Chapter 9
Assessing Mechanisms in Public Health

Abstract Further considerations need to be borne in mind for evidence appraisal in areas beyond clinical medicine, such as public health. This chapter looks at how public health has treated associations and correlations. Then it examines the importance to public health of mechanisms operating at the group and individual level, concerning social interactions and support, access to socio-sanitary infrastructures, psychological factors, and so on, which have to be explored in the appraisal of public health evidence. Finally, the chapter considers the relationship between biological and social factors, and the difference between mechanisms of disease and mechanisms of prevention.

9.1 Introduction

When applying the ideas described in this book to areas other than therapeutic clinical medicine, a number of further considerations need to be borne in mind. The arena beyond clinical medicine where most thinking has been done relating to methods of evidence appraisal is public health (NICE 2012). Public health is concerned with actions, interventions and policies designed to protect the public from hazards, to prevent disease, and to promote good health (Tannahill 1985). In different countries, specific institutions were given the task of developing methods for the assessment of evidence and for the formulation of guidelines in public health. These individual efforts have been brought together into a European initiative, led by the European Centre for Disease Prevention and Control (ECDC). In their 2011 synthesis report, they show how public health should adopt and integrate the methods of evidence-based medicine, specifically the GRADE system, for the assessment of evidence (European Centre for Disease Prevention and Control 2011). In this chapter the focus is on one particular sub-issue, namely mechanisms of causation and, given the concerns of this book, how to deal with mechanisms conceptually and then practically in the appraisal of evidence.

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9.2 Public Health and Evidence-Based Medicine in the UK

Public health in the UK has been working within the evidence-based paradigm formally since 2000, and much has been learned (Kelly et al. 2010; Kelly and Moore 2012). In 2001 the English Department of Health published its Research and Development Strategy. Amongst other things it made the case for using the principles of evidence based medicine in public health (Department of Health 2001). Organisations such as the Centre for Reviews and Dissemination at the University of York, the Cochrane Collaboration, the Campbell Collaboration, the Health Development Agency and NICE took up the challenge. These organisations have confronted in various ways the methodological, theoretical, practical, epistemological and ontological problems of applying EBM principles to the very broad church of public health. Since then other policy areas have gone in the same direction of taking an evidence based approach. So social care, education and criminal justice, amongst others, have all had agencies created to move these arenas onto an evidence based footing (Paisley et al. 2018).

9.3 Statistical Associations and Correlations in Public Health

Statistical associations and correlations have been at the heart of progress in public health for many years. A number of landmark studies show just how important finding statistical associations can be. The investigations by Doll and Hill (1950, 1952, 1964) into the connections between smoking and disease are the original benchmarks. Their initial observations showed that there was an association between exposure to cigarette smoke and carcinoma of the lung (an association which had not hitherto been noticed). This led, in the long run, to public health policies which have reduced the prevalence of cigarette smoking in the population and greatly reduced the number of deaths from lung cancer, and also heart disease, stroke, and various other cancers, which were subsequently found to be associated with exposure to cigarette smoke too.

These pioneering works are often thought to be purely statistical, but in fact Hill was concerned with biological plausibility, and hence mechanisms (Hill 1965). Since the early 1950s when the first statistical observations were made, the biological mechanisms operating in the interaction between the contents of cigarette smoke and the tissues in the lung, as well as the mechanisms relating to the effects on blood circulation, heart functioning, arterial disease, and many other pathologies have been described. Considerations about biological plausibility also led to investigations of the relation between asbestos and mesothelioma (Doll 1955; Newhouse and Thompson 1965). Scientific discoveries relating to these mechanisms continues to the present. The basic mechanisms are well understood in individual human

beings, and public health policy has developed in such a way that smoking in the European Union is now a minority habit and protection from unwanted exposure to cigarette smoke is the norm.

So cigarette smoking was identified as what public health practitioners have come to call a risk factor. In the wake of this great public health success, statistical associations have emerged over the years pointing to risks from other things, notably a lack of physical activity, being overweight and obese, over consumption of alcohol (Sytkowski et al. 1996), certain types of sexual activity (Dougan et al. 2005), ingesting certain non-prescribed drugs (White and Pitts 1998) as well as toxins in the environment, although the dangerous consequences of exposure to certain substances used in industrial processes like asbestos, phosphorus and radium had been known long before the discoveries about smoking (Gochfeld 2005).

