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Systemic Materialist Realist Ontology, Epistemology, Semantics, Axiology and Ethics: Why Pearl's Counterfactuals and Pawson's methods are insufficient to Causation in Social Epidemiology and Public Health

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Abstract

We evaluate counterfactual empiricism, the dominant philosophy of science in contemporary epidemiology and public health from a scientific realist perspective. Building on our earlier work, we critique the dominant counterfactual/potential outcomes epistemology in epidemiology and public health, based on its neglect of ontology and exclusion of causal mechanisms which are confused with statistical inference methods (e.g., mediation). We argue that a realist systemic materialist ontology of levels, scientific realist epistemology, and realist semantics, axiology and ethics could constitute a best philosophical system for the disciplines of social epidemiology and public health, and their social inequalities in health subdivisions.

Keywords: Scientific Realism; Empiricism; Semantics; Values; Ethics; Counterfactuals; Potential Outcomes; Epidemiology; Public Health; Social Determinants of Health; Social Inequalities in Health; Social Epidemiology

Abbreviations: CF: Counterfactual; PO: Potential Outcomes; CCL: Contradictory Class Location; IBE: Inference to the Best Explanation.

The renewed interest in causality in epidemiology in the last two decades [1-3] has led to a debate around the counterfactual/potential outcomes approach [2-10]. Without questioning the necessity of causal inference investigations, the sole focus on causal inference (e.g., potential outcomes/counterfactual approaches) are missing aspects of causality that are necessary to understand social (economic, political, cultural) systems. Drawing from debates on causation in social epidemiology [11-19] we challenge the exclusive emphasis on debating (counterfactual/potential outcomes) [2] as well as constructivist [3] (empiricist) epistemologies in social epidemiology and public health. As an alternative, we propose a unified "Systemic Scientific Realist" approach [18,20] that includes all features of philosophy of science (Table 1) as follows:

- A systemic realist materialist ontology of levels: the world is external to the observer and made up of concrete systems with emergent properties.
- A scientific realist epistemology: this objective world can be known, at least in part and gradually, with the use of the scientific method and the uncovering of explanatory causal mechanisms.
- Realist semantics: there are true factual hypotheses or some propositions about the world are true.
- Realist axiology/values: at least some values are

objective.

- Realist morals: at least some behavioral prescriptions, or moral principles are true.

Philosophies of Science Compared					
Philosophy of Science	**Ontology**	**Epistemology**	**Methodology**	**Semantics**	**Values/Morals**
Scientific realism	The external world exists independently of our sense experience, ideation, and volition/ Systems and levels	The World can be known, yet partially and progressively / casual mechanisms	The best way to understand the world is the scientific method	Some factual hypotheses are true	There are objective values & true moral Precepts
Counterfactual Empiricism	The world is a set of experiences	The source of knowledge is experience with the imagination of counterfactual worlds/ associations and casual inference	The best way to understand the world is using the counterfactual imagination of non-existing worlds	Some factual hypotheses are close to being true/ truth is subjective	Values and ethics are Neutral
Critical Realism (most)	The external world exists independently of our senses, cognition and behavior	All we can know mostly is subjective/scientific indeterminism	The scientific method is not superior to other forms of knowledge (intuition, personal experience, mysticism)	We cannot know whether an hypothesis is true	Values are subjective/ Morals are relative

Table 1: Three Philosophies*of science in Epidemiology and Public health compared in terms of their Ontologic, Epistemologic, Methodologic, Axiological and Moral stances.

***Empiricism** shares with counterfactual empiricism its ontology, axiology, and morals, yet its epistemology and methodology does not include the imagination of counterfactual worlds. Its methodology relies on the scientific method exclusively.

Crucially, in affirming the priority of ontology over epistemology, we overcome the impasse of empiricism and its limited success in bringing about explanations via uncovering causal mechanisms in social epidemiology (Muntaner, 2003) [14]. Our focus on social epidemiology stems from our familiarity with its subject matter, yet our critique and proposal may be generalizable to other area of epidemiology and beyond.

