

Noncommunicable diseases: more things in heaven and earth than are dreamt of?

Doenças crônicas não transmissíveis: há mais no céu e na terra de que se possa sonhar?

Enfermedades no transmisibles: ¿más cosas de las que pensábamos entre el cielo y la tierra?

Eduardo Faerstein ¹

doi: 10.1590/0102-311X00239221

Macinko & Mullachery ¹ analyzed data from Brazil's first two *Brazilian National Health Surveys* (PNS), conducted in 2013 and 2019 and coordinated by the Oswaldo Cruz Foundation (Fiocruz), in partnership with the Brazilian Institute of Geography and Statistics (IBGE). The authors sought to test two relevant socio-epidemiological hypotheses. The first hypothesis was that over the six years between the two surveys, economic recession, deteriorating socioeconomic conditions, and weakened social policies increased the prevalence of noncommunicable diseases (NCDs) in Brazil. The second was that this increase mostly affected the poorer population, thus widening social inequalities for these diseases and risk factors. Although they are not comparable regarding data sources and analyses, previous work ² had shown mixed results for an earlier period (1998-2013), when economic and social indicators were improving in Brazil: a decrease of educational disparities in the prevalence of hypertension and heart disease, but an increase in those prevalence disparities for diabetes.

For a defined group of physician-diagnosed NCDs that were self-reported by survey participants at in-person interviews (with body mass index calculated based on measured weight and height), the authors observed an increase in prevalence rates from 8% (arthritis) to 24% (obesity) between 2013 and 2019. However, health inequality indexes (slope index of inequality – SII, relative index of inequality – RII, population attributable fraction – PAF, all of which are still underused with Brazilian data) showed no meaningful increase in social gradients for those diseases and risk factors.

The authors acknowledged several limitations of their analyses, including the self-reported nature of most data, their cross-sectional nature, the restricted number of outcomes, and the use of education as the single marker of social position. They speculated about possible causes for the absence of growing health inequalities for the analyzed NCDs, such as detection or survival bias. However, both PNS datasets do not seem to indicate those biases. Further analyses might clarify this important issue if they explore already available data from the PNS regarding the inclusion of additional NCDs, sex-specific trends, health care details, and case stratification by clinical severity and age at diagnosis.

If perhaps the next PNS is conducted 10 years after the 2013 PNS, it may give more time for the induction and latency periods needed for several NCDs to develop. Sadly, consequences of the current COVID-19 pandemic in Brazil – where, as in most countries, incidence and case fatality are higher among the poor – will most likely increase health inequalities as they will for NCDs, with sev-

¹ Instituto de Medicina Social
Hesio Cordeiro, Universidade
do Estado do Rio de Janeiro,
Rio de Janeiro, Brasil.

Correspondence

E. Faerstein
Instituto de Medicina Social
Hesio Cordeiro, Universidade
do Estado do Rio de Janeiro.
Rua São Francisco Xavier
524, Bloco E, 7º andar, sala
7009, Rio de Janeiro, RJ
20559-900, Brasil.
efaerstein@gmail.com



eral mechanisms. However, the relationship between economic cycles and health is rather complex, sometimes even counterintuitive – as an example, European recessions, on average, showed beneficial short-term effects on adult mortality ³.

I hope to contribute to the discussion of more issues related to causes and control of NCDs, with the same objective as Macinko & Mullachery, that is, to stimulate a debate on the most effective ways to combat the causes of these diseases. I believe that this arrangement of views should also address two long-standing shortcomings of current research and practice, as follows.

Firstly, it is outdated and no longer useful to divide a major share of human morbidity between communicable and noncommunicable. Worse, it tends to be followed by an objectionable segmentation of health programs.

In fact, abundant evidence shows a complex overlap between NCDs and infectious agents. Some agents are causally related to various neoplasms (e.g., *Helicobacter pylori* – gastric cancer, hepatitis virus B and C – liver cancer, *Schistosoma haematobium* – bladder cancer, human papillomavirus – several cancer sites, among others). Regarding cardiovascular diseases, several pathogens are associated with atherosclerotic plaque (bacteria – e.g., *Chlamydia pneumoniae*, *Mycoplasma pneumoniae*; viruses – e.g., cytomegalovirus, hepatitis C, Epstein-Barr) ⁴.

Most neglected tropical diseases (NTDs) show a chronic clinical course, so it is difficult to distinguish them from NCDs, with Chagas disease in Latin America as an emblematic example. Bidirectional associations between tuberculosis and diabetes, for example, also occur. Above all, the growing field of human microbiome research has shown evidence of gut bacteria contribution (with as many as 20 million genes!) in several NCDs ^{5,6}.

