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The relation between the social and the biological and COVID-19

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ABSTRACT

Social factors have been linked to disease severity and mortality in COVID-19. These social factors are ethnicity, social disadvantage, age, gender and occupation. Pre-existing medical conditions have also been identified as an increasing risk. This paper explores the relationship between these social and biological factors using a syndemic frame of reference. The paper argues that although the associations have been very well documented, the mechanisms linking the social factors and disease outcomes are not well understood. An approach that seeks to find commensurability between the social and the biological, is suggested.

Introduction

The coronavirus disease 2019 (COVID-19) pandemic has revealed an important gap in the evidence about the links between the disease and socially defined vulnerability, susceptibility and risk. This paper considers this gap and points to a way to re-think the relationship.

Why do some groups get sicker and die in greater numbers?

This paper begins with the perhaps surprising assertion that our understanding of the relationship between social and biological phenomena is limited. We, of course, know a great deal about biological mechanisms and pathology, and that some are triggered by, and often associated with, social phenomena.[1–5] However, the way the interface between the social and the biological realms actually works, is not well understood mechanistically.[6,7] So, although factors like chronic stress and inflammation linked to the social position are sometimes pinpointed, the specification of stress as a social phenomenon, involving a transaction between the person and the environment is not elaborated, other than in quite general terms.[8] Most of the focus is on the biological mechanisms, which follow the triggering stressor. The triggering phenomena are treated as if they were quite separate from the biological consequences, rather than in an interactive relationship with them.

Early on in the COVID-19 pandemic and then repeatedly during 2020–2021, in the United Kingdom and elsewhere, the data showed that some social groups were at greater risk of severe infection and mortality than were others. These were 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.[9–20] There is a clear association across different jurisdictions between COVID-19 disease severity and death and social factors broadly defined.

It may, therefore, seem perverse to suggest that we have limited understanding, as the associations are so well established. However, beyond the associations, what of causal mechanisms operating within dynamic interacting systems?[21,22] Public health science has an excellent record of unravelling mechanisms linking toxic environmental exposures and biology, and proximal risky behaviour and disease.[23–25] Its record in respect of the *mechanisms* linking *social life* and the biological is much patchier, even though the associations have been well known since the nineteenth century.[26–28] The descriptions of the precise mechanisms by which the social determinants exert their malign influence is largely absent from the scientific, including the social scientific, literature. This gap is apparent in the respect of COVID-19.

During the pandemic in the UK, there have been numerous policy and managerial admonitions for the system to orient itself to take remedial action in respect of the groups most at risk and the social factors involved.[15] There is good advice about what might be done.[29] However, these efforts have been less successful than they should or could have been. To remedy the situation, we need to

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understand the social better and to learn more about its interactions with human biology.

Understanding the social better

In the UK, and beyond, there are important differences within and between social groups along the dimensions of class, gender, ethnicity, age, geography, sexual orientation, religion and disability.[30–32] These differences in turn intersect and interact with each other through the social practices in which people engage.[33] This produces the rich diversity that is contemporary society. That behavioural variegation—normatively, culturally, socially, economically and geographically—means that there will be differences in access to power and the resources available to people, their abilities to shape their own lives, and to control their lifeworlds. The spread of infection is just one function of the different social practices in which these highly differentiated groups engage.

The complex overlay of social differences and the intersections between them, make up the richly nuanced heterogeneity of the populations. It is at best naïve, and at worst ignorant, to try to think about, model, and act upon the population without reference to this. There is not one British or English, or white, or Scottish, or South Asian, or Black sub-culture. There are myriads of them. It is undoubtedly complex, but not unknowable. Although far from complete, a lot is known about the nature of the different communities and their characteristics in the UK.[34–39] This rich literature demonstrates that it is important not to treat the different ethnic and social class groups as if they were homogenous. Categories like 'BAME' or 'socially disadvantaged' are unhelpful as they imply that the factors at work in the vulnerability, susceptibility, risk and severity are the same for everyone in that group. The data suggest this to be inaccurate. For example, there were differences in the risk of death between the first and second waves of the UK pandemic between the Black African and the Caribbean and the Bangladeshi and Pakistani communities. It was higher in the latter two groups.[40] We have to be much more granular and nuanced.[41] We must be mindful of the rich variegation within, as well as between, communities.

