The 'lifeworld' of health and disease and the design of public health interventions

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Abstract

In the middle the COVID-19 pandemic, we reflect ever more deeply about the nature of health and disease, and about how to design and implement effective public health interventions. For numerous diseases and conditions, as well as for COVID-19, our knowledge base is rich. We know a lot about the biology of the disease, and we have plenty of statistics that relate health to socio-economic factors. In this paper, we argue that we need to add a third dimension to this knowledge base, namely a thorough description of the lifeworld of health and disease, and of the mixed bio-social mechanisms that operate in it. We present the concepts of lifeworld and of mixed mechanisms, and then illustrate how they can be operationalized and measured. Finally, we explain their complementarity with the biological and statistical dimensions of health and disease for the design of public health interventions.

Prelude: the biology, statistics, and sociology of COVID-19

At the time of writing, we are (still) in the middle of the severest public health crisis of the 21st Century thus far. The COVID-19 pandemic has caused many deaths globally, has disrupted economies and trade word-wide and has become a dominant political as well as a medical problem in many countries as they struggle to contain the virus. The WHO declared a pandemic in early 2020. The immediate response in some societies was to lock down very hard. Others, notably in the West, were much slower to act to contain the spread of infection by strict social controls. A remarkable success was the very rapid genetic sequencing of the virus, which opened the way for the development of vaccines at pace and on a scale that has been unique in drug development.

In spite of the sequencing, much remains to be discovered about the virus. It affects some people very severely, others remain asymptomatic; its methods of transmission are variable – including droplets, aerosol, and touch. It has been both more common and more deadly in some sections of society than others; and, its mutations seem to be more virulent than the original. Novel therapeutics have also emerged, but there remain significant gaps in the treatment armoury.

The epidemiological data about COVID-19 show that some social groups are at greater risk of severe infection and mortality than others. These are the black and minority ethnic community, the relatively poor and disadvantaged, men, certain

occupational groups, people with some pre-existing medical conditions and the elderly. There is a clear association across different jurisdictions between COVID disease severity and death and social factors (M. G. Marmot et al. 2020; SAGE 2020; Bhaskar et al. 2020).

The sociology of COVID-19 has been less prominent than the virology and epidemiology as the pandemic has proceeded. However, the fact that social groups have been so differentially affected, and that the patterning of death has followed closely the contours of existing health inequalities, invites a more considered response, than mere admonitions to do something about the death toll. What are the features of the practices that are embedded in cultural and economic distinctiveness? Or that are at the root of the observed differences? The social practices, which make up the lifeworlds of ordinary people, provide a potential clue to the patterning, which has been so visible. The social spread of infection is a function of people's abilities to shape their own lives and to control their lifeworlds. These dynamics interact with biology, but we contend that it is vital to understand that interaction to elucidate the mechanisms of the spread of the infection. We must get inside the lifeworlds of the various sub-groups in the population. We use the concept of lifeworld to explore this. The concept of the lifeworld allows us to identify the physical, social and subjective space where the real time experience of recursive social practices which shape people's lives in a general sense, but also in the very specific ways that interact with human biology and hence on health and disease. If we want to tease out the mechanisms linking social and biological life, we can find them in people's lifeworlds, as we explain in the paper.

1 The bio-social dimension of health and disease

Health and disease are neither solely or merely biological phenomena. Health and disease are intrinsically social *and* biological and therefore their conceptualisation and explanation should incorporate social context, factors, and practices, as well as biomedicine. In order to do this, an appropriate method is needed to get beyond the conventions of biomedicine and the epistemic divide between biological and sociological accounts of the world. The idea that the biological and social realms are linked is a very ancient one (Meloni 2019). None the less, the practice of both contemporary clinical and public health medicine could be enhanced by a greater recognition of this idea and a more explicit integration of the two. This in turn would assist in the implementation of more effective public health measures, and would have helped enormously in the COVID pandemic.