There is now a very large and important scientific literature originating in the observation of statistical correlations and subsequently strengthened into causal understandings based on the mechanisms at work in the human body following exposures. Policies designed to protect the public have flowed from this scientific knowledge. New risks regularly appear and currently the role of air pollution and toxins from emissions from vehicles are under scrutiny. This debate mirrors events in the 1950s when the dangers from smog in urban environments caused by the burning of coal led to the Clean Air Act and the phasing out of coal as a primary domestic fuel in the UK (Brimblecombe 2006). In public health there is a long history of bringing together correlations and mechanisms to understand the processes which can cause a number of very common diseases and which potentially offer a platform to take action to mitigate the risks and harms, and, as with the Clean Air Acts of the 1950s and action against tobacco, have been highly effective and successful.

9.4 Recurrent Public Health Problems —Non-communicable Disease in the Present

However, notwithstanding the successes with smoking and clean air, deaths from preventable causes which are known and well understood have not gone away. Deaths from non-communicable diseases associated with excess calorie and alcohol consumption and lack of physical activity continue to increase steadily in most countries around the world (Beaglehole et al. 2012). Type 2 diabetes, cardiovascular disease, and certain cancers all have rising prevalence even though the statistical associations between the diseases and the risk factors are well known and the mechanisms operating at the individual level are well understood (though in some diseases better than others).

This is very important as far as appraising evidence of mechanisms is concerned. It is fundamentally important in ethical terms too, because the rising prevalence, while affecting the whole of the population, affects those in poorer and more disadvantaged

circumstances to a far greater extent than the well to do and the privileged (Wilkinson and Marmot 2003). There is a sharp gradient in health inequities that shows a strong correlation between poor health and early death from non-communicable disease and disadvantage. This holds whether disadvantage is measured by income, occupation (or lack of it), housing tenure or educational level or qualifications (Buck and Frosini 2012). The fact is that there are a number of mechanisms which are conceptually and practically distinct from the mechanisms describing the processes of disease causation following exposure to a pathogen or toxin of some kind. Such mechanisms operate at the group and individual level, and concern social interactions and support, access to socio-sanitary infrastructures, psychological factors, etc. It is these mechanisms as well as the biological ones, which have to be explored in the appraisal of public health evidence (Kelly et al. 2014).

9.5 The Individual Level and the Population Level

The first thing to note is that mechanisms operate at different levels. In almost all of the investigations referred to above, the mechanisms that have been subject to most scrutiny are those operating at the level of individual human biology. So, after association were found in the population data, the focus shifted to understanding what was actually going on in the human body when it was exposed to cigarette smoke, ethanol, high levels of sugar, asbestos, particulates in the atmosphere and so on. And this approach of course has shown why these exposures are harmful and how they operate on the human biology. These investigations have been extremely successful and we now have plausible biological mechanistic explanations.

But what about the mechanisms operating at the population level? What about the mechanisms that produce the patterning of health between the rich and the poor, between different parts of countries (Graham and Kelly 2004)? In the United Kingdom, for instance, health on average is much worse in Scotland and the North of England than in the South. How can we explain that? What are the mechanisms which explain the fact that, on average, baby boys born in Guildford will live much longer than baby boys born in Shettleston in Glasgow? What are the mechanisms which link poverty to early death? And what are the relationships between the mechanisms going on biologically and in the wider social and physical environment (Kelly et al. 2014)?

