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Cognitive biases in public health and how economics and sociology can help overcome them

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ABSTRACT

Objectives: The objective of this study was to identify important gaps in the public health evidence base and consider the implications of these for public health and public health economics.
Study design: This was a review and critique of public health policy in the UK.
Methods: Using two key psychological concepts relating to cognitive biases, viz. cognitive dissonance and heuristics, the shortcomings in public health approaches to confronting the prevalence of non-communicable diseases are described. The implications are drawn out.
Results: Two cognitive biases in public health thinking are identified. (i) A dissonance between what is known and what is done, resulting in the repetition of solutions that have previously been shown to have had little or no effect. (ii) The habitual use of set of heuristics which mean that simple solutions to complex problems are preferred to undertaking the detailed assessment of how to bring about change. These biases mean that the evidence about the dynamics of populations and the ways that the mechanisms of prevention actually operate seldom feature in the way interventions, policy and practice are undertaken. The evidence base is consequently highly skewed.
Conclusions: Health economics combined with sociological reasoning has potentially an important role to play in developing the ideas that will overcome the problems attaching to the cognitive biases.

Introduction

Two cognitive biases in public health thinking are barriers to effective policy implementation: a dissonance between what is known and what is done and the habitual use of short cuts in thinking involving finding simple solutions to complex problems. This article argues that the consequence of these biases is that evidence-based interventions are not put to work effectively. It is suggested that a coalescence of ideas about complexity from economics and sociology will help to begin to break down these barriers.

The evidence base in public health has expanded and developed considerably in the last two decades. The knowledge base is large and growing, and these advances look set to continue (See, for example, https://www.nihr.ac.uk/about-us/documents/NIHR-Annual-Report-2015-16.pdf). The evidence base provides a potentially important platform for policy and practice development. Yet, there is a gap between what is

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known and what is done. In spite of the strength and depth of this evidence and in spite of the existence of Cochrane (formerly the Cochrane Collaboration), the National Institute for Health and Care Excellence (NICE) and the infrastructures created by the National Institute for Health Research (NIHR), the School of Public Health Research, the Medical Research Council (MRC), Public Health England (PHE) and the Department of Health and Social Care, as well as similar developments in Scotland, Wales and Northern Ireland, there is a paradox. Some of the most important research undertaken about primary, secondary and tertiary prevention in the world is funded and conducted in the United Kingdom. There are excellent research centres and arm's-length bodies producing primary research and reviews of the evidence about risk and causal pathways to disease at individual or population level, as well as about the cost-effectiveness of interventions. There are well-argued and extensive data about the patterning of health at population level and in particular about health inequalities. The Research Excellence Framework 2014 (REF) results in Unit of Assessment 2 (Public Health) showed that the research outputs in that unit stand comparison with, and in many cases surpass, the best from the top centres in the US and elsewhere.[1] The quality of British research in public health is world class. However, much of what is known, discovered and revealed by the research is not, or is only partially, implemented in policy and practice. The reasons for this, it will be argued, are two cognitive biases that are found in policy, practice and interventions. First, a dissonance between what is known and what is done, which results in the repetition of that which has previously been shown to have had little or no effect.[2–4] Second, the habitual use of set of heuristics or short cuts in thinking which mean that simple solutions to complex problems are preferred to undertaking the detailed assessment of how to bring about change.[5,6] Therefore, what is known, and indeed what is known to be not known, is not subject to detailed analysis and scrutiny. A number of consequences follow from this state of affairs.

The fact that there are gaps between research and policy and research and practice is not, of course, a new observation.[7,8] It has often been noted and commented on, not just in public health and not just in the UK. There are many models, which purport to describe and to explain the problem or which seek to find solutions. There are linear ones such as that proposed by Cooksey,[9] cyclical models,[10] explanations couched in terms of complexity theory[11–13] and political models.[14] There are journals devoted to implementation science and a system of university performance management in the UK – the REF – designed to bridge the gap in various ways.[15] However, Public Health England (PHE) suggests that there is still a 17-year gap between initiation and uptake of research.[16] Eerily, all of this echoes the observation made by Archie Cochrane back in 1972 about the failure of the medical profession to keep up to date with emerging evidence and innovation.[17] (For an overview about public health see Orton et al.[18])