To realise this ambition requires going beyond conceptualising the social as 'context' or 'background', and instead recognizing that the two spheres - biology and society - interact and are intrinsic to one another. This is difficult because on the one hand, medical science has made considerable progress in measuring, describing, and curing disease, by elucidating mechanisms down to the molecular and sub-molecular level. Biomedicine is demonstrably successful in very many spheres, and without much help from the social sciences. On the other hand, the social sciences, with a few exceptions (Warin et al. 2015), have not really addressed the 'social' and 'biological' as *integrated processes* and have often tended to eschew the biological altogether. We seek to bridge the divide.

If there is to be integration, both sets of disciplines need to seek consilience and we contend that the mixed 'socio-biological' mechanisms that happen in the lifeworld of individuals provide an avenue to do this, and that the methods to achieve this – mixed methods- are already available. Mixed methods combine qualitative and quantitative approaches, but we additionally argue that these have to be focused on what we conceptualise as the lifeworld. We further suggest that such an endeavour potentially could contribute to better policy design and implementation in public health.

In this paper, we make an attempt to contribute to the design of public health interventions by explaining how the concept of lifeworld can be made operational and actionable. We proceed as follows. In section two we present the concept of life-world and contrast it with sociological accounts of structure which do not deal with mechanisms. In section 3, we operationalize the concept of lifeworld. Our proposal is two-fold. On the one hand, we need better to understand the import of the concept of lifeworlds on the phenomena we may wish to consider and measure. On the other, we need to go beyond the qualitative-quantitative divide and synergistically use both approaches to operationalize and measure it. By adopting a mixed-methods frame of reference, we suggest we may properly study and detail the bio-social mechanisms of health and disease that operate in the lifewordl. In section 4, we discuss the complementarity of our approach with leading accounts in the field, notably biologically and aetiologically-oriented ones. Evidential pluralism, we finally argue, is what allows us to build a bridge between the social and the biological spheres of health and disease.

2. The sociology of health and disease

2.1 The concept of 'lifeworld' and the mixed mechanisms of health and disease

The concept of lifeworld is central to our argument and for this reason we present it first. Our approach is derived, but distinct, from the work of Alfred Schutz (1972; 1975). We also draw on the social theory of Anthony Giddens (see Giddens (1979); Giddens and Dallmayr (1982); Giddens (1986)), and Pierre Bourdieu (2000; 2008). The basic idea is that each and every one of us inhabits our own lifeworld. It is deeply subjective in the sense that it is constituted of the assumptions, understandings, and taken for granted aspects of our everyday existence. It is the seat of our sense of self, and the ideas we have about who and what we are, and who and what others are. We anticipate the actions of others and we anticipate the effects that our actions will have on others in our lifeworld. But the lifeworld also consists of the things we do, the actions we take, the practices in which we engage on a day to day basis. It is what we do and our bodies are like a book in which we inscribe all the many things we do and we experience. Any aspect of our life can be described in terms of the lifeworld experience, including health and disease. For health and disease, in particular, our point is that all this information is crucial in order to decide what factor to intervene upon,

whether to target specific individuals, groups, or the whole population, and to identify the most suitable media to promote an intervention.

It is important to note that our individual lifeworlds abut with others' lifeworlds, particularly others who share the same or similar life circumstances to us, and share our placement in the social world. Our practices, our actions, have consequences at a supra-individual level. While we engage in practices in our own apparently singular lifeworlds, simultaneously billions of other people are likewise engaged in practices. Social patterning at population and community level arises from the intersecting practices of different social groups producing the rich variegation in the social world. In the context of public health, this explains why one size does not fit all, and why some interventions will be more effective if targeting specific groups, or the whole population. The aggregate consequence of billions of people engaged in trillions of practices, is not random chaos; rather, it produces patterns at the social level. It is by detailing these patterns that we can intercept the social practices that are likely to influence health in the intended way.

But there is more. Actions have health consequences. The patterns of people's lives, their lifeworld across time - the life course - will inscribe on the body, or get under the skin not just metaphorically, but biologically through human 'omics' as a timeline of our life (Kelly and Kelly 2018). To understand this, we need to supplement the concept of lifeworld with that of mixed mechanism, in which biological and social factors are on a par to explain health and disease, and on which to intervene. Biological factors may have an effect on social factors; social factors may have an effect on biological factors; and the two are *de facto* interacting all the time. Ongoing research in exposure science, epigenetics, allostatic load, and the life-course approach to the 'social-to-biological transition' tries to detail aspects of these mixed mechanisms (Delpierre et al. 2016; Kelly-Irving, Tophoven, and Blane 2015).

