Agent, host and environmental factors offer a powerful approach to elucidating the causes of an outbreak of infectious disease after it occurs. They are less helpful in predicting when an outbreak will occur and do not explain why the conditions for the outbreak arose in the first place. As public health turns its attention toward non-infectious health problems such as obesity or adolescent suicide, other limitations of this traditional approach become clear. The agents involved in non-infectious diseases are usually non-specific and there may be no necessary causal factors; host susceptibility cannot be measured in terms of an immunological test, and the environment takes the form of complex, interacting layers of influence. In such situations, causal explanations in terms of agent, host and environment show significant limitations. This discussion reviews alternative approaches to thinking about public health problems and discusses the connections among causes, explanations and understanding.

Epidemiologists who study health issues such as obesity or suicide refer to risk factors for the conditions, in tacit admission that there are no specific causal agents. Population health researchers speak in terms of health determinants, which include a broad swath of factors from genetics to social inequities. In each case, the precise causal status of such factors is frequently left unclear; the published lists of odds ratios provide little in the way of an overall explanation. Interventions should be based on coherent explanations, and the lack of progress in resolving these problems suggests that our explanations are lacking.

This is not new. Public health practitioners faced similar problems in the early 19th century, when an extended debate pitted the miasma explanation for diseases such as cholera (attributing them to general environmental factors) against the contagionist explanation which argued for specific factors. The debate was not merely scientific; conservatives favoured the contagionist explanation, which would conveniently divert attention away from the need for social reform. Radicals (Virchow in Germany, Villermé in France and Farr in England) saw poverty and general social conditions as the underlying culprit. But, should they condemn industrial conditions entirely, tear down slums and press for reforms that would slow economic growth, or should they, as Chadwick did, try to improve health by sanitary engineering while not shaking the foundations of economic wealth that coincidentally drove the patterns of disease? This debate was played out in venues such as the first International Sanitary Conference (Paris, 1851). Neither side could demonstrate precise mechanisms of causation, but a crucial advantage of the contagion theory was that it led to practical interventions. Removing the handle from the Broad Street pump quickly arrested the outbreak of cholera, just as Lind's supply of oranges and lemons to sailors in 1750 had prevented scurvy. Those who blamed socioeconomic circumstances could offer no simple fix, diminishing their credibility. When the germ theory later filled in the missing link in the contagion theory, this did not disprove the relevance of poverty, but it gave conservatives a winning argument: anyone who argued for general improvements rather than proven interventions such as water treatment, sewers or prohibitions on spitting was guilty of cynicism and of damaging economic livelihood. Hence, specific causal factors came to form the central approach of aetiological thinking in public health. Then, as infectious disease gave way to non-infectious conditions, it was slightly adjusted to speak, instead, of risk factors.

Risk factors as an explanatory framework

Risk factors are useful in explaining disease at the individual case level; they may have less value at a population level.
Most of the critiques of the risk factor concept are epistemological in nature: it is often not clear exactly what a risk factor contributes to understanding and explanation. Risk factors are identified via statistical analysis and so may mix risk markers with true causes. They lie intermediate between underlying determinants and the biological process of disease, and are often presented in unstructured lists of disconnected entities. Risk factors do not distinguish the ‘How?’ from the ‘Why?’, as Pearce noted: ‘Epidemiology has become a set of generic methods for measuring associations of exposure and disease, rather than functioning as part of a multidisciplinary approach to understanding the causation of disease in populations.’ Risk factors provide an incomplete picture of the overall process: looking downstream, the connection to pathology is often represented by ‘black boxes’. Looking upstream, the links to underlying determinants also seem vague. Regression analyses tend to focus on the unique contribution of each factor rather than on the overall aetiological processes involved; adjustment for upstream variables diverts attention away from underlying determinants, narrowing our perspective on the overall process. Risk factors are the most successful in identifying the mechanisms: the ‘How?’ questions; they do not address ‘Why?’ questions—why did this situation arise? Nor are they suited to explaining competing causes (why did the person not get some other condition?). To achieve these goals, we need to broaden our explanatory net to gain a more complete picture.

Understanding and explanation

Understanding and explanation lie in a cyclical and symbiotic relationship. An explanation involves linking a fact, such as rising suicide rates, to a concept or idea, commonly embedded in a theory; the goal is to link the fact to prior facts and establish causal connections among them. I can observe and record the rising suicide rates, but understanding comes when a cognitive process connects them to concepts, such as Durkheim’s anomie or fatalism, that are fitted into a broader theory. This may be only one of many potential explanations, which may lie in competition and represent different paradigms: attributing suicide to a lack of social norms, or to mental instability, for example. Explanations lie in layers, radiating outwards with each explaining the preceding; the more layers, the broader or deeper the understanding.