Social variation in the population in the UK, US, much of Europe and beyond is great. Policy actions must reflect that, avoid thinking in categorical terms and start thinking in relational terms.[31,41] Moreover, we have the information, or at least some of it, in the UK and elsewhere. The fact that there is no single pattern within the diverse groups seems to be clear in the data and is actually well-understood.[15] However, even when we have the data, turning that knowledge into action is difficult. Linking it to causal mechanisms remains to be developed.

It is not just the official data and the academic descriptions of communities to which we should turn. Much of the knowledge about the rich variegation and the social dynamics resides in the communities themselves and the local municipalities, the primary care and the public health services that support them, as well as the many small and larger businesses that serve them. The well-worn precepts of health promotion would be the obvious route to follow.[42–44] We need to work with people, learn from them, get to understand their preferences, likes and fears and try to get to grips with their practical knowledge about why they do what they do. We should not 'just do stuff to them', even if that 'stuff' is done with the best of intentions.

This, actually, is also well understood in the official record within the UK. Community asset-based approaches to health promotion were advocated in response to COVID-19 and resources exist to help with this endeavour from the Local Government Association, for example.[12,14,16] There was an official acknowledgement that there might have been systemic service delivery failures to ethnic minority communities.[12] Many Directors of Public Health worked with their local populations and centrally there were well-intentioned aspirations to engage.[15] At the end of July 2020, in a letter to all NHS Trusts, Clinical Commissioning Groups, Community Health Services and NHS 111 and Primary Care providers, Sir Simon Stevens and Amanda Pritchard (NHS Chief Executive, and Chief Operating Officer respectively) urged the NHS to engage with local communities. They advocated protecting the most vulnerable, ensuring services were used by those in greatest need, and targeting long-term conditions.[45] This was followed by a set of detailed instructions on 7th August 2020.[46] The need for preventive efforts, because of health inequalities linked to class and ethnicity and COVID-19 was widely acknowledged, officially and beyond. However, although strong on aspiration, the system-wide changes that were canvassed have not been easy to discern in action. The obvious exception occurred at the end of 2020 when the Joint Committee on Vaccination and Immunisation in the UK (JCVI) identified the oldest members of the population as the first candidates for immunisation.[47]

Putting it together—the syndemic concept

Public Health England identified the factors leading to a greater risk of infection as increased exposure, transmission, susceptibility and vulnerability.[48] The phrase 'underlying medical conditions' entered the lexicon of newsreaders and journalists early on in the pandemic, as a catchall to explain the apparent clustering of cases and mortality, especially for ethnic minorities and older people. The implication was that these groups had more underlying medical conditions and that this raised their level of risk and hence their probability of dying.

Anyone with a passing familiarity with the literature on health inequalities would scarcely have been surprised by the data on severe infection and mortality and social disadvantage that emerged in the first months of 2020. The somewhat mysterious 'underlying medical conditions' turned out to be the principal killers of the last 70 years linked to social disadvantage.[48] The conditions were diabetes, hypertension, cardiovascular disease, chronic obstructive pulmonary disease (COPD), chronic kidney disease, obesity, dementia and Alzheimer's disease.[9] There were marked regional variations reflecting different levels of wealth across the country.[49] The data provided a grim vindication of decades of research linking disadvantage to ill health, and particularly the patterning of non-communicable diseases. The data were a chilling reminder of repeated policy failures to deal with health inequalities, or worse, the deliberate pursuit of policies that inevitably made inequalities in health more pronounced. In this regard, policies pursued in the UK since 2010 stand out as ones that have exacerbated inequalities in health.[50]