During the ongoing coronavirus pandemic, COVID-19 has interacted with several pre-existing noncommunicable conditions in COVID patients (e.g., obesity, chronic respiratory disease), increasing the risk of adverse clinical outcomes. Moreover, the post-acute sequelae of COVID-19 (PASC) have been increasingly recognized; according to a *Fair Health White Paper* ⁷, one-fifth to one-half of all COVID-19 cases had at least one PASC symptom, and several organs and systems can be affected by the virus. As stated by *The Lancet* ⁸ (p. 649): “COVID-19 and NCDs form a dangerous relationship, a syndemic that is exacerbating social and economic inequalities”.

Secondly, several environmental determinants of NCDs have already been identified, and challenging hypotheses seek to identify others. Nevertheless, those environmental determinants and novel hypotheses have been largely neglected or ignored.

As an example, strategies from both the Brazilian Ministry of Health ⁹ and the World Health Organization ¹⁰ to prevent and control NCDs focus on four risk factors: tobacco use, harmful use of alcohol, unhealthy diets, and physical inactivity – with barely any mention to the exposure to environmental hazards. These imply that the main causes of NCDs are individual lifestyle choices, even though those health-related behaviors are, in fact, strongly influenced by external events, especially by social determinants such as education, wealth, and access to quality health care.

Moreover, NCD effects of widespread environmental exposure, caused by global societal changes, are still clearly overlooked. A comprehensive appraisal of all of these issues was conducted by an international, multidisciplinary team of researchers gathered by The Lancet Commission on Pollution and Health ¹¹ (p. 465), from which we quote:

“Pollution is one of the great existential challenges of the Anthropocene epoch. (...) Pollution [i]s unwanted, often dangerous, material that is introduced into the Earth’s environment as the result of human activity, that threatens human health, and that harms ecosystems. (...) The burgeoning problems of air, water, and soil pollution produced by modern industry, electricity generation, mining, smelting, petroleum-powered motor vehicles, and chemical and pesticide releases in low-income and middle-income countries have received almost no international attention or resources. (...) In 2015, all forms of pollution combined were responsible for 21% of all deaths from cardiovascular disease, 26% of deaths due to ischaemic heart disease, 23% of deaths due to stroke, 51% of deaths due to chronic obstructive pulmonary disease, and 43% of deaths due to lung cancer. (...) Heads of government who successfully confront vested interests, bring agencies together, reduce environmental injustice, control pollution, and prevent pollution-related disease can reap great praise, build a legacy, [and] help the world achieve the S[ustainable] D[evelopment] G[oa]ls...”

The neglect of established NCD environmental risk factors is exemplified by mainstream guidelines (resulting in few successful stories, if any) to prevent obesity, a condition with higher risk of

several NCDs. Most guidelines^{9,10} focus on avoiding excess energy intake and sedentary lifestyle. However, many researchers consider that those “Big Two” cannot resolve the explosive gain of excess weight in many societies over the last few decades¹². The documented weight gain of various animal species over the same period – in laboratories, near humans, and in the wild – shows compelling evidence that common environmental factors play etiologic roles¹³. As discussed above, such factors have been investigated, and challenging causal hypotheses have been proposed.

As an example, persistent organic pollutants (POPs) – including pesticides and plastic products – may act as endocrine disruptors, influencing weight gain with several mechanisms^{14,15}. Riley et al.¹⁶ had another plausible hypothesis: that intestinal microbiota can capture more energy from food after being disrupted by widespread chronic exposures to antimicrobial residues, which are increasingly entering the food chain and the environment because of massive antibiotics use in agribusiness and in animal husbandry.

Relationships between climate change and obesity have also been discussed. While some authors suggest that obese individuals emit more greenhouse gases linked to global warming¹⁷, others have considered possible mechanisms that suggest the opposite – but to our knowledge, they are still quite ignored. Hersoug et al.¹⁸ proposed that increased atmospheric CO₂ can reduce blood pH, thus deregulating orexin neurons in the hypothalamus, which controls appetite and wakefulness, and leading to weight gain. Zeutlin et al.¹⁹ observed ecological associations between carbon dioxide emissions and changes in obesity and diabetes prevalence in the United States. Cotner²⁰ had a different hypothesis: excess CO₂ in the atmosphere could cause excess carbon in food supply, thus contributing to “obesogenic” changes in metabolism.

In the near future, exposure to microplastics and nanoplastics (MNPs) will likely be one of the most researched causes for obesity. MNPs are common persistent pollutants and the most representative material of the Anthropocene epoch. The effects of MNPs on the environment, especially on marine and freshwater animals, are well documented. However, research of their impact on human health by food and/or water consumption is still incipient. The possible toxic effects of MNPs could be caused by the potential toxicity of plastics themselves, combined with leachable additives and adsorbed contaminants, both acting as endocrine disruptors^{21,22}.

In conclusion, regarding the causes of noncommunicable diseases, that famous Danish prince might as well tell his friend: “*There are more things in heaven and earth, Horatio, than are dreamt of in your philosophy*” (Shakespeare, *Hamlet*).

Additional information

ORCID: Eduardo Faerstein (0000-0002-4027-4896).

