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Causal Pluralism and Public Health

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Abstract

In this chapter, public health (PH) is considered as a diverse field in which a number of disciplines and approaches contribute to establishing a knowledge base for the design and implementation of interventions. I aim to explain why causality is central, yet not always explicit, in both the generation of the knowledge base and in the design process of interventions. I point to the importance of this diversity in PH and thus motivate for a pluralistic approach to causality, which has the potential to strengthen our knowledge base for PH interventions and the communication from PH scholars to decision makers and the general public.

1. Introduction

Public health (PH) is a diverse field in which a number of disciplines and approaches contribute to establishing a knowledge base for the design and implementation of public health interventions (Winslow 1920; WHO 2021; Tannahill 1985; Beaglehole and Bonita 2004; Brownson 2003; Guest 2013; Killoran and Kelly 2009). While epidemiology is arguably the main generator of evidence to feed the knowledge base, to a lesser extent many other disciplines from the health and social sciences are part of this joint enterprise: from bio-chemistry to sociology of health (Haveman-Nies and Jansen 2017; Mackenbach 1995; Pearce 1996). The diversity of PH is not just in terms of its "composing disciplines," but also in terms of the "people that make PH." PH scholars are academics from different fields, as well as officers in various non-academic organizations. I mention this kind of diversity too, as it may sociologically explain the difficulty in transferring academic knowledge outside the

walls of academia, including using jargon and highly specialized vocabulary. The notion of causality, with its conceptually and historically loaded baggage, is a case in point.

Despite its inter- and multidisciplinary approach, PH appears to be dominated by one narrative about health and disease, one that owes its origin to the success of the biomedical approach, and that traces the causes of health and diseased down to the (alleged) measurable biological realm (Engel 1980; Kelly and Russo 2017; Meloni et al. 2018). Thus, while few in PH will question nowadays the importance of social factors, and the strong and steady correlation between health (inequalities) and social factors, the vast majority of interventions still tackle biological factors, rather than social ones. This may be due to the fact that it is easier to conceptualize causality between biological factors and biological outcomes, rather than across factors and outcomes of different natures.

I will return to this point later in the chapter, but for now it is important to note that causality *is* central, albeit not always explicit, in the generation of the knowledge base as well as in the design of PH interventions. The centrality of causality should be obvious to see from the fact that PH interventions aim to *bring about change:* in individuals' behavior, in populations' health, in risk exposures, in the burden of disease, and so on.

But given the multidisciplinary nature of PH, it is also easy to imagine what creates a barrier to thinking productively about causality: each and every domain works with different concepts of causality and holds different epistemic norms or methodological standards to establish causal relations. However, just as PH is diverse in its knowledge base, goals, and implementation, so is the philosophy of causality. As I shall argue in the remainder of the

chapter, causal pluralism can be of help, in order to keep diversity and pluralism, and to understand what concepts can help advance the field and design good interventions.

The chapter is organized as follows. In section 2, I introduce six causal challenges in PH. These six causal challenges are meant to motivate the need for a pluralistic approach to causality. In section 3, I explain why monistic approaches to causality are bound to fail and present causal pluralism, and in particular the causal mosaic approach. This approach has the peculiar feature of distinguishing philosophical and scientific questions of causality, for which specific concepts are needed, also depending on the scientific or policy context under investigation. In section 4, I return to the six causal challenges and explain how the causal mosaic approach can help at both the stage of establishing the knowledge base and at the stage of design and implementation of PH interventions.

2. Causal challenges in PH

In this section I present six causal challenges that PH faces in the process of establishing a knowledge base and then designing interventions.

The first challenge [Challenge 1] is a most general issue in PH: Choose the most effective causal narrative to explain disease and then intervene (direct paths versus very indirect paths). This is distinctively a causal problem because, arguably, narratives of aetiology, of intervention, and of prevention are different, as they identify different factors as the relevant ones (Kelly and Russo 2017). Any PH intervention is subject to this challenge. But Covid-19 will serve as a handy example. While it did not take very long to isolate the virus and understand (some of) the bio-chemical aspects of infection (aetiology), these are not the immediate "actionable" factors to contain the spread of the virus (prevention and

intervention) (Greenhalgh 2020; Khalatbari-Soltani et al. 2020; Marmot et al. 2020). In other words, they provide only some clues, but insufficient to design effective strategies to control infection control.