It is precisely because the mechanisms of health and disease are mixed, that some public health interventions should target social factors in early life, even if the health outcome is expected much later in the life course. Some people have greater ability and resources to control their lifeworlds across the life course, because of access to resources, power and so on, but all of us are the product of the recursive nature of the timelines of our lives and we suggest that the mechanisms that bridge the body and the social sphere are to be located in the recursive nature of the practices in which we engage, or in which we are enmeshed.

Once we agree that the concepts of lifeworld and of mixed mechanisms hold a potential to explain the phenomena of health and disease in their many facets, namely as biological *and* social phenomena and as individual- *and* population-level phenomena, then the next challenge is how to make them actionable, rather than just explanatory. And to make them actionable, lifeworld and mixed mechanisms need detailing and quantification. In other words, these lifeworlds can be detailed, operationalized, and measured with the concept of mixed mechanism. Our constructive proposal proposes a synergistic use of qualitative

and quantitative methods in the sociology of health. This combined methodology will, in turn, allow us to appropriately incorporate the social dimension in the understanding of the mixed mechanisms of health and disease and in the design of policy intervention (see Greenhalgh (2020)). Before we explain our proposed methodology, it is important to further qualify the concepts of lifeworld and mixed mechanisms with respect to some existing, similar approaches in the sociology of health.

2.2 Structures without mechanisms

Over the years, sociological theories and methods applied to medicine have developed approaches close to the idea of lifeworld sketched above. These approaches have paid attention to the role of institutions, social support, power structures and communication. These are all important elements for a proper understanding of the social dimension of health. These sociological approaches have not generally been dominated by quantitative analyses. They have tended towards the role of these elements at a high level of abstraction – social structures or systems. These are all very important, but not very actionable for the purpose of the design of public health interventions.

Knudsen and Vogd (2015), for instance, revive and develop the system theory approach. At its basis we find the concept of 'polycontexturality'; this means studying health/ patients from different perspectives (of the patient, of the doctor, of the health care provider, etc.). A system theory approach (especially the one of Niklas Luhmann) is then applied to the sociology of health and illness. Here, various aspects of the many 'contextures' involved in health, how they are arranged, and how they interact are considered. The main focus is communication as a fundamental element of society (and therefore of medical social systems too). Communication is used to shed light on decision-making processes, which may involve science-based decisions about diagnosis or treatment as well as the economic and financial aspects of health care management. In this way one can, for instance, account for the tensions between the (scientific) principles of evidence-based medicine and their implementation in health care management.

The main difference with the lifeworld and mixed mechanism approach of section 2.1 is the following. The tendency in the structure or systems approach is to obscure or deliberately eschew the specific *explanatory power* of the bio-social mechanisms affecting health, and their usefulness for policy design or intervention effectiveness. Individual and group behaviour (including preferences, choices and values) become somehow secondary to higher-level sociological structures and while we grasp – at this higher level – the extent to which medicine and medical practice are fundamental social activities, we might miss more concrete, down-to-earth explanations of how and why certain socio-economic factors impact on health and disease.

In sum, while much potentially important policy and practice data and evidence has emerged, what is missing in this literature is that the *mixed mechanisms* underlying the phenomena of health and disease remain largely undefined. This is still more surprising because since the 1980s there has been a vibrant sub field, which emerged in medical sociology, about precisely the sociology of the body

(Turner 1992; Shilling 1993/2012). Once again, an important and dynamic literature, but one that, in our view, paradoxically has not established a bridge with the fields studying the biological sphere. Our view is that a system approach is useful to the extent that the role of high-level social structures is also explained in terms of the mixed mechanisms in which health and disease happen, at the individual and group level. In the next section, we attempt to explain how this can be done.

