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The epistemic values at the basis of epidemiology and public health

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Abstract: In the middle of the severest public health crisis of the 21st century thus far, scholars in the humanities and health and social sciences have accelerated their reflections about public health, its evidence base, its normative dimensions, as well as its successes, failures and pitfalls. In this paper, we aim to contribute to this global thinking about public health by focusing on one particular aspect, namely its dependence on epidemiology as the main generator of evidence for public health interventions and policy. Our argument is that while it is undeniable that epidemiology (and its sub-fields) have made very many significant contributions both to enlarge our knowledge of disease and its causation, prevalence and incidence, and to channel public health interventions, it also has its limits. The limitations of epidemiology lie in the (implicit) epistemic assumptions which involve focusing on (aggregates of) individuals, and obscures the key role of the social level of analysis. These limitations, however, are also an opportunity to highlight and rethink a more distinctly social approach to health and disease, and one that is genuinely population in character. Our argument applies broadly, but the COVID-19 pandemic makes it an urgent topic to address.

Keywords: COVID-19; mixed-mechanisms; disease causation; epidemiology; social epidemiology; social-to-biological transition; public health interventions; epistemic values

1. *Introduction*

In recent years a number of philosophers have turned their attention to ethical, epistemological and methodological questions arising from the

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practice of epidemiology and public health[1]. The definition of public health is broad – the science and art of preventing disease, prolonging life, and improving quality of life through organized efforts and informed choices of society, organizations (public and private), communities and individuals[2]. It involves disease prevention, health protection and health education and promotion[3]. This embraces a wide number of disciplines from virology and bacteriology, through the social and behavioural sciences, to management and political and policy science. However, epidemiology and its associated statistical methods, have been pre-eminent in the public health canon. Training in epidemiology is at the heart of public health education and the academic activity called public health. The practice of public health in the field and the community, has to pay close attention to the behavioural, management, and other disciplines, but the dominance of epidemiology in the academy creates a paradigmatic world view, the weakness of which has been revealed in the current COVID-19 pandemic. It has been argued that the science that has guided governments during the pandemic has been driven mostly by epidemic modellers and infectious disease specialists[4]. We suggest that, in the severest public health crisis of the twenty first century thus far[5], one of the reasons why the voice of public health has not been as powerful as it should have been has to do with the epistemic frameworks and evidence base coming from epidemiology.

This is not to propose that epidemiology is unimportant. Quite the contrary. However, we do argue that there is an epidemiological way of

[1] A. Broadbent, *Philosophy of epidemiology*, Palgrave Macmillan, Basingstoke 2013; O. Dammann, *The etiological stance: explaining illness occurrence*, "Perspectives in biology and medicine", 60, 2017, 2, pp. 151-165; A. Dawson, M. Verweij (eds.), *Ethics, prevention, and public health*, Clarendon Press, Oxford-New York 2009; F. Russo, *Causal webs in epidemiology*, "Paradigmi", XXIX, 2011, 1; T. Schramme, *Health as notion in public health*, in *Handbook of the philosophy of medicine*, T. Schramme, S. Edwards (eds.), Springer Netherlands, Dordrecht, pp. 1-10; T. Schramme (ed.), *New perspectives on paternalism and health care*, Springer International Publishing, Switzerland 2015; J. Stegenga, *Medical nihilism*, Oxford University Press, Oxford 2018; D. Teira, J. Reiss, *Causality, impartiality and evidence-based policy*, in *Mechanism and causality in biology and economics*, H.K. Chao, S.T. Chen, Roberta L. Millstein (eds.), Springer Netherlands, Dordrecht 2013, pp. 207-224; S.A. Valles, *Philosophy of population health science: philosophy for a new public health era*, Routledge, New York 2019.

[2] C.E.A. Winslow, *The untilled fields of public health*, "Science", 51, 1920, 1306, pp. 23-33; World Health Organization, *WHO definition of public health*, 2021.

[3] A. Tannahill, *What is health promotion?*, "Health Education Journal", 44, 1985, 4, pp. 167-168.

[4] R. Horton, *Offline: COVID-19 is not a pandemic*, "The Lancet", 396, 2020, 10255, p. 874.

