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The Problem with the Social Determinants of Health: Moving from Determinism to Structuration in Social Epidemiology

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Abstract

Social epidemiology has long focused on the social determinants of health, often employing a deterministic paradigm. This article critiques this approach by identifying five key conceptual pitfalls: the over-determinist bias; the focus on policies not designed for health; the conflation of statistical causation with social mechanisms; the false dichotomy of ecological and individualistic fallacies; and an unexamined biopolitics. To address these limitations, we propose a theoretical shift toward a structuration approach, which reframes health as a structured process occurring through the interplay of agency and the meso-level institutions that mediate structural factors. This shift is incremental: it calls not for new methods, but for reframing research questions within existing analytical tools. We illustrate the relevance and application of this framework through three empirical domains: the neglect of collective bargaining in research on work and health; the oversight of community resilience in studies on ethnic discrimination and mental health; and the conflation of causal association with mechanism in analyses of retirement. We argue that social epidemiology must replace the essentialist concept of "social determinants" with the dynamic notion of "social factors" and refocus its analytical lens on the institutions where structure and agency interact.

Key words: Public Health, Structuration Theory, Determinism, Causation, Social Determinants

Introduction

Social epidemiology is the study of the social determinants that shape the distribution of health, disease, and well-being within populations (Krieger 2001a). Over recent decades, significant efforts have been made to identify and refine these determinants, highlighting their crucial role in health disparities and positioning social justice as a vital response to adverse health outcomes (Marmot 2005). This importance was solidified by institutions like the World Health Organization (WHO), notably in its 2008 report "Closing the gap in a generation," which called for action on the social determinants of health to achieve health equity (Frank *et al.* 2020; WHO and Commission on Social Determinants of Health 2008).

While social epidemiology has gained immense importance in the scientific community over the past twenty years (Braveman *et al.* 2011) and produced a wealth of evidence (Kaplan 2004), its core findings are part of a long continuum of research on health inequalities that began more than a century ago (Krieger 2001b). The field now faces a fundamental challenge: it primarily focuses on quantifying associations between social structures and health outcomes, without sufficiently addressing the institutions that perpetuate or alter these structures (Farmer *et al.* 2006). This methodological focus limits its ability to instigate meaningful change.

This problem is evident in the field's theoretical foundations. Social epidemiology has long been influenced by sociology, drawing on works such as Durkheim's studies on suicide or Marx's analysis of class (Berkman and Kawachi 2014). However, this influence is somewhat misleading. While often viewed as an offspring of sociology, it actually pre-dates sociology as a formal discipline (Krieger 2001b). Moreover, it has remained largely insulated from broader intellectual shifts in the social sciences (Krieger 2001b). Consequently, much of its framework remains anchored in a reductive lens, framing disparities in terms of a simplistic opposition between groups. This leads to an overemphasis on categorizing populations and establishing statistical causality, rather than critically examining the social processes and institutional mechanisms that sustain inequality.

Thus, while it is well evidenced that socio-economic inequalities have profound health consequences (McLaren *et al.* 2010), social epidemiology frequently positions individuals only as exposed. Studies targeting at-risk groups can inadvertently perpetuate the status quo by neglecting structural roots, while research advocating systemic reform often has limited impact (Raphael 2006) because health is rarely the primary goal of social and economic policy. Dominant frameworks fall short of addressing the root causes it so effectively describes.

A growing body of critique has highlighted this limitation. While social epidemiology has successfully demonstrated that socio-economic inequalities have profound consequences for health, its dominant focus on structural determinants has tended to overlook human agency and the role of institutions that mediate between macro-level structures and everyday life. As a result, research has often struggled to translate empirical findings into actionable public health interventions, particularly when health is not the primary objective of the policies under study.

This article argues that addressing these limitations does not require abandoning quantitative methods or adopting highly abstract theory. Rather, it calls for a shift in perspective: from a predominantly determinist paradigm toward an institutional and relational approach that

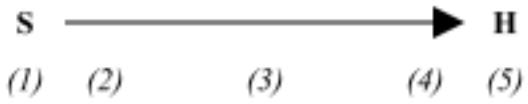
foregrounds the dynamic interplay between structures, systems, and agency. Drawing on structuration theory (Giddens 1984; Lamsal 2012; Whittington 2015), the paper proposes conceptualising health as a negotiated process shaped within meso-level institutions such as workplaces, unions, community organisations, and families.

The article proceeds in three steps. First, it critically examines the determinist assumptions that underpin much contemporary social epidemiology and identifies five recurring conceptual pitfalls. Second, it outlines a structuration-based framework that re-centres institutions as key sites where social inequalities are enacted, mitigated, or transformed. Third, it illustrates the analytical value of this approach using empirical examples from research on work and employment, ethnic discrimination and mental health, and retirement and health.

