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SCIENTIFIC CONTRIBUTION

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Abstract The paper addresses the question of how different types of evidence ought to inform public health policy. By analysing case studies on obesity, the paper draws lessons about the different roles that different types of evidence play in setting up public health policies. More specifically, it is argued that evidence of difference-making supports considerations about 'what works for whom in what circumstances', and that evidence of mechanisms provides information about the 'causal pathways' to intervene upon.

Keywords Causation · Disease causation · Evidence · Evidence-based public health · Obesity · Public health policy

The fat generation in the era of 'evidence'

Health is a concern for particular individuals as well as for policy makers. My interest in this paper goes to the worries of policy makers, who are committed to finding effective ways to improve the health of populations. No wonder public health policy is concerned with questions about evidence, that is with questions about the very basis of public health interventions. There are of course a plethora of issues looming here; in this paper I shall restrict discussion to the roles that different types of evidence ought to play in setting up public health policies. Studies on obesity are an exemplar, as we shall see, exhibiting the complexity of the quarrels about policy, evidence and causation; at the same time, these studies also provide fruitful insights for answering questions about evidence and causation in the public health policy debate. Let me then open the paper with an outlook of the studies on obesity.

Obesity is a chronic disease. The *prevalence* of obesity—i.e., its percentage with respect to the total population—is increasing in various countries, for adults as well as for children. Interest in obesity is not in its being a new disease, as obese people existed in the past too, but rather in the fact that the percentage of obese people is growing, and very fast. In more and more Member States of the European Union, over 20% of the adult population is obese, over 50% of the adult population is overweight, and up to 20% of children are overweight.[1]

Figures show that obesity is now an *epidemic*. Evidence collected in numerous studies shows that diseases related to excess adipose tissue—e.g., cardiovascular diseases—have drastically increased. Moreover, those diseases are amongst the most significant factors of morbidity and mortality *worldwide*; the contention of those studies is that, ultimately, prevalence in obesity and its associated comorbidites is likely to continue to increase in the near future.

The question easily arises: how to explain such an increase in the percentage of obese people? There are, broadly speaking, two main factors: biological and genetic factors on the one hand, and nutrition and lifestyle on the other hand. These two broad categories of factors correspond to two perspectives on the disease: biological and

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[1] This data refers to statistics published on open access websites and databases of the European Commission (http://www.hopeproject.eu/index.php?nav_id=), the UK National Health Services (http://www.ic.nhs.uk/statistics-and-data-collections/health-and-lifestyles/obesity/statistics-on-obesity-physical-activity-and-diet-england-2010), and the World Health Organisation (http://www.who.int/mediacentre/factsheets/fs311/en/index.html), all accessed in March 2010.

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socio-economic. Not only there exist different perspectives *on* the disease, but also different ways in which different people involved—overweight people, health professionals, policy makers—perceive the problem (see e.g. Greener et al. 2010; Galea et al. 2010). Later in "*Causally*-based public health" I will discuss how these different perspectives on obesity may lead to different public health interventions. I will use, as a paradigmatic example, a programme called MEND (standing for 'Mind, Exercise, Nutrition, Do it!'). This is a course for families with overweight children aged between 2 and 13. MEND officers teach both children and parents how to live more healthily in order to manage their weight better.[2]

Also, in understanding obesity, there seem to be two distinct albeit related questions at stake: (i) what are the *causes* of obesity and (ii) what are the causes of the *increase of prevalence* of obesity. Whilst the first question is concerned with having a thorough understanding of the disease, it is in fact the second that is most relevant for public health purposes. Both questions concern causal assessment and we will see in "Public health and epidemiology" that different different types of evidence—evidence of difference-making and of mechanisms—are needed to establish the causes of disease in general, and of (the increased prevalence of) obesity in particular. In "*Causally*-based public health" it will be further argued that these two types of evidence are also at stake in public health policy.

The line of reasoning that I will endorse (mainly in "*Causally*-based public health") is that the action taken in response to the second question above (about the causes of the increased prevalence of obesity) also depends on the answers given to the first question (about the causes of obesity). In other words, the better our understanding of the disease and of its development, the better our actions to reduce its burden on the population. Nevertheless, this is easier said than done. It is in fact the complexity of disease causation in the case of obesity that makes interventions so hard to put in place. Research suggests that most human obesity probably reflects complex interactions between genetic, environmental, and social factors often mediated through nongenetically derived changes in metabolism. The phenomenon is so complex that some are suggesting computer simulation using the so-called 'agent-based models' to get a better grip on it (see e.g. Galea et al. 2010).

An interesting aspect of obesity is that both 'health' and 'economic' considerations motivate public health interventions. I mentioned earlier consequences on health such as cardiovascular problems. But there are consequences other than health: standard seat width increased, as well as office furniture, revolving doors and all equipment in hospitals (beds, wheelchairs, operating tables, etc. altered), average passengers weight implications for fuel used by airlines …We thus want to reduce obesity rates for its effects on health, but not only that.