The next two challenges I call "vertical" and "horizontal,"' respectively. The vertical challenge [Challenge 2] is about the level of aggregation of individuals into groups and population. The issue at stake is to pitch the right or best level of intervention: individual versus population, different types of social aggregation (family versus school versus peers …). This is distinctively a causal problem because causal relations may work differently at different levels of aggregation, and also because levels interact and interfere with each other. This "unit of analysis" problem is well known in social science, as instantiated in the debates about methodological individualism versus holism (Zahle and Collin 2014). Smoking prevention programs are a good example of Challenge 2, because such programs need tailoring to different groups, for example, targeting teenagers in schools, or certain types of workers (Santiago, Talbert, and Benoza 2019; Strickland et al. 2015). It is well-known that what works for one group may not work for another, and this is true for individuals as well.

The horizontal challenge [Challenge 3] is about choosing the right or best factor(s) to intervene upon. In part, such choice will depend on one's metaphysical views about disease causation. Simply put, one can reduce disease causation to bio-chemistry or hold the view that socio-economic factors (broadly construed) are also real causes. This is distinctively a causal problem, and for multiple reasons. To begin with, it has been (and still is) a struggle of some strands of social epidemiology and of medical sociology to establish the "social" as a legitimate, real causal factor, as opposed to a useful and informative classificatory device with respect to health and disease (Kelly, Kelly, and Russo 2014; Kelly and Russo 2017).

Moreover, and relatedly, it is not obvious that the best factor to target is always biological even when we hold thorough knowledge about the biochemistry of health and disease. In part, this traces back to Challenge 1, about distinct causal narratives in place, but in part this is a distinct metaphysical question about disease causation. A good example of this challenge is the MEND (Mind, Exercise, Nutrition...Do It!) program against child obesity.[1] Part of these interventions, in fact, target parents rather than children directly, exploiting remote, behavioral and social (rather than proximate, biological) factors, and on a quite indirect (rather than direct) path.

The next two challenges concern methodological issues in establishing the knowledge base. Challenge 4 is about understanding the complex conceptual relation between cause and risk. This is clearly a causal problem because the conceptual borders between "risk" and "cause" are not so clear-cut, but the problem also pertains to the actionability of risks and causes, and to the communication to general public (Covello, Von Winterfeldt, and Slovic 1987; Giroux 2011; Gigerenzer et al. 2007; Schooling and Jones 2018). The debate around the classification of red and processed meat as carcinogenic to humans (Group 1) by the International Agency for Research on Cancer (IARC), illustrates well the subtle difference between cause and risk, and the potential for misunderstandings, from the perspective of the general public (IARC 2018).

Challenge 5 concerns the assessment of evidence and the choice of methods to form the knowledge base and to design interventions. More specifically, one question is to assess how much evidence of correlation and/or of mechanisms is needed to have a solid enough knowledge base. Another issue, related to the previous one, concerns the use of quantitative and/or qualitative approaches to generate evidence of correlation and of mechanisms. These

are distinctively causal problems because, on the one hand, it is contested that we always have enough of these types of evidence. On the other hand, qualitative and quantitative methods (and of different kinds) come with specific assumptions, and especially about which causes are the "right" ones. Some methodological debates in epidemiology and public health illustrate this challenge. For instance, the use of statistical methods such as the potential outcome model have been considered by some as a gold standard method because of their rigor and for their requirement to work with "well-defined" interventions (Hernán and Robins 2020; VanderWeele 2018). However, some scholars have been critical of the approach precisely because of the impossibility of defining interventions "well enough" and for the assumption that causes be manipulable factors, thus excluding gender, ethnicity, or other key social factors (Vandenbroucke, Broadbent, and Pearce 2016). Similarly, the recent rise of qualitative methods in epidemiology testifies to the need for a plurality of methodological approaches that nonetheless come with very different epistemic assumptions (Bannister-Tyrrell and Meiqari 2020).

The final challenge [Challenge 6] is that concepts of health and disease are not "causally neutral." The problem here is really about the way health and disease are to be conceptualized. And this is distinctively a question about causation, because depending on how we conceptualize them, this will impact what causes we look for and what actions could/should follow (Russo 2021). Concepts of health and disease that reduce them to their biology are not just conceptually different from bio-social ones. They are accompanied by different *epistemologies* about how we can find out about health and disease, and they are also accompanied by a different *normative framework* about which interventions should (not) follow from said conceptualization. Differently put, in the context of PH, the concepts of health and disease that are part of the knowledge base are not just value-laden, but they are

also *value-promoting.* A good example of this challenge are the European Union (EU) directives to fight obesity, which explicitly mention that social and behavioral factors are a key target, but in fact only regulate food labeling (European Commission 2007; WHO 2014; Erixon 2017). Arguably, these directives do not target the social aspects of obesity in a structural and systematic way (as stated in the documents), but instead implicitly appeal to the biology and chemistry supporting certain food labelling recommendation rather than others. Additionally, they leave the burden of choice to individuals, without introducing any structural changes for instance at the level of food industry.