1. Social determinism and agency in social epidemiology

In this section, I intend to show that the common representation of a direct, deterministic link from society (S) to health (H) is flawed by five key misconceptions. **Figure 1** schematically represents the relationship between the so-called social determinants (S) and possible health outcomes (H) and identifies, at key points along this pathway, the following conceptual pitfalls: (1) the over-determinist bias, which erases human agency; (2) the focus on policies not designed for health, which creates a paradox for research translation; (3) the conflation of statistical causation with social mechanisms, which mistakes a model for a mechanism; (4) the false dichotomy of ecological and individualistic fallacies, which reifies analytical levels; and (5) the unexamined biopolitics of health, which accepts health optimization as a primary imperative without critical scrutiny.

Figure 1. Five pitfalls on the relationship between society and health



1.1. The over-determinist bias

The dominant paradigm in social epidemiology conceptualizes a deterministic relationship from society (S) to health (H). In this model, 'S' represents the ensemble of variables that reflect macro-social structures. This S → H framework operates on the underlying perception that individuals possess little to no agency, being primarily shaped by determinants beyond their control (Kirkbride *et al.* 2024). In such a perspective, the social determinant of health may be defined as "the conditions in which people are born, grow, live, work and age that shape health" (Artiga and Hinton 2018).

This deterministic logic is concretely exemplified in mainstream explanatory frameworks. For instance, Bartley's typology of five causal pathways – material, cultural-behavioural, psycho-social, lifecourse, and political-economic – systematizes this S → H logic (Bartley 2004). While analytically useful, the framework presents health as the predetermined output of these macro-mechanisms. The stated aim is policy relevance; accurate structural explanations are

deemed necessary for intervention. Yet, as Bartley notes, evidence of policy impact from such studies is lacking.

It is true that the medical field has long overlooked socio-economic inequalities and that emphasizing structural dynamics is crucial for shifting from an individual focus to a more structural perspective – moving from sick individuals to sick populations (Rose 1985). Not so long ago, some authors positioned themselves against the very name of the discipline arguing that "Epidemiology is part of medical science and rests on a human-biological (scientific) background," (Zielhuis and Kiemeney 2001) thereby rejecting any potential complementarity between social sciences and epidemiology. Although this perspective was criticized (Muntaner 2001) and ignores the psycho-social dynamics explaining physical and mental health outcomes besides established pathologies (Kasl and Jones 2002), it reveals some truth: despite its now-established status, there remains a degree of resistance to a field that seeks to combine medical and social sciences but is often accused of doing both poorly, for example by limiting the root causes of diseases to social and economic factors (Kaplan 2004) However, one must not fall at the other side of the spectrum where all health outcomes are analyses through the lens of the social determinants of health.

Since the WHO 2008 report, the nature of these so-called determinants has been questioned using different theoretical approaches.

For instance, addressing the duality between structures and individuals, Abel and Frohlich have argued for the Sen's capability approach (Abel and Frohlich 2012) to take into consideration both the structural conditions and the active role of individuals in shaping their health. However, the problem in this type of approach is that it locates agency – and the possibility of social change – at the individual level, reproducing a duality between the structures and the individuals.

Another type of critique comes from The Latin American Social Medicine that has pointed out the necessity to better understand the mechanisms of domination and the power structure that shape health rather than indicators of such inequalities (Harvey *et al.* 2022). The same criticism was found in the debate surrounding Wilkinson's income inequality hypothesis. Wilkinson posits that beyond a certain threshold, relative inequality and the psychosocial stress of social hierarchies most damage population health (Pickett and Wilkinson 2015). While pivotal, this model has been critiqued for not going far enough. Wilkinson's model would only treat a symptom (Scambler 2012a, 2019) rather than identifying the root causes, i.e. the political-economic forces of neoliberalism that empowers capital and weakens labour (Coburn 2004), following a traditional Marxist perspective that looks at the production and distribution of wealth across populations (Harvey 2021).

Whilst these approaches challenge the social determinant framework by underlying both the roles of individuals in shaping their health and the social process and power relationships that explain health inequities, they struggle to translate into proper empirical evidence because they recast the structure-agency relationship as a binary division, a framing that ultimately validates a deterministic view of the social determinants.