Ultimately, the difficulty is to decide what policies will be effective, and for whom. In other words, two issues are at stake here: (i) *what* factors—i.e., biological/genetic or social—to intervene upon to reduce obesity; and (ii) *who* should take part in public health policies to reduce obesity (that is, what level to target the intervention at: children, adults, poor people, …).

Thus, the question arises: *what* evidence ought to inform public health policy? The answer I will give is that two types of evidence broadly conceived ought to inform public health policy: evidence of difference-making and of mechanisms. Whilst the former supports considerations about 'what works for whom and in what circumstances', the latter provides information about the 'causal pathways' to intervene upon. The argument will run in three steps.

Section "Public health and epidemiology" states the importance of epidemiology for public health because of its population-level perspective on disease causation. It is then argued that in epidemiology (and in medicine more generally) causal assessment requires evidence of difference-making and of mechanisms. Arguments in favour of this thesis can also be found in Russo (2009, 2011) and in Russo and Williamson (2007, 2011).

Section "*Evidence*-based public health" praises the efforts of public health practitioners to address questions of evidence. It is argued that although they work towards developing criteria for evidence assessment, the most crucial question, that is *what* evidence is needed for public health policy, is by and large left unanswered.

Section "Causally-based public health" pleads for *causally*-based public health on the grounds that a better *understanding* of disease causation will result in better *action and decision*. Thus, if we accept the idea that disease causation requires two types of evidence—of difference-making and of mechanisms—so does public health policy. Evidence of difference-making, mainly in the form of risks coming from descriptive epidemiology, supports considerations about 'what works for whom in what circumstances'. Evidence of mechanisms, mainly coming from analytic epidemiology, cashes out the 'causal pathways' upon which we have to intervene. In sum, these different types of evidence serve different roles in deciding about public health policy.

[2] Information about the programme, the services, the follow-up, related scientific publications, etc. can be found at the sable URL= http://www.mendprogramme.org/hom.

Public health and epidemiology

Public health aims to prevent disease, prolong life, and promote the health of populations. A first peculiar characteristic of public health is that actions to reach those aims are based on *population-level* analyses. Thus, public health is concerned with preventive rather than curative and individual-level interventions; individual-level measures are in fact a concern of medicine (and, within the broad evidence-based perspective, a concern of evidence-based medicine).

A second characteristic of public health, as practitioners notice, is its interdisciplinarity. The decisions about what actions to undertake rely on findings in other areas, such as epidemiology, biostatistics, behavioural sciences, health economics and health care management. Given the *population-level* dimension of public health, I am most concerned with the importance of epidemiology, for reasons that will become clear throughout the discussion.

Epidemiology studies how the distributions of diseases and their biological or socio-economic determinants vary within a population and across different populations. This happens in two, typically subsequent, kinds of analysis: descriptive and analytic epidemiology.

On the one hand, *descriptive epidemiology* seeks to answer questions about 'who' in the population is affected by 'what' disease and under what circumstances ('when' and 'where'). The main goal of descriptive epidemiology is thus to work out risks of disease and exposure for a given population.

For instance, Reilly (2005) describes trends in paediatric obesity, relating a number of health consequences to early obesity both in childhood and in adulthood. Hu (2008) also provides a comprehensive collection of trends and risks of obesity in different categories of people in the United States. These types of studies are mainly epidemiological descriptions of the obesity phenomenon: they provide a 'snapshot' of what parts of the population are obese or overweight, and what are the corresponding risks, exposures, and health consequences.

On the other hand, *analytic epidemiology* seeks to answer questions of 'how' the disease operates and 'why' it develops and spreads. Measures of associations are the main focus of analytic epidemiology, and from those measures analytic studies attempt to draw inferences about the *causes* of disease. Thus, analytic epidemiology is certainly more 'causally-oriented' than descriptive epidemiology. The causal character of analytic epidemiology is not always made explicit (see for instance Porta 2008) on this point). Nevertheless, in asking *how* and *why* questions, analytic epidemiology goes beyond the mere measurement of associations and contributes to the evaluation of *mechanistic* explanatory hypotheses.

Consider for instance the following study on the *effects* of obesity. Obesity is recognised to be an important risk factor for type 2 diabetes and for cardiovascular diseases as well. Constantin et al. (2010) are interested in measuring *associations* between leptin G-2548A and leptin receptor Q223R gene polymorphisms; the motivation behind this, however, is to test a *mechanistic* hypothesis. Researchers, in fact, focus on the mechanism by which leptin acts to reduce weight by regulating energy intake and energy expenditures. But things get more complicated when subjects affected by type 2 diabetes are considered. Research showed, in fact, that whilst serum leptin level is indeed higher in obese people, in people affected by type 2 diabetes leptin levels appeared to be unchanged or reduced. The mechanisms of energy intake and energy regulation thus need further investigation and comparisons between samples from different populations. In this study, researchers used a sample from a Romanian population. Here, they did not find significant differences in leptin, leptin receptor and genetic variants in obese and non-obese people and therefore they cannot be considered as risk factors in the analysed sample. However, they also report that in the Chinese population, leptin and leptin receptors are indeed positively correlated with incidence of type 2 diabetes.