Where we have data from the modern period, pandemics have always affected the poor more than the well-to-do. There were major inequalities in the 1918 Spanish Flu pandemic within and between countries, with the richer faring better. This pattern was repeated in the 2009 H1N1 influenza epidemic.[51] Villermé, one of the French sociomedical investigators of the nineteenth century, studied the links between social conditions and disease. Villermé suspected working and housing conditions, nutritional status and pre-existing disease were the culprits. He wrote in 1833 'epidemics everywhere strike the destitute or miserable classes much more than they do persons of quality'.[26] Not perhaps the language we would use today, but the point is that the pattern we have seen with COVID-19 is not new. The current links between disadvantage and epidemic mortality were in fact highly predictable, and given the ways that life expectancy had stopped increasing in recent years in

the UK—a proxy for worsening health inequalities—it was probably predictably worse than it might otherwise have been.

However, it was not just that the pandemic was an overlay on and an exacerbation of existing inequalities, the link to existing medical conditions is important. The pattern of pre-existing morbidity in the populations where mortality has been highest from coronavirus was already reported in the literature. The link between non-communicable diseases and viral infection has been called a syndemic one '… the concentration and deleterious interaction of two or more or diseases or other health conditions in the population, especially as a consequence of social inequality and the unjust exercise of power'.[52] The connection between SARS—a coronavirus—and age, diabetes, hypertension, coronary artery disease and COPD, involving a *three-way interaction* between the viral and other pathologies *and* social disadvantage was well known.[52] That the same process was at work in COVID-19 was noted early on.[49,51,53] The syndemic intertwining of interactive, cumulative consequences, which exacerbated population and individual disease burden, was clear to see.[54]

11 years before the current pandemic, Singer had described the relationship between a coronavirus, non-communicable disease and disadvantage *in a textbook*. The published papers on which he based his argument were widely available.[55–59] The evidence that there was a highly predictable additive effect of disadvantage, pre-existing non-communicable disease and SARs viral infection was in plain sight. Modellers and commentators were well aware of the interaction with the virus[60] and with ethnicity.[10] Moreover, a lot was known about health inequalities, which *a priori* would have suggested that the population would be differentially affected. The clustering of negative syndemic factors in the black and ethnic minority community, the poor and socially disadvantaged, and the elderly created a perfectly predictable storm. Worryingly during the current pandemic, a lot of effort has gone into re-describing this problem, rather than getting to the root of it.

Nevertheless, the idea of syndemic is a helpful way to frame our future thinking and link back to the points above, about the relation between the social and biological. Singer's explanation is useful. "Human environments, including the prevailing structures of social relationships (such as social inequality and justice) and also sociogenic environmental conditions (for example hazards of the built environment, sales of toxic commodities, pollution, species loss and climate change) contribute enormously to both disease clustering and interaction'.[52] The important thing is not only to conceptualise these as *risk factors*, but also as phenomena interacting with the virus. The interactions between the virus, other diseases and inequalities need to be conceptualised in terms of the simultaneous interactive causal mechanisms involved. Associations statistically and epidemiologically and the identification of risk are the starting points, but further elaboration of sociobiological mechanisms is necessary.[61]

The syndemic idea is that human biology is not only affected by interactions between viral infection and pre-existing medical conditions, but the manifestation of the disease is a function of *simultaneous interactions* with the social environment. That social environment consists of social practices in local communities, the material and economic conditions in which people live and work, as well as their subjective experience of all of these things. The key idea is *simultaneous interactions in an open system.*[62] The individual pathology of course attracts attention, but the other dimensions in the system are not merely context. They are part of the multiple pathways of the origins and manifestations of the disease, of vulnerability susceptibility and protection.

We also need to be mindful of the salutogenic possibilities.[63] In other words, what were the protective factors? What are the things that seemed to have conferred social as well as biological immunity? Why is the pattern of the disease so different between different people, so severe in some cases but less so in others? What are the mechanisms at work for those who came out relatively unscathed? Furthermore, are the protective mechanisms merely the inverse of pathogenesis? Or, do the protective measure processes operate through a different causal pathway? Gender, class and ethnicity and age may well be protective for some, but why and how? Broad ecological associations do not explain the phenomena.