1.2. Social epidemiology addresses policies that do not primarily target health

Another pitfall of the impact of the society on health is that the policies implicated in the analysis of the social determinants of health are often not designed with explicit public health objectives in mind (Rothstein 2002). Developing models that extend beyond the traditional public health spectrum may leave little hope of achieving tangible policy impact. Rothstein, for example, advocated for a narrow vision of public health, arguing that only activities falling within a strict definition of public health should be considered as potential areas for government intervention (Rothstein 2002). This perspective, however, was criticized, as the question of which policies can be enacted is distinct from the question of what is ultimately good or bad for health (Goldberg 2009).

For instance, whilst positive public health outcomes depend on reducing socio-economic disparities, limiting public health to government interventions directly targeting health would ignore a significant part of what actually affects health outcomes. In this context, policies promoting a fairer distribution of wealth would also qualify as public health interventions. Given that the majority of diseases worldwide are attributable to "the social conditions in which people live and work" (Stonington and Holmes 2006), a public health approach that ignores social epidemiology and focuses exclusively on health-specific government interventions might achieve some impact, but it would fall far short in addressing the broader causes of population health.

This is the paradox of much research in social epidemiology: it often examines the unintended effects of policies that were neither designed to impact health nor prioritized for their health consequences. In other words, it often seeks to quantify effects that were neither anticipated nor, to put it bluntly, deemed important enough to predict. This is a true paradox. While social epidemiology identifies the root causes of population health and is essential for understanding public health dynamics, it must also critically examine its own impact.

The consequences are profound. When Bartley's five pathways (material, cultural-behavioural, psycho-social, lifecourse, political-economic) are analysed, their interactions are reduced to a mechanistic flowchart. Education, for instance, appears as a fixed "determinant" rather than a contested field were institutional racism, parental agency, or policy shifts might mediate its effects. The bidirectional reality – where health shapes social trajectories as much as *vice versa* – is erased.

By conditioning its lens on health disparities as the inevitable collision of structure and vulnerability, social epidemiology might echo the very dominant logic it opposes. Individuals are rendered calculable, their suffering predicted but not prevented, while the state and scientific institutions retain authority to define both the problem and (not very often) its solutions.

1.3. Statistical causation and social mechanisms

One of the key feature of social epidemiology is that it aims to address causal relationships. Causal statistical methods provide powerful tools for identifying the effect of a variable S on an outcome H over time. They often achieve this by leveraging the temporal dimension of the data or test counter-factual scenarios to control for unobserved, time-invariant characteristics

of the units being studied. In essence, these models ask a refined question: for a given entity, does a change in *S* lead to a subsequent change in *H*, after accounting for all the stable but unmeasured factors that make that entity unique? When applied rigorously, such methods can produce a robust estimate that is consistent with a causal effect of *S* on *H*, providing strong evidence that the relationship is not spurious. The development of longitudinal methods in social epidemiology has led to a frenetic quest for causation (Goldberg 2009; Parascandola and Weed 2001).

However, establishing statistical causation is not synonymous with capturing the full reality of the social mechanisms. A model might demonstrate that *S* has a genuine effect on *H*, but this finding exists within a severely simplified representation of the world. The social world is an open system characterized by immense complexity, interdependency, and meaning (Krieger 1994). In this reality, the effect attributed to *S* is always embedded within a dense web of other influences that are never fully accounted for in any statistical model. These unobserved variables represent the myriad social forces, cultural contexts, historical contingencies, and individual interpretations that constitute the true mechanisms of social life; these are the factors social epidemiology should look at.

It has been argued that public health, and specifically social epidemiology, has overemphasized methods in recent years (Smith 2016). Increasingly complex methodologies are being used to address causality and analyse intricate datasets, often facilitated by the growing availability of linked administrative data. The development of computing tools and artificial intelligence has and will transform public health into a highly specialized field. While it is undeniably beneficial to explore causality through quantitative research, and the influx of new data and methods certainly enhances our ability to investigate health issues, one must ask: What is the point if the questions we ask remain unchanged?

1.4. Ecological fallacy, individualistic fallacy and determinist fallacy

A common, though insufficient, response to the over-determinist bias in social epidemiology is methodological rather than theoretical: shifting the level of analysis. Here, the simplistic S → H model is expanded into a multilevel framework. This approach distinguishes between individual-level analyses (focusing on personal attributes and outcomes) and ecological or group-level analyses (focusing, for instance, on the averages or characteristics of neighbourhoods (Olagnero *et al.* 2005), or areas of material deprivation (Robinson 1950)).

The well-known ecological fallacy warns against inferring individual-level relationships from group-level data. Conversely, the atomistic or individualistic fallacy occurs when analysis restricted to individual data omits the genuine effect of macro-social contexts (Marmot 1998). This analytical duality creates a persistent challenge. Researchers have long argued that the solution lies in integrating data across levels and accounting for both the composition of groups (the types of individuals within them) and their context (the features of the environment) (Macintyre and Ellaway 2000; Subramanian *et al.* 2009). The goal is to avoid both fallacies by interrelating individual and contextual variables (Scheuch 1967).