3. The operationalization of the lifeworld and mixed mechanisms

In order to detail, quantify, and measure – in one word, *operationalize* – the lifeworld and mixed mechanisms, we do not need a brand-new methodological approach. Instead, to do so we need to integrate synergistically what already exists. In this section, we explain how, in our view, we can operationalize lifeworld and mixed mechanisms, using both quantitative and qualitative methods.

3.1 What to measure and how to measure it

The lifeworld of health and disease is very much about social factors and their role in health and disease of individuals and groups. But the problem is that the level of description of lifeworld does not lend itself easily to the quantification of these factors. The temptation is therefore to default to statistical approaches which take a social factor (education, socio-economic status, access to health infrastructure), and measure it as precisely as possible. However, we don't think this solves the problem. We call this 'the problem of more measurement': increasing the granularity of measurement of social factors doesn't carry explanatory power on its own. Our solution does not rely on quantity but on quality. In a nutshell, we need to consider more closely which (social) variables to measure and to specify what aspects of health and disease these variables are in fact supposed to measure or be proxies for. Providing theoretical underpinning to measurement of the social hitherto, provides partial descriptions of the lifeworlds in which mixed mechanisms operate. Let us explain further.

It is not a new idea that measurement in social science is complex and challenging. There is a well-established tradition in social science that reflects on how to measure social characteristics (Blalock 1989; Pawson 1989; Zeller and Carmines 1980). This section builds on this tradition, drawing specific attention to the following. There are variables / factors that are easy to measure but problematic to interpret (e.g., age), and there are variables / factors that are difficult to define but then apparently easier to measure (e.g., socio-economic status). In trying to establish a connection between 'the social' and 'the biological' dimension of health and disease, within a complex mixed mechanisms approach, difficulties and challenges in measurement exacerbate. There are also some aspects of social life where measurement is extremely problematic, and the question arises whether dynamic social processes are amenable to conceptualisation as variables at all (Blumer 1962). There is also the risk that conceptualising things mechanistically resurrects naïve positivistic attempts to apply some of the methods of the natural sciences to the social world – this is not the line of reasoning we advocate.

Take the example of age. Nowadays, chronological age is very easy to measure, especially in Western countries, where demographic registries are long established and properly working. Age may provide very useful information to classify / stratify the population according to given socio-economic-demographic characteristics to be mapped onto health conditions (and vice-versa). But what kind of information is age really giving? This is where a quantitative-oriented approach to social measurement needs to be complemented with more qualitative-oriented approaches that go into the details of habits, culture, and behaviour – basically all key aspects of a lifeworld. Is it the same to be 15 years-old in industrialised Western countries and in rural developing countries? It certainly isn't. Thus, an appropriate description of the lifeworld should be able to make this distinction visible and meaningful, exploiting not only the 'quantitative' information about age (easy to retrieve), but also the 'qualitative' information about what it means to have a given age in a given culture, context, and environment. This qualitative information would constitute the kernel of the lifeworld, providing the details of the mechanisms through which the social operates to affect health.

The quantitative measurement of chronological age carries another problem. The rate at which we age biologically is determined not by our chronological age but by the interaction of our biological inheritance with the experiences of exposures to microbiological, toxicological and traumatic insults across the life course as well as social exposures including, diet, nutrition, and broader environmental and socio-economic phenomena. The impact of these factors is strongly patterned by social class, ethnicity, education, income and gender. Thus, although chronological age has a deceptively easy and quantifiable character, its meaningfulness, as a way of understanding the social dimension of health and disease, is highly contingent. This becomes clear when considering current research done in molecular epidemiology and epigenetics, where the concept of 'allostatic load' is meant to capture a 'biological' state of the body that is caused by a number of stressors, from the environment to important events in life or continuous stressful situations (see e.g. Delpierre et al. (2016); Vineis and Russo (2018)).

Conversely, numerous socio-economic variables and factors are difficult to measure and yet their effect on health is known to be pervasive. The challenge is then to explain the mechanisms through which given socio-economic conditions get under the skin. Numerous studies exemplify this point. For instance, Case and Deaton (2015) ran a thorough analysis of midlife mortality and morbidity in non-Hispanic Americans in the 21st century. In their work, they clearly relate the rise of mortality in the said group to a rise in (self-reported) morbidity. Their analysis includes aspects of the lifeworld of the said group that while are amenable to a quantitative assessment (e.g., how much respondents were able to walk, whether they could socialize with friends) require deep explanation of the social dynamics and how these directly affect health.