In the next sections we develop our critique of what we consider the standard model of contemporary social epidemiology, the counterfactual/potential outcomes empiricism (Pearl, 2018). The counterfactual definition causation (or "third rung in the Ladder of Causation" according to Pearl) abandons the regularity account of empiricists "A causes B" for the counterfactual "B would not have occurred if not for A". Thus, causality requires the imagination of alternative potential worlds. The potential outcomes version of counterfactual causation states that "the Potential Outcome of a variable Y is the value that Y would have taken for individual (u) had X been assigned the value x" or $Y_{X=x}(u)$ (Pearl, 2018). We also point to the shortcomings of two approaches to causation in social epidemiology and public health respectively, the constructivist/empiricist approach of inference to the best explanation (a form of idealism, like counterfactual empiricism) and Ray Pawson's critical realism, an incomplete realist ontology and epistemology [21], which we address first.

The distinction between Systemic Scientific Realism (SSR) and Pawson's Critical Realism

In table one we present the differences between the major epistemology in epidemiology and public health (counterfactual empiricism) and we contrast it with scientific realism and critical realism (popular mostly in public health via the work of Ray Pawson) which we want to differentiate with our proposal. Pawson's critical realism is a method

(Context Mechanism Outcome configurations) to uncover unobservable psychosocial mechanisms (e.g., a person's attitudes, motives, intentions, interpersonal trust). This realist method is consistent with ontological realism (the view that there is a world outside us (the observers) and that our senses are too limited to understand it). But Pawson's critical realism has a narrow focus on psychological and psychosocial levels, therefore does not encompass the ontology of levels of scientific realism (Aristotle's). Furthermore, Pawson's critical realism is not systemic. Its ontology is concerned with "Context" "Mechanism" and "Outcomes". The systemic ontology of scientific realism on the other hand, defined the world as formed of systems (with boundaries, components, relations, mechanisms, emergent properties, and levels). While Pawson's incomplete realism finds unobservable mechanisms, it does not tell us what elements (e.g., persons) and what relations bind those elements. Therefore, its realist ontology is incomplete. For example, uncovering as that a mechanism of racial discrimination produces ill health does not tell us who takes part in this mechanism (e.g., a White and a Black person), what is their relation (e.g., supervisor-worker) and in what system they are (e.g., a work organization). Most critical realism (including Pawson's) is not only at odds with systemic scientific realism with regards to ontology, but also with regards to epistemology. Scientific realist epistemology claims that only science helps us to overcome the gnoseological limitations of our senses (we do not see societies, but we can understand them with survey samples). Most critical realism, on the other hand, does give science a privileged status as a way of knowing the world, falling close to skepticism.

Counterfactual/Potential Outcomes Empiricism vs. Scientific Realism: causal inference vs. causation

The key features of scientific realism stem from its ontology, epistemology, semantics, values, and ethics. Realist ontology means that "the world exists independently of our knowledge of it" (Sayer 1992); for our purposes, a population, and the social phenomena we are examining exist independently of the epidemiologist or public health interventionist who sees it. Ontological realism also claims that our senses are too limited to understand the world and that we need theories, which refer to unobserved explanatory causal mechanisms, not only experience (i.e., data), to make sense of it. The scientific realist epistemology states that at least parts of the world can be known with the scientific method. Scientific realism [22] is preferable to critical realism [23] because the later does not emphasize the scientific method as means to know about the world [18].

The philosophical antecedents of the counterfactual/potential outcomes (CF/PO) popularized by Pearl (2018) (or the modern epidemiology of Rothman earlier) are British empiricists (Hume and the empiricist cluster of epistemologies that he inspired). In addition to his empiricist epistemology Hume was a defender of social inequality as per his elitism and attitudes about on race, indigenous peoples, and colonialism (Prescott 2018). Such observation might appear an opportunistic attack on the epistemologist, but it is our claim that ontology and epistemology set constraint on values [14,18]. On the other hand, modern epidemiology and public health neglects the realist approach to medicine starting with Claude Bernard and his follower Ramon Turro i Darder, up to contemporary French scientists such as the biologist Francois Jacob[1].