In sum, establishing a solid knowledge base goes well beyond establishing the alleged biological, "hard" facts of disease causation. Instead, establishing the knowledge base should be done trying to make the complexity of the phenomena of health and disease intelligible and tractable. There is a sheer diversity in the type of causal challenges that PH faces. For this reason, it is a wrong move to assume any monistic or monolithic view about causation. In the next section, I will explain why monistic approaches to causality are bound to fail, and why we should seriously look into pluralistic options to advance the production of the knowledge base or the design process of interventions.

3. Conceptualizing causality and the prospects of causal pluralism

3.1 The search for the-one-theory of causality

The philosophy of causality has a long history in Western and non-Western traditions. It is clearly beyond the scope of this chapter to retrace these histories, and the reader may want to refer to the work of others (see, for example, Beebee, Hitchcock, and Menzies 2009; Illari and Russo 2014; Rabins 2015) to understand the development of causality across time and disciplines.

I instead focus on the debate on causality in the past 50 or 60 years, and particularly the one that mostly happened in English-speaking circles. In the reconstruction of this recent history of the philosophy of causality, a main strategy has been to analyze the concept of causality, as it occurs in natural language, and as is used by competent (English) speakers (Illari and Russo 2014). Such a strategy is rooted in the analytic tradition of philosophy of language. The goal was to reach *The-One-Definition* of causality, capable of resisting all kind of counterexamples. A main limitation of this approach was to appeal to competent speakers' intuition, and also to tie the concept to its linguistic formulation. Another limitation was that ordinary language analysis may be quite unilluminating when it comes to the specific meaning a concept like causality acquires in, say, physics, biology, and in PH.

A first important way to liberalize the philosophy of causality was to relocate the analysis within proper scientific domains. Philosophers of science thus investigated causality in specific contexts such as physics, and over the years, in biology, social science, and very recently also in medicine. Nevertheless, these investigations remained very "discipline-oriented," while trying to reach *The-One-Definition.* The philosophy of causality managed to produce an impressive variety of accounts that cashed out causality in terms of processes, mechanisms, capacities or dispositions, inferential practices, etc. It is not difficult to understand that the challenge of making *one concept* fit *all* scientific domains is real and bound to fail. For this reason, some philosophers of causality turned their attention to causal pluralism. When the community started investigating causal pluralism, philosophers of science had not yet paid attention to public health as a legitimate scientific domain worth of philosophical investigation. The situation has now changed, and the six causal challenges

presented in section 2 offer further ground for exploring causal pluralism, which I present next.

3.2 Pluralistic approaches to causality

Simply put, causal pluralism is the view that causality cannot or should not be reduced to one notion or kind of thing only. In the philosophy of causality, this is a strategy that has already been explored, partly as a reaction to the difficulty of finding *one* notion or concept that fits any domain, context, or problem. Phyllis Illari and Federica Russo (2014) also note that there are different variants of causal pluralisms. I lack the space here to undertake a comprehensive and exhaustive discussion of all the approaches that adopt a pluralist strategy. I will therefore offer a simple roadmap through this literature and motivate for the approach I favor, namely, "causal mosaic."

It should be noted that while pluralism is becoming an increasingly popular option in philosophy of causality, the Aristotelian theory of four causes (formal, material, efficient, and final) was already pluralistic about *types of causing.* Also about types of causing, but more related to the analysis of ordinary language, is Elizabeth Anscombe's idea that causality is (linguistically) couched into transitive verbs such as pulling, pushing, or binding, that all express ways in which causes act (Anscombe 1975), an approach that has been further developed by e.g. Nancy Cartwright (2004).

More recently, philosophers of causality tried pluralistic strategies, for instance, with regard to *concepts* of causation. Ned Hall (2004), for instance, famously held the view that we have two concepts, one to be cashed out in terms of "dependence" and one in terms of "production," which are applicable in different contexts. The proposal of Erik Weber (2007),

instead, is to use different concepts of cause/causation, depending on the scientific context (for example, natural science versus social science). Other accounts centered on the idea that *causal inference* can be of different types; thus, for instance, Julian Reiss (2012) thinks it is important to distinguish inferences about the model and the target. Another strategy is pluralism about *evidence* for causal relations. This was initiated by Federica Russo and Jon Williamson (Russo and Williamson 2007), who argued that to establish causal claims in medicine, one typically needs evidence of difference-making and evidence of mechanism. The original paper sparked lively debate and a research program, to which I will return later in this section.