Methods

This article seeks to identify the cognitive biases specifically as they apply to public health. The author worked for 14 years before his retirement at senior national policy level in public health in England. The observations reported here are based on that experience. The focus is the relationship between what is known from the evidence and why it is applied suboptimally. The discussion takes place within the framework outlined by Smith.[19] She argued that the relationship between research and action/policy/practice is best understood as a constant interaction between many actors, which include, but is not restricted to, researchers, policy makers and practitioners. It also embraces opinion formers, journalists, politicians, officials, pressure groups, vested interests, industry, commerce and members of the public. The interplay of ideas between these various actors has emergent properties that include policy and practice outcomes. The argument in this article is that (i) within that commerce of ideas, the cognitive biases play a significant role in shaping the discourse and (ii) we need to develop economic ideas to better meet the consequences of these biases.

It has been noted for many decades, prefiguring the arguments in this article, that the nature of economic and social arrangements are the product of the multitude of different actions and the knowledge of countless individual players acting according to their own volition. Furthermore, the collective consequence of these actions is the structure of economic and social systems. Hayek, for example, argued back in the 1940s that the price mechanism operates because of the actions of many players in the market and that it would be impossible therefore to plan an economic system because no single economic actor or the state could command sufficient knowledge and understanding of the complexities involved.[20] Hayek saw the price system as both the determinant and product of individual actions. The sociologist Antony Giddens used a similar argument about the social structure which he argued was the product of the interactions between the many millions of individual human actions. These many millions of individual actions produce structured patterns at the level of society. The patterned structure in turn constrains and limits the choices open to individuals.[21] The importance of this coalescence in economic and sociological thinking will be considered in the following sections, but the fact is that the failure to acknowledge the importance of the multiplicities of human actions is fundamental to the cognitive biases with which this article is concerned.

Results: systematic biases in the evidence base

What is known about populations and the way public health policy and practice generally describe populations

The first bias is the dissonance between what social scientists have discovered about how populations work[22] and the way that populations are conceptualised in public health.[23,24] The understanding of the effectiveness of interventions, and how to put them into practice for example, is seriously hindered by a lack of understanding of the mechanisms by which interventions work in different segments of the population. This in turn is compounded by a lack of use of knowledge and evidence about population dynamics themselves.

The way that populations work as dynamic entities and the implications of this for public health policy and practice is neither well understood nor theorised very adequately.[25] At its most basic, the categories used to describe the population are crude and coarse (socio-economic status [SES], ethnicity, gender, education and income). These categories are associated with health and illness, but the categories themselves conceal the enormous diversity in the population – its social variegation. They are coarse because they are unidimensional – people are not just their occupation, or their educational background or their ethnicity. People are members of multiple social groupings with distinctive lifeworlds; they engage in diverse social practices, and their behaviour and their health are not the product of, nor can be reduced to, single categorical variables. They are crude because they are reductionist – focussing on bringing things down to the individual level of analysis. SES, for example, is treated as a characteristic of an individual, usually with reference to occupation, education or income. However, the collectivities to which people belong and the relationships between those collectivities are drivers of the dynamics of society. In turn, these relationships, and particularly power relationships in the workplace, communities and markets, are fundamental to the patterning of health and disease.

However, this relational dynamic is ignored. Instead, the categories are assigned to individuals and then used to count individual events cross sectionally before being aggregated up to population level and then sometimes plotted longitudinally. The intersections between the complex dynamic social variations in the population are seldom pressed into service in public health thinking.[26] The interactions and synergies between age, gender, ethnicity, occupation, tribe, caste, geography and housing tenure and the ways these change across time profoundly shape people's lives and their health. The fluctuations and nuances in the intersecting lifeworlds of the populace and the ways these interact with each other and change over time, all of which would allow us to understand better the differential effectiveness of interventions, are simply not usually part of the discourse of disease prevention or health protection and promotion.