Although analytic epidemiology does not state in such clear terms that the evaluation of *mechanistic* hypotheses is what is at stake, it is hard to think of answers to 'why' and 'how' questions that are not causal and mechanistic. In addressing the 'why' question, analytic epidemiology must be looking for the *causes* of disease, rather than mere associations between risk factors or exposure and disease. To follow Wesley Salmon, successful explanations are those that put "the cause into be*cause*" (Salmon 1984). As for the 'how' question, here a satisfactory answer must consider the functioning of, or the modes of organisation and interaction within, the phenomenon being analysed. This is the core idea behind mechanistic explanations, which is defended, in slightly different ways both by scholars in the 'mainstream' literature on mechanisms (see e.g. Bechtel and Abrahamsen 2005; Illari and Williamson 2010) and by scholars interested in the explanatory import of models in social science (see e.g. Mouchart and Russo 2011).

This emphasis on mechanisms seems to be at odds with the 'risk factor epidemiology' tradition, according to which the isolation of significant risk factors is sufficient basis for policy. Whether and how mechanisms ought to enter epidemiology at all is a vexata quaestio. Yet, some voices advocating the complementarity of the risk factor approach and search for mechanisms do exist (see for instance Perry (1997). Putting mechanisms at the forefront, as I urge here should be done, is not consensus yet. Rather, the received

view trusts randomised trials (as campaigned for by EBM partisans), which do *not* explicitly involve the evaluation of mechanisms for causal assessment nor for policy. The plea for a *causally*-based approach (see later "*Causally*-based public health") contrasts with this dominant view.

As mentioned above, epidemiology is perhaps the main source of information for public health. Yet, it is worth noting that the public health dimension of epidemiology is a matter of controversy amongst epidemiologists themselves. Although many epidemiologists share the view that their discipline underpins public health, there is still a wide gap between public health practice and academic epidemiology (on this point see for instance Bophal (1997, 1999) and references therein).

Research in epidemiology is arguably driven by two distinct but nonetheless complementary goals: (i) to *understand* and learn about disease and (ii) to *take action* in order to reduce the burden of disease at the population level. This second, more controversial goal will be thoroughly discussed in "*Evidence*-based public health". Let us now focus on the first one. Understanding and learning about disease consists in drawing conclusions from evidence, more particularly conclusions about disease *causation*. Therefore, epidemiology brings to the fore questions about *what evidence* is needed to draw conclusions about disease causation.

We have seen in "The fat generation in the era of 'evidence'" that epidemiological studies on obesity aim to reach a *comprehensive* understanding of the disease. This is done by seeking answers to questions about 'who-what-when-where', identifying the relevant risk factors, viz. by making considerations about difference-making, *and* by seeking answers to the 'how-why' questions which concern mechanisms. Blass (2008) offers, in this perspective, an integrated account of the different causes, underlying mechanisms, and consequences of obesity. This aetiological knowledge is used to set principles for prevention.

This thesis—that causal assessment typically needs evidence of difference-making and of mechanisms—has been defended for the health sciences in general, and for epidemiology and autopsy in particular, by Russo (2009, 2011) and by Russo and Williamson (2007, 2011). In "*Causally*-based public health" I defend the idea that the two types of evidence are *also* needed for public health purposes. I do not reiterate here the arguments already given in favour of the thesis, but let me give a taste of what these two types of evidence amount to. The characterisation hereby offered is very general and the interested reader may also want to have a look at Gillies (2011) and Illari (2011) for a critical discussion.

Simply put, *evidence of difference-making* is evidence that a putative causal factor 'makes a difference' to the putative effect. In other words, evidence of difference-making helps decide whether the putative causal factor is relevant for the occurrence of the putative effect. This type of evidence can be cashed out in different forms: probabilistic, statistical, or counterfactual relations. Evidence of difference-making is especially needed for description and for prediction of disease causation. For instance, Reilly et al. (2005) provides (difference-making) evidence that that children in early life with obese parents, having very early (by 43 months) body mass index or adiposity rebound, watching television for more than eight hours per week (on average) at 3 years old, etc. are more likely to become obese already in childhood. *Evidence of mechanisms* is about plausible or confirmed enough mechanisms that are meant, in turn, to support results of difference-making. In Kahn et al. (2006), for instance, the mechanism for insulin resistance in obese people involves the increased release of non-esterified fatty acids, glycerol, hormones, pro-inflammatory cytokines and other factors by adipose tissues. Those factors tend to develop insulin resistance. This, together with other pancreatic dysfunctions, results in failing to control glucose levels in the blood, which may lead to the development of type 2 diabates. Evidence of mechanisms is especially needed for explanation and control.

Evidence-based public health

Consider now the second goal of epidemiology mentioned above, i.e. taking action in order to reduce the burden of disease. Although some epidemiologists do think that this is their concern too, in recent years public health has worked towards establishing itself as an autonomous scientific domain (with respect to the many other disciplines that inform public health policy) within the so-called evidence-based movement.