[5] R. Horton, *Offline: a global health crisis? No, something far worse*, "The Lancet", 395, 2020, 10234, p. 1410.

seeing the world, a discourse, which is closely linked (not surprisingly) to mainstream bio-medical science. We need to get inside that way of seeing the world to understand not just some interesting philosophical issues, but also to help to do things better. We think this will also lead to a more policy-friendly approach. We aim to show that epidemiology rests on very specific epistemic assumptions which determine what is (and is not) regarded as admissible knowledge, evidence and proof. These assumptions sideline a population or social perspective proper, reducing them to background and context, rather than part of the explanatory and actionable process. We claim that epidemiology is an *individual-level* science, despite the widespread claims to be population level. We will explain this thoroughly below, but our position is that epidemiology makes measurements of health and disease in *individuals*, and then aggregates them into populations[6]. This may seem an odd, not to say eccentric, statement given the work of Geoffrey Rose[7]. He distinguished between the causes of a case (individual level), and the causes of incidence (the changes in numbers of cases – population or social level). Notwithstanding the seminal position of Rose's work, and the importance of his distinction between the individual risk and population risk, our contention is that the thinking in epidemiology remains locked into an individual-level epistemology. Therefore, and despite the huge knowledge base produced in social epidemiology, we still miss informative and tractable accounts of complex relational interactions and *mixed mechanisms* between the individual and the social and between the biological and social sphere.

The paper is organized as follows. In section 2, we reconstruct the historical development of public health interventions, leading to epidemiology becoming its main evidence base. In section 3, we explain that this evidence base is, contrary to a widespread claim, an aggregate of individual-level observations, thus neglecting the proper population or social dimension of health and disease. This is not to criticize epidemiology, but it is to identify a fertile terrain for exploring the synergies between epidemiology and social science approaches to health and disease – this exploration we undertake in section 4, and we exemplify the potential positive impact on public health interventions in the case of the COVID-19 pandemic.

[6] M.P. Kelly, *The individual and the social level in public health*, in *Evidence based public health: Effectiveness and efficiency*, A. Killoran, M.P. Kelly (eds.), Oxford University Press, Oxford 2009, pp. 425-435.

[7] G. Rose, *The strategy of preventive medicine*, Oxford University Press, Oxford 1992; *Sick individuals and sick populations*, "International Journal of Epidemiology", 30, 2001, 3, pp. 427-432.

2. *A brief historical reconstruction of the evidence base of public health interventions*

Public health is an interdisciplinary field tasked with the design and implementation of interventions, the goal of which is to prevent and reduce the burden of disease, protect and improve health and to reduce health inequalities in populations[8]. Public health interventions may target specific biological processes or behavioural, social, commercial and industrial factors, playing a role in the development and spreading of disease. They may target a population as whole, or specific subgroups, or individuals. The design process of public health interventions does not always follow strict protocols and it may vary depending on the norms of the institutions in charge in different countries. Yet, while there is no single protocol for the design, it is fair to say that the main evidence base of public health interventions is generated by research done in epidemiology. Epidemiology is considered the main generator of the evidence base of public health interventions. It is usually defined as the study of variations in health and disease within and across populations[9]. The field is organized in numerous subfields, focusing on specific aspects of the health and disease, for instance clinical epidemiology, social epidemiology, epidemiology of occupational health, and its most recent addition, i.e. molecular epidemiology.

Public health interventions take many forms, and an historical lens is revealing of the way things have evolved. From the mid-fourteenth century, public health interventions involved efforts to control infections, such as Bubonic Plague (the Black Death). The only option available to authorities over millennia were measures of isolation, quarantine, and various efforts at social control[10]. The idea of contagion is very old, with ref-

[8] R.S. Bhopal, *Concepts of epidemiology: Integrating the ideas, theories, principles, and methods of epidemiology*, Oxford University Press, New York 2016; R.C. Brownson, *Evidence-based public health*, Oxford University Press, Oxford-New York 2003; R.J. Donaldson, *Essential community medicine including relevant social services*, Springer Science & Business Media, 1983; S. Griffiths, D.J. Hunter, *New perspectives in public health*, Radcliff Medical Press, Abindon 2017.

[9] R.C. Brownson, *Evidence-based public health*, cit.; R.J. Donaldson, *Essential community…*, cit.; S.A. Valles, *Philosophy of population health science*, cit.