All of that is very complex, but is not unknowable. The implication is clear. Unravelling the mechanisms at the *social and biological interf*ace is critical. The frameworks describing social practices, of the interactions between human agency and social structure, are very well-established in the social scientific literature and are potentially helpful.[64,65] The way people live their lives—their actions, habits and skills—becomes deeply ingrained socially *and biologically*. These everyday actions have biological consequences with respect to good or poor health. Human biology or human health, in turn, constitute the capacity and capability to shape and constrain one's own life.[66] The dynamic interactions between agency and structure interact for good and ill with human biology. The constantly recurring interactions between the social, biological and physical worlds and the human subjective experience of them, affect everyone but in different and patterned ways.[7] The consequences of the social imprint on the biology of the body and of the constraints, which, the biology places on social life, are not metaphors. They are phenomena, which are biologically and socially real with mechanisms of interaction. Although beyond the scope of this paper, it is not in principle difficult to map these.

Discussion

There have been no shortage of critics of the policies pursued in the UK and elsewhere and the manner of their implementation. The purpose here is not to add to that chorus, but rather to consider the kinds of ways we should develop the idea of syndemic interactions; use the evidence we already have to do so; and use our scientific knowledge and that of communities, to get it into practice.

The social—biological interaction and in turn its interaction with health services and social care provision may be thought of as a complex open system with continuous interactions between the physical, material and ecological environment, social relations and practices, subjective experience and cognitive processes, and human biology. Systems have emergent properties.[67] In the interactions in the system, no single dimension has precedence. To grasp this requires a commitment to non-reductionist thinking.[68] There also needs to be an acknowledgement that these social factors are not mere background or context, but are intrinsic to the causal mechanisms and interactions involved, and that we have ways of describing those interactions. This is important. The biological, whether microbiological, genetic or atomic, is usually foregrounded in medical research, while social, economic and cognitive processes are viewed, at best, as mere context or the origin of risks or stressors, while the primacy of the microbiological is retained. When thinking syndemically, it is more useful to conceptualise the various elements in the system as in constant interaction with each other.

So, for example, although there is clear evidence that certain aspects of the way the pandemic has evolved are associated with structural racism, the epistemic bases (the knowledge admissible as an adequate explanation), for accounting for the phenomena of racism and its effects on health, are quite different to, for example, the grounds of proof in a biological model of way T cells respond to infection. If we stay in our silos and do not try to understand the mechanisms involved in each of these domains *and how they*

interact with each other, the only outcome is likely to be the feeling that there is epistemological incommensurability—a high sounding way of describing mutual incomprehension. Soft systems thinking, instead of silo thinking, allows the totality of the problem to be conceptualised as a whole. It involves investigating interactions that are not yet, well understood.

And despite everything, I sense from the public record that we are almost there. There was clearly considerable effort across the UK government, its Arm's Length Bodies, and the research community to draw together the data relating to ethnicity and to inequality more generally.[69] Much research was commissioned to support this and to fill gaps in the evidence base. However, it mostly remained locked in the language of describing the problem and identifying risk with a strong biomedical primacy.

Conclusion

Several researchers working in a syndemic frame of reference have begun to hypothesise the pathways and mechanisms between COVID-19 (and other viral infections), pre-existing disease (especially diabetes and cardiovascular disease) and social structures including ethnicity.[70–74] This work is important, as the absence of detailed understanding, especially of the interaction with the social, has significantly hindered the ability of jurisdictions across the world to respond forensically and effectively to the pandemic. Future responses to similar viral infections will be considerably enhanced, by such understanding. These hypothetical pathways will need to be explored in full, including systematically interrogating the extant literature as well as new primary research to test them.

In scientific terms, so far so traditional! However, the arguments presented in this paper carry a health warning about the conceptual structures to be used in hypotheses development and testing. With respect to COVID-19, the syndemic account must involve several elements.