Understanding is a personal and internal representation of reality, using facts, concepts and theory as its inputs. It gives the capacity to explain, communicating understanding to another person. Explanation is the external transmission of understanding, using concepts linked into theory as the vehicle for communication. Every teacher knows the interaction: there are many ways to understand and to explain a given idea, and understanding is deepened by having to explain it to others.

Most explanations involve causal analysis; the exceptions are trivial. For example, one might explain a rising suicide rate in terms of errors in data collection; no causal influence is involved. Other ‘explanations’ may refer to chance occurrence, but this should probably not be seen as an explanation, so much as a failure to find one. Finally, some may ‘explain’ a phenomenon in terms of an act of God. This, also, is often dismissed as an explanation, as it does not invoke a mechanism of action.

Determinants and Explanation in Public Health. The transition to explain why something occurred often invokes the notion of determinants. And yet ‘determinants’ is also a term in need of clarification; it is not entirely satisfactory in that it implies a stronger, deterministic, causal force than generally applies. Perhaps ‘environmental forces’ would be more appropriate, including climate, place and politics. But the term ‘determinants’ is firmly established and refers to a wide array of factors that give rise to risk factors: the causes of the causes. The contrast between determinant and cause was illustrated by philosopher Peter Lipton’s metaphor of why a fox was killed: the explanation is not that a particular hunter shot it, because another would have come along later. The explanation lies upstream in terms of determinants such as liberal hunting laws that created the context for any of a range of hunters to kill the fox.

Public health explanations may focus at the individual, or the population, or even the global level. Global forces may influence population determinants that affect local environments and, ultimately, individual risk factors. This reflects Geoffrey Rose’s distinction between causes of cases and of patterns of incidence. Herein lies a potentially useful terminological distinction: cases have causes, while determinants drive incidence rates in a population. Determinants predict how many will fall sick, but not who. The latter is the role of risk factors which are driven by determinants, but also incorporate modifying influences of personal characteristics and behaviour. Pursuing Rose’s distinction one step further, academic disciplines cleave along levels of investigation and hence explanation: microbiologists explain at the cellular level, while sociologists focus on processes among groups of people. Explaining public health patterns involves many levels; pieces of the picture are supplied by individual disciplines, but explanations within any level refer to mechanisms and ‘How?’ rather than ‘Why?’ questions. In discussing suicide, for example, sociology can discuss culture and acculturation, describing support and obligations. But to understand how these have emerged requires broader reference to
history and perhaps ecology. From this broader perspective, much of the discussion over culture represents classification rather than explanation, a distinction that is often missed. To explain that a person behaved in a certain way because he is from a particular culture omits crucial steps. It is equivalent to observing that the wood grain of my desk looks a particular way because it is made of oak; this is a classification rather than an explanation. Explanation begins from classification, but in addition requires a sequence of causal statements that record influences from different levels of investigation. This is asymmetrical, however. Explanations for variance within a given unit of investigation (e.g. for a person over time) are drawn from lower levels of investigation (e.g. endocrine changes); variance between people may sought from lower or higher levels of investigation (e.g. genetics or cultural differences). Causes of cases are typically drawn from lower levels of investigation, while causes of patterns of incidence typically invoke higher-level phenomena. The historical contagion explanation invoked variables at a lower level but also presented these within a social context; the miasma approach focused only on higher-order variables, so held less explanatory power. Assembling adequate explanations in public health will involve both perspectives.

**Causal models in public health explanations**

If cause is central to the explanation and if we seek explanations that cross levels of investigation, what causal model forms an adequate basis for understanding and explanation in public health? Sidestepping an endless debate over defining cause, I take cause in a counterfactual sense, as a variable without which a given case would not have arisen. For convenience, I also admit the idea of a probabilistic counterfactual: ‘a factor without which the probability of occurrence would have been diminished.’

Metaphors offer a valuable way to simplify and represent complex reality, focusing attention on key features and often drawing attention to limitations in the underlying thinking. The traditional metaphor of a causal chain highlights the multi-stage nature of causal processes, but it is one-dimensional, ignoring contributory causes or protective factors. Chains are in tension, which may not be entirely apposite; causal pressure might work better. The web metaphor improved on the chain by illustrating the interactions between protective and noxious factors. The web also captures the image of concentric layers of influence. However, a web is static, so does not capture the dynamic causal interactions. Nor is it clear whether the web image implies that every factor is equally influential in every case and, finally, webs are extremely robust, suggesting that preventing disease may be a daunting task.