With the stunning progress in understanding the biochemistry of disease since the nineteenth century, the tendency has been to focus on mechanisms operating at the biological individual level. As noted above this is usually relatively straightforward, as the biological processes have been well understood in broad terms for decades and the detail is constantly developing as the science progresses. But what are the social and behavioural mechanisms involved? The behavioral mechanisms are also reasonably well described in the psychological literature (see Table 9.1 for some examples). Models and theories explaining why, on average, humans are likely to do this or that, are plentiful (Conner and Norman 2005). However, why when following

Table 9.1 Behavioural mechanisms

Hazard	Behavioural mechanisms	Disease/morbidity
Exposure to cigarette smoking	Teenagers imitating peers or smoking parents	Lung cancer, cardiovascular disease
Exposure to ethanol, binge drinking	Socializing	Liver disease, certain cancers
Exposure to HPV	Unprotected sex, socializing	Cervical cancer
Exposure to mosquitos	Sanitation, clothing	Zika
Workplace posture	Incorrect posture while sitting or working with a computer, poor lifting practices	Lower back pain
Work overload	Organizational structures, management practices	Anxiety, depression

the same intervention based on the same information about the dangers of smoking, one individual does "this" (say, decides and successfully quits smoking) and one does "that" (doesn't even think about quitting smoking) is less well understood in a mechanistic sense (Marteau et al. 2015).

However, where the biggest gaps in mechanistic understandings exist, is at the social or population level. The associations between poverty and poor health have been known since at least the middle of the nineteenth century and for probably much longer than that in a non-statistical sense. But how it works mechanistically is much less well defined. From an evidence appraiser's point of view there is no easy solution to these problems and neither will there be till primary studies examining the mechanisms have been conducted. But it is important nevertheless to ask the questions. And to ask the questions in a way that acknowledges that we do indeed know with a very high degree of certainty that there is a relationship between wealth, education and employment and health, but we do not know with sufficient clarity what the mechanisms are and in such a way as to target interventions and policies in a directed way to be maximally effective (Kriznik et al. 2018).

There have been many attempts around the world to tackle inequalities in health and while overall the health of populations has improved decade on decade, the relative inequities remain a stubborn fact of life (WHO 2008). Although the lack of political will to do something about it has been a major barrier everywhere, one of the other important reasons for failure has been an absence of mechanistic studies at the population level studies and therefore of the ability to know what to do based on mechanistic understandings of the causal pathways involved.

9.6 The Biological Level and the Social Level

In recent years, the relationship between the individual biological level and the social level has come under scrutiny as a consequence of developments in biology itself, particularly developments in developmental programming, epigenetics and metabolomics. While each of these topics is different, what they have in common is that they show how the human phenotype is the product as much of its environment physically and socially as it is of its genetic inheritance (Kelly and Kelly 2018). Human (and animal) biology is much more plastic in the face of environmental exposures than had been previously thought. DNA doesn't change, but the way that it is expressed does. The metabolic structure of our bodies reveals a timeline of the various exposures we have been subjected to across the life course. Factors affecting the health of our grandmother when she was pregnant with our own mother may have a fundamental effect on our own health in adulthood. The mechanisms here are now quite well developed (Hanson and Gluckman 2011; Ozanne and Constância 2007) and they show that our health is not just a metabolic response to toxins; it is about a complex social and biological interaction—a relational process or mechanism. These mechanisms are critically mediated by the social worlds that people inhabit.

This science is still developing at a rapid rate and along with it, the understanding of the human genome and the therefore of individual biological differences between humans. It is highly likely that new and better mechanistic models and understandings will emerge including ones incorporating the social factors. The implications for the evidence appraiser at this stage are that the question should be asked—are mechanisms relating to the relationship between biological and social factors being described, used, and articulated? A further important epistemological consideration is the degree to which the approach taken by the researcher is a genuinely a relational one—in other words, one that sees the process as a dynamic and interactive one rather than a deterministic one. This is important because if the new understandings of the plasticity of biology are to be useful in public health, the models need to move away from a reductionist approach and should instead be about elucidating the interactive nature of the process. Again this is a question to be asked by the evidence appraiser: what is the nature of the interaction?

9.7 Mechanisms of Disease and Mechanisms of Prevention

There is another question to be asked about the evidence of mechanisms in public health matters and that is about the difference between the causes of disease and the causes of prevention (Kelly and Russo 2018). So far in this chapter we have focused on the important difference between the causes of disease in individuals and the mechanisms involved and the causes of the patterning of disease at population

Table 9.2 Public health mechanisms for tackling obesity

Generic, population level	Targeted
Food advertising, e.g., max amount per day, recommended amount per day, amount of lipids or carbohydrates contained in food portions	MEND programmes in the UK http://www.mendfoundation.org/, e.g., targeted training for school children about diet and healthy lifestyles, targeted training for parents about psychological risks related to obesity

level and the mechanisms involved in this patterning. We have also discussed the mechanisms involved in the relationships between the two.