One motivation to embrace the evidence-based framework is to provide decisions and actions with a stronger basis. For instance, Brownson et al. (1999, p. 87) notice that

> [i]deally, public health practitioners always incorporate scientific evidence in making management decisions, developing policies, and implementing programs. However, in reality, these decisions often are based on short-term demands rather than long-term study, and policies and programs are developed frequently around anecdotal evidence.

Consequently, the advent of *evidence-based* public health is certainly welcome. But, at the same time, evidence-based public health (EBPH) brings to the fore specific questions about the basis upon which policy ought to be based. Moreover, EBPH inherits from epidemiology

crucial questions about evidence and causal assessment. This can be extracted from the way EBPH theorists define their own discipline. We read, for instance:

> Evidence-based public health is defined as the development, implementation, and evaluation of effective programs and policies in public health through application of principles of scientific reasoning, including systematic uses of data and information systems, and appropriate use of behavioral science theory and program. (Brownson et al. 2003)

> Evidence-based public health is the process involved in providing the best available evidence to influence decisions about the effectiveness of policies and interventions and secure improvements in health and reductions in health inequalities. (Killoran and Kelly 2010, p. xxii)

From the quotes above it emerges that the main concern of EBPH is to provide a solid, scientific basis for the decisions and actions taken to reduce the burden of disease. *Multiple* sources of evidence are said to be necessary for this end. Indeed, the focus on evidence concerns many aspects (Guyatt and Drummond 2002): what evidence one's practice or policy is based upon, the soundness of evidence, the strength of inference permitted by evidence, etc. Put it in more general terms, what is key is the *assessment* of evidence.[3]

There is a broad consensus that assessing scientific evidence involves evaluating peer reviewed publications by conducting meta-analyses in order to minimise problems of bias and to decide about the quality and generalisability of studies. Evidence assessment is also usually taken to involve quantitative risk assessment, economic evaluation of the prospective interventions, and consultations with expert panels. Brownson et al. (2003, ch. 2), a classic in EBPH, explicitly mention issues related to causality. They start with the usual caveat that causality is almost impossible to establish with certainty; they then discuss well-known criteria and guidelines used in the health sciences such as Henle-Koch postulates and Bradford Hill's guidelines. They also review pioneering and influential methods put forward by a number of epidemiologists that overtly embraced a causalist perspective (most famously, Rothman's 'pie' and Susser's 'Chinese boxes'). However, according to Brownson et al. (2003), causal issues are eventually resolved by following Hill's guidelines (Hill 1965). No more, no less.

[3] It goes without saying that, when discussing policy, questions about the ethical principles and values that ought to guide interventions inevitably come up. Those are certainly important. However, I want to focus here on a theoretical issue, namely on what constitutes *evidence for* policy in public health contexts.

It is worth emphasising that the worries of evidence-based proponents concern the *best* evidence to license inference and action. However, in the discussions about how to assess what evidence is *best*, a clear statement about *what* is the evidence that undergoes assessment is missing. This is the point I am most concerned with. Consider for instance Bradford Hill's guidelines for causal assessment. Even if we agree that they provide a comprehensive enough list for causal assessment, we need to make clear *what* these guidelines help us assess. According to Russo and Williamson (2007), the guidelines assess evidence of mechanisms and evidence of difference-making. Once we make clear *what* evidence we are assessing, we can discuss about the *quality of evidence*. For instance, causal assessment is certainly more reliable if the underlying mechanism has been 'confirmed' by current molecular epedemiology and biology, rather than just 'plausible'. Or, it is certainly better to have statistically significant correlations (one possible form of evidence of difference-making) that are consistent across various studies, rather than just a weak association. But again, the question 'what evidence is *best*?' comes after the clarification of *what* evidence is being assessed.

Granted, there is an effort to provide a positive, more specific account of *what* evidence is to undergo evaluation for public health purposes. Notably, some EBPH practitioners distinguish between Type I and Type II evidence. Type I evidence points to a particular health condition for which some preventable risk factors have been identified. Such evidence tells us that *something* must be done. Type II evidence points to specific interventions that proved to be relatively efficacious in order to improve a particular health condition. Such evidence tells us that *this particularly* must be done. The evaluation of either type of evidence is done through systematic reviews of findings in well-conducted studies, especially with attention to their internal and external validity (Brownson et al. 1999). This brings us straight onto the next point about evidence assessment.

EBPH theorists, just like EBM theorists, oft appeal to the so-called 'evidence hierarchy' and claim that the best evidence comes from randomized clinical trials—to be sure, from systematic reviews of several RCTs. There are a number of arguments that may be developed against this claim or against the 'received' hierarchy altogether. I will not endorse this line of argument here, though. I just want to flag the issue and report that critical views come from the scientific community itself, not just from the philosophical community. For instance, Rychetnik et al. (2004) admit that RCTs are important for causal assessment but make the point that the complexity of public health requires that RCTs be accompanied by other forms of causal assessment, e.g. observational studies using

adequacy or plausibility design. Likewise, Glasziou et al. (2007) make the point that sound causal conclusions may be reached through observational methods too; consequently RCTs are mistakenly considered *the* gold standard of causal inference. However, as I said, I will not pursue this line argument here.

