur J Public Health* 2009; **19**: 568–69.
- 6 Dietz WH. The response of the US Centers for Disease Control and Prevention to the obesity epidemic. *Annu Rev Public Health* 2015; **36**: 575–96.
- 7 Hernán MA, Taubman SL. Does obesity shorten life? The importance of well-defined interventions to answer causal questions. *Int J Obes* 2008; **32** (suppl 3): S8–14.
- 8 Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA* 2013; **309**: 71–82.
- 9 Glass TA, Goodman SN, Hernán MA, Samet JM. Causal inference in public health. *Annu Rev Public Health* 2013; **34**: 61–75.
- 10 Galea S. An argument for a consequentialist epidemiology. *Am J Epidemiol* 2013; **178**: 1185–91.