[10] P.E. Pormann, *The mirror of health: discovering medicine in the golden age of Islam*, Royal College of Physicians, London 2013; P.E. Pormann (ed.), *"Epidemics" in context: Greek commentaries on Hippocrates in the Arabic tradition*, De Gruyter, Berlin 2012; S. Halliday, *The great stink of London: Sir Joseph Bazalgette and the cleansing of the Victorian metropolis*, History Press, New York 2013; M. Susser, Z. Stein, *Eras in epidemiology: the evolution of ideas*, Oxford University Press, Oxford-New York 2009; S. Johnson, *The ghost map: the story of London's most terrifying*

erences in Greek mythology and the Bible. Over time, protective measures changed little. Foucault's account of French responses to epidemics in the seventeenth and eighteenth centuries is interesting because he describes two things happening: first, the control of state (and church) on people's everyday lives, and, second schisms and controversies within the medical profession over different understandings of the disease[11]. The part of the medical profession which gained the upper hand was the one that allied itself with the state, as the state took for itself the right to interfere in the private sphere of people's lives[12].

Infection had been a part of the human condition since time immemorial, but industrialization and urbanization in the nineteenth century seems to have greatly highlighted the problem, forever crystalizing the idea of the patterning of disease with deprivation and poverty[13]. Cholera, typhus, typhoid, scarlet fever, diphtheria, tuberculosis, and influenza killed millions of people and infected many others; it is only by the end of the 19th century, with the eventual understanding of the role of microbes in infection, as well as with improvements in nutrition and general living conditions, that the tide of infectious disease seemed to ebb[14]. Antibiotics, vaccination, improvements in maternal and child health, maternity services and screening programs also provided tools allowing public health to make significant inroads on the profile of disease by the time we reach the 1950[15].

What is sometimes called an 'epidemiological transition' is said to have occurred after the mid-20th century with the gradual diminishing number of deaths from infectious disease (in the Western World) and the gradual increase in the numbers of deaths from non-communicable diseases linked to cigarette smoking, alcohol intake, calorie consumption and physical inactivity[16]. The simple notion of an epidemiological transition is

epidemic – and how it changed science, cities, and the modern world, 2008; A. Kessel, *Air, the environment and public health*, Cambridge University Press, Cambridge-NewYork 2006.

[11] M. Foucault, *The birth of the clinic*, Taylor & Francis, London 1973.

[12] L. McCray Beier, *For their own good: the transformation of English working-class health culture, 1880-1970*, Ohio State University Press, Columbus 2008; A.F. La Berge, *Mission and method: the early nineteenth-century French public health movement*, Cambridge University Press, Cambridge-New York 2002.

[13] W.T. Gairdner, *Public health in relation to air and water*, Edmonston & Douglas, Edinburgh 1862.

[14] T. McKeown, *The role of medicine: Dream, mirage, or nemesis?*, Princeton University Press, Princeton 2014.

[15] L. McCray Beier, *For their own good*, cit.

[16] A.R. Omran, *The epidemiologic transition: a theory of the epidemiology of population change. 1971*, "The Milbank quarterly", 83, 2005, 4, pp. 731-757.

now seen as a gross oversimplification; infectious diseases never went away even in advanced societies, and in developing societies they remained endemic, and famine likewise[17]. Moreover, the simple distinction between infectious and diseases of lifestyle is not itself scientifically robust, as the 'syndemic' approach suggests[18]. However, the findings of early epidemiological studies associating disease with social behaviour and social and economic conditions, remain fundamentally important. Even if there was no epidemiological transition strictly speaking, there was most certainly an epistemological consolidation. The really important epidemiological investigations by Doll and Hill into the causes of lung cancer and other diseases with exposures to cigarette smoking and industrial toxins was a watershed scientifically[19]. Pinpointing particular toxins, like cigarette smoke, allowed for a new way of tackling certain diseases which were all about the identification of risk factors, often linked to behaviour and what is sometimes called lifestyle[20].

There were two elements in the epistemological consolidation. First, those public health reformers who linked disease to social conditions in the 19th century – Chadwick, Snow, Villermé – set out an approach to public health which was social in orientation[21]. Their foci, in various ways, were on social and industrial life and its health damaging consequences. In the European and North American contexts for example, the targets for intervention were frequently local municipalities, slum clearance, housing and sanitation. However, the star of the social approach faded towards the end of the century, with the various discoveries of Pas-

[17] A.J. Mercer, *Updating the epidemiological transition model*, "Epidemiology and Infection", 146, 2018, 6, pp. 680-687; A. Mercer, *Infections, chronic disease, and the epidemiological transition: a new perspective*, University of Rochester Press, Rochester 2014; M. Vaughan, K. Adjaye-Gbewonyo, M. Mika (eds.), *Epidemiological change and chronic disease in sub-saharan Africa: social and historical perspectives*, UCL Press, London 2021.