First, we must focus on the repetitive, recursive features of people's lives *pre-pandemic(s)*. These are important in two different respects in understanding the dynamics involved. (i) The recursive nature of social interactions across the life course leave their marks on the human body. Metabolomics and other omics show how life literally gets under the skin.[7] The conditions, which have been so deadly in COVID-19 (diabetes, heart disease, dementia etc.), follow this process. (ii) The same practices that operate across the life course also operate in the here and now; at work, in the home, in the community, on public transport and so on. They are the vectors of viral infection. The practices are the gateway to the molecular structure of the human body, already damaged and made susceptible by those same practices. The social practices of the communities most at risk must be an urgent research priority. Importantly, academic researchers must not assume that they know how these communities live; they must let the communities themselves tell them.

Second, to understand practices, attention must be directed to the relational nature of social phenomena. The focus must not be on individuals and their behaviour, but on collective activities. The social phenomena, which the epidemiology has so clearly revealed as risks and vulnerabilities, must not be treated as people's individual characteristics. To do so easily leads to victim blaming and often ill-considered strategies to bring about individual behaviour change.[75] They must be conceptualised instead as dynamic relationships and interactions, operating at the group level.[41] The essence of ethnicity, gender, sexual orientation, class, occupation and age is not that they are things or labels that we can attach to individuals. Relationally, they are about the experience of being black, gay, a woman, poor and old. It is not being Bangladeshi or Black that is important. It is the nature of the social relationships involved in these social placements, at work, at home, in the community—that defines the noxiousness or otherwise of the lives people lead and their abilities to control their own lifeworlds. Those experiences involve power, conflict and social exclusion, as well as more nurturing relationships. What makes us human is not our individuality, but our social relations with others. So, we must move away from concepts that are individually oriented to ones that are relationally oriented, and are dynamic not static. In research terms, we must move beyond variables and factors and instead look at the lives that human beings lead with other human beings.

Third, we must identify the competencies and capabilities people have, the sense they make of their lives and the physical infrastructures around them, which constrain and facilitate the things they are able to do. In empirical terms, this means going granular and going local, and foregrounding the kinds of community studies that can do precisely this. We will need to understand the history of locales and communities and, very importantly, the understandings that communities have of their own histories. This may sound like a tall order, but in the UK at least, the kinds of things referred to here are the meat and drink of local public health and primary care teams. This kind of knowledge must not be relegated to second place behind epidemiology, modelling, virology, immunology and clinical medicine; it must instead be the starting point and at the very least as an equal partner with the biomedical sciences.

Fourth, we must think upwards to the complex open system that is human health and disease. The COVID-19 pandemic vividly illustrates that human health and disease involve multiplex interactions and relations between physical, material, political, economic and ecological environments, social and cognitive life and human biology. If we ignore this, and seek to analyse these things in a reductionist and isolated way, our understanding will only ever be partial, and we will never unravel the complexity nor see that complexity from the point of view of the people whose lives have been so desperately perturbed in the pandemic. Unfortunately, much of the science about COVID-19 has been highly discipline specific. It represents the viewpoint of the scientists, policymakers and politicians—not the communities themselves. The starting point is to be in, and work with, the communities whose lifeworlds have been cruelly ransacked by COVID-19. We need to work to enhance capabilities—biologically, socially and economically—capabilities that are the source of current and future social and clinical vulnerability.

Finally, we must stop using terms like wider determinants or social causes, carelessly. This is because too often these terms are operationalised heuristically—as shortcuts in understanding and explanation.[76] We must instead use the clues revealed by the wider determinants and social causes literature. These signpost the mechanisms that reveal the pathways to individual and community health outcomes. We know a lot biologically about some of the mechanisms involved in COVID-19. We know a good deal about social life too. But the two lots of knowledge remain in separate domains. Metaphorically, many of the pieces of the jigsaw puzzle are there already. We have yet to fit them together. The problem is that unless we conceptualise things along the lines suggested in this paper, we will forever keep the jigsaw puzzle, not just in its box, but in separate boxes of scientific silos. We will continue to spend our time staring at the underside of the box, rather than its top. The top of the box, of course, has the picture to guide the way we fit the pieces together. The picture is the reconceptualising suggested here.

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