In spite of the valuable attempts to give rigour to evidence assessment, the evidence hierarchy—just like the distinction between Type I and Type II evidence—leaves largely unanswered the question of *what* evidence is in fact needed for policy. To be sure, this is a concern shared by theorists of EBPH too. Witness for instance Rychetnik et al. (2004, p. 538):

> In the broadest sense, evidence can be defined as "facts or testimony in support of a conclusion, statement or belief".[4] Such a generic definition is a useful starting point, but it is devoid of context and does not specify what counts as evidence, when, and for whom.

What evidence is needed for policy is likewise left unspecified in official documents of agencies such as the European Food Safety Authority (EFSA).[5] Consider for instance the "Regulation (EC) No 1924/2006 of the European Parliament and of the Council of 20 December 2006 on nutrition and health claims made on foods".[6] The first chapter of the document provides precise definitions of the terms that will be subsequently used, such as 'nutrient', 'health claim', 'reduction of disease risk claim', etc. Throughout the document we are repeatedly told that evaluation of food is made on the basis of scientific evidence, yet *evidence* is never defined.

A charitable interpretation would be that what is meant by evidence in this context is exactly the evidence discussed in scientific publications of evidence-based public health theorists, as the ones mentioned above. Here, evidence is typically understood as evidence of difference-making in the form of statistics and risks. In fact, randomised trials use statistics to establish, to put it very roughly, that treatment (rather than placebo) makes a difference to recovery in chosen samples of individuals. The importance of evidence of difference-making is clear already since the 'manifesto' of the EBM movement (Evidence-Based Medicine Working Group, 1992), where knowledge of the basic mechanisms of disease is said to be "necessary but not sufficient". However, given (i) that EBM theorists don't explain *how* to use knowledge of mechanisms and (ii) that they trust the results of randomised trials based on their formal characteristics (namely, randomisation), evidence of difference-making (in the form of statistical evidence) definitively overrules evidence of mechanisms.

Thus I contend that EB proponents, even if they theorised the Type I and Type II distinction or the 'evidence hierarchy', did not adequately answered the question of *what* evidence is needed for public health in a satisfactory way. Moreover, what is left unanswered is what roles different types of evidence serve in making policy decisions. This, I shall examine next.

Causally-based public health

So far I argued that two types of evidence—evidence of difference-making and of mechanisms—enter causal assessment in epidemiology ("Public health and epidemiology"). This thesis, I argued in "*Evidence*-based public health", goes against EB proponents, as they give more importance to difference-making at the expense of mechanisms. To the contrary, I now argue that public health policy needs evidence of difference-making *and* of mechanisms to plan effective interventions, able to reduce the burden of disease at the population level.

Understanding and acting: *causally*-based scientific practices

Let the following idea guide the arguments below. As a general rule, making good decisions and taking good actions depend on having a good understanding of the phenomenon or situation. To be more specific about public health policies, the better our understanding of the disease, the better too the interventions to reduce the burden of disease. Thus, if we accept the idea that understanding disease causation involves considerations about different types of evidence (see "Public health and epidemiology"), then those different types of evidence should feed the design of public health policies too.

Killoran and Kelly (2010, pp. xxii-xxiii) make a list of what they take to be the key features of EBPH. Two of them, in particular, matter for the present discussion. One is "conceptual plausibility: an understanding of causal pathways defining the factors influencing health and the potential for intervention"; the other is "use of different types of evidence to determine what works for whom in what circumstances".

[4] Trumble W.R., Stevenson A. (eds), *Shorter Oxford English dictionary on historical principles*, Oxford:Oxford University Press, 2002.

[5] Information about EFSA, its goals, organisation, documents, campaign and events can be retrieved from its portal, URL= http://www.efsa.europa.eu/.

[6] The document may be accessed at the following stable URL= http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2006:404:0009:0025:EN:PDF

This patently brings causal questions upfront, such as those that I mentioned at the end of "Public health and epidemiology", and in the discussion of evidence-based public health in "*Evidence*-based public health". Those questions, recall, concern what kinds of evidence are typically involved in causal assessment. I argue below that evidence of mechanisms cashes out Killoran and Kelly's 'causal pathways' and difference-making backs up their 'what works for whom in what circumstances'.

Thus, this section pleads for the idea that public health policy should be *causally*-based, not just evidence-based. Some may immediately retort that causality is too strong a requirement for policy. Not only this is an area where a good grip on causal relations is rarely obtained; moreover, as a matter of fact, decisions and actions are normally based on sole knowledge of risk factors and their strength. To this I counterargue that intervening on factors that are not the *causes* of disease would turn out to be useless. Consequently, to ensure (as much as possible) that policies are effective, we have to intervene on the *causes* of disease, not on factors *merely associated* to the disease. Moreover, even if it is common practice to design public health policies on the basis of mere risks, this is bad practice notwithstanding their strength. I explain below why evidence of mechanisms, beside evidence of difference-making cashed out in the form of risks, offers a more solid basis to public health policy.