This is in spite of the fact that there is an evidence base to draw upon. The work of sociologists such as Savage,[27] which pick up the nuances of life in contemporary Britain in a highly sophisticated way, seems to have passed by the public health community at large, in spite of the fact that arguments for embracing this type of thinking have been around in the literature for more than a decade.[28] Consequently, the categories do not capture the huge amount of individual and collective variation, and they fail to treat the population as a dynamic entity. Populations or societies are dynamic and constantly changing. They are not the mere aggregation of the individuals which make up the population.[29] Ontologically, populations are real in themselves and can be analysed in themselves and understood in that way.[30]

In short, society and the communities and neighbourhoods within them are not treated as complex entities with recursive interactions occurring continuously between the social, subjective, biological and physical phenomena.[31,32] In general, the public health community does not seek to make sense of the complexity. There is a lot of rhetoric about complexity in the literature, although none of this really has a social or economic theory of complexity embedded in it. The result is that actions designed to help to protect the population from disease, to protect it from hazards and to promote good health are largely complexity-free zones. The default is to fall back on cross-sectional accounts of the categories, which conceal as much as they reveal. This is true even of the landmark attempts by MRC to deal with complexity.[33–35] These contributions were very important in moving the argument forward but, to date, have been deficient in providing a social theory or an empirical account of complexity themself.

There is a collective cognitive dissonance.[36] On one side is the knowledge base about the complexity to be found in sociology, psychology, anthropology and economics and in some of the landmark statements by physicians such as Engel[37] (and even in the rhetoric of public health[38]). On the other side are high-level descriptions of health inequalities and the continuation of interventions that fail to deal with the prevalence of non-communicable disease.[39,40] The mechanisms operating at the population level are not described in ways that are useful to do interventions or to change things.

Guideline developers and others constructing interventions are in effect hamstrung because they do not have empirical or theoretical accounts of dynamic social mechanisms.[41] They are armed only with data about associations.[42] It is very difficult to get down to a level of granularity to help to develop interventions that would be fit for purpose in different sections and segments of populations and to tailor to the needs of specific groups and communities. Data about associations or correlations do not explain cause, although cause may sometimes very helpfully be inferred from associational data. From a causal point of view, however, we need to describe and understand the mechanisms to be able to describe causes. By articulating the mechanisms involved (by taking the dynamic approach to the social, economic and biological advocated here), we are able to identify points for intervention more forensically and with greater granularity. My argument is that this obvious point is habitually ignored in policy, which remains fixated on individually behaviourally based solutions when individual human behaviour is at best only one part (and not necessarily the most important part) of the mechanisms at work. The necessary knowledge of mechanisms is simply missing. In clinical medicine, the fact there is biological variation between individuals is a *sine qua non* of practice – at its most basic, not everyone responds to the same drug or treatment in the same way. The implications of the fact of social variation are just as important, but the efforts to explore and understand that variation are, in public health terms, in their infancy.

The argument is that the categories such as SES, ethnicity, gender and so on are not used in ways that capture complex dynamic processes and the mechanisms involved. This is because the way they are habitually conceptualised and used in the public health literature, and more specifically in policy, is reductionist.[43] The categories do not in themselves imply reductionism, but the way they are used is reductionist, with the consequent concept and policy default to individually based solutions.[44] It is however possible to conceptualise the social level or population level separately from in the individual one, in a way analogous to the economic level of

analysis of the market. This helps to get through the cognitive biases. It is as if this idea of the dynamic social level is hidden in plain sight. The individual is so obviously and commonsensically the focus because the world is made up of individuals. But, individuals interacting with each other produce a reality that is at once both economic and social, as Hayek and Giddens in their different ways make very clear.

Mechanisms of prevention

The second bias refers to the focus on the wrong mechanisms and evidence about prevention.[45] In public health policy at large, there is a focus on cause of disease and the origins of health inequalities.[46–48] There is much less attention paid to the mechanisms of prevention. In essence, the heuristic or cognitive bias is that the assumption is made that if you know the former (the cause), you will be able to do the latter (prevention). The empirical fact that the mechanisms of aetiology may be quite different from the mechanisms of prevention (and that this has been known for decades) is seldom brought into sharp focus. This has become a default bias.[49] The same decisions are made instead of taking note of the consequences of those decisions previously. This is habitual and almost automatic.[50]