[18] M. Singer, *Introduction to syndemics: a critical systems approach to public and community health*, Jossey-Bass, San Francisco 2013; M. Singer *et al.*, *Syndemics and the biosocial conception of health*, "The Lancet", 389, 2017, 10072, pp. 941-950.

[19] R. Doll, *Mortality from lung cancer in asbestos workers*, "British Journal of Industrial Medicine", 1955, 12, pp. 81-86; R. Doll, A.B. Hill, *Smoking and carcinoma of the lung*, "BMJ", 2, 1950, 4682, pp. 739-748; R. Doll, A.B. Hill, *Study of the aetiology of carcinoma of the lung*, "BMJ", 2, 1952, 4797, pp. 1271-1286; R. Doll, A.B. Hill, *Mortality in relation to smoking: Ten years' observations of British doctors*, "BMJ", 1, 1964, 5396, pp. 1460-1467.

[20] T.R. Dawber, W.B. Kannel, *The Framingham study: An epidemiological approach to coronary heart disease*, "Circulation", 34, 1966, 4, pp. 553-555; P.A. Sytkowski *et al.*, *Sex and time trends in cardiovascular disease incidence and mortality: The Framingham heart study, 1950-1989*, "American Journal of Epidemiology", 143, 1996, 4, pp. 338-350.

[21] S.W.P. Chave, *John Snow. The broad street pump and after*, "The Medical Officer 99", 1958, pp. 347-349.

teur, Lister and Koch and specifically of germs and microbes. The demonstrable efficaciousness of protecting people from bacteria by providing clean water and good sanitation, as well as the ability to target the microbes in various ways, shifted the focus down from the social to the individual. Second, the work of Doll and Hill and the identification of cigarette smoke and asbestos as causes of disease to all intents and purposes treats the toxins like microbes. Toxins precede pathology just like viruses and bacteria, and reducing people's exposure has proved effective. The focus has been on the individual, quintessentially with remonstrations to stop smoking as the most effective way to avoid (individual) exposure to cigarette smoke. All the various risk factors which have since been identified for non-communicable disease likewise find their principal vector in the individual's behaviour, while they should *also* be located at the level of their social environment[22].

During the 20th century, therefore, epidemiology has brought to light some of the most important medical discoveries the application of which has had enormous benefits to the health of the population. The understanding of the origins of many diseases owes a huge amount to epidemiological investigation. The role of cigarette smoking in lung cancer and heart disease, the discovery of the role of diet, exercise and alcohol consumption in early mortality and the dementias, the unravelling the pathways to infection in HIV and AIDS as well as earlier observations about diseases with their origins in industrial toxins like asbestos, lead and phosphorus, and more generally the links between poverty, deprivation and disadvantage and ill health and early death, all owe a large debt to epidemiological science. The epistemological consolidation is complete, with epidemiology the handmaiden of this shift. But this very success has helped to conceal some important problems, as we explain next.

3. *Epidemiology as science of health and disease of individuals*

Despite the widespread claim that epidemiology is a population science, we contend that it is in fact a science of the individual, but at an *aggregate* level: it adds up all the individual cases and, in this way, it manages to create a solid evidence base about health and disease in populations, meant as aggregates of individuals. However, epidemiology does not conduct its analysis at the level of population or of society, in the sense of so-

22 P.A. Sytkowski *et al.*, *Sex and time trends*, cit.

cial groups with proper and distinct social dynamics, which would constitute a level of explanation and analysis in its own right. It is in this sense that we claim that epidemiology remains largely individual and non-relational. The dynamic and relational interaction between human agency and social structure, and the emergent social practices in which people engage, is almost entirely invisible in the epidemiological paradigm, while it has been described in detail in sociology[23].