However, this focus on navigating between fallacies at different "levels" is itself a determinist fallacy. It does not resolve the core theoretical issue but merely displaces it. Whether the

exposure variable is an individual's income (micro) or a neighbourhood's poverty rate (macro), the underlying logic remains deterministic. The explanatory variable, now situated at a higher level, is still conceptualized as an external, structural force acting upon individuals to determine health.

Consequently, even sophisticated multilevel modelling often perpetuates the determinist paradigm. It creates an artificial divide, suggesting that "levels" are ontologically distinct rather than co-constitutive. From this perspective, an individual's unemployment status and their community's unemployment rate are merely different methodological points of entry for analysing the same overarching social force. The framework of opposing fallacies presumes a choice between the individualistic fallacy and the ecological fallacy, when the more fundamental error is the determinist fallacy: the reification of social relations – whether measured at the micro or macro level – as fixed determinants of health. The task is not to choose the correct "level" of determination but to dismantle the deterministic framework itself.

1.5. Why health?

This leads to a more fundamental, and often unexamined, question: why is population health itself such an important object of concern? To interrogate this is to move beyond the discipline's internal debates and into the realm of biopolitics. As developed by Michel Foucault, biopolitics names the historical moment when political power began to take as its object the biological life of the population, aiming to manage and optimize its health, longevity, and reproductive capacities (Foucault 1997).

This biopolitical rationality manifests at distinct yet interconnected scales. At the national level, people's health ceases to be merely a moral good and becomes an economic and political resource. A productive workforce, a robust military, and a stable social body are the implicit goals that have long justified state intervention in public health, from sanitation systems to vaccination campaigns. Concurrently, at the international level, this logic expands. Global health initiatives, metrics and health targets represent a transnational biopolitics (Bashford 2006). Here, the health of populations becomes a key indicator of human development, a prerequisite for economic growth, and a matter of global security and governance. For instance, the recent interest for the notion of "active ageing", "healthy longevity", "successful ageing" (Leveille *et al.* 1999) or "healthy ageing" in social epidemiology (Scott 2021) reflects international institutions – such as the United Nations or WHO – concerns and have entered the field of social epidemiology without being very much discussed.

Placing social epidemiology within this biopolitical framework forces a critical reflection on its foundational premise. The field's compelling documentation of the social gradient in health and its advocacy for health equity can be seen as a progressive form of biopolitics. It argues, in essence, for a more equitable and inclusive administration of life, challenging the state and other actors to optimize health outcomes for all. However, this very positioning risks reinforcing the core biopolitical tenet: that life must be managed, and that individuals and populations are, above all, subjects whose health must be measured, assessed, and optimized for the proper functioning of the social and economic order. Social epidemiology, for all its critical intent, must therefore confront this paradox: in its quest to demonstrate how social

structures make us sick, it may take for granted why health is an imperative, potentially reinforcing the very systems of valuation it seeks to challenge.

The critic of social epidemiology does not have to adopt a radical or oppositional stance. The real challenge lies in avoiding the current fragmentation between two largely disconnected worlds: on one side, those who critically analyse biopolitics; on the other, those who, often inadvertently, operate within and reproduce its logics. There is a middle ground. This is where social epidemiology could renew itself – by subjecting its own concepts, methods, and measures to reflexive scrutiny, and by aligning them with aims that extend beyond the technical objectives of researchers or the rationalities of the state. This is therefore not an argument against public health action, but for a more reflexive engagement with the ways in which appeals to health can simultaneously enable care, regulation, and social ordering. To find this middle ground requires acknowledging that every analytical choice – what we define as "determinants", what we measure, what we seek to improve – carries implicit political commitments. These questions invite a social epidemiology that remains empirically rigorous yet philosophically aware, critical yet constructive – a discipline capable not only of explaining why inequalities make us sick, but also of interrogating the broader systems that define what it means to be healthy, and for whom.

2. A Structuration Approach

To move beyond the determinist approach, social epidemiology requires a fundamental recalibration of its perspective, not a change in methods. The field is rich with sophisticated tools, but they are too often deployed within a framework that reifies social structures. The solution lies in reframing research questions through a lens that can accommodate the dynamic, recursive nature of social life. Anthony Giddens' structuration theory may provide this necessary corrective, directly addressing the core pitfalls identified in this article.