This collective cognitive bias or heuristic in thinking is understandable. It is an unintended consequence of two great public health advances. First, with the eventual discovery at the end of the nineteenth century that germs present in dirty water could cause disease – and the coincidental realisation that the provision of clean water and sewage disposal was an effective prevention strategy – cause and prevention were coupled together. Certainly, protecting people from insanitary conditions is a highly efficacious public health strategy, but knowing which germs are in the water, does not tell you how to build a sewer. Very importantly, we must remember that the engineers who built and designed the sanitary systems did not do so originally because of knowledge about water-borne microorganisms – they were miasmatists and were trying to improve air quality.[51] More generally, knowing of the existence of germs and other microorganisms does not necessarily tell you how to prevent their spread. If it did, we would have stopped hospital acquired infections and influenza epidemics long ago.

Second, the discovery that exposure to cigarette smoke causes lung cancer in some smokers and a range of other serious illnesses and the observation that if people stopped smoking, then the risks dropped dramatically also coupled cause and prevention. Once again preventing exposure to cigarette smoke is a highly effective public health strategy, but critically, knowing the statistical association or indeed understanding the biology of the aetiology of lung cancer and heart disease does not tell you *how* to help people to stop smoking. The latter requires knowledge of a whole range of evidence from other sciences such as the elasticity of the price of tobacco products, the psychology of addiction, the influence of advertising and the way peer-group pressure operates for example.

In other words, successful tobacco control and sanitation require a quite different evidence base to that which is about the aetiology of carcinoma of the lung or cholera. Understanding cause is the necessary but not sufficient condition – it tells you what to do but not how to do it. There is a gap between what we know about the mechanisms of the causes of the public health dangers facing us and our ability to turn that knowledge into practices that will facilitate how to halt or reverse or slow down the epidemics relating to obesity, alcohol misuse, lack of exercise and common infections.

A lopsided evidence base

A consequence of this is that the evidence base is lopsided and we continue to make it more lopsided. The evidence is heavily skewed towards details about proximal risk factors for communicable and non-communicable disease, towards elegant expositions of the way the wider determinants of health are associated with patterning of mortality and morbidity at population level and towards details about the effectiveness and cost-effectiveness of an array of interventions designed to prevent disease to improve population health or protect the public from various hazards. The evidence base for translating this information into accurate descriptions of the mechanisms needed for effective prevention strategies is scant, and the economic evaluation of these matters underdeveloped. In each of the areas – proximal risk factors, wider determinants and the interventions which are derived from the evidence, the mechanisms at work as against the associations and correlations between the various factors, are unexplored and underdeveloped. Therefore, with respect to proximal risk factors, for example, of alcohol consumption and liver disease or of calorie consumption and obesity, the associations are well established – as are the biological mechanisms involved. But, the social mechanisms are much less well described.

The variations in why, where, how, when, with whom and for how long people engage in swallowing food or alcohol is obviously enormously variable. That is because eating and drinking alcohol are not single behaviours but are embedded in webs of social practice which are the products of individual human agency, on the one hand, and in the social structures which are the product of human agency and also constrain and limit individual choices, on the other.[52] Clearly, there are mechanisms at work – but, for sure, the mechanism will be very different in the many different circumstances in which people eat and drink. Yet, seeking to make sense of these different mechanisms linking proximal risk and outcomes, for the most part, remain unexplored, whereas the default position is that changing behaviour is the answer.[53] That has been the direction of policy for decades, even though the results have been, at best, disappointing.

The picture is just as dismal with respect to the patterning of disease contingent on the wider determinants of health. The associations between the coarse categories of SES, income, education and gender and patterns of health and illness are very well established. However, what are the actual social mechanisms that link poverty and disadvantage and wealth and health, and what is the nature of the links between the social phenomena and the biological phenomena, is another great chasm in the public health armoury.[54] In recent years, developments in epigenetics and metabolomics have begun to demonstrate some of the plausible biological mechanisms

involved and the possible transgenerational effects of exposure to poverty and other noxious agents in the environment.[55] Similarly, historical scholarship demonstrates that these processes have a very strong historically recursive pattern of reproduction over generations. Notwithstanding epigenetic transmission across generations, independently the recursive nature of the consequences of local social and economic arrangement over time reinforces, generation on generation, the disadvantages and advantages of social arrangements. Much is known, but the gap between the biological and the social, and more particularly the nature of the mechanism between the two, remains at the level of statistical association, and not mechanistic understanding. Yet, it is a scientific question which John Snow, William Duncan and Rudolf Virchow would surely have recognised. The precise links between the biological and social and how it works are yet to be elucidated.