Concerns about the relations between evidence and policy have also been raised by Nancy Cartwright. Cartwright (2009) argues that *causal* knowledge is valuable for policy and planning. In particular, she is interested in the connection between causal knowledge and the ability to predict results of manipulations or interventions. To this end, what is most relevant, according to Cartwright, is whether 'invariance' and 'modularity' are able to deliver *causal* relations, as many people believe. In fact, invariance (i.e., that model parameters exhibit some kind of stability across changes of environments or interventions) and modularity (i.e., that we can intervene on one factor to see its effects without affecting the whole system) are usually taken to be the fundamental characteristics that allow structural models (i.e., the models customarily used in economics and econometrics) to correctly represent causal relations. Whilst I am concerned with the general problem of the *types of evidence* to establish whether a relation is causal, Cartwright, as I see it, is more particularly concerned with the assessment of evidence of difference-making, cashed out in terms of invariance and modularity in structural models.

But Cartwright (2008) is also concerned with what claims are *relevant* for policy and, she argues, the relevant claims are *causal* claims. It is along those lines of reasoning that I will next make the case for the claim that the two types of evidence involved in causal assessment ought *also* to be involved in public health policy, because of the specific role each of them serves.

It is worth making clear that the normative claim is that evidence of difference-making and of mechanisms should *explicitly* enter EBPH guidelines, such as the ones of the European Food Safety Authority mentioned earlier. My critical target is the way evidence of mechanisms appears in EBPH guidelines: it rarely figures in those documents and, when it does, it is *just implicitly*. My arguments are precisely motivated by this omission. The need, use, and role of evidence of difference-making and of mechanisms have to be explicitly stated in working documents, textbooks and scientific contributions. The danger, if these different evidential components are not made explicit, is to have a deficient causal assessment or policy.

Notice, though, that I am *not* claiming that no action is possible without detailed evidence of each of the two types. Rather, my point is that *better* action can be planned *if* those are explicitly taken into account, as much as possible and subject to their availability. There certainly are situations in which some decision must be taken even in the absence of full evidence (most typically, in the absence of good evidence of mechanisms). It does not follow, though, that research to collect further evidence (of mechanisms) is not needed anymore.

Evidence of difference-making and of mechanisms

I now argue that public health policy needs back-up coming from evidence of difference-making and of mechanisms, broadly conceived; each serves a specific role in public health policy.[7]

Evidence of difference-making

Public health policy needs information coming from evidence of difference-making. Evidence of difference-making may be in the form of e.g. statistical relevance relations, probabilistic dependencies, risks. This is typically provided by descriptive epidemiology, which answers questions about who, what, when, where.

Such information is helpful in deciding whether policy interventions have to target the whole population (e.g., in food labelling), or only subgroups (e.g., families) or individuals that fall under certain categories. Thus the role of evidence of difference-making is to back up considerations about *what works for whom in what circumstances*. Part of

[7] For clarity of exposition I draw a neat distinction between evidence of difference-making and evidence of mechanism. However, in practice this distinction is much more blurred and in fact the two types of evidence are often entangled. For a discussion, see for instance Russo (2011).

the job is to make it plausible that what worked for some population will also work in another one based, by and large, on arguments by analogy.[8] Such information is collected, for instance, by local and national cancer registers, by obesity databases,[9] or by a number of research projects promoted by the Executive Agency for Health and Consumers.[10]

Here are two examples of how policies can target different groups, according to what is supported by evidence of difference-making. Food labelling is a policy intervention that targets virtually the whole population in order to induce a global change in food consumption, or at least more awareness of dietary issues. Other policy actions are instead more specific. The MEND programme, established in 2004, aims to teach children and their families how to live more healthily. By targeting children in the age ranges of 2–4, 5–7, and 7–13, MEND programmes also target the parents of obese children thus aiming to positively change their and the children's habits about nutrition and lifestyle. But MEND can be even more specific, as it is fact possible to report an obese child to MEND officers, therefore getting the whole family involved in the programme. MEND proved to be quite successful in reducing obesity. Targeting the right groups and individuals, that is having and using the right difference-making information, is certainly part of the success.

On a different level, evidence of difference-making may also help whether it is more efficient to intervene on the social or biological factors of disease (or on both simultaneously). In this respect, it will be interesting to compare the 2008 report on obesity policies in the United States published by the Trust for America's Health and the Robert Wood Johnson Foundation (Trust for America's Health 2008) and, again, the MEND programme. Many policies in the US, according to the report, target the following behaviours: physical activity, fruit and vegetable intake, breastfeeding, consumption of sugar-sweetened beverages, intake of high energy density food, television viewing. We further read in the report (p. 37) that

> [o]ne objective common to almost every state is the urgency to get people involved on all levels; this is known as the Social-Ecological Model. This model aims to affect behavioral change by engaging all levels of influence—individual, interpersonal, organizational, community, and public policy. [. . .] Some states focus exclusively, or to a large extent, on childhood obesity.