Giddens' central concept, the *duality of structure*, offers a powerful antidote to the determinist fallacy. It posits that social structures are not external, immutable forces that unidirectionally determine action. Instead, they are both the medium and the outcome of the practices they recursively organize (Giddens 1984). Structures provide the rules and resources that shape action, but they have no existence independent of the human agents who, through their daily practices, continually reproduce and sometimes transform them. This dissolves the false "macro" versus "micro" dichotomy that sustains both the ecological and individualistic fallacies. It reveals that a "social determinant" is not a static entity but a constantly evolving product of human action. In other words, it goes beyond than an oppositions between agency and structures, as approached by a public health (McLaren *et al.* 2010) that has constantly omitted what links them (Frohlich and Potvin 2010). In that sense, the agency is neither "a matter of individual will and skill" (Whittington 2015), it is rather the control individuals have over resources and how this control is exercised.

Building on the duality of structure, Giddens introduces the notion of the system to describe how social relations are organised and reproduced. Systems represent the patterned interactions and institutions that emerge from the ongoing flow of social practices. They are not structures in themselves, but the observable outcomes of structuration – i.e., the recursive process through which agents draw on structural rules and resources in their actions. Through repeated social

practices, individuals contribute to the formation and stabilisation of systems such as family, economy, education, or health care, which in turn provide the context within which future actions take place. In this sense, systems embody the continuity of social life: they persist because agents continually reproduce the relations that sustain them. However, systems are also sites of change, as modifications in practice or shifts in access to resources can transform the rules and relations that underpin them. By articulating the concept of system, Giddens provides a way to connect the everyday activities of individuals with the larger, enduring configurations of social life without reducing one to the other.

Applying this to health leads to the concept of the *structuration of health*: the dynamic process through which health outcomes are shaped by and shape social structures, via the agency of individuals and collectives and within social systems. Health is not a terminal endpoint in a causal chain, but an ongoing negotiated process. This negotiation occurs through meso-level institutions (e.g., workplaces, community organisations, families). These institutions are the very arenas where the abstract force of a macro-structure meets the concrete reality of human life, and is thereby enacted, resisted, or altered. Unlike approaches that locate agency primarily at the individual level (e.g. capability approaches) or privilege structural causation, structuration explicitly theorises the institutional processes through which structure and agency are recursively linked.

This shift directly tackles the pitfall of essentialism. The term "determinant," which reifies complex social phenomena into static, one-dimensional constructs, must be retired in favour of *social factor*. A factor is an element in a dynamic system, not an ultimate cause. It allows for bi-directionality and mediation. For instance, "precarious employment" is not a determinant of poor health; it is a social factor whose relationship to health is mediated by the presence or absence of effective institutional buffers, such as collective bargaining agreements. This reframing solves the problem of reducing individuals to passive vectors of structural forces by explicitly building agency and negotiation into the analytical model.

This shift is not theoretically demanding and does not require new statistical techniques; it requires embedding our existing methods within a more sophisticated theoretical framework that addresses the systems that produce health outcome. The same modelling that currently estimate the "effect of X on Y" can be repurposed to investigate processes of structuration. The research questions, however, must transform to avoid analysis that solely focus on exposure and damage.

Concretely, we could operationalize a structuration-informed approach by explicitly incorporating meso-level institutions as mediators or moderators of social factors in our analyses. Existing quantitative methods – such as multilevel models, structural equation models, or longitudinal designs – can be repurposed to capture how these institutions shape, buffer, or amplify the effects of social factors on health outcomes. This approach allows researchers to move beyond deterministic effect estimates, reframing health as a dynamic, negotiated process in which agency and structure continuously interact. By embedding analyses within this perspective, social epidemiology can shift from documenting static disparities toward understanding the mechanisms and conditions through which health outcomes emerge and change.

Figure 2. The explanatory power of structures, systems and agency to understand health outcomes

