Epidemiological Reasoning

Comments on ‘Popper’s Philosophy for Epidemiologists’ by Carol Buck

COMMENT ONE

Lest thou make a covenant with the inhabitants of the land, and they go a whoring after their gods, Exodus 34:15

And we are right, I think you’ll say,
To argue in this kind of way,
And I am right
And you are right
And all is right—too-looral-lay!

W. S. Gilbert: The Mikado, Act I (1885)

The two quotations summarize my reactions to Dr. Carol Buck’s stimulating paper on ‘Popper’s philosophy for epidemiologists’. There would be little disagreement as to the desirability of enriching the scope and discipline of epidemiology by applying the ideas of several of the philosophers of science. On the other hand, might there not be a danger of rigidity, of too strict an application of principles derived from the exact sciences to the detriment of the broad development of our discipline? In the present case, why pick on Popper? To play down inductive reasoning and to demote the value of forming hypotheses based on observation could be detrimental to the art, if not to the science as well. If Carol Buck’s intention is to make a plea for stricter scientific methods in epidemiological research (and who would disagree?) are there not sufficient lessons to be learned from the masters of epidemiology themselves?

Karl Popper (1) is not easy to understand although it is generally accepted that ‘the first strongly reasoned and fully argued exposition of a hypothetico-deductive system is unquestionably Karl Popper’s’ (2). Dr. Buck agrees with this but, as she also points out, the importance of attempting tests of disproof of hypotheses was already stressed by several of his forebears. Susser (3) paraphrases Sir Francis Bacon, whose system of inference in science was published in 1559, as ‘in other words, the most cogent test of a hypothesis available to scientists is to attempt disproof’, citing Popper and Platt (4) in support. Whewell (quoted by Medawar (2)) wrote, as his ninth aphorism in 1840:

‘The truth of tentative hypotheses must be tested by their application to facts. The discoverer must be ready, carefully to try his hypotheses in this manner . . . and to reject them if they will not bear the test.’

Other forebears, including Lord Acton, Karl Pearson, de Bono and Sherlock Holmes are quoted in Dr. Buck’s paper.

Perhaps the classics of epidemiology are the two papers of John Snow, reprinted in 1936 (5), where, in his studies on cholera, the author formed a hypothesis, repeatedly tested it and systematically searched for exceptions such as cases who resided away from Broad Street. Thus, he recorded cases in children and adults who were exposed during their school or working hours and noted the absence of cases among brewery workers. In this search for exceptions, Snow anticipated Popper by 100 years. Snow himself was preceded by Sir George Baker, whose 1767 classic on the endemic abdominal colic of Devonshire deserves to be better known (6). At the time, the current hypothesis was that this condition was due to excessive consumption of tartaric acid in apple cider. Supporting evidence was adduced from the existence of a similar condition in the West Indies ascribed to excessive intake of lemon juice. Further ‘proof’ was the fact that ‘precisely the same disease’ had been described in 1617 in Poitiers in France among heavy drinkers of the local acid wine.

But, stated Baker, unless the ‘colic of Poitou’ (Poitiers) was also produced by Rhine and Moselle wines, the analogy does not hold. Was this not seek-
ing a refutation of the hypothesis? Moreover Turks who drank large quantities of acid sherbert and jockeys who drank much vinegar to lose weight, did not suffer from the colic. The cause could not be consumption of acids.

Nor could it be the cider itself, as inhabitants of other counties of England also drank large quantities of cider but did not have the colic, except on occasion. The clue came from the serendipitous discovery of the fact that in Wurtemberg, adulteration of wine with litharge led to widespread colic while drinking of unadulterated, very acid, wine did not. The symptoms of colic were similar to those suffered by lead workers and an outbreak had occurred in Worcester in those who drank cider which had been stored temporarily in a lead cistern. The cause could not be the cider itself, as inhabitants of other counties of England also drank large quantities of cider but did not have the colic, except on occasion. The clue came from the serendipitous discovery of the fact that in Wurtemberg, adulteration of wine with litharge led to widespread colic while drinking of unadulterated, very acid, wine did not. The symptoms of colic were similar to those suffered by lead workers and an outbreak had occurred in Worcester in those who drank cider which had been stored temporarily in a lead cistern. The clue came from the serendipitous discovery of the fact that in Wurtemberg, adulteration of wine with litharge led to widespread colic while drinking of unadulterated, very acid, wine did not. The symptoms of colic were similar to those suffered by lead workers and an outbreak had occurred in Worcester in those who drank cider which had been stored temporarily in a lead cistern.

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Finally, Baker proposed the theories that the Devonshire colic was due to lead contamination of the cider and that cider must be prepared differently in Devonshire than in other counties. The former theory he proved by laboratory demonstration of the presence of lead in cider and the latter by examining cider presses and showing that in Devonshire lead was more commonly used to line them or to repair them when cracked.