The CF/PO approach does indeed centers around causal inference, and the empiricist epistemology has a well know reluctance to talk about causation (but not about causal inference). Note that even the epidemiologic critics of the CF/PO approach are reluctant to talk about causation preferring the constructs of "lack of theory" (Krieger 2000) or Lipton's "inference to the best explanation " (abductive reasoning) (Krieger and Davey Smith 2017). True, most problems in epidemiology, just like in clinical medicine and nursing, are of the reverse kind, where the researcher begins with the effect, in search of the cause. Yet, we find problems with Krieger and Davey Smith's ontology and epistemology, namely with 1) its constructivism an instance of anti-realist idealism; and 2) its (reverse) empiricism which cannot lead to the trans-empirical constructs (i.e., a denial of meta-physics) which we need in any advanced scientific explanation [14]. Thus, the crucial influence of empiricism in current epidemiology is its neglect of ontology, causation as an unobserved real process. Pearl's causality "revolution" in epidemiology and AI is de facto only a renaissance of (idealist) causal inference in epistemology since causation occurs in the subjective thoughts of scientists who can imagine counterfactuals. Pearl's causality starts late in the scientific process, when we need to decide whether, say, whether data supports that a virus causes respiratory symptoms. Pearl's revolution is silent on the earlier ontological step where theories are developed by scientists consisting of trans-empirical concepts and mechanisms inaccessible to direct observation with our senses: how the HIV virus via a biological mechanism generates the AIDS clinical syndrome[2,3].

1 Our limitations prevent citing scientific realists from countries with which we are not familiar. Among them we find Santiago Ramon y Cajal,

2 Note that this example is a direct problem, not a reverse problem using abduction, which is associated with Lipton's inference to the best explanation.

3 Note as well that counterfactual's antecedent (Pearl, 2018), Bayesian statistics, and Bayesian Networks are irrelevant to causality, when expressed

Regarding semantics, realism states that at least some propositions are true, yet empiricism shies away from this position, with induction, Popperian Falsationism (all we can say is that theories are not false) and now with counterfactual thinking. Thus, *a counterfactual cannot be assessed for its truthfulness because it is a conditional*. Let us consider the counterfactual "If Michael had cited Marx in his papers, he would have been ostracized" is not a proposition, therefore is irrelevant to causation, or causal inference.

Finally, ethics, either in axiology/values ("what is good") and morals ("what is the right action"), realism states that with science we can affirm that at least some values a good and that there are moral precepts. For example, "Economic, political, and cultural inequality is bad because it leads to human suffering and sickness, and therefore egalitarian social policies should be implemented".

How Empiricism Thwarts Social Epidemiology Explanations and Public Health Interventions

The empiricist tradition ignores ontology because most causal mechanisms cannot be experienced with our senses, and thus does not distinguish between indicators and their referents – the referents being the causal processes in the real world outside the observer. For example, the socio-psychological causal mechanisms underlying "residential racial segregation" or "institutional racism" [11] which we have an imperfect ability to observe (i.e., via indicators such as the "number of mortgages denied to Blacks in a given area" or "wage inequality between Blacks and Whites in a firm"). Thus, in empiricist epidemiology (e.g., the "racial disparities" research program), the indicator (race) and the referent (racial discrimination) are collapsed and the account of the analysis is merely about "variables" and "indicators" and not the real individuals, structures and causal mechanisms that they represent. Indeed, the term 'indicator' is telling – it is an indicator *of something,* and a large part of causal analysis should be identifying what that something it and how it behaves in the real world.

Let us consider another example. Economic inequality is the referent of indicators such as "income" or "occupation" and the gradient in health is the mere association between the social indicator/variable and the health indicator/variable, devoid of any reference to causal mechanisms (e.g., the Whitehall studies; Marmot 2016). Thus, it is crucial to realize that in the Whitehall studies *there is no mechanism linking "occupation" to "autonomy", "job control", "participation" or other psychosocial mediators, because occupation refer to the technical attributes of work and says little about social relations at work* [24,25]. Marxian theoretical frameworks [26,27] or the contradictory class location (CCL) hypothesis include mechanisms linking class relations of domination and exploitation at work to psychosocial exposures such as lack of control or high job demands (Muntaner, et al. 2015). In fact, support for the CCL hypothesis refutes the alleged generality of gradient and supplies an explanatory social mechanism (Muntaner, et al. 2015). If proponents of the gradient where consequent empiricists, they would consider the gradient hypothesis rejected with a single "Black Swan". That such simple association has been, and still is, considered sufficient to epidemiologists for so many decades is remarkable, and suggests, whether investigators are aware of it or not, that there is little interest in explaining (understanding via causal mechanisms) health inequalities.