Discussion: implications and challenges for public health economics

Aside from the public health community generally, there are some interesting challenges for public health economics generated by these cognitive biases. With respect to population dynamics and especially with respect to the reductionism and individualism inherent in current thinking about populations, utility theory is individualistic *par excellence*. The reliance in cost-utility analysis and on data derived from randomised controlled trials, for example, mean that in a paradigmatic sense, the application of alternative ways of thinking are seldom entertained. The challenge mounted by behavioural economics[56] does not really change the paradigm. It simply suggests that individuals may be motivated by a range of factors in the environment and notes that their actions are not driven solely by utility maximisation. This is of course true but is still about individuals. Notwithstanding the success and power of the individualistically based economic theories, there is another very interesting issue in play.

There is in utility theory (as noted previously) a recognition of dynamism which when linked to social theory might be very useful. The notion of the market is at the social or supra-individual level of analysis. The ideas of structure and agency at the heart of social practice theory[57] strongly resonate with the idea of the market as a supra-individual structure that exists as a consequence, but independent, of individual decision-making.[58] The arguments proposed by Etzioni[59] in what he called socio-economics, are helpful in this regard. His idea that variegated communities are the expression of agency-structure interaction and that identity and self are critical to understanding people's practices offers an interesting potential theoretical contribution. It might also help to move the arguments in health economics beyond the distinction between efficiency and equity. In agency – structure sociological theory, societies are not conceptualised as inherently equitable or efficient and neither are efficiency and equity seen as goals that the system itself should or will maintain. Rather efficiency, equity, inefficiency and inequity are emergent properties of the dynamics of the complex system. To draw together the social and economic theory might be a very productive avenue for health economics and would help to overcome the bias described previously about population dynamics. The writings of the economists Piketty[60] and Varoufakis[61] are important too, not because they major on socio-economics but because they get into the detail of complexities and mechanisms, although different in each case. Both develop economic ideas in which the dynamism and detail of social systems are paramount and from which public health implications flow.[62]

Empirically, there are undoubtedly opportunity costs arising a consequence of the way that society or populations are presently conceptualised and the public health policies that flow from that conceptualisation. Of course, wrestling with the problems of heterogeneity has been intrinsic to much thinking across epidemiology and evidence-based medicine, and economic modellers, in particular, have been cognisant of this.[63] At its most basic, the fact that the different incremental cost-effectiveness ratios will apply to the same intervention introduced into different segments of the population has been acknowledged and was, for example, intrinsic to the way NICE public health models were constructed. However, this was a theoretical rather than an empirical exercise because mostly there were no data on differential outcomes in different population groups because such data were simply not collected in the primary studies.

From a heath economics point of view, so long as the primary studies in the public health population sciences do not operate with the levels of granularity required to describe population dynamics, economic modellers will have to fall back on a different strategy – but this itself may be an opportunity. The opportunity is to require that economic models take the fullest account of the best social science models about human behaviour. In this regard, in the psychological sciences, the advances being made in researching the mechanisms of behaviour change and the ontologies of the components of behaviour change interventions will provide a firm basis on which to develop future economic models.[64,65] Similarly, from sociology, the use of social practice theory in which simple determinism is eschewed in favour of theoretical understandings of the interactions between structure and agency, and the emergent properties in complex systems of human conduct provide another important platform on which to build models.[66] Greater interdisciplinary working between economists, psychologists and sociologists would undoubtedly help this process along. As Horton put it in a recent editorial in the Lancet 'public health science needed to pay more attention to the lived experiences of people in societies. Public health needed to recognise the importance of identity, reasoning and voice. Public health today is crudely reductionist, often ignoring or denying the lives of those it purports to defend.'[67] The social sciences have much to offer here to improve this state of affairs, and public health economics has a central place potentially.