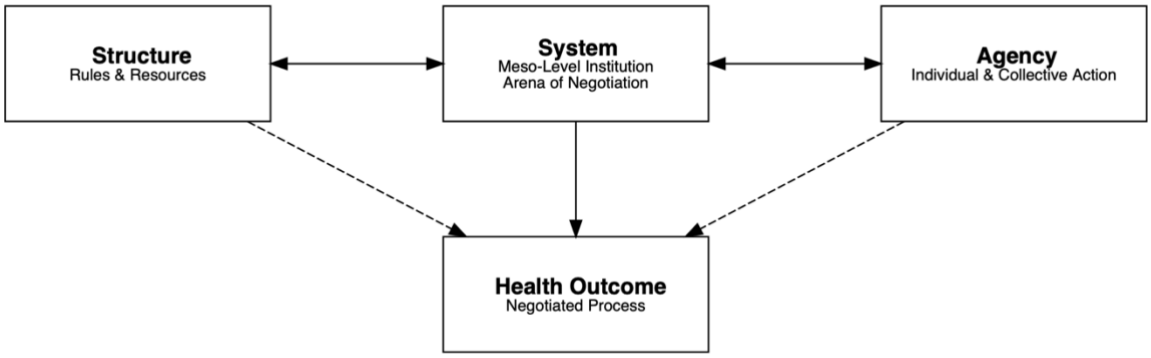


Figure 2 shows that health is the result of a process involving three key elements: structure, system, and agency. For a long time, most research in social epidemiology has focused only on structure – the large-scale social and economic forces that shape health outcomes. Some studies have also looked at agency, the power of individuals and communities to act. In both cases, the relationship with health outcomes is not direct (as shown by the dashed lines). Structure and agency do not directly affect health outcomes – they rather contribute to shape them via systems where the everyday life is negotiated on a daily basis. This middle element has been largely forgotten. Systems are the real-world institutions, like workplaces, unions, or community organizations, where structure and agency actually meet and interact. This is where the rules are applied, resources are distributed, and people can push back. By ignoring these systems, we miss the most important part of the story: the actual process through which health outcomes are created. To truly understand and improve health, we must stop looking just at the large forces or just at individual actions and start focusing on the systems where their negotiation takes place, succeeds or fails. Such a perspective avoids both an over-determinist approach where the structures act on health independently of individuals (Bartley 2004; Scambler 2012b) and an over-individual approach where capabilities are seen as a key ingredient of health (Abel and Frohlich 2012).

3. Examples

3.1. Example 1: The work and employment determinants of health

The neglect of negotiation as a central paradigm is starkly visible in social epidemiology's approach to work and health. For decades, extensive research has meticulously documented how precarious employment – characterized by job insecurity, irregular hours, and low wages – correlates with a spectrum of adverse physical and mental health outcomes (Anon 2024)·(Vahtera *et al.* 1999). Yet, this vast body of literature has largely reproduced the determinist fallacy, framing precarious workers as passive victims of broader economic forces. The very etymology of 'precarious' means 'obtained by begging or prayer', which reveals a profound power imbalance and a state of dependency that the research inadvertently reinforces by focusing solely on the damage inflicted.

This lens presents precarious employment as a monolithic determinant, a fixed structural position to which individuals are condemned. It systematically overlooks the critical distinction between flexible work, which can be a voluntary and empowering arrangement, and genuinely precarious work, which is defined by a lack of agency and security. By conflating the two, the field erases the possibility of choice and negotiation, reducing all non-standard employment to a singular health risk. This perspective ignores the reality that for some, flexibility is a desired adaptation to life's demands, while for others, precarity is an imposed condition of exploitation.

Crucially, this deterministic framework has led to a glaring omission in the research: the role of trade unions and collective bargaining as fundamental institutions of negotiation. After forty years of studying work-related health inequalities, only a very small number of studies have considered unions as a central variable (Benach and Muntaner 2007). When unions are mentioned, it is often within the same reductive framework of precarious employment, as another variable in the struggle between labour and capital, rather than as a dynamic meso-level institution that actively mediates this relationship. The primary focus remains on quantifying the health deficits of the 'precariat', rather than on investigating how collective action can successfully transform precarious conditions.

By ignoring these institutions of negotiation, social epidemiology has underemphasised why similar structural positions can lead to vastly different health outcomes. It cannot account for the workplaces where collective agreements have mitigated hazards, ensured living wages, and provided dignity – effectively shielding workers from the health-damaging effects of potential precarity. The field's focus on exposure and damage, to the exclusion of agency and collective resistance, has thus produced a body of evidence that is strong on diagnosing problems but weak on identifying viable pathways toward their resolution. It has documented the consequences of having to *pray* for one's livelihood, while paying little attention to the institutions that allow workers to *demand* it.

In research on the relationship between work and employment, the determinist fallacy of framing precarious workers as passive victims is countered by shifting the research question. Instead of documenting the damage of job precarity, we could ask: "To what extent does union membership moderate the relationship between job precarity and psychological distress?". This introduces institutions or groups, like trade unions, as a meso-level institution of negotiation, explaining variance in health outcomes by focusing on the collective agency that can transform structural conditions.