In public health, the counterfactual/potential outcomes approach to causality has a censorship effect and conservative bias since social interventions and policies that cannot meet the criteria for a potential outcome (e.g., [8]).[4]

An Epistemological Revolution in the Social Health Sciences? More on the Counterfactual Approach to Causation

From all the above we conclude that the epistemology of contemporary epidemiology is still empiricism, which states that scientific knowledge stems from experience ("observations" in epidemiologic terminology). A major flaw of empiricist epistemology, including the dominant potential outcomes framework [2], is to conflate epistemology (what can be known) from what it is (ontology, in reference to the world outside the observer). This narrow focus on the criteria for proof of a causal relation has limits our understanding of social systems. A narrow focus on the causal relation (a relation between events [28], not between things or properties as it is often stated in contemporary epidemiology textbooks (Rothman, Greenland, and Lash 2008)) neglects the role of uncovering non observable causal mechanisms. Absence of explanatory causal mechanisms limits the depth of what can be known (i.e., how things work), reducing epistemology to observable associations. The counter-factual revolution in the epistemology of epidemiology does not offer a solution to the lack of ontology (Pearl 2018). In fact, counterfactual/potential outcomes empiricism supplies an added part of idealism (causality relies in the subjective imagination of alternative worlds that do not exist). For

as subjective probabilities. When conditional probabilities stem from earlier research, the superiority of Bayesian statistics in prediction and causal inference, *ceteris paribus*, is undeniable.

4 The effects of the potential outcomes approach to causation might have had, in specific instances, shaped the kinds of public health policy advice that epidemiologists might have provided to governments during the Covid10 pandemic.

example, its reliance on Bayesian statistics for causal inference (Pearl 2018) makes up important epistemological and ethical problems. Radical Bayesian approaches to causal inference focus on subjective probabilities: they reduce epistemology, and therefore causal inference, to individual subjectivity (psychology). The ethical consequence is moral relativism, which deters public health action since there is no way to settle between different subjectivities. Other versions of Bayesian approaches to causal inference are mere distractions from the quest in finding explanatory causal mechanisms (Howson and Urbach 1993; Pearl 2018). This presents another problem of scientific ethics since scientists should not engage in activities that deviate from the search for truth.

Beyond these problems of empiricism, there are other reasons why the counterfactual/potential outcomes approach is ill-equipped to inform causation and causal inference. This stems from the notion that counterfactual sentences are not propositions whose truth can be figured out. For example, "If Michael had studied methods in genetic epidemiology, his impact factors would have been even higher" cannot be a hypothesis since "If Michael had studied methods in genetic epidemiology" is not a proposition, thus it cannot be tested for its factual truth. In other words, counterfactual sentences are not hypotheses whose truth content can be found empirically. Therefore, counterfactuals have no significance to causal inference. We conclude that counterfactual comparisons, a foundation of observational epidemiology is a sheer imaginary that cannot be tested for its truthfulness and is therefore irrelevant to causation and causal inference.

As an illustration of this point, let us consider how the current use of counterfactuals in the epidemiology of "racial disparities" [29]. Counterfactual approaches to "race" take the direct observation of skin color or its self-report as observations from which causal inferences are made thus impeding explanations via causal mechanisms involving non observable trans empirical constructs concepts such as, for example, institutional racism [11,14,18]. This construct refers to mechanistic hypotheses in a social structure (that is, the sum of all social relations in a society). For example, we may hypothesise that institutional racism reduces access to labor markets, leading to chronic unemployment, lack of psychological control and high rates depression. Thus, testing hypotheses involving causal mechanisms would require data gathering with indicators such as hiring practices, promotion and wage differentials in firms, and individual behaviors and attitudes at the workplace. The empiricist counterfactual approach would not uncover any of those mechanisms, as it would be satisfied with inferences obtained from associations between "race" (Black vs White categories) and health (e.g., Pearl 2018). It is not surprising that Pearl's Book of Why contains a defence of an author who advanced an essentialist/organismic concept of race [11]. The price for conflating causality with the proof for causality (criteria for causal inference) as seen in the counterfactual/potential outcome approach is the lack of explanations with mechanisms.