Many, non-specific factors are associated with chronic disease, suggesting that not all need be involved in the development of every case; there may, in effect, be multiple, parallel causal chains. Philosopher JL Mackie proposed the example of a house fire, attributed by the firemen to an electrical short-circuit. He noted that the short circuit was not a sufficient explanation, the fire also required combustible material close to the location of the short, and the absence of effective fire sprinklers. Explaining these circumstances would invoke upstream causes, such as building codes. This discussion considers the cause of a particular fire, viewed retrospectively; looking prospectively, house fires in general can evidently be caused by many different chains of events, each offering a sufficient explanation, but none of which is necessary. This led Mackie to define a cause as an ‘Insufficient, but Necessary component of a set of factors or events, which are Unnecessary but jointly Sufficient’, abbreviated as ‘INUS’. Applied to explain a particular suicide, for example, the ‘I’ suggests that social conditions such as job pressures formed a Necessary ingredient, but did not give the whole picture. The ‘U’ captures the idea that there are always other ways that the suicide could occur, so (as with the web metaphor) removing any one element might not have prevented it; the ‘S’ argues that the set of factors offer an adequate explanation for the suicide.

Rothman incorporated the INUS idea when he portrayed causes as a series of segmented circles, like the slices of a pie shown in Fig. 1. Each pie portrays the idea that explaining a case requires multiple components; the set of pies suggests that explaining disease patterns will require considering many alternative causal paths. Applied to public health, Rothman’s approach is plausible, but focuses on accounting for individual cases rather than for patterns of cases or incidence. The pies also seem static and do not portray interactions among the pie pieces; they also omit the levels of causal influence that were implied in the web metaphor. Bhopal extended the web metaphor into concentric

![Fig. 1 Rothman’s pie diagrams illustrating alternative causal routes. Each slice represents an insufficient cause; in aggregate, these form a sufficient, but not necessary, set of factors to produce the condition.](image-url)
circles with a spiral component. This incorporates the layers of the web, running from broad determinants on the outer orbit through intermediate layers to risk factors and immediate causes towards the centre; there can also be a dynamic indication that the levels influence each other, as shown in Fig. 2. Like Rothman’s pies, the circles can be segmented to suggest alternative pathways at the same causal level. Compared to the pie diagram, the combination of layers of causal influence with alternative pathways of mutually interacting factors helps us to move from cause to a fuller explanation.

The idea of interaction was long ago represented by Waddington’s notion of epigenetic landscapes, a precursor of the complexity perspective. The classic diagram shows a ball at the top of a sloping valley whose uneven terrain would enable the ball to randomly take several possible pathways, each pathway dividing again. The metaphor is that features of the landscape will influence which route the ball takes, but these features change over time, perhaps from external forces such as erosion and perhaps also with the impact of the balls rolling down, just as lifestyle patterns produce repetitive insults. Waddington also drew subterranean forces beneath the surface that could also modify its shape, thereby altering the path of the ball. Somewhat mixing his metaphor, he held that these could represent genes, so the landscape is affected by nature and by nurture.

The limitations of the ‘black box’ metaphor may be overcome by referring to probabilistic neural networks. These illustrate the types of connection that may exist within the black box (see Fig. 3). Inputs are processed through multiple, hidden nodes which have multiple connections among them. But the links between the nodes are variable and can change over time; this reflects the idea that it is the interactions between people (and not merely the people) that are predictive. The effect of each factor can change according to the status of others in the system. Networks evolve characteristic pathways of connections, and these constitute our identity. The prediction of the outcome derives mainly from the pattern of interconnections between nodes, not from the complexity of each. This seems inherently relevant to thinking about a condition such as suicide.

**Moving from analysis towards explanation**

Developing more adequate explanations in public health will begin from an overall explanatory structure that might be represented graphically by a blend of as Bhopal’s concentric circles and the neural networks. Each causal layer contains separate yet equivalent pathways, including both harmful and protective mechanisms. The neural network diagram may be inserted between each of the layers of influence, to suggest how influence transfers from one level to another. This could indicate the processes that might occur at the interface between, say, economic conditions and housing environment, and the resulting family tensions that may lead to adolescent suicide. Moving beyond the black box, the intent is to draw the connections between existing disciplinary islands of understanding. The concentric layers of the overall structure...
would tackle the ‘Why?’ questions by combining several approaches. Teleological and reason-giving explanations represent a first level that is useful for understanding actions and behaviours. This needs to be placed within contextual explanations: social, economic, geographical environments, which themselves have to be understood in the context of historical explanations.

**Criteria for judging an explanation**

How do we conclude that we actually understand, or can explain something? As with Popper’s demonstration that the only proof of a hypothesis lies in failure to disprove it, we cannot prove understanding. An explanation is good only until we find a superior one. The minimal criteria are that explanations should span several layers of causal processes. They should incorporate a plausible theory, or theories, that trace the connections between the levels. There should be an analysis both of the dynamics of the process (‘why did this occur?’); and of its functioning (‘how do the processes operate?’). Desirable features include parsimony; presentation in the form of a mathematical model to permit empirical testing, and the ability to cover individual cases as well as patterns of cases.

**References**