Decision theoretic approaches also offer a potential solution to the biases. Threlfall et al. have argued that the combination of robust theory, causal understanding and observation are able to provide sufficient evidence of the direction of effect in public health interventions.[68] They propose moving from what they call the dominant hypothesis-testing approach that is based on the individualism and reductionism

in the current evidence base. The same team has argued that in many cases, it is simply not possible to conduct trials that are large enough to capture meaningful effect sizes because the nature of populations is such that the randomised controlled trial (RCT) cannot cope with the heterogeneity in any meaningful way. RCTs pick up individual effects and only get to population effects by aggregating the results – not by conceptualising the population as an entity in its own right.[69]

A decision theory approach utilises all relevant knowledge, theory and data. Building the links between the psychological and sociological ideas identified previously is a way to help develop the decision theoretic approach. The beauty of such a strategy aside from its comprehensiveness with respect to evidence and theory and its intrinsic interdisciplinary nature is that it releases the thinking from the constraints of the hypothesis-testing approach at the heart of the reductionism attached to RCTs and the individualism of epidemiology and utility theory.

In respect of mechanisms, there is much work to do. There are two dimensions to this: first, the mechanisms operating between the social and the biological. As noted previously, while plausible biological mechanisms have been described about the links between social exposures and biological consequences and this is a fast-moving scientific arena, the gap is between the social and the biological. Sociologists for the most part have been taciturn on this matter (but see Meloni et al.[70]), and biologists have been seemingly content to see the social as a factor that kick starts the biological processes without saying with any precision as to when and how it happens.[71] This is true even when there are known toxins in the environment. It is assumed that exposure occurs but how social factors interact to produce biological outcomes is not explored. Perhaps the most productive way to conceptualise the process is one in which physical, biological, social and subjective processes (which are normally treated as analytically distinct) are synthesised into a single complex system and in a dynamic and changing process. Within that process, the connections between social and subjective phenomena and the biological and social still need to be elaborated. And here, utility theory, or that part of it that describes market mechanisms as the outcome of individual decision-making but having an economic reality *sui generis,* can be conjoined with structure and agency in the social theories as suggested previously. In other words, it is out of the very repetitive nature of social and economic life, the social and economic practices in which people engage in their everyday lifeworlds that offer a clue. This is an exciting possibility for future development as mentioned previously.

Second, the links along the causal chains might be given some further attention. In general, even in the most sophisticated economic models, the mechanisms in complex causal chains are treated in a predictive way and as existing in time and space. This is the sense that event A at time T1 has consequences B at time T2; T2 occurs chronologically after T1. This allows the prediction that if A happens, then B will also likely occur. Most public health interventions are premised on this assumption, and in a world governed by Newtonian physics, that is a reasonable assumption. However, it is also perfectly possible to flip the coin metaphorically and to think about the question the other way round. In everyday life, most of us think the other way round a lot of the time. Therefore, when we ask the question why did something happen, at time T, we generally commonsensically answer that question by attributing it to a preceding causal event *that happened in the past*. We then may ask another question which is why did that preceding event occur. And, we could continue to ask why the event that preceded it occurred and so on in a potentially never-ending process of regress.

However, this way of thinking can actually be very helpful in model building because it allows us to disassemble phenomenon and see them for the multiple complexities that they may involve. This form of regressive forensic inference can be very helpful as we build and construct models and may be very useful in terms of economic thinking. Prediction can indeed be very seductive, although in the social sciences, it is often highly inaccurate. However, if we think about our models or build our models in this way and ask the question about the dynamic economic process at each of the stages, this may prove to be very fruitful. It would certainly assist in articulating the mechanisms of prevention and examining the economic processes at each of the stages. The theoretical and methodological means of articulating those mechanisms have been described and provide a useful platform on which to build.[72]

All of this would of course help to get beyond the problem of the skewed evidence base as we build the knowledge base in the arenas where the evidence cupboard is presently pretty bare. Novel economic thinking might also help to get past the cognitive biases and the cognitive dissonance. But, this comes with a health warning. The policy arena is much more than a set of cognitive biases and government failure and ineptitude, unanticipated consequences, internal and external power relations, vested interests, incentives and disincentives in the system all play their parts and in ways that no single agent has the ability, knowledge and wisdom to know.[73] Therefore, even if we can overcome the cognitive biases, these other realities of political and policy making life will not go away, although along with ways of implementing policy, they should form part of the evidence as we seek to gain leverage.