We suggest that neither a quantitative nor a qualitative approach can on its own, provide us with an explanation for how and why the social turns into something biologically visible and quantifiable and real. Measuring the social is an important

exercise in quantification that has delivered many useful results and that needs to be further pursued. Ghiara and Russo (2019a) argue that to understand how the social gets under the skin, we need to attain a finer level of granularity and measure at the individual level socio-markers, in a way analogous to bio-markers. They suggest that this can be a step in understanding how mixed mechanisms work, how the social and the biological should not be considered dichotomously but as part of a same whole. Ghiara and Russo's approach, while pushing for the need of identifying markers of social factors, also makes the point that their identification rests on the possibility and ability to understand people's lifeworlds. In the remaining part of the section, we explain how, in our view, qualitative and quantitative research can, together, contribute to specifying and measuring salient aspects of the life-world, thus 'measuring the social' in a useful way.

3.2 Beyond the qualitative and quantitative divide

In order to elucidate mixed mechanisms, we must develop a detailed and precise understanding of the characteristics of lifeworlds and the life course which goes beyond broad statistical generality and which provides enough details to act. One of the most significant gaps in public health understanding, we contend, is an inability to describe social variation in populations –in other words the lifeworld in which mixed mechanisms operate – in sufficient detail to act effectively. Measures such as class, education, income, gender, and ethnicity are of course to be found in the literature, but the way these social differences intersect with each other in the lifeworld to produce patterning at local level and differences at individual level is hard to find in the public health canon. Yet medicine is very familiar with, and works with, the grain of individual biological variation – at its simplest individual people respond differently to biological interventions, like the ingestion of a pharmacological agent. That social variation manifested by the variation in lifeworlds has the same variable effect on interventions, and yet it is neither properly theorised nor properly empirically investigated. Not surprisingly, then, the facility to elaborate the mixed mechanisms both of aetiology and prevention are hidden way – a problem that seems to be too complex to even try to unravel.

Nevertheless, methodologically, unravelling mixed mechanisms may actually be within our grasp, and policy makers need not fall back on simple heuristics to deliver effective interventions. There are a number of existing approaches that try to move beyond the quantitative vs qualitative dichotomy. The most paradigmatic is perhaps the so-called Mixed Methods Research (MMR). Applied researchers in MMR combine both quantitative and qualitative approaches to address the same research question. Methodologists in MMR, at the same time, also reflect on how this combination of methods ought to happen (Guba and Lincoln 1994; Johnson, Onwuegbuzie, and Turner 2007). Some, for instance, prefer calling it 'multi method' (rather than mixed method) to emphasise plurality and to keep distinct aims and goals when applying one or the other approach in a study. There is an on-going debate in the field as to whether MMR constitutes a new paradigm in social science research or whether MMR blends different existing paradigms, in a Kuhnian or post-Kuhnian sense. We build here on the work of Ghiara (2020), according to which MMR combines different, existing paradigms in social science. This has a rather specific meaning, for instance combining

epistemologies as different as those underpinning qualitative and quantitative studies, or different ontologies, such as single-case or generic causal relations. We also build on a recent contribution on the foundations principles of MMR by Johnson et al. (2017), who point to the importance of adopting a pluralist stance, notably concerning the notions of causation, evidence, and mechanism. In the remaining part of this section, we further explain how both quantitative and qualitative approaches can contribute to elucidating the mechanisms through which the social affects health and disease.

The general idea is that quantitative and qualitative approaches contribute to understanding mechanisms, but at different levels. On the one hand, with qualitative, small-scale studies we can typically grasp the *details* of the lifeworld: we can describe in detail what happens between actors, and why, and under what conditions, etc. We can also compare different qualitative, small-size studies and enhance our understanding of cultural differences, or similarities. On the other hand, with quantitative large studies we can *generalize*, or test how stable certain correlations are across cultures, geographical differences, and even times.