The use of the race variable is only one example where the empiricist conflation of ontology with epistemology in social epidemiology leads to limited explanations via causal mechanisms and ultimately contributes to maintain the social order (i.e. racial, class and gender inequalities in health). Sayer's distinction between "indicator" and "referent" is important here (Sayer 1999). The social sciences and psychology have substantial theoretical work on the relationship between a causal process (referent) and the way it is measured (indicator) (Sayer 1999). The ubiquitous gradient (Marmot 2016) referring to associations between income (or occupation) and health illustrates the consequences of this absence in epidemiology. There is no social relational mechanism that might explain the occupational class gradient in health [25,30].

We can obtain data on income and observe and association with health, but we do not explain either the social mechanisms leading to income differentials, or the mechanisms linking income to poor health in doing so. There is no mechanism in gradient studies that could explain how occupation or income strata might be differently associated with health indicators. We can be confident that the health effects of income are not about eating it or rubbing it on our skin, so we can dismiss these causal mechanisms, but what are the mechanisms that matter? These tend to be lacking *a priori* theories. What are the causal powers and liabilities of money? Under what conditions are these exercised? How might these factors lead to the repeated pattern of health differentiation by income level? We claim that all the evidence across time and place of associations between income gradients (e.g., [31]) and health could not shed light on the social mechanisms that might explain why say, a high-income person might be healthier than a low-income person. In fact, from an empiricist epistemology, once when a single explanation based on social and psychological mechanisms such as the contradictory class location is confirmed (e.g., Muntaner, et al 1998), the universally accepted generality of the gradient [31] should be refuted (see Muntaner, et al. 2015 for a review). From an empiricist epistemology all it takes is a single "Black swan" to refute the law of the social gradient. Therefore, lack of explanatory causal mechanisms such as class exploitation or domination will ensure that no political or policy intervention emerging from social epidemiology might be formulated to reduce class inequalities in health.

Considering the above, the call of empiricists, both

proponents [8] and critics [10] of the CF/PO approach, for better defined exposures and interventions, is mistaken. What social epidemiology needs are not definitions but theoretical models that, upon empirical confirmation, reveal explanatory causal mechanisms on which to base political/policy interventions [18]. In other words, if the experiment/clinical trial with well-defined interventions is the "gold standard", the experiment /clinical trial with explanatory mechanisms is the "rhodium standard".[5]

An Appraisal of the Constructivist/Empiricist Inference to the Best Explanation Alternative

In their critique of the CF/PO approach some researchers [3] have proposed a causal inference alternative, the Inference to the Best Explanation (IBE). The author's approach is also empiricist, as their epistemology begins with observations/data but advocates for a plurality of methods of causal inference (including the CF/PO approach) and Inference to the Best Explanation (IBE) in particular. IBE's inference begins with observations and compares a set of competing hypotheses to decide which of them is best supported by the evidence.

Inductivism, the building block of IBE, implies that we "start with data". Therefore, it can never lead to deep explanations, for example those requiring theoretical constructs such as gravitational mass or racist ideology, because our senses are too limited [14].

In fact, CF/PO and IBE are incompatible since IBE begins with facts, objective occurrences, while CF relies on imaginary scenarios (see section above). Therefore, the authors could have rejected the CF/PO, rather than considering CF/PO one of their approaches to causal inference. Consistent with constructionism pluralism calls for consensus between contending views of causal inference and causation. There is also a veil of constructionism in the subjective nature of choosing among hypotheses (i.e., the "loveliest explanation"). IBE similarities with CF/PO are nonetheless large. There is no distinction between epistemology and ontology, observations are not informed by theory testing, and explanations are post-hoc. One big advantage of the IBE methods over the CF/PO approach is that IBE highlights the importance of mechanisms, a feature of advanced explanations, albeit post-hoc, that is, as heuristic or sound speculation. In sum the IBE cannot supply an alternative to the development of social epidemiology as it is burdened by many of the shortcomings of the CF/PO framework.