All these pluralistic strategies capture something true about causality and causal methods, as they are used and developed in different (scientific and philosophical) contexts and settings. But it is the approach of the "causal mosaic" developed by Illari and Russo that, I submit, can be of help in the case of PH. The approach of causal mosaic also starts from the observation that, until now, no single concept of causality fits all domains and contexts. This approach is maximally liberal: it allows for different kinds of causing as well as a variety of causal methods and concepts. But this approach goes a step further in motivating the project philosophically: no single concept of notion or causality can simultaneously answer the following different philosophical questions about causality:

(i) Metaphysics (or ontology): What is causality? What are causal relata?
(ii) Epistemology: What concepts guide causal reasoning or govern causal knowledge?
(iii) Methodology: What methods to use to discover/explore/confirm causal relations?
(iv) Semantics: What is the meaning of "cause" / "causality" in natural or scientific language?

(v) Use: What can we do (or not do) in the presence/absence of causal knowledge?

The approach of causal mosaic is also motivated *scientifically:* causality is not "one thing," or "one problem," as the sciences deal with different types of causal problems, notably:

(i) Inference: Does C cause E? To what extent?
(ii) Explanation: How or why does C cause or prevent E?
(iii) Prediction: What can we expect if C does (not) occur?
(iv) Control: What factors should we hold fix to understand the relation between C and E? Or to modify C so that E accordingly change?
(v) Reasoning: What considerations enter into establishing whether / how / to what extent C causes E?
Causal reasoning is arguably the broadest of the scientific problems, as it concerns all the ways we think about causality in science, whether explicitly and implicitly, and it lies at the intersection of science and philosophy.

To remain within the metaphor of the "mosaic", causal theories, notions, or concepts developed in the literature thus far constitute the "tiles" to juxtapose to one another in order to form an image. We choose the tiles as they help us address philosophical questions and scientific problems. For instance, the concept of (causal) mechanism may help with explanatory practices in e.g. biology (epistemology, methodology), while concepts of capacities or dispositions may instead help address ontological questions about biological phenomena. Or, the concept of (causal) process may help with tracing "world-line" trajectories in physics contexts or in social science (metaphysics), while some explanatory practices in the social domain may need a concept of "function" rather than mechanism. The

image generated by choosing the tiles and by selecting which philosophical questions and scientific problems are at stake can be different for different problems in different fields, and even within a same field it may change substantially over time. It is worth clarifying that, in the mosaic approach, once the appropriate "tiles" are chosen and placed next to each other, an image will appear. The approach to causality thus produced is not static, rigid, or immutable. In many ways, it is a pragmatic approach to causality, rather than a substantive one that seeks to nail down "The One Concept" of causality. However, causal mosaic is not an "anything goes" strategy, but instead it is about selecting and choosing appropriate notions for appropriate contexts, keeping in mind that while we can ideally distinguish elements within the philosophical questions and scientific problems above, they are in practice intertwined. What makes a concept more or less appropriate is its fitness to a given goal. For instance, probabilistic theories of causality fare pretty badly with explanation, but they were never intended to be explanatory in character, but are rather about inferences of different kinds. Thus, the ultimate goal of the causal mosaic approach is to select and use "compatible" notions across philosophical questions and scientific problems. The approach of causal mosaic can offer a pretty sophisticated way of synergistically using many of the existing accounts, always specifying the philosophical question(s) and scientific problem it intends to address at any given time. It is part of the philosophical and scientific debate to establish which tiles best satisfy the intended function – i.e. whether and to what extent they address the selected philosophical questions and/or scientific problem – and to keep the search open for always better suited accounts in case none of the available ones works.

In an applied context such as PH, philosophical questions about causality are clearly phrased mainly in terms of use; namely, using causal knowledge for the purpose of designing an intervention. Yet questions of use are not independent of other philosophical issues. For

instance, Challenge 6, which is about defining health and disease, is clearly an ontological or metaphysical problem, but one that has important links with causal epistemology/methodology, and with use. As for the other scientific challenges, PH may be concerned with any of the scientific problems of causality mentioned above, but possibly at different moments or stages of the process. Consider the different scientific problems first. Most of the time, we will need *some* level of explanation of a phenomenon of interest in order to make decisions about intervention and control. But in some cases, descriptive and correlational claims will have to serve as a basis for intervention and control, even in the absence of firm and sound explanations, according to the knowledge base established at any given time. Consider now the different philosophical questions. While defining health and disease (a question of metaphysics or ontology) are likely to remain, implicitly or explicitly, central through the whole process, from forming the knowledge base to the design of intervention proper, other questions, such about epistemology, methodology, or semantics can be of variable relevance.