The value of keeping both quantitative and qualitative studies, and actually foster a mutual support in these research traditions is twofold. First, methodologically, quantitative and qualitative approaches really are complementary: detailed qualitative studies may give hints about what to test at large scale, population level; conversely, quantitative studies may allow unexpected correlations (or lack thereof) to emerge deserving an in-depth qualitative study. Second, at the level of policy making, we need to know and understand which mechanisms are really culture-specific and which mechanisms are instead more general. This should help us design public health policies that are very specific and tailored to a population, or group, and decide when to export successful intervention to different contexts.

4. On the complementarity between biological and social determinants approaches to health and disease

Scholars already versed into interdisciplinary and transdisciplinary approaches to health and disease, from epidemiology, sociology, or public health, may find themselves at ease in the approach sketched above. But as we hope to show in this section, there are still two dominant approaches, largely in competition with each other. Our own approach complements, or bridges, rather than compete with them.

4.1 The biological and aetiological approach

The dominant paradigm in public health is based on a biological and aetiological understanding of health and disease, focussing on the biological causes of disease. For communicable diseases, this is allegedly the most appropriate approach, using 'the social' as classificatory, but not an explanatory or aetiological factor. In the case of non-communicable diseases, the approach takes 'the social' to be factors associated with risk, again largely in classificatory rather than explanatory or mechanistic way.

The scientific literature is enormous, ranging over heart disease, cancer, obesity, alcohol problems and sexual health, for example. Consequently, we know a great deal about the origins of these diseases – the biological causes – and the things that put people at risk of them. These risks are typically located in behaviour and lifestyle. However, the mechanisms linking the risk factors to disease are much better defined biologically than the mechanisms linking the social to pathology. Consequently, the way to explain which aspects of the social are relevant in which specific context, and how to act on the social for public health purposes, is not clear. Most policy therefore falls back on high-level generalisations about, for example, the relationship between social disadvantage or ethnicity and poor health, or on simple recipes for behaviour change. In practical terms, this does not take us very far, because it does not specify what precisely needs to be done to change or improve things. These models also assume that if aetiology is sufficiently well understood, then effective preventive action can follow (Kelly and Russo 2017).

The efforts at prevention have been geared, across the world, either to trying to change the behaviours that lead to exposure to risk, or less commonly at the so-called wider or social determinants – education, social class, income, poverty, disadvantage and social exclusion (Rutter et al. 2017). Neither approach has been conspicuously successful. This, we suggest, is because the linkages between the social and the biological remain ill-defined and under-described in mechanistic, and particularly *mixed* mechanistic ways. The logically important fact that the mechanisms involved in aetiology and in prevention are different is sidestepped. This is also the case for communicable disease. The aetiology from microorganism to infection is well understood in principle, and usually in its specifics, for most communicable diseases. However, the importance of human social affairs in the spread of infection, and the acceptance or rejection of vaccines, while known about, are not subject to anything like the detailed mechanistic understanding of the virology or the bacteriology. Although the emphasis in what has been called 'population health' is leading towards re-defining of epidemiology as population health science, this in itself does not address the specific point with which we are concerned (see e.g. Keyes and Galea (2016); Valles (2019)). In part, this is due to the fact that this approach, while explicitly focusing on social factors, is not explicitly explanatory or aetiological, as we explain next.

4.2 The social determinants approach

The biological, behavioural, and social factors are conventionally correlated and associated with disease outcomes. Part of this literature shows that socio-economic inequalities *correlate* with poor disease outcomes (see e.g. Bambra (2016); Bartley (2017); House (2002); Marmot (2005); Wilkinson and Pickett (2010)).