One interpretation of the failure of obesity policies in the US is that evidence of difference-making, that supports considerations about 'what works for whom in what circumstances' is not properly used and consequently policies don't target well-selected groups of individuals. Consider the choice of privileging children, for instance. The success of the MEND programme lies in the recognition that, to reduce child obesity, we may need to intervene on the child's eating behaviour and physical activity *and* on the parents of the child: both on proximate and remote factors, both on biological and socio-psychological factors (see also below).

Let me emphasise that the importance of *social* determinants of health has been long discussed both in epidemiology and public health. Social epidemiology experienced ups and downs in the last decades. The point at stake here is that determinants—whether social or biological–are *difference-making components*. No matter how important they are, we also need a mechanism that explains *how* social determinants can have effects on health. Russo (2011) makes the point for epidemiology, and in this section I further argue for a complementarity of socio-biological determinants *and* mechanisms for public health purposes.

Evidence of mechanisms

Public health policy needs evidence of mechanisms too; this is provided by analytic epidemiology, which is mainly concerned with explanatory causal hypotheses and answers questions about 'how' and 'why'. Recall, the goal of analytic epidemiology is to design studies to test hypotheses that come from descriptive epidemiology. The scientific literature stresses the use of measures of associations (e.g., relative risks and odds ratios) and hypothesis testing. However, arguably, this is just half of the story. In fact, measures of association and hypothesis testing need to be underpinned by evidence of mechanisms in order to be explanatory (or otherwise they just restate evidence of difference-making).

A good example is provided by the studies on obesity and type 2 diabetes, more particularly on the mechanisms regulating insulin resistance. Those investigations are motivated by descriptive studies that reveal neat correlations

[8] I say 'part of the job' and 'by and large' because this leads us straight into problems of external validity, which are far from being settled. For a novel and thought provoking account of external validity see Steel (2008); for a discussion of the insufficiency of statistics for external validity, see Cartwright (2011).

[9] See for instance the National Childhood Obesity Database 2005–2006 (http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsStatistics/DH_063565).

[10] See for instance http://ec.europa.eu/eahc/projects/database.html?prjno=2003305, or http://ec.europa.eu/eahc/projects/database.html?prjno=2004313. Notice that, although those projects are listed under policy actions, they are in fact highly exploratory, in that they are meant to provide relevant difference-making information for actions such as prevention in children or particular classes of professionals.

between increased numbers of overweight or obese people and numbers of people affected by diabetes.[11]

Thus, the role of mechanistic evidence is to provide information about the *causal pathways* upon which to intervene.

Notice, though, that such pathways need not be sharply nor only biological. There is in fact substantial investigations on social determinants and health inequalities in general and for obesity in particular.[12] I shall develop this point more thoroughly below when discussing 'ecological' views of obesity). A number of remarks are in order.

First, although there is no 'conceptual' priority of one type of evidence over the other, it is true that different types of evidence may have unequal weights in assessing different hypotheses of disease causation. For instance, in addressing the question of what caused the increased obesity prevalence, evidence of difference-making is more important for the 'fast-food hypothesis'. According to this hypothesis, the drastic changes in dietary habits, and in particular the increased consumption of greasy meals prepared in fast food restaurants explains the rapid increase of obesity in the last fifty years or so. In this case evidence of difference-making—namely about 'who' eats 'what' and 'when'—is more important to pick out the right targets for intervention, as the mechanism is pretty obvious: a much easier access to food, which is in turn richer in fat than it used to be, makes people get fat very quickly. On the other hand, when investigating the genetic hypothesis, evidence of mechanisms is the primary focus and difference-making will play an auxiliary role. According to this hypothesis the main factor explaining obesity is some particular genetic make up that in turn regulates insulin resistance. Thus, what is most important is to work out the 'causal pathways', and difference-making may help in singling out similarities and dissimilarities across different groups of individuals. Therefore, the heavier weight given to difference-making or mechanisms has to do with the specific research questions at hand, not with alleged conceptual superiority of one type of evidence over another.

Second, biological and genetic factors are not enough to explain obesity. Biological factors *alone*, including genes playing specific roles in the mechanisms underlying obesity, do not wholly explain the rise of obesity because it has happened too quickly in evolutionary terms. Obesity, instead, is arguably the result of interactions between biology and environment. This thesis is supported, for instance, by Power and Schulkin (2009). Notably, Power and Schulkin (2009, p. 5) hold that "much of the increase in human obesity is due to a mismatch between adaptive biological characteristics of our species and the modern environment, which has changed dramatically from the one under which we evolved".

What is put forward is then an *ecological* view of regulation of food intake: beside biological and genetic causes, there are also *socio-economic* causes of obesity. Before, life was hard and food scarce. Improvements of general living conditions, including access to food, resulted in more elevated intakes of food, especially of fats. The switch from agricultural economies to ones based on manufacturing also meant reduced costs of food and less calories to consume; also, exercising stops being part of normal working time and is confined to leisure time. These socio-economic changes explain the cross-sectional and time-series patterns of obesity better than biological factors, addiction, and cultural changes (Philipson and Posner 2008). This is again an example showing that the available evidence of biological mechanisms supporting the hypothesis of a 'biological' increase in obesity is deficient.