We also question the adequacy of the "pluralist" approach to causal inference and causation [32]. Primarily, pluralism in causal inference and methods is suggestive of constructivism (researchers construct their own proof for causation). Historically, pluralism is suggestive of Berkeley's subjectivist empiricism. Subjectivism makes science unnecessary since there is no objective truth. Truth is in the eye of the beholder, and "your truth is as good as mine."

The Causal Inference Bubble

The popularity of counterfactual models in causation seems a distraction from finding models with causal mechanisms that can be empirically tested [14]. Indeed, a focus on counterfactuals does not require knowledge of the mechanisms at play (as in "Black and white disparities" or "SES gradients in health". Therefore, it ensures that its findings will not be relevant for policies or interventions, which require the precise cause-effect relationships that only mechanisms can provide. That is why it is politically innocuous to avoid theories and causal explanations via mechanisms and rely on associations (e.g., focus on differences between Black and White categories or groups of occupations). Mechanisms are necessary to understand in depth, and this is a major challenge, but even when the mechanisms of causation of a phenomenon are well understood, an adequate understanding of the mechanism of amelioration (or intervention) may be quite different. We cannot just ram the causal mechanism into reverse as we would a car transmission and expect the phenomenon to be reversed. Lack of well-developed accounts of the causal mechanisms that produce health and an account of the mechanisms by which possible interventions to ameliorate inequalities in health might work is what is lacking in social epidemiology, not a lack of causal inference.

Social epidemiologists, content with associations between Black/White comparisons and ranks of income/occupation and health indicators should consider that for social epidemiology to progress we need to generate unobservable constructs integrated in theoretical models (e.g., to uncover the social mechanisms that explain why a person with a supervisory/managerial occupation might present worse health than one with a worker occupation). Unobservable relations and mechanisms between social constructs (e.g., the 'social structure') help us make sense of the world because our experiences are too restricted. For example, although we cannot directly observe "nations" or "black holes" (constructs), we can observe them indirectly with population surveys and visible light, respectively. It is it

5 Banerjee's and Duflo's [33,34] approach to poverty reduction with "one experiment at a time" cannot be a role for social epidemiology because of its lack of a priori systemic multilevel theory (eg political economy that accounts for the role of the state, international North-South neocolonial or imperialist influences in local development) and micro-social approach, even considering its success in generalizing specific interventions.

not possible to advance causal models without moving away from an empiricist epistemology bounded by observations and repeated associations. The number of times something happens has nothing to do with why it happened, although sometimes repetition of patterning of events can point to the existence of causal processes that we still need to theorize. WWII only happened once, but we can still develop an account of the causal mechanisms that led to it.

Social and Public Health Policy Consequences

A positive heuristic of this debate has been the acknowledgment advanced by potential outcomes proponents of the link between causal inferences in epidemiology with public health programs [2]. Yet without confirmed causal models it is difficult to design useful public health interventions or policies, since we may not know "what component of the intervention causes the desired effect". If the experiment/clinical trial is the empiricist's Golden Standard, we need to strive for the realist's Painite/ Titanium Standard with mechanisms.