This is all the more true in the era of *molecular* epidemiology[24]. Molecular epidemiology is in fact the latest frontier in the field of epidemiology. It tries to understand the development of disease as part of a broader understanding of exposure: we are exposed to hazards, outside our bodies, but then this exposure continues inside the body too; it is the *total* exposure that we need to study in order to understand disease onset and development[25]. A molecular approach to exposure will help understand – at the biological and biochemical level – exactly how exposure works. This approach has given us invaluable understanding about the onset and development of health and disease, and yet, there have been controversies about its legitimate place within epidemiology. To begin with, in the first days of molecular epidemiology, some raised the concern that shifting the unit investigation down to the molecular level, effectively meant betraying the inherent principles of epidemiology, namely to study individuals not populations. The danger, as we see it, is that shifting the unit of analysis down at the molecular level paves the way to reductionist accounts of health and disease. As the field has advanced and managed to

[23] P. Bourdieu, *Pascalian meditations*, Stanford University Press, Stanford 2000; Id., *The logic of practice*, Stanford University Press, Stanford 2008; A. Giddens, *Central problems in social theory: action, structure, and contradiction in social analysis*, University of California Press, Berkeley 1979; A. Giddens, F.R. Dallmayr, *Profiles and critiques in social theory*, University of California Press, Berkeley 1982; A. Giddens, *The constitution of society: outline of the theory of structuration*, University of California Press, Berkeley 1986.

[24] F.P. Perera, I.B. Weinstein, *Molecular epidemiology and carcinogen-DNA adduct detection: New approaches to studies of human cancer causation*, "Journal of Chronic Diseases", 35, 1982, 7, pp. 581-600; E.J. Radford, *Exploring the extent and scope of epigenetic inheritance*, "Nature Reviews Endocrinology", 14, 2018, 6, pp. 345-355; P.A. Schulte, F.P. Perera, *Molecular epidemiology: Principles and practices*, Elsevier Science, Saint Louis 2012.

[25] G.M. Slavich, S.W. Cole, *The emerging field of human social genomics*, "Clinical Psychological Science", 1, 2013, 3, pp. 331-348; G.M. Slavich, M.R. Irwin, *From stress to inflammation and major depressive disorder: A social signal transduction theory of depression*, "Psychological Bulletin", 140, 2014, 3, pp. 774-815; C.P. Wild, *Complementing the genome with an "exposome": The outstanding challenge of environmental exposure measurement in molecular epidemiology*, "Cancer Epidemiology Biomarkers & Prevention", 14, 2005, 8, pp. 1847-1850; C. Wild, S. Garte, P. Vineis, *Molecular epidemiology of chronic diseases*, Wiley, Hoboken 2013.

generate groundbreaking, new knowledge, the problem of the unit of analysis has arisen again. In fact, no matter how well we understand the biology and bio-chemistry of health and disease, from a public health perspective, molecules and biomarkers are hardly 'actionable'. The next challenge of molecular epidemiology is therefore to climb up the ladder again and relate the biology and bio-chemistry to the social dimension of health and disease. It is in fact at this level we can find actionable factors.

To be sure, this is a direction taken in the latest projects in molecular epidemiology, for instance *Lifepath* (https://www.lifepathproject.eu), in which biochemical knowledge about health and disease is studied and contextualized in a life-course approach. A life-course approach, in turn, is not only open to, but essentially includes information about socio-economic factors through the whole life of individuals[26]. In this approach, specifically, recent attempts to understand the 'social to biological transition', or how social environments may act on the biology, investigate, for instance, the mechanisms of epigenetics and the role of allostatic load[27]. Notwithstanding the importance, relevance, and timeliness of this line of research, the 'social' is studied for its possible causal effect on the 'biological', but there are in fact many other relations that would be of relevance: from the biological to the social, and any other path in between and across (i) individual level and group level, and (ii) social, psychological, political, cultural factors and the biological factors proper. Thus it would be important to study, for instance, how certain medical conditions influence one's life, and depending on the different socio-economic environment one lives in, or the different health infrastructure one has access to.

The contribution of epidemiology to establishing important evidence base for public health has been great well before epidemiology became more 'experimental' and oriented towards the biochemistry of health and

[26] M. Kelly-Irving, S. Tophoven, D. Blane, *Life course research: new opportunities for establishing social and biological plausibility*, "International Journal of Public Health", 60, 2015, 6, pp. 629-635; A. Sacker *et al.*, *Never too early, never too late: social and biological influences on health and disease over the lifecourse*, 2016.