Causal mosaic has been developed as a general pluralistic approach to causality. In the next section, I use PH as "stress test" for causal mosaic: how does causal mosaic really help PH with its tasks and challenges?

4. What is causal pluralism good for?

In this section, I seek to explain how causal mosaic can help to address the general task of PH (how to pass *from* conceptualizing/understanding a causal relation *to* acting on the causal relation), and the different causal challenges in PH.

4.1 The two stages of PH and causal pluralism

I begin with the general task of PH. The inference from conceptualization/understanding of causes to action/intervention is clearly not an easy one. For the sake of clarity, I cash out this inference as corresponding to the (conceptual) distinction of two following stages: (i) establishing the knowledge base and (ii) designing and implementing interventions. Though highly intertwined in practice, this conceptual distinction is directly related to Challenge 1: choosing the most effective causal narrative to explain disease, or to intervene, or to prevent, and this has to do with the identification of direct or indirect causal paths. Causal mosaic helps with Challenge 1 because it distinguishes between different scientific problems of causality, each having their proper formulation of the question, and appropriate methods. Explanation, intervention/control, or prevention are different, and their difference is conceptually and methodologically acknowledged in causal mosaic. With the causal mosaic approach, we can nuance these different scientific tasks, their import toward forming the knowledge base, and consequently their import toward the design and implementation of interventions.

A problem that cuts across the two stages is Challenge 6: the conceptualization of health and disease will influence a great deal how we form our knowledge base or how we design interventions. Causal mosaic helps with this challenge because it takes the most liberal stance about the metaphysics of causation, and for that matter about disease causation. This means that disease causation can be as inclusive as possible, to incorporate social and biological factors (I return to this later), and to open a space for the direct inclusions of values (epistemic, moral, political), into the formation of concepts such as health and disease (Russo 2021; Schramme 2017; Valles 2019). Let me elaborate further. The conceptualization of health and disease as primarily or solely biological phenomena, or as bio-*social* ones has very

important consequences at the methodological and ethico-political level. To begin with, a reduction to health and disease to "the biological" sphere restricts causal methods to the ones used in bio-medicine, in epidemiology, and in evidence-based medicine. But it largely excludes direct contributions of sociology and anthropology of medicine or of narrative medicine. Such choices are underpinned by different epistemic values and norms in selecting methods and concepts. But all this also has profound consequences at the ethico-political level. Again, reducing health and disease to the biological sphere excludes socioeconomic, sociocultural, and sociopolitical factors not just from the *explanation* of health and disease, but also from the basket of *intervenable* factors in a PH intervention. For instance, if obesity is a bio-social (rather than just biological) phenomenon, intervening on the socioeconomic, structural factors that favor the obesity epidemic is *also* a clear normative standpoint—for instance, about the role of governments with respect to individuals or to the food industry, and these stances are de facto ethico-political in character.

In the rest of the section, I provide further explanation of how causal mosaic helps thinking about causality in these two moments and through the six challenges of PH, and how it may help with communication outside specialist circles, for example, from PH scholars to PH officers, and from PH officers to the general public.

4.2 Establishing the knowledge base

As mentioned earlier in this chapter, establishing the knowledge base may need different disciplinary and methodological approaches. This is captured by Challenge 5: assessing evidence and choosing methods to form the knowledge base and to design interventions. Specifically, we are interested in assessing how much evidence of correlation and/or of mechanisms is needed, and in choosing between and from quantitative versus qualitative

approaches. In conceptual (philosophical) terms, the most general way of expressing this is that we need to admit, generate, and evaluate a whole variety of evidence. This is precisely the point of *evidential pluralism,* which is one of the tiles of causal mosaic, and a distinct pluralistic strategy.

