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Pourquoi la santé et la maladie sont-elles distribuées socialement dans les sociétés humaines ? L'incorporation du vécu et sa dynamique au cours de la vie humaine

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Cette note fait suite à une communication invitée présentée lors des 1847^{es} journées de la Société d'Anthropologie de Paris dans le cadre de la session “Corps malades”

Abstract – This paper is based on a keynote address at the Société d'Anthropologie de Paris conference in 2021. It refers to research stemming from the field of social epidemiology, specifically addressing the issue of health inequalities. The unequal distribution of health across social-structural groups in the population is nothing new, and continues to be a persistent problem in public health. Understanding how social inequalities relate to health inequalities, how processes and mechanisms operate over the life course to create health inequalities, is an interdisciplinary endeavour that combines the social and biomedical sciences. In this paper I outline recent theoretical and empirical work that aims to unravel how ‘the social becomes biological’ through the embodiment dynamic, leading, at least in part, to social and socioeconomic gradients in many health outcomes.

Keywords – embodiment, health inequalities, life course, social epidemiology

Résumé – Cet article est basé sur une conférence plénière lors du congrès de la Société d'Anthropologie de Paris en 2021. Il porte sur des recherches issues du domaine de l'épidémiologie sociale, abordant spécifiquement la question des inégalités sociales de santé. La distribution inégale de la santé entre les groupes socio-structurels de la population n'est pas un phénomène nouveau et reste un problème persistant de santé publique. Comprendre comment les inégalités sociales sont liées aux inégalités de santé, comment les processus et les mécanismes opèrent au cours de la vie pour former les inégalités de santé, requiert d'un travail interdisciplinaire qui réunit les sciences sociales et biomédicales. Dans cet article, je présente les travaux théoriques et empiriques récents qui visent à comprendre comment “le social devient biologique”, à travers une dynamique

d'incorporation qui conduit, au moins en partie, à des gradients sociaux et socio-économiques dans de nombreux états de santé.

Mots clés – incorporation, inégalités sociales de santé, parcours de vie, épidémiologie sociale

Introduction

The links between medical anthropology and social epidemiology are obvious. Both disciplines scrutinise the relationships between human societies, cultures, activities, etc. and human health. Having taken an undergraduate degree in anthropology myself at the University of Durham, I gradually migrated towards the field of social epidemiology. In doing so, my main interest was to understand how social inequalities affect health inequalities over human life courses. In this talk I will discuss the question of why health and sickness are socially patterned across human societies from the perspective of social epidemiology. Hopefully, the parallels drawn between anthropology and epidemiology in this brief overview will highlight the need for approaches that combine their respective theoretical, methodological and empirical traditions.

Health inequalities are “systematic, avoidable and unfair differences in health outcomes that can be observed between populations, between social groups within the same population or as a gradient across a population ranked by social position” (McCartney et al., 2019). They are by no means a new phenomenon, with a large body of evidence bearing witness to their existence and persistence. Many scientists from across disciplines have asked the same question over the years as the one I am raising here. The field of social medicine has been especially prolific in showing

the relationship between social conditions and health or mortality in the mid-1800s, which was observed by a variety of wealthy European men at the time. These include Engels and Marx in Great Britain, Virchow in Prussia and Villermé in France. The evidence for these historical health inequalities is all around us: Davey Smith, for example, showed socioeconomic differentials in mortality using data from graveyards in Glasgow, where the height of obelisks was associated with age at death, obelisks being a symbolic demonstration of social status. The higher your status/obelisk, the longer you had lived (Smith et al., 1992). The historical record from England also shows that social differences in life expectancy may have become especially visible from around the 1750s, when the titled upper classes ('ducal' males and females) had a longer life expectancy than the rest of the English population (Kendall et al., 2021).

To me, the key research question is to examine how health inequalities are constructed, to understand how social inequalities lead to health inequalities. This relationship between social and health inequalities has been laid bare during the Covid-19 pandemic, during which the social dynamics of exposure to risk and differential susceptibility (Diderichsen et al., 2019) have affected *who* was most likely to get ill, and *who* was most likely to develop severe forms of disease or to die. None of the population categories making up the '*who*' is random. The risk of infection is socially patterned, by working or housing conditions for example (Vandentorren et al., 2022), while susceptibility to disease is socially patterned due to material, behavioural and psychosocial exposures over the course of life leading to susceptibilities (Bambra et al., 2020). I will return to this crucial point further on, because the social and economic consequences of a syndemic reach well beyond Covid-19-specific health inequalities.

The UNDP has projected serious consequences of the pandemic for human development (UNDP, 2020), due to the closure of education infrastructures, an economic downturn and the effect on mortality worldwide, predicting a regression to levels of poverty not seen since the human development index was established in the early 1990s. Based on the body of literature describing the relationships between education, work, income and other socio-economic measures, and health, this regression is likely to lead to worrying increases in both social and health inequalities. In order to understand why socioeconomic and structural factors have an impact on health, we need to examine how the socially structured environment becomes embodied biologically.