What is instead needed is an 'integrated' mechanistic picture where the mechanism explaining obesity involves at the same time biological and socio-eco-psycological factors. Consider again Greener et al. (2010). What emerges from this type of study is that there isn't just one mechanism in place, but several mechanisms, according to whether we consider the overweight people's, the health professionals' or the policy makers' perspective on the disease. And even within these perspectives, mechanisms may involve more biological or psychological or socio-economic factors. The success of a policy thus depends on identifying the right group target and the right factors to intervene upon. A thorough multi-faceted mechanistic understanding of obesity can therefore offer means to set up effective policies.

Third, the question still looms as to why mechanistic evidence is needed after all, given that policy makers almost exclusively use information coming from risks. The answer to this question is that the more detailed the knowledge of the pathways, the more accurate the decision on what factors to intervene upon. Public health can hardly intervene on the genetic causes of obesity. Instead, significant results can be achieved if the patterns of behaviours of different types of obese people are identified. The MEND programme exemplifies this again. They aim to reduce child obesity by targeting families and parents. The key is to make parents realise that being overweight can cause their children not only health problems now and in the future, but also unhappiness, lack of confidence, depression. Thus, MEND appeals to psychological mechanisms (both in obese children at risk of depression and in the parents worried to avoid such a situation) in order to

[11] See for instance http://ec.europa.eu/research/leaflets/diabetes/index_en.html. Here is a list of projects funded under the 5th and 6th Framework Programme: http://cordis.europa.eu/lifescihealth/major/diabetes-eu-funding.htm.

[12] See for instance http://ec.europa.eu/health/social_determinants/policy/index_en.htm.

improve health conditions. What supports this type of action cannot simply be risk factors and difference-making considerations, as those are by and large correlational, and although they indicate *possible* factors for intervention, there is no guarantee that policy will work. What is needed, and what indeed supports MEND-like actions, is exactly an understanding of the various pathways to obesity. Setting up policies taking mechanistic considerations explicitly into account is likely to increase the effectiveness of policy. Granted, no evidence will give us certainty that some action will work. Yet, everything else being equal, adding mechanisms brings an added value.

Let me also stress the following point. To this stress on mechanisms, it may be objected that if we have found an effective intervention, then understanding the relevant mechanisms adds very little, if nothing at all, to the implementation of such an intervention. Reasoning, however, should go the other way round. Effective interventions are found *on the basis* of a relevant mechanism. The MEND programme discussed above exemplifies this again. Their interventions proved effective *because* they had a good understanding of psychological mechanisms, whereby by intervening on the parents we can obtain effects on their children's health.

Finally, the thesis that evidence of difference-making and of mechanisms are typically needed for causal assessment has the nice consequence that they usually help and support each other. This somehow goes against traditional views according to which to establish a causal claim *different and independent* sources of evidence are needed in order to triangulate. But the 'tangle' of difference-making and mechanisms on the one hand and of biogenetic and socio-economic factors on the other hand exactly mirrors the complexity of disease causation (on this point, see especially Russo 2011). Again, the phenomenon of obesity is an exemplar in this respect. It is hard to separate out completely difference-making considerations about biological and social factors from the corresponding mechanistic considerations. A synoptic view, where those aspects are all simultaneously present, can help isolate, successively, more precise targets and factors for intervention. But it is the interplay between the difference-making and the mechanistic considerations that advances our understanding of obesity (and of disease in general) and of our actions to reduce its burden.

Conclusion

Public health aims to prevente disease, prolong life and promote health of populations. *Evidence-based* public health, more specifically, seeks to reach such goals by means of population-level interventions that are decided on the basis of the *best* scientific evidence. Nevertheless, despite all this emphasis on *evidence*, *what* evidence ought to inform public health policy has not received a satisfactory answer yet. Studies on obesity are paradigmatic as they show the complexity of the issues behind public health policy and therefore help us in drawing useful lessons about what evidence is needed.

In a nutshell, I defended the idea that public health policies ought to be informed by two types of evidence, broadly conceived: evidence of difference-making and of mechanisms. The argument runs in three steps. First, public health policy strongly relies on findings coming from epidemiology because it provides a population-level perspective on disease *causation*. Drawing on other works, I concluded that causal claims in epidemiology (and in medicine more generally) are established on the basis of difference-making and mechanisms. Second, evidence-based public health heavily relies on evidence *assessment* but, I argued, this does not fully answer the question of *what* evidence backs up public health policy. Third, I pleaded for *causally*-based public health on the grounds that evidence of difference-making supports considerations about 'what works for whom and in what circumstances' and that evidence of mechanisms indicates where to intervene in the identified causal pathways.

The main objection is that it is common practice to set up public health policies only on the basis of risks, that is on the basis of difference-making. Far from urging that action is taken *only* with full mechanistic knowledge of the disease, the argument is that *better* policies can be envisaged *if* evidence of mechanisms is explicitly taken into account, whenever available.

The advantage of adopting such a view is to have a coherent account where causal assessment, based on evidence of difference-making and mechanisms, contributes to *understanding* disease and to *take action* to reduce its burden in population.

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