This approach addresses the fact that socio-economic factors, and especially inequalities, map onto health patterning at societal level; it is closely linked to social epidemiology. The literature is extensive and has been around a long time (Gairdner 1862; Kadushin 1964; Marmot et al. 1978; Antonovsky 1965; Mackenbach 2006; Erikson and Goldthorpe 1993). Since the 1950s, this literature

has been enlarged to include the idea of risks linked to social factors especially risks associated with practices like smoking, diet, alcohol, consumption, sex, drug use, and physical inactivity (Doll and Hill 1964; Sytkowski et al. 1996)). Globally, all forms of disease are patterned socioeconomically and some infectious diseases are unequivocally diseases of poverty (Kelly and Doohan 2012). The original impetus for this type of work was in the nineteenth century when infectious disease was the largest cause of mortality particularly associated with deprivation. The links between infection and disadvantage have been most recently underlined in data from the US and UK on COVID-19 deaths.

The results of this line of work have been relevant and ground-breaking, and has shed much light on the social dimension of health and disease and in the case of smoking led to declining rates of smoking and corresponding disease prevalence. The results of this research (and especially that of Marmot) are routinely endorsed and used by the World Health Organisation, which recognises the tight relation between social factors and health and urges the implementation of public health interventions that specifically tackle social factors related to health (WHO 2008).

There are several important points to note about this approach (see e.g. Kriznik et al. (2018)). Relevant to our argument, in particular, is that this approach establishes *that* socio-economic factors are crucial to health. However, it does not establish *why / how* it is so, or how to go about changing things. In other words: while the social determinants approach does establish meaningful and robust correlations between social and behavioural factors and health, it does not elucidate the mechanisms or the causal pathways through which 'the social' affects health. In this 'social determinants' approach, the tendency, especially in recent years, has been to go as granular as possible in the measurement of the social. Thanks to progress in sampling, statistical techniques, availability of medical records, geographical information systems, data linkage and so on, we can establish such correlations at increasing levels of granularity. This means, for instance, being more precise about the social and geographical groups involved. Alternatively, refining socio-economic characteristics with more precise definitions and measurement tools. Or, relating not just 'classes' of pathologies but finer grained types of disease: think, for instance, of how many types of breast cancer we can now differentiate.

Our main concern with this approach is that, after all, correlations remain non-actionable. Instead, for the purpose of public health interventions, we need to elucidate the mechanisms, or rather the *mixed* mechanisms at work in the lifeworld of individuals. Our own approach, instead, is in line with evidential pluralism, a line of research in the philosophy of causality and of medicine, in which both elements (correlations and mechanisms) are important (Clarke et al. 2014; Parkkinen et al. 2018), and that can help bridge the biological and the statistics-based approaches.

4.3 Why evidential pluralism can bridge the biology and the social dimensions of health and disease

The two approaches just presented have been successful but unfortunately remain largely distinct, and therefore unable to bridge the world of 'the biological' and the world of 'the social'. Instead, we think that adopting 'evidential pluralism' will help us make a significant step further.

Evidential pluralism is a position developed in philosophy of science and in philosophy of bio-medicine, according to which, in order to establish a causal claim, we typically need evidence of correlation *and* evidence of mechanisms (Russo and Williamson 2007; Illari 2011; Parkkinen et al. 2018). This is an epistemological and methodological thesis about disease causation, rather than an ontological account. In the field of (philosophy of) biomedicine, evidential pluralism has been used to argue, against evidence-based medicine (Sackett et al. 1996), that evidence of mechanisms is also important, and in a way that is not reflected in evidence hierarchies (Clarke et al. 2014) or even in the GRADE system (Parkkinen et al. 2018).

Interestingly for our argument, evidential pluralism is not only about exploiting mechanisms and correlations synergistically for a more solid evidence base. It is also about *enlarging* the scope of what we take evidence *of*: both mechanisms and correlations can be about the biological *and* about the social dimensions. In particular, evidential pluralism has, until now, largely focused on how bio-chemical mechanisms should be part of the evidence base to establish causal claims, besides correlations. But our argument is that mechanisms of health and disease are not just bio-chemical but also inherently social, or better said: bio-social (see also: Kelly and Kelly 2018; Kelly, Kelly, and Russo 2014; Kelly and Russo 2017; Ghiara and Russo 2019).