Evidential pluralism is the epistemological and methodological view according to which, in order to establish a causal claim, we need different sources of evidence, and notably evidence of correlation and of mechanisms (Clarke et al. 2014; Illari 2011; Parkkinen et al. 2018; Russo and Williamson 2007). The thesis has been developed in the philosophy of causality and of medicine, partly as a reaction to evidence-based medicine and the use of evidence hierarchies, which put too much emphasis on evidence of correlation (especially in the form of randomized controlled trials and meta-analyses), at the expenses of any other form of evidence, from the ones generated by observational studies, or by experimental studies (for example, lab studies about mechanisms), or expert opinion. Evidential pluralism, it is important to note, is not about what causation is, but about what is needed in order to deem a relation causal—it is in this sense that the thesis is epistemological and methodological. Slightly different forms of pluralism about evidence and methods have been in recent times supported by, for example, Alex Broadbent, Jan Vandenbroucke, and Neil Pearce (2016); Vandenbroucke, Broadbent, and Pearce (2016), and Susan Haack (2009), and are certainly part of the history of epidemiology (Hill 1965).

Evidential pluralism holds that we need to establish *some* correlation and *some* mechanism in order to have *some* level of explanation of the phenomenon to address. The big problem is that none of these "some" can be fixed, as we cannot provide exact thresholds for when we have enough (or not) of these correlations and mechanisms. But the whole process of

reasoning (one of the scientific causal problems of the causal mosaic) about the "quality" and "quantity" of mechanisms and correlations available *is* valuable to return a balanced report on what is the available knowledge base. For instance, the EBM+ group designed a number of tools for evaluating knowledge bases (Parkkinen et al. 2018, chap. 4).[2] Using these tools, we can clarify how much evidence of correlation is available, and assess its quality, and how much evidence of mechanisms is available, and assess its quality. In a similar vein, the CauseHealth group has explored the philosophical underpinnings (for example, causal complexity or individual variation) of several aspects of the medical profession evidence of mechanisms (Anjum, Copeland, and Rocca 2020), and the role of evidence of mechanisms in clinical practice, notably in establishing claims about safety or about efficacy (Pérez-González and Rocca 2021).[3] These important contributions notwithstanding, a question that deserves further discussion is how quantitative and qualitative methods can contribute to generating evidence. In evidence-based medicine, it is an established view that randomized controlled trials (RCTs) and meta-analyses are the gold standard for the generation of evidence of difference-making, but other methodologies—from lab research to observational methods to qualitative-oriented research—likewise contribute to generating both evidence of difference-making and of mechanisms.

The principles of evidential pluralism, as developed by the EBM+ group, have been in part implemented in the United Kingdom's National Institute for Health and Care Excellence (NICE) and IARC methodologies (IARC 2019; NICE 2014; Samet et al. 2020),[4] and further work should tailor evidential pluralism to the specific needs of PH. Evidential pluralism, when properly understood, is likely to help with communication from academia to the policy world. In fact, the conceptual separation between evidence of difference-making and evidence of mechanism can be cashed out in terms of "knowing that" versus "knowing how,"

which is possibly easier to grasp than the categories of evidential pluralism. For instance, we have long established *that* smoking causes lung cancer, but understanding *how* smoking causes cancer required more time and resources. Additionally, the "how" part of smoking is not reducible to the biochemistry of the inhalation of substances such as tar, but also involves a whole variety of social practices, from imitation to stress release to "social smoking." "That" and "how" would, in *ideal* situations, license different inferences and interventions. Increasing smoking taxation targets the "that" part. But targeted interventions may instead focus on specific parts of the "'how," for instance, prevention programs in schools.

Yet we all know that the design and implementation of intervention is done in far from ideal situations or conditions. Very often, we need to intervene on the basis of some established "that" part, and without much understanding of the "how" part. This is exactly what happened with general lockdown strategies in 2020, at the beginning of the Covid-19 pandemic. But now that we have gathered more information about the mode of transmission, and also data about the exposure, morbidity, and mortality in different socioeconomic and age groups, arguably we are in a position of better exploiting the "how" part for targeted interventions (for example, in designing specific protocols for schools).

So, at the very least, we can design interventions with more awareness about what is solidly grounded in the "how" and what is instead solely grounded in the "that." Again, in ideal situations, it would best to have as much of "that" and of "how" in our knowledge base, but in practice we need to be able to say why "that" can be enough to act, and this may be justified with arguments that do not necessarily appeal to the knowledge strictly speaking, but to arguments of precaution, urgency, or other values.