Conclusion

A number of writers have developed ideas that resonate with the arguments here. Rychetnik et al.[74] called for the recognition of the importance of evidence about *how* something should be done and argued that would include information about design, implementation and context and how the intervention was received. Brownson et al.[75,76] have noted that the evidence about what should be done based on risk is extensive but also stresses the importance of evidence about the relative effectiveness of specific interventions. Taking our cue from this, we must obviously continue to build the evidence for the *necessary* conditions for effective prevention, i.e., the evidence about aetiology, proximal risk factors and cost-effectiveness. However, we must also build the evidence base about the *sufficient* conditions for prevention. This is the evidence about how to create population level changes, how to implement policies that benefit all segments of the population and how to implement policies in ways that recognise how interventions are experienced (and resisted) by different communities. With the exception of tobacco control policy –

which incorporates evidence about sufficient conditions such as the price mechanism, the psychology of quitting and addiction and how to manage misinformation from big tobacco etc. – policy in many areas, including dementia, obesity, alcohol and physical activity, has been implemented sometimes without robust evidence but more usually with little consideration of the evidence about the mechanisms likely to produce optimal effectiveness. Economics needs to be front and centre in working with psychology and sociology in particular to develop new ways of thinking and new models to make this evidence clear.

In practical terms, this means that research funders should devote more resources to develop the evidence about the mechanisms of prevention,[77] and they should rebalance the portfolio away from aetiology and risk.[78] This would involve a focus on the politics of implementation for example and in particular on the power struggles that go on once the evidence of causation and risk have been established. It would involve building the evidence about how complexities of real-world systems (not imagined simple linear ones) operate. Practically, the argument also means that policy makers should become much more cognisant than they usually are, of the cognitive biases which with which they themselves operate. It is interesting to note that most government officials are acutely aware of, and sensitive to, the political nuances of every action they take and propose. But, in my experience, they are for the most part blindsided to the cognitive processes involved in their own thinking. And, my own tribe, academic researchers, should similarly not imagine the job is done once research findings are published. Frequently what happens now is that authors complete their work and leave it at that, hoping others will take up the baton, assuming that it is self-evident from their published work what ought to happen. More actively minded colleagues, however, sometimes get involved in advocacy. This is seldom an effective strategy as governments, decision-makers and industry are mostly impervious to academic advocacy, and in any event, telling people that they are wrong is seldom an effective way to change their behaviour. Instead, the political, social and economic processes that affect the ways that findings are received, interpreted, acted upon and implemented, in the way that Smith[79] has described, should be the subject of much greater academic and scientific scrutiny, than is currently the case. These findings should then be used to gain the leverage that is needed. And, it is not as if we do not know this as an academic community, it is just that we choose not to do it, preferring, very often, political gesturing, rather than applying our scientific skills to the processes that we seek to influence. Finally, as noted previously, economists and sociologists have long understood the dynamics of complexity, although most recent accounts of complexity pay almost no attention to this classic literature. But, there is clearly an opportunity for sociologist and economists to work together in that conceptual and theoretical space of markets and structure and agency. We need a strategic research programme to develop this, and a consilience between the disciplines should be encouraged. Furthermore, together, the theoretical insights of the two disciplines will, if applied properly, help shift the rather vacuous rhetoric that currently surrounds notions of complexity to replace it with some properly social scientific empirical and theoretical work.

The biases described here are no doubt to be found in many other fields of policy and indeed in other jurisdictions. Public health is not unique in this regard. However, it does seem likely to me that those government departments have a longer history of grappling with evidence, and here I single out transport and health in England as exemplars of good practice, will be more likely to able to get into an understanding of the bias problems than those departments where down the years, evidence has been less prominent in the decision-making process.

Author statements

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Competing interests

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