3.2. Example 2: Ethnic discrimination and mental health

Another pertinent example concerns the health of discriminated ethnic groups, which has been highlighted in the context of the United Kingdom's hostile environment policies. This approach, promoted by the Conservative Government from the mid-2010s to the early 2020s, had far-reaching implications for the health and well-being of ethnic minorities and migrant populations. Initially, these policies specifically targeted Black British citizens from the so-called Windrush Generation, treating documented citizens as though they were illegal migrants (McKee 2018). This led to devastating consequences, including loss of access to employment, housing, and healthcare, as well as wrongful detentions and deportations. Over time, the hostile environment policies were expanded into a broader framework that encompassed tougher

immigration laws, rising visa fees, and increased costs for accessing the National Health Service (NHS). These measures were further compounded by the socio-political effects of Brexit, which exacerbated anti-migrant rhetoric and reinforced barriers to healthcare access for migrant and minority groups.

Research has demonstrated that these policies have not only intensified structural racism but also contributed to widening health disparities (Jeffery *et al.* 2024). While documenting this damage is a necessary empirical task, the prevailing analytical framework often remains trapped in a deterministic paradigm. Minority groups are perceived not as complex agents but as a problem to be studied, their lived reality and intellectual potential circumscribed by the damages caused by some exposure.

This may overlook the crucial meso-levels (Bailey *et al.* 2017): the community organizations that provide legal support and solidarity, the religious groups that offer sanctuary, the informal networks that share resources and information, and the individual acts of resilience that constitute a daily defiance of a hostile system. By focusing solely on the damage, social epidemiology inadvertently reproduces the very power dynamics it seeks to critique, presenting a picture of a monolithic state acting upon a vulnerable and undifferentiated population. Addressing the impact of the social determinant of health framework, Irwin and Scali has indicated that "policy action on social determinants has generally emerged in response to demand from civil society organizations and communities mobilized to give political expression to their needs"(Irwin and Scali 2007) – this is where impact can be found.

A more robust approach would shift the focus from determination to structuration. It would examine not only how the hostile environment produces mental ill-health but also how the capacity of individuals and communities to negotiate its pressures – through collective action, legal challenges, and cultural resilience – can protect mental well-being. The question is not merely to prove that structural racism harms mental health, a fact that is known, but to understand the conditions under which this harm is exacerbated or attenuated. This requires moving beyond a framework that conditions analysis on victimization and towards one that investigates the active, albeit constrained, processes of institutional and individual negotiation that ultimately shape the mental health outcomes of living within a discriminatory social order.

3.3. Example 3: Health and Retirement

In social epidemiology, a significant body of research has focused on the relationship between retirement and health, often motivated by the national policy concern of increasing the state pension age. This literature typically asks a direct and apparently simple question: what is the causal impact of retirement on subsequent health and mortality? Studies using advanced longitudinal methods have reported associations between retirement and negative outcomes such as increased post-retirement mortality and accelerated biological ageing (Furuya 2025; Furuya and Fletcher 2024). From these statistical correlations, a deterministic policy conclusion is frequently drawn: to protect population health, the retirement age should be raisexd.

This line of inquiry and its policy translation are problematic on several grounds. Methodologically, while studies control for factors like education and occupation [50], they

often inadequately address the issue of health selection – the fact that poor health is a major cause of retirement (Han 2021) (Miah and Wilcox-Gök 2007). This creates a persistent risk of reverse causality, where pre-existing health decline is misattributed to the act of retiring.

More fundamentally, this research exemplifies the conflation of statistical causation with social mechanisms. It reduces the complex, multifaceted social institution of retirement to a binary or timed exposure variable. Retirement is not a singular event but a major life-course transition that reconstitutes an individual's daily structure, social networks, economic status, and access to services. The transition can alter healthcare access (Dor *et al.* 2006), increase risks of loneliness or social isolation (Tanaka *et al.* 2018) (Mansfield *et al.* 2023), disrupt sleep patterns (Vahtera *et al.* 2009), change dietary habits (Lagström *et al.* 2025), and redefine social roles and care responsibilities (Potočnik and Sonnentag 2013). A statistical model identifying an "effect" of retirement cannot capture how these interwoven mechanisms are actively negotiated by individuals, thereby mistaking a simplified correlation for a comprehensible social process.

Ultimately, this approach engages in an unexamined biopolitics. It subjects the social right to retirement—a institution created to protect vulnerable populations (Kohli 2007) – to a cost-benefit analysis based on population health metrics. The deterministic question ("Does retirement cause harm?") implicitly makes the legitimacy of a social right contingent upon its health outcomes, reinforcing a managerial logic that optimizes life for economic productivity rather than human flourishing.

Applying a structuration paradigm transforms this inquiry. It replaces the search for a monolithic causal effect with an investigation into processes of structuration. A more pertinent question becomes: "How do access to social networks, community participation, and institutional support mediate the relationship between retirement transition and health outcomes?" This reframing shifts the focus from retirement as a health determinant to post-retirement life as a contested field where structure and agency interact. It aligns social epidemiology with its emancipatory potential, seeking not to restrict social rights based on statistical risks, but to identify the institutional conditions that enable a healthy and agentic life after work.