4.3 Designing and implementing an intervention

Ideally, in order to design and implement effective PH interventions, one should base them on the best available knowledge base. We have just seen that, even if we are typically far from being in ideal conditions, evidential pluralism offers tools to assess what evidence is (not) available. From a causal perspective, in the process of designing an intervention, we may need to distinguish two questions, which also maps onto specific causal problems:

1. How much do we really need to know about the target population to properly intervene? (Explanation, control)
2. How likely it is that implementing X we'll get the sought result Y? (Inference, prediction)

The two questions are not independent, but they are nonetheless distinct. Arguably, the first question is really about explanation, while the second is about inference, and notably about prediction. Being clear about these two questions will be of great help in the design of an intervention, because the link between the two may or may not be essential. The "Semmelweis case" provides a useful illustration. The case is debated in the literature, and in no way I will be able to do justice to the wealth of its historical and philosophical scholarship (Gillies 2005; Broadbent 2011; Tulodziecki 2013; Kadar and Croft 2020) in this chapter. Briefly put, the case is about Ignaz Semmelweis, a doctor active in Vienna in the first half of the nineteenth century. He was hypothesizing that puerperal fever was caused by some kind of infection; he proposed hand disinfection with chlorinated lime solutions for doctors in wards of the obstetrical clinic. As the story goes, Semmelweis's recommendations encountered lots of resistance from the medical community of the time, and in part because there was no scientific theory to support the proposed intervention, as the germ theory of disease was yet to be developed. The exact historical reconstruction of this case does not

matter here, and its basic facts are not disputed, but the philosophical lessons to draw from the case are. It is contentious, in philosophical circles, whether the community at that time was right in rejecting Semmelweis's proposed intervention. Yet, it is to be hoped that one lesson to learn from this story is that, at least in some cases, we can accept to carry out an intervention, even in absence of a good enough explanation of its underlying mechanism. Making masks mandatory during the Covid-19 pandemic is another good example of the difference between the two questions above, and would be supported by arguments from precaution, rather than from a fully established knowledge base in terms of both difference-making and mechanisms (Greenhalgh et al. 2020).

The approach of causal mosaic is pluralistic about metaphysics or ontology too, and for this reason it helps address Challenge 2, that is, picking the right/best level of intervention, depending on the types of social aggregation, and Challenge 3, that is, targeting the right/best factor to intervene upon, social or biological. Let me explain further. Causal mosaic does not take the biological level as prior or foundational to the social level: health and disease can be caused by bio-chemical *and* by socioeconomic factors. Much of medicine (broadly construed) since the second half of the nineteenth century has been about opening the opaque box of health and disease, down to the molecular level. But this has been at the expense of socioeconomic factors, which gradually lost their status as causes proper. The development of epigenetics and of the life-course approach in epidemiology require a non-reductionist ontology for disease causation (Blane et al. 2013; Kelly-Irving, Tophoven, and Blane 2015; Lock 2015; Sacker et al. 2016; Castagné et al. 2018). It is in this sense that causal mosaic opens the doors for exploring the (causal) relations between the biological and the social sphere in health and disease, and for an ontology of disease that is not reductive in character. Likewise, causal mosaic does not fix that causation is primarily a token or type thing, or that

it has a privileged level of aggregation at which it happens. With causal mosaic, we can legitimately talk about causal relations happening "inside the body," at the bio-chemical level, or at the level of the individual, or at some level of aggregation—for instance, social or working environment—and across any of these levels. These different levels may call for very different types of interventions, from the individual, clinical one, to a public health level proper; for instance, in occupational health. These are *all* legitimate levels at which *real* causal relations operate, but levels that arguably need different methods of analysis and likely different types of interventions.

Let me give an example of different levels of PH interventions, using different types of interventions in the case of Covid-19. A general lockdown, for the whole country, some would argue, does not need not much "local" knowledge of different groups, environments, or other. The likelihood of reducing infection rates solely depends on reducing to a maximum any contact as a vehicle of infection. Partial lockdowns, for instance, targeting specific professions, or schools, or other targets, need a lot of local knowledge about the target populations or groups. This argument has something going for it, but also masks some important reasons why, after all, general lockdowns have been less effective than hoped for. In fact, general lockdowns target the whole population, without making distinctions about living conditions of specific areas or households, or which socioeconomic groups actually benefit, to a greater or lesser extent (Broadbent and Smart 2020). Similarly, consider banning smoking in public places versus designing prevention programs in schools. The same reasoning applies here, and actually traces back to the two key elements of evidential pluralism: "how" versus "that." In any of these situations, socioeconomic and sociocultural factors loom large, in terms of explaining exposure, identifying more or less direct pathways to disease outcomes, and for prevention. If it is true that even very general interventions, such

as the ones just mentioned, do require "local" knowledge, the right question to ask is not in absolute terms about their efficacy, but in terms of their usefulness, for some specific purposes. A general lockdown may still be helpful, in the state of emergency, to gain time to develop strategies that are more tailor-made. The evaluation of generic interventions such as taxation of tobacco, sugar, or alcohol is different, and should weigh in also the simultaneous implementation of several other "local" programs.