Perhaps one of the most important ideas that has contributed to our understanding of why health inequalities are so ubiquitous over space and time was developed in social epidemiology by Krieger, who referred to it as 'embodiment'. According to Krieger "at the most general level, embodiment, as an idea, refers to how we, like any living organism, literally incorporate, biologically, the world in which we live, including our societal and ecological circumstances" (Krieger, 2005). This concept allows us to move from abstract observations of associations between

social-structural phenomena and health, to a concrete understanding of the dynamic processes over the course of life that underlie this observation. In social epidemiology, Krieger's work on embodiment and ecosocial theory melds with the literature on the developmental origins of adult health and disease, herein Hertzman refers to biological embedding, which is intricately linked to what he describes as the omnipresent socioeconomic gradient in health (Hertzman, 1999). This work also connects with research from the social sciences, including social and developmental psychology through Elder's principles of life course patterns (Elder Jr. and Shanahan, 2007), and Bronfenbrenner's ecological systems theory of child development (Bronfenbrenner, 1977). I have outlined the relationships between these concepts across disciplines from empirical evidence, as well as their influence on our understanding of how social inequalities become health inequalities, in my recent essay (Kelly-Irving and Delpierre, 2021). Based on the model developed in that paper, I will now briefly describe how social-structural factors become embodied.

The embodiment dynamic

The social gradient in health, whereby social circumstances are related to health conditions along a graded association, has been observed over time, across different contexts and types of pathological processes (Hertzman, 2012). In other words, socioeconomic variables, such as education, occupation, income, etc., have been associated with a wide variety of diseases including cardiovascular diseases, diabetes, some cancers, mental health conditions, infectious diseases, etc. This association usually takes the form of a gradient whereby socioeconomic disadvantage is associated with a greater risk of poor health, while socioeconomic advantage is associated with being relatively protected from adverse health outcomes. We also know that the different pathologies described as being socially patterned are inter-related, involving higher rates of co-occurring disease but also shared behavioural risk factors, and potentially shared aetiological processes, including evidence at the molecular level (Barabasi et al., 2011; Goh et al., 2007). The question that arises is, are we responding biologically to our socially structured environment?

Our definition of the embodiment dynamic:

"[a] set of social and biological processes and interactions between individuals within a population and their environments over time. It is a dynamic that is socially stratified representing the past environmental landscape and an ongoing response to the present environment. Humans thus come to physically represent their past environments in their present state through a constant process of change." (Kelly-Irving and Delpierre, 2018).

Based on the theoretical and empirical literature, we formulated a model to show explicitly how socially stratified exposures elicit biological mechanisms leading to a biological response. This dynamic is ongoing throughout

the course of life and is sensitive to the timing of exposures. We suggest that two broad types of biological mechanisms are solicited by the social environment, those of exogenous and endogenous origin. Those of *exogenous origin* involve entities or conditions external to the body that, entering the body or interacting with it, elicit physiological responses leading to physical harm or exertion. These entities include inert or living entities such as foodstuffs, asbestos, viruses, bacteria, pollutants, etc. On the other hand, our perception of situations, experience of relationships and other daily psychosocial experiences induce biological mechanisms of *endogenous origin*. These occur when sensory interpretations of socially patterned interactions with the environment elicit responses from the central nervous system, as well as cognitive and psychological functions. Both broad types of biological mechanisms interact and are subject to the historical social structures and stratifications that characterise human environments and underlie the lived experiences of populations (Kelly-Irving and Delpierre, 2021). There is plenty of evidence for the embodiment dynamic in the scientific literature.

Social-to-biological mechanisms

We need look no further than to the Covid-19 pandemic to see evidence of socially patterned mechanisms of exogenous origin. Through housing conditions, occupations and population density, socially differentiated exposure to the Sars-Cov-2 virus, an entity external to the body, has led to more socially disadvantaged people being at increased risk of infection (Vandentorren et al., 2022). The everyday lives led by different social groups within the population have caused them to be more or less likely to encounter the respiratory virus, and to be infected by it. Mechanisms of endogenous origin, however, are harder to observe, largely misunderstood and thus under-appreciated within the health care disciplines, although they are crucial if we are to understand how social inequalities become health inequalities over the course of life. I will therefore provide more insight into them here, based on previous empirical work.