The counterfactual/Potential Outcomes and other empiricist approaches are politically and policy conservative as they tend to favor the "status quo" [18]. If we do not know what causal mechanisms produce health inequities, how can we change them? The recent history of social epidemiology is full of programs that generate vast amounts of data on associations ("the gradient", income inequality, race/ ethnic disparities) but few causal mechanisms, therefore no interventions. The potential outcomes framework is used to find what interventions are feasible on the basis of variables (e.g., race, income). Yet the lack of hypothetical causal social mechanisms limits both our knowledge and the possibility of interventions. Hernan [8] recently noted that an intervention on "race" [35] is not possible because "race" is "ill defined". Yet the problem here is not one of definition of race [8] or "lack of well-defined exposures [10] or lack of theory (a hypotetico-deductive system, not a narrative convention (e.g. Krieger 2000), it's that the epidemiological literature on "race disparities" does not provide evidence of causal mechanisms to explain the associations between "race" and health. Testing theoretical causal mechanisms of interpersonal racism, institutional racism, labor market discrimination, or residential segregation could lead to successful interventions since we would know what cause (e.g., denying mortgages to dark skinned persons) produces what effect (e.g., dark skinned persons do not have access to health promoting residential neighborhoods). Using scientific constructs such as "racial discrimination" and "social causal mechanisms" integrated in theoretical models about how the world works is an earlier step to confirming a causal mechanism. Once a causal mechanism is confirmed empirically with the help of indicators (number of mortgages denied to Latino families, socioeconomic indicators of Latino families' residential neighborhoods) we can apply this knowledge about causal mechanisms to design effective interventions. Therefore, explanations via causal mechanisms (i.e., happening in the world independently of epidemiologists) are both distinct from proof of causation (e.g., Hill's criteria) and essential for effective interventions. Thus, interventions explained by causal mechanisms are more effective than those stemming by trial and error, "black box"/association generalizations, or personal experience.

On saving FACE (or do not shoot the statistical messenger)

In a recent article Daniel, De Stavola and Vansteedlandt [36] take on the recent critiques of the formal approach to quantitative causal inference (FACE) [32] and Krieger and Davey Smith [3]. DDV question the claim by these critiques that the FACE approach restricts the study of causal inference to humanly *possible* interventions. The authors in our view correctly assert that FACE does not limit the type of interventions possible:

"*We view the FACE as precisely offering formal tools to investigate cause–effect relationships. They are always guided by what KDS call IBE (inference to the best explanation). Indeed, IBE is often how one comes to investigate the specific cause–effect relationship in the first place.*"

The absence of theories with causal social mechanisms (an ontological problem) is what is missing in social epidemiology and public health, which is not a question of causal inference. The formal approach including DAGs are valuable tools to increase the exactness of theories and serve as a map for testing hypotheses and continuing to causal inference with data analysis. The limitation of DAGs is not DAGs themselves, but the unwillingness of social epidemiologists to create, imagine causation as it occurs in the world – to theorize about the behavior of referents (real world phenomena) and not just variables (indicators). Before we go ahead with causal inference (epistemology) we need to propose causation (ontology). Empiricists are reluctant to generate social causal mechanisms because they are skeptical of what cannot be directly observed (causal mechanisms involve constructs such as exploitation, sexism, racism that can only be inferred via indicators, not directly observed) therefore they prefer to stick to observations of skin color, sex, income, occupation, education and their associations. Absence of social causal mechanisms precludes causality upon which policies can intervene. Societies don't change. Empiricism thus leads to conservatism (or vice versa)[1]. The implicit conservatism of the epistemology in social epidemiology reminds us of the embrace of another

conservative epistemology in the 80s and 90s, namely Popperian Falsacionism. Falsationism states that all we can know is that a proposition is not false. In that epistemology truth is elusive, therefore interventions postponed ad infinitum.

A Discipline in Denial: Fear of Reality in Social Epidemiology and Public Health

It seems that social epidemiologists are willing to accept the existence of parallel universes before willing to look at social mechanisms that might point to value driven political change in their societies**.** Because epidemiology is dominated by an empiricist epistemology that shuns explanation via causal mechanisms in favour of associations, the early adoption of methodological innovations in causal inference are considered most important, as in Pearl's "causation revolution" (Pearl 2018). Unfortunately, in epidemiology and public health we still suffer from a strong adherence to British empiricism as in the incongruous cult of David Hume (Pearl 2018) [2,8] where all we can know are subjective observations, and in the counterfactual case, not even that. On the other hand, scientific realism [18], which separates ontology (causation as it exists in the world) and epistemology (causal inference), remains overlooked, as in the seminal work of Claude Bernard on internal medicine. Yet most developed sciences progress thought the understanding of systems, their components, relations, and their mechanisms Mazzarello [37]; (e.g., Kandel, et al. [38]). This shortcoming seems particularly damaging to social epidemiology, which suffers from both a dearth of theories proper (hypothetic deductive systems) and explanatory social mechanisms, and which cannot or does not want to generate major egalitarian social policies to curb social inequalities in health [39-49].

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