The approach of causal mosaic can help with formulating clearer expectations and more transparent reasoning in the design process, which may in turn help with the communication from PH officers to politicians and general public. This is because, within this approach, we are prompted to specify which scientific problem we want to tackle, whether it is inference and prediction, explanation, or control. Moreover, assuming that the whole process is carried out with the least vexed interests and with the highest level of intellectual honesty, making clear what the expectations are and being transparent about the evidence base should lend support to the choices made, and find a middle ground between two equally deleterious attitudes: scientism and skepticism towards science. In fact, while not having "The Truth" all the time or for everything, science still has *some* base to act. Also, decisions are not the simple product issued from the knowledge base, but incorporate other dimensions too, including values, economic priorities, cultural aspects, or other elements. There must be a way in which we can succeed in communicating these subtleties about the scientific and policy process beyond the ivory towers of our specialisms.

Another pressing issue in PH is captured by Challenge 4: the complex conceptual relation between cause and risk. Causal mosaic helps with addressing Challenge 4 because it allows for a conceptual distinction and use of "cause" versus "risk," and also of "probabilistic cause"

versus “deterministic cause.” Grasping the difference between a “cause” and a “risk” is a well-known problem, and not just for the general public, but also for doctors (Gigerenzer et al. 2007). Thus, if causal mosaic can return a useful semantics of these concepts, together with fine-grained analyses of the concepts that will be of immense help to PH (Giroux 2011, 2013). It is important to acknowledge that these notions are complex from a philosophical and scientific perspective and their meaning is not univocally fixed (yet) in the sciences (of health and disease). At the same time, these terms *are* part of the technical jargon and their precise meaning ought to be better conveyed to the general public. Thus, any quick slip from the language of correlation and association into the language of *deterministic* cause should be avoided. It should be noted that, in epidemiological circles, causal talk is not always well received. Arguments to drop the term “causality” are regularly made (Lipton and Odegaard 2005; Hernán and Robins 2020). Adopting an explicit causal perspective does not imply that we have to always find causes, or that these causes are deterministic, but that we *can* distinguish between causes and risks, without reducing everything to the nebulous and dubious claim that, ultimately, we can never establish causes. In this sense, concepts of probabilistic causality, and the distinction between generic and single-case causality can be of much help to PH in recovering a “healthy” causal talk.

An effort can and should be made to better communicate the difference between causes and risks, notably by making clear what kind of studies have been conducted on problem X, whether there is experimental evidence, whether meta-analyses exist, whether similar interventions have already been done, and so on. It is not just a matter of degree. Partly, it is a matter of conceptually distinguishing (again) the “that” and “how” of causal relations, and partly it is a matter of distinguishing other dimensions of causality, notably between individual- and group-level. In this way, we can hopefully better explain the difference

between risk and cause to the public, disclosing, whenever possible, elements of the knowledge base and of the rationale behind interventions. It goes without saying that nothing can replace good literacy in general, but the idea is that causal pluralism can help the quality of science communication.

5. Concluding remarks

Public health interventions face a number of challenges,. In this chapter, I have identified six of them, all inherently causal, and I have introduced a pluralistic approach to causality to address them. Specifically, the form of pluralism I advocate is "causal mosaic" because of its distinction between philosophical and scientific questions of causality, and for its explicit stance that we need to select concepts of cause/causation that suit specific problems, questions, or domains. Causal mosaic can help to establish a more solid knowledge base, to better specify the rationale behind PH interventions, and also to improve on the communication between different actors and stakeholders involved in the process (scholars, officers, the public).

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[1] See, for example, https://healthyweightpartnership.org.

[2] (https://ebmplus.org)

[3] (https://causehealthblog.org)

[4] The 2019 version of the IARC Preamble explicitly references Parkkinen et al.(2018), and changes to the methods are also described in Samet et al. (2020). These changes resulted from a collaboration during the AHRC-funded project 'Evaluating Evidence in Medicine' (https://blogs.kent.ac.uk/jonw/projects/evaluating-evidence-in-medicine/). Update in the NICE manual since 2018 include, inter alia, a more explicit recognition of

the importance of evidence of mechanisms in identifying sub-populations, using the terminology of ‘mechanisms of action’ rather the more generic ‘pathophysiological basis’.