The stress response system is one of the main ways through which we embody our external environment. Overall, stress tends to be loosely defined as “a process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk of disease” (Cohen et al., 1995). To capture the physiological aspect of stress response we have used the Allostatic Load concept. This was described by McEwen and Stellar in 1993 as the impact of wear and tear on multiple physiological systems and organs of the body as it is put under strain by repeated responses to external stressors. Allostatic load is the price paid by the organism over time to adapt itself to environmental challenges (McEwen and Stellar, 1993; Seeman et al., 1997). It is measured in humans through a list of biochemical biomarkers, extracted from blood tests and clinical examinations, which represent as many physiological systems as possible (cardiovascular, metabolic, inflammatory, endocrine, etc). We have described the measurement of allostatic load and debates around its validity elsewhere (Delpierre et al., 2016). One of its characteristics is that it is socially patterned: an extensive literature on the subject has shown that socioeconomic disadvantage is associated with a higher allostatic load, and thus with being at greater risk of subsequent morbidity and mortality. For example, a 2012 paper by Gruenewald et al. concluded that a higher allostatic load may be one pathway through which greater life course socioeconomic adversity leads to greater risk of morbidity and mortality in later adulthood (Gruenewald et al., 2012). An overview of the evidence on the life-course social patterning of allostatic load is available here (Kelly-Irving, 2019). From our own work, we have shown that a disadvantaged socioeconomic position at birth, using mother’s education and father’s occupation, was associated with a higher allostatic load at the age of 45 (Barboza Solís et al., 2016). We also ascertained that a higher allostatic load was associated with a greater risk of subsequent premature mortality (Castagné et al., 2018), and that a potentially interesting physiological system to consider separately, using available biomarkers, was the inflammatory system. Figure 1

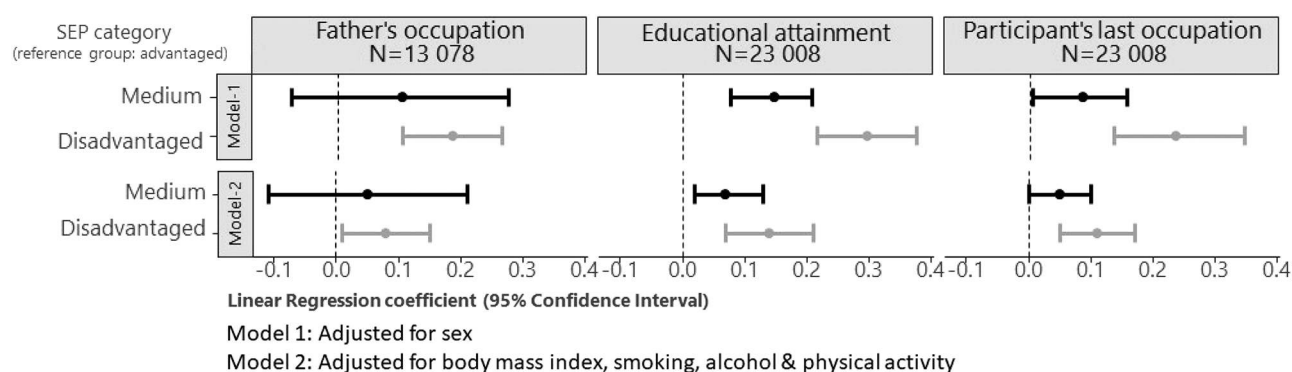


Figure 1. Association between social position (SEP) at three stages of the life course and C-reactive protein level (CRP) across six European cohort studies (adapted from Berger et al., 2019) / Association entre la position sociale (SEP) à trois étapes du parcours de vie et le niveau de protéine C-réactive à travers six études de cohorte européennes (adapté de Berger et al., 2019)

shows findings from a study carried out using data from six European cohorts. Here, we showed that socioeconomic disadvantage at three different points in time over the life course, measured using father's occupation, educational attainment and the participant's last occupation, was associated with C-Reactive Protein (CRP), a marker of overall inflammation (Berger et al., 2019). We adjusted for body mass index, smoking, alcohol and physical activity level, which may be potential mediation variables through which SEP affects inflammation. While this attenuated the strength of the association, we observed that controlling for these variables did not 'explain' the relationship.

Further evidence also points to inflammatory biomarkers being sensitive to the social environment from childhood and adolescence (Fraga et al., 2019), and to the relationship between the social environment and human internal biology throughout the life course and across biological layers (Vineis et al., 2020).

Concluding remarks

Understanding the mechanisms through which our social-structural environment affects our internal biologies and thus our health allows us to understand why people who experience disadvantage, discrimination, exclusion, marginalisation and domination are not only most likely to be exposed to risk of illness, but are also more likely to be susceptible to severe forms of "dis-ease". We return therefore to the differential exposure and disease susceptibility described above, and the Covid-19 example. The embodiment dynamic is implicitly involved in the effects of socially differentiated exposures and the processes involved in constructing disease susceptibility over the life course. In turn, this helps us to understand why minoritized people, poorer people and people in positions of social domination have been observed to have higher rates of the severe forms of Covid-19 and to be at greater risk of death. Social groups with similar health outcomes are far more likely to have experienced similar socio-historical embodiment mechanisms over the course of their lives than to have a common genetic heritage of disease risk.

The physical embodiment of social-structural experiences is widely misunderstood or under-appreciated, especially in medicine, although it is one of the most important ways in which social inequalities become health inequalities over the course of life. Like all frameworks and models, the embodiment dynamic described here is a work-in-progress allowing us to pursue our scientific research and interpret our findings.

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