In Baker’s study therefore we have the basic elements of the scientific method as applied to epidemiology. Formation of hypotheses by observation and inductive reasoning: testing by deduction and experiment.

Two hundred years later, Yerushalmy (7) was pointing out that the first requirement for epidemiological studies seeking causal associations is that all groups or a random sample of the groups be included, not only those which support the hypothesis. He recalled that the lack of correlation between dietary fat intake and coronary heart disease in certain African tribes and Eskimos, which did not support current hypotheses, was ignored. Yerushalmy’s observations on maternal smoking habits and low birthweight, quoted by Carol Buck, have never been satisfactorily explained, although many attempts have been made to dismiss them.

One explanation for our unwillingness to face up to aberrant results is suggested by another philosopher of science, Gerald Holton (8). As Merton puts it in a discussion of Holton’s concept:

‘The themata of scientific knowledge are tacit cognitive imageries and preferences for or commitments to certain kinds of concepts, certain kinds of methods, certain kinds of evidence and certain forms of solutions to deep questions and engaging puzzles. Implicit . . . is the notion that they are unevenly accessible to observation’ (9).

In Holton’s definition, based, according to Merton, on inductive reasoning, there are underlying elements in the concepts, methods and hypotheses of scientific work that function as themes that motivate or constrain the scientist and consolidate or the cognitive judgements appearing in the community of scientists.

But all this has been well stated, in a form relevant to epidemiology, by Susser in a recent book (3); a book that should be required reading for all students and practitioners of the discipline.

At this point, I should state that I benefited greatly from ‘Popper’s philosophy for epidemiologists’ and support Dr. Carol Buck’s plea for focusing on the exclusion of hypotheses. But this should only be part of our armamentarium: we must retain inductive methods of reasoning along with deductive methods. Consider the need for imagination and the search for generalization as expressed by Einstein:

‘There is no logical way to the discovery of the elemental laws. There is only the way of intuition, which is helped by a feeling for the order lying behind the appearance’ (10).

Finally, while we must never forget the need for the strict application of scientific method to epidemiology, we may recall the advice given by Merton to his students.

‘“In general, it’s a good thing to know what you are doing and why you are doing it.” The qualifier “in general” is designed, of course, to warn against the danger of that premature fault finding which stifles ideas that need to be played with before being subjected to systematic and rigorous examination. There is a place, as Max Delbruck and Dickinson Richards have severally reminded us, for “the principle of limited sloppiness”.

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REFERENCES


COMMENT TWO

Whether or not one can accept Dr. Buck's implied thesis that Popper represents the last word in scientific philosophy, her paper is a crucially important contribution to the epidemiological literature. The most important reason why this is so, is that she rejects wholeheartedly the restrictive view of epidemiology that it is primarily concerned with data gathering. Too many epidemiologists have accepted the view that epidemiology is not a domain of enquiry but simply an assembly of methods capable of application to a wide range of medical problems. This view is not only depressing to the would-be epidemiologist but also naive in its implied assumption that the methods of epidemiology are specifically characteristic of that science. They are, of course, the relevant methods of almost any science that is concerned with the study of aspects of human populations.

I like to define epidemiology as the branch of medical science that is concerned to study the health of human communities just as community medicine (or public health in the old nomenclature) is the branch of medical practice that is concerned to promote the health of human communities. Since medicine is a profession and not a trade it is incumbent upon its practitioners to contribute to its science, and upon its scientists to accept some responsibility for its practice. Thus, epidemiology, as the science upon which community medicine practice is based, must seek to provide the body of understanding necessary before decisions in the field of community medicine practice can be other than arbitrary. The epidemiologist must therefore commit himself to explanations that help the practitioners rather than to the illusory pursuit of 'truth' which, if he is honest, results in a perpetual unhelpful agnosticism.

The explanatory function of epidemiology rests, like that of any other science, not upon its body of data but on the set of hypotheses that have survived serious attempts to refute them by data. The explanatory power of such a set of hypotheses depends not only on the rigour of the data testing that they have survived but even more importantly on their mutual coherence. If the set of surviving hypotheses exhibit a high degree of interrelatedness they may be thought of as a body of theory. All well established sciences have such a body of theory out of which new hypotheses arise which lead to purposeful data gathering in the attempt to refute them. If the body of theory is coherent and consistent it may provide a powerful guide to decision and action for practitioners of related fields of professional practice. Thus, for example, we may diagnose and prognosis, in clinical medicine, with a confidence based on a body of general understanding of disease processes. In the nineteenth century practitioners of public health (who were usually also practising epidemiologists) could base their recommendations for general environmental hygiene on a general theory of the communicability of diseases without the need to repeat field studies in each locality and on each occasion that a general measure was contemplated. We lack
such a body of general theory