Let us return to Covid-19 to exemplify the usefulness of evidential pluralism and of bio-social mechanisms. On the aetiological side of the equation, the vector of transmission involves a large number of social factors. These include socializing, touch, sneezing, coughing, working environments and work practices, domestic circumstances, numbers of people and generations in shared households, and locality, age, ethnicity, sex, occupational, educational and income composition of communities. On the preventive side of the equation, these same factors were equally important, although the mechanisms involved are not the same as the aetiological ones. The problem is that the knowledge base about the preventive and the aetiological social mechanisms are not known about in sufficient detail to enable jurisdictions to act forensically, and instead blanket, rather than targeted interventions, were the best that most authorities could do – and then hope for the best. Most governments favoured non-targeted and unspecific action based on the known social co-ordinates, and opted for heuristics – short-cut thinking to provide answers to complex problems, without knowledge of unknown or intended and unintended consequences. Heuristics allow fast thinking, but they are usually biased and often wrong (Kahneman 2013). The fact that high-risk groups such as the BAME community and the elderly that should have been the target for protection, was ignored. And, of course, the epidemiological models used to justify the decisions taken by governments, were invariably about aetiology not prevention and did not deal in mechanistic social evidence about either aetiology or prevention (Aronson et al. 2020).

More generally, many preventive efforts relating to non-communicable disease in particular, have not produced results, which have made much impression on the mortality and morbidity associated with the patterning of inequalities or the prevalence and incidence of disease (excepting smoking related disease). This had led to commentators raising two issues. First, given what is known about aetiology and risks, many diseases ought to be by now preventable, and yet some epidemics are on the rise – obesity and some cancers for example (Cavalin and Lescoat 2017; Horton 2017). And, second, despite the deep understanding of the biology of non-communicable disease, public health interventions mostly target either whole populations or behaviour in ways that have been less than optimal (Rutter et al. 2017; Marteau, Hollands, and Kelly 2015). It is not clear why.

The interactions between different social dimensions are complex, self-evidently, but they are not unknowable. Very clear accounts exist in the sociological literature such as social practice and structuration theory (Giddens 1979; 1986; Giddens and Dallmayr 1982; Bourdieu 2000; 2008), which provide high level, but very informative frameworks to understand the interactions between people's actions and the social structures that they inhabit, and the ways that these interactions ingrain themselves in human biology. At the very least, they provide a route map with the key co-ordinates for forensic action to prevent transmission and facilitate preventive action.

5. Conclusion

Health and disease are not solely biological or social phenomena. The social and biological spheres are deeply intertwined and interconnected, one cannot be reduced to the other. This has long been recognized by epidemiologists and medical sociologists alike. And yet, far too many public health interventions do not succeed in making the most out of the vast body of knowledge documenting the relations between social factors and health outcomes. Why is it so?

We think that what is missing in the vast and very valuable knowledge base of public health is an explicit recognition and use of two concepts: lifeworld and mixed mechanisms. We presented these concepts in section 2, and we offered an approach to operationalize them in section 3. In section 4, we explained how, in our view, lifeworld and mixed mechanism can bridge two dominant approaches, namely the 'biological and aetiological' and the 'statistics-based and classificatory' one. By adopting evidential pluralism, we can combine in the evidence base both mechanisms and correlations, and especially correlations with social factors, which can hitherto be given a proper place in a bio-social mechanistic understanding of health and disease.

Our approach applies to communicable and non-communicable diseases alike (Khalatbari-Soltani et al. 2020). We contend that a detailed description of the lifeworld and of the mixed mechanisms within which health and disease happen, can help in designing more effective interventions even when the biology of the disease is well understood. This is because social factors are not remote, background factors, but are proximal factors, just like biological ones. But the

decision to intervene at the level of the social, at the level of the biological, targeting specific individuals, groups, or the whole population is very much contextual. We are not here to provide magic recipes or rigid checklists; we are here to offer a theoretical framework, but one that can be used in practice, to make important choices in the design of public health interventions.

We are also aware of the fact that, more often than not, conflicting values, vested interests, or other ethico-political factors play a role in how policies are designed and implemented. In this paper, we set aside this aspect not because we think it is secondary, but because we believe that, if we can get a firm grip on the epistemology and methodology of interventions, it will be easier to isolate and effectively deal with any non-epistemic factor influencing policy.

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