[27] R. Castagné *et al.*, *Allostatic load and subsequent all-cause mortality: which biological markers drive the relationship? Findings from a UK birth cohort*, "European journal of epidemiology", 33, 2018, 5, pp. 441-458; D. Blane *et al.*, *Social-biological transitions: how does the social become biological?*, "Longitudinal and Life Course Studies; Vol. 4, No. 2, 2013: Longitudinal and Life Course Studies; M. Lock, *Comprehending the body in the era of the epigenome*, "Current Anthropology", 56, 2015, 2, pp. 151-177; M. Meloni, *The social brain meets the reactive genome: neuroscience, epigenetics and the new social biology*, "Frontiers in Human Neuroscience", 8, 2014.

disease. In fact, its proper observational method, as exemplified by the Bradford Hill guidelines[28] and through the analysis of evidence as is done in institutions such as NICE or IARC, are exemplars of solid scientific method, despite regular attacks on it as being 'junk science'[29]. Our line of argument is *not* that epidemiology is junk science. Instead, we aim to shed light on how the largest evidence base for public health interventions is about the biochemistry of health and disease and about *individual* factors of many kinds. Defenders of epidemiology will rebut and argue that social epidemiologists *do* study the role of social factors. But these defences actually miss the point: the study of social factors still happens at the individual level, which is then aggregated into populations.

It is in this sense that we claim that a main epistemic value at work in epidemiology is the reliance on the 'individual' as the unit of analysis. We think that *more* is needed: detailing the *mixed mechanisms* of health and disease that happen not only across social and biological factors, but also in the relational social dynamics across individuals and groups. Below we will illustrate how we think this populational and social perspective can be added to epidemiology. But before we proceed, let us turn to COVID-19 as the latest prominent case in which the individual is at the centre of the analysis and of many interventions that have been put in place.

The global response to COVID-19 has been twofold. First, to prevent the spread of infection and second to develop vaccines. This is logical and, in the circumstances, the best and probably only options initially available. Progress on vaccines has been encouraging. However, efforts to enforce social restrictions to prevent spread, markedly less so. Those jurisdictions which experienced SARs in the early part of the twenty first century were very swift to institute very strict, to total lockdowns, involving major limitations of civil liberties. In the UK, most of Europe, and North and Latin America, such action was much less restrictive. Speed was clearly important, but in varying ways the restrictions in the Global West have not been sufficient to curb infections quickly. If measures can be introduced which effectively stop the spread, then since the virus needs a continuous series of human bodies to infect in order to survive, without that chain, it would be contained. There has been much

[28] A.B. Hill, *The environment and disease: Association or causation?*, "Proceedings of the Royal Society of Medicine", 58, 1965, 5, pp. 295-300.

[29] P. Boffetta *et al.*, *False-positive results in cancer epidemiology: A plea for epistemological modesty*, "JNCI: Journal of the National Cancer Institute", 100, 2008, 14, pp. 988-995; P. Vineis, *Viewpoint: The skeptical epidemiologist*, "International Journal of Epidemiology", 38, 2009, 3, pp. 675-677.

criticism of governmental action in the West in this regard. But this is not our argument.

For us, the more important point relates to those members of the community who were most prone to serious infection and to dying from the virus. There was a clear association between social factors and disease severity and risk of death. Early on, the data made clear that the most vulnerable to serious infection and mortality from COVID-19 were the socially disadvantaged, black and minority ethnic groups, men, certain occupational groups (mostly those involving close contact with the public and many of which are low paid and low skilled) and the elderly[30]. Another important early finding was that those who suffered the most severe infections were likely to already have a preceding medical condition, such as type 2 diabetes, coronary heart disease, obesity, and dementia. As early as 2003, there were already papers in the peer reviewed literature highlighting the interaction between these conditions and coronaviruses[31]. The two biological pathologies – the pre-existing disease and the virus – interact with each other, to greatly exacerbate the disease process. The data also show that there is a third party involved in the interaction – social position and specifically social disadvantage. The epidemiology has been particularly good at describing the virus' pathological associations with existing disease[32], and the biological processes are readily hypothesized[33].

However, the nature of the interaction with the third dimension – the social – while clear in the associational data, remains undescribed in explicit mechanistic or causal terms, as instead suggested by advocators of evidential pluralism[34]. The nature of the three-way interaction, the connection between the biological and the social remains a significant gap in the evidence. This is an important research area, we would contend, and one that, in the event of a future pandemic, we ought to be ready for. It

[30] M.G. Marmot *et al.*, *Build back fairer: The COVID-19 Marmot review. The pandemic, socioeconomic and health inequalities in England*, 2020.