1. Macinko J, Mullachery PH. Education-related health inequities in noncommunicable diseases: an analysis of the *Brazilian National Health Survey*, 2013 and 2019. *Cad Saúde Pública* 2022; 38 Suppl 1:e00137721.
2. Beltrán-Sánchez H, Andrade FCD. Time trends in adult chronic disease inequalities by education in Brazil: 1998-2013. *Int J Equity Health* 2016; 15:139.
3. Tapia-Granados JA, Ionides EL. Population health and the economy: mortality and the Great Recession in Europe. *Health Econ* 2017; 26:e219-35.

4. Campbell LA, Rosenfeld ME. Infection and atherosclerosis development. *Arch Med Res* 2015; 46:339-50.
5. Shivani S, Chattopadhyay A, Chuang EY. Targeting the gut microbiome for non-communicable diseases: present and future. *Ann Transl Med* 2021; 9:361.
6. Wilkinson JE, Franzosa EA, Everett C, Chengchen L; HCMPH Researchers and Trainees; HCMPH Investigators, et al. A framework for microbiome science in public health. *Nat Med* 2021; 27:766-74.
7. FAIR Health. A detailed study of patients with long-haul COVID. https://s3.amazonaws.com/media2.fairhealth.org/whitepaper/asset/A%20Detailed%20Study%20of%20Patients%20with%20Long-Haul%20COVID--An%20Analysis%20of%20Private%20Healthcare%20Claims--A%20FAIR%20Health%20White%20Paper.pdf?utm_source=mahoning%20matters&utm_campaign=mahoning%20matters&utm_medium=referral (accessed on 15/Jun/2021).
8. The Lancet. COVID-19: a new lens for non-communicable diseases. *Lancet* 2020; 396:649.
9. Ministério da Saúde. Plano de ações estratégicas para o enfrentamento das doenças crônicas não transmissíveis (DCNT) no Brasil 2011-2022. Brasília: Ministério da Saúde; 2011. (Série B. Textos Básicos de Saúde).
10. World Health Organization. Global action plan for the prevention and control of NCDs 2013-2020. Geneva: World Health Organization; 2013.
11. Landrigan PJ, Fuller R, Acosta NJR, Adeyi O, Arnold R, Basu NN, et al. The Lancet Commission on pollution and health. *Lancet* 2018; 391:462-512.
12. Keith SW, Redden DT, Katzmarzyk PT, Boggianno MM, Hanlon EC, Benca RM, et al. Putative contributors to the secular increase in obesity: exploring the roads less traveled. *Int J Obes (Lond)* 2006; 30:1585-94.
13. Klimentidis YC, Beasley TM, Lin HY, Murati G, Glass GE, Guyton M, et al. Canaries in the coal mine: a cross-species analysis of the plurality of obesity epidemics. *Proc Biol Sci* 2011; 278:1626-32.
14. Thayer KA, Heindel JJ, Bucher JR, Gallo MA. Role of environmental chemicals in diabetes and obesity: a national toxicology program workshop review. *Environ Health Perspect* 2012; 120:779-89.
15. Petrakis D, Vassilopoulou L, Mamoulakis C, Psycharakis C, Anifantaki A, Sifakis S, et al. Endocrine disruptors leading to obesity and related diseases. *Int J Environ Res Public Health* 2017; 14:1282.
16. Riley LW, Raphael E, Faerstein E. Obesity in the United States – dysbiosis from exposure to low-dose antibiotics? *Front Public Health* 2013; 1:69.
17. Magkos F, Tetens I, Bügel SG, Felby C, Schacht SR, Hill JO, et al. The environmental food-print of obesity. *Obesity (Silver Spring)* 2020; 28:73-9.
18. Hersoug LG, Sjödin A, Astrup A. A proposed potential role for increasing atmospheric CO₂ as a promoter of weight gain and obesity. *Nutr Diabetes* 2012; 2:e31.
19. Zheutlin AR, Adar SD, Park SK. Carbon dioxide emissions and change in prevalence of obesity and diabetes in the United States: an ecological study. *Environ Int* 2014; 73:111-6.
20. Cotner JB. How increased atmospheric carbon dioxide and ‘The Law of the Minimum’ are contributing to environmental obesity. *Acta Limnologica Brasiliensia* 2019; 31:e113.
21. González-Acedo A, Recio EG, Illescas-Montes R, Ramos-Torrecillas J, Melguizo-Rodríguez L, Costela-Ruizab VJ. Evidence from in vitro and in vivo studies on the potential health repercussions of micro- and nanoplastics. *Chemosphere* 2021; 280:130826.
22. Facciola A, Visalli G, Pruiti Ciarello M, Di Pietro A. Newly emerging airborne pollutants: current knowledge of health impact of micro and nanoplastics. *Int J Environ Res Public Health* 2021; 18:2997.

Submitted on 05/Oct/2021
Approved on 11/Oct/2021