But there is another very important distinction to draw out which is especially important in public health. This is the difference between mechanisms causing the disease (either in individuals or in populations) and the mechanisms involved in preventing disease (e.g., Table 9.2). The question simply is this. Does knowing the cause of a disease (an exposure to something which is risky) and knowing that by reducing exposure that disease will be prevented, tell you how to reduce exposure? The short answer is that it doesn't, though many public health policies proceed as if it did. The biology of the aetiology of lung cancer, of liver disease, of type two diabetes and the metabolic syndrome tell you nothing about the mechanisms involved in helping people to stop smoking, to consume less alcohol, to eat fewer calories or take more exercise. Knowledge of the cause tells us what people should do, but it doesn't explain how to do it. The mechanisms involved in smoking and giving up smoking, the mechanisms involved in the practices of eating and drinking (and for that matter, sexual conduct, bad driving, or going jogging) belong to a quite different realm of evidence than microbiology. The relevant evidence is social and psychological. The mechanisms involved are social and psychological and there is a considerable amount of evidence, some of which has been around for a long time, describing both associations and mechanisms—see Becker et al. (1977) and Kelly and Russo (2018). For the most part, however, public health policy (with the very significant and successful exception of smoking) pays scant attention to the social and psychological evidence, mechanistic or otherwise. We suggest that the evidence appraiser begins by asking the question: what evidence is available about the aetiology of the disease? And what evidence about effective preventive measures? The distinction between aetiology and prevention should then guide the appraisal of correlations and of mechanisms. Specifically, are only mechanisms at biological level invoked, or also social mechanisms?

Finally, for both mechanism of disease and mechanism of prevention, the evidence sources will be heterogeneous. The disciplines of psychology, sociology, economics, anthropology, organisational behaviour, political science, history, and the public health sciences all have, and have had, things to say on these matters. Unfortunately, it is not the case that we can simply cheerfully agree that the evidence for these things is heterogeneous so we should just pull it all together, synthesise it and out will come a nice clear set of mechanisms. The reason for this is that each of these disciplines,

and the many sub-disciplines within each of them, operate with a variety of epistemological, methodological and ontological assumptions about the nature of human life and its place in the world. Sometimes these veer toward highly individualistic accounts sometimes to more socially oriented accounts. So the task is not to try to adjudicate, but to acknowledge the differences, to articulate them (even if the researchers don't themselves do that), and to consider the degree to which the different positions really matter in terms of the substantive problem (Kelly 2017). Intriguingly, all these disciplines are dealing with the same basic concern—humans in the physical and social world and what is going on in their heads as they go about their business. They each construct ways of seeing and describing the same phenomena differently and in ways that sometimes defy any kind of commensurability. However as long as the appraiser keeps in mind that the basic thing under consideration is the same, and there are just lots of different ways of looking at the phenomena, then the task is not an impossible one. But as ever the first step is to ask the appropriate question, to describe what is there in terms of evidence and to determine to what extent this allows us to understand the mechanisms with clarity.

Here are some simple questions that one can ask in order to structure the search for relevant mechanistic studies, in the context of public health interventions:

Checklist of questions:

1. What disease is the intervention targeting? Infectious or non-communicable?
2. What biological mechanisms are known?
3. What socio-economic or psychological mechanisms are known scientifically?
4. How can behavioural mechanisms reduce exposure?
5. Why might public health interventions targeting the pathogens fail?
6. What is the public perception of the disease in terms of risk, seriousness and personal vulnerability?
7. What mechanisms come into play as a population or different segments in the population react to an intervention?
8. Are there sub-groups within the population that should be specifically targeted? How can they be reached and what specific mechanisms might come into play?

Users interested in carrying out structured searches for relevant mechanistic studies should refer to the Public Health and Social Care tool in Sect. 4.7.

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