[31] M. Singer, *Introduction to Syndemics: A Critical Systems Approach to Public and Community Health*, Jossey-Bass, San Francisco 2009.

[32] M.G. Marmot *et al.*, *Build back fairer*, cit.

[33] K. Suzuki, *The developing world of DOHaD*, "Journal of Developmental Origins of Health and Disease", 9, 2018, 3, pp. 266-269.

[34] M.P. Kelly, F. Russo, *Causal narratives in public health: the difference between mechanisms of aetiology and mechanisms of prevention in non-communicable diseases*, "Sociology of Health & Illness", 2017; V.-P. Parkkinen *et al.*, *Evaluating evidence of mechanisms in medicine: Principles and procedures*, Springer International Publishing, Cham 2018; F. Russo, *Public health policy, evidence, and causation: lessons from the studies on obesity*, "Medicine, Health Care and Philosophy", 15, 2012, 2, pp. 141-151.

requires a full elaboration of the mixed mechanisms at work, socially and individually. In practical terms, special measures to protect the especially vulnerable and susceptible *could* have been employed, but in fact they were not. Instead, the potentially most vulnerable were mostly the ones who had to continue to work as drivers, carers, warehouse operatives, as well as front line nursing and medical staff. The latter of course are not normally thought of as socially disadvantaged (though many front-line care staff are among the lowest paid), but nevertheless they were subject to greater exposure.

If we reflect on why the third social element is absent, other than in associational terms, the answer, we suggest, is that the social operates in a different epistemic frame. It is about relationships between individuals and groups, it is about what they do and how they spend their time, it operates in different epistemological universe to epidemiology, with its focus on the individual in biomedicine. It is not uncommon to find the social being articulated in medical investigations, but it is seldom foregrounded and usually plays a role as context, or in epidemiology variables like age, gender, occupation are used to test for confounding. There is in other words and epistemic divide.

This is an old problem. The pioneers of public health reform in the nineteenth century, such as Gairdner, Snow, Duncan and Villermé, had a very clear understanding that the social environments in which people lived profoundly influenced the individual cases as well as the patterns of disease, which they observed. They didn't know how it worked. And arguably we still don't, at least with enough precision to intervene effectively. Amongst other things, contemporary epigenetics and metabolomics underpins an urgent need to explore these mechanisms, and not just in the case of COVID-19. Contemporarily, there are competing explanations of the biological pathways explaining epigenetic and metabolomic actions. The broad argument however – that there is a clear and mechanistic pathway from social exposures to biological manifestations in the body – is not in doubt, but what it actually consists of *is* in doubt. Just imagine if in this pandemic these mixed mechanistic pathways had been known, or even hypothesized, how much better public health interventions might have been.

4. *What else does (social) epidemiology need?*

Our argument so far is *not* that epidemiology has not studied social factors and socio-economic determinants of health. Quite the contrary:

social epidemiology has done a stunning job in documenting that socio-economic determinants are correlated with different health outcomes and with health inequalities. Some key references will suffice to establish the relevance and importance of this field[35]. Our point is that, despite social epidemiology being part of knowledge base of public health interventions, arguably, (i) social factors are not really targeted in far too many interventions and (ii), we still lack the 'why' of the well-established correlations of social epidemiology.

We contend that the root cause of these two shortcomings lies in the individual-level epistemology of (social) epidemiology described in section 3. What (social) epidemiology does not provide is information about group dynamics, contexts, behaviour and social practices, which can be very local, or very specific to particular groups. It is there, implicitly in much of the work, but it remains obscured by a dominant epistemic viewpoint.

One may rebut that this is exactly what medical sociology studies, and in great detail: this field has extensively explored how socio-economic structures influence the concept of disease, or how power structures interfere in the (good) delivery of health services etc.[36] But medical sociology is part of the evidence base of public health intervention only to a limited degree. This is in part because the social dynamics described and studied in medical sociology are often not about the *mixed mechanisms* of health and disease, in which *both* social and biological factors play a role, and that we think would be the key to improve significantly on the knowledge base of public health.