Conclusion

The central argument of this paper is that social epidemiology, despite its emancipatory ambitions, has become constrained by a predominantly deterministic analytical framework. While this framework has been highly effective at documenting health inequalities and establishing the social patterning of disease, it has also narrowed the kinds of questions the field is able to ask. By prioritising the identification of structural determinants and their effects on health outcomes, social epidemiology has tended to privilege explanatory models that emphasise exposure and damage, often at the expense of understanding the social processes through which these effects are mediated, negotiated, or transformed.

This limitation is not the result of a lack of critical intent. On the contrary, the field's commitment to social justice and health equity has driven much of its most important empirical work. However, the cumulative effect of a determinant-focused paradigm has been to position individuals and groups primarily as recipients of structural forces, rather than as agents

embedded within institutions that can modify, resist, or reconfigure those forces. As a result, social epidemiology has excelled at describing the consequences of inequality but has been less effective at systematically theorizing the conditions under which change occurs

A potential solution to these pitfalls is to consider health through the lens of structuration theory, as a negotiated process. This means understanding health not as a predetermined endpoint, but as the dynamic and bidirectional product of the interplay between individual agency and the meso-level institutions (like unions or community groups) that mediate the influence of macro-structures.

It is entirely possible to adopt a progressive perspective while rejecting simplistic interpretations of social inequalities. The change required is profound in its implications yet minimal in its practical demands. It does not necessitate to give up on sophisticated quantitative methods, quite the opposite. It rather calls for reframing the questions we ask of our data. The point is not to discard our models, but to ensure they are deployed in the service of better questions. The structuration theory might provide the framework for this shift, moving us from asking "What is the damage?" to "What are the conditions of successful structuration?". By doing so, we realign the purpose of social epidemiology with its original, emancipatory goal: to understand the world not merely to document its injustices, but to illuminate the pathways through which it can be changed.

The critique developed throughout this article is not intended as a dismissal of the social epidemiology project, but as a necessary recalibration to reach its emancipatory potential. To move beyond the determinist fallacy, the field could embrace a new set of foundational principles. The following recommendations outline a path for this essential renewal:

Reframe core research questions from "what" to "how" and "under what conditions”. Instead of asking only "What is the effect of social factor X on health outcome Y?", social epidemiology must prioritize questions like: "Through what social processes is this effect mediated?" and "Under what institutional conditions is this effect amplified or mitigated?" This shifts the focus from documenting deterministic relationships to understanding the dynamic mechanisms and contingencies that shape health inequalities.

Adopt a structuration paradigm to replace the determinist fallacy. Conceptualising health as a structuration process would allow social epidemiology to move beyond viewing health as a predetermined outcome of social forces. This perspective invites researchers to reflect on how their research objectives and questions are framed, and to situate empirical analyses within a structuration framework that can enrich interpretation while remaining compatible with established quantitative methods.

Make meso-level institutions the central unit of analysis for understanding structuration. Research should explicitly identify and investigate the institutions – such as trade unions, community organizations, family networks, and healthcare systems – that serve as the primary arenas where macro-structural forces are translated, resisted, and negotiated. Understanding how these institutions buffer or exacerbate social determinants is key to identifying actionable levers for intervention.

Replace the concept of "social determinants" with "social factors" to avoid essentialism. The term "determinant" implies a fixed, unidirectional, and reified causal force. Adopting the more neutral term "social factor" acknowledges that these elements operate within a complex, open system of bidirectional relationships, mediation, and moderation, thereby opening the analytical space for agency and negotiation.

Methodologically, shift from estimating main effects to modelling effect modification by institutions, systems or agency. The sophisticated quantitative methods already in use should be repurposed. The primary analytical goal should become testing how the relationship between a social factor and a health outcome is modified on the presence and strength of negotiating institutions or expressions of agency.

Questioning health outcomes as a policy target. Social epidemiology must engage in critical self-reflection to ask *why* population health is such a paramount object of concern. This involves scrutinizing whether the field's efforts to optimize health inadvertently reinforce a managerial logic that serves state and economic interests and to ensure that its aims align with genuine human liberation rather than administrative rationalities. Above all, research should critically engage with policy notions such as healthy or successful ageing.

The change social epidemiology needs is incremental. The field's interest in structural determinants has provided a wealth of evidence but redirecting our focus to the crucial middle ground, i.e., the everyday systems and institutions where structure and agency actually meet, would be a way not to repeat ourselves. By finally studying the systems, we stop just proving that the world makes people sick and start understanding how people, through their collective institutions, can make a healthier world.

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