Thus, for instance, over and above collecting statistics about COVID-19 infections and deaths per age group, we need a lot more information about the key social mechanisms *and* thick descriptions of the dynamic and relational conditions in which the virus spread. What kind of social practices and material conditions facilitate infection in different home and working conditions? Or in schools? Or in places of worship? Or in households with very different age groups? Why can we allow up to X number of people per square meter in a bus but Y in a shop? Detail-

[35] M. Bartley, *Health inequality: an introduction to concepts, theories and methods*, Polity, Malden 2017; C. Bambra, *Health divides: where you live can kill you*, Policy Press, Bristol-Chicago 2016; M. Marmot, *Social determinants of health inequalities*, "The Lancet", 365, 2005, 9464, pp. 1099-1104; M. Marmot, *Fair society, healthy lives: Strategic review of health inequalities in England post 2010*, 2010.

[36] E. Annandale, *The sociology of health and medicine: a critical introduction*, Polity, Malden 2014.

ing the socio-biological mechanisms at work in different contexts will help immensely with designing tailor-made preventive measures for different social groups, and also to effectively communicate to the public. But this requires an important shift in the values driving research, re-habilitating qualitative and small-scale social science research to enrich the knowledge base of public health.

The description of the mixed mechanisms of health and disease is however not an easy task. *Methodologically*, this would require integrating standard, quantitative-oriented social epidemiology approaches with qualitative, small-scale studies in social science[37]. *Conceptually*, this would require adopting the notion of 'mixed mechanism'. This means (i) that mechanisms are not reduced / reduceable to biology, (ii) that social factors are *causal* factors on par with biological ones, and (iii) that social and biological factors interact in some way to be detailed and described. Relatedly, this also means adopting a broader view on causation, in which we allow causal relation to happen across levels (individual-group) and across different types of factors (biological-social)[38].

A different methodological and conceptual approach is needed because public health interventions are more likely to succeed when tailored to specific groups, contexts, or environments. Many measures did not have enough policy traction during the pandemic because they were too vague and high level, instead of being local. We also think that the rationale behind local and tailored interventions should take advantage of a clearer and more effective science of communication, hopefully increasing compliance with safer conduct.

For instance, as we mentioned earlier, a general lockdown aims at reducing infection rates in the overall population, but does not actually protect the most vulnerable groups, taking advantage of specific social dynamics proper to these groups. Selected closure of e.g. pubs and restaurants is instead targeting specific groups of the population, where infection risk may not have been uniformly very great. The reason to forbid gatherings after say 6pm is not that the virus is more infectious in the evening, but because *specific social dynamics* happen at that time of the

[37] F. Russo, M.P. Kelly, *The 'lifeworld' of health and disease and the design of public health interventions*, 2020.

[38] P. Illari, F. Russo, *Information channels and biomarkers of disease*, "Topoi", 35, 2016, 1, pp. 175-190; M.P. Kelly, R.S. Kelly, F. Russo, *The integration of social, behavioral, and biological mechanisms in models of pathogenesis*, "Perspectives in Biology and Medicine", 57, 2014, 3, pp. 308-328; M.P. Kelly, F. Russo, *Causal narratives in public health…*, cit.; F. Russo, M.P. Kelly, *The 'lifeworld' of health and disease…*, cit.

day and within specific groups. And the choice to close some places (e.g. pubs) rather than others (e.g. schools) does not necessarily have a scientific base (only), but is also a question of priorities at the ethical level and/or of social values. The tailoring of public health measures to contain a pandemic such as COVID-19 would have had a better chance if we had more detailed understanding of the mixed mechanisms of health and disease, because these would contain many more actionable factors, tailored to specific environments, groups and their dynamics.

5. *Conclusion*

COVID-19 is the severest public health crisis of the 21st Century thus far. The pandemic has (yet again) shown us how much we are vulnerable, despite the very advanced level of our best bio-medical and epidemiological knowledge. Isolating the virus did not take that long. But the spread of infection is not just a matter of biology – it is also, if not foremost, a matter of dynamic and relational social practices and behaviours.

Reflecting on the knowledge base of public health measures against COVID-19 helps us restate our arguments even more forcefully. Public health interventions have better chance of success if the social factors are analysed properly. Work in this direction exists[39], and we should further contribute to it by complementing the individual-level epistemology of (social) epidemiology with population-level epistemology that describes social dynamics as part of the knowledge base of public health interventions.

[39] N.M. Kriznik *et al.*, *Moving beyond individual choice in policies to reduce health inequalities: The integration of dynamic with individual explanations*, "Journal of Public Health", 40, 2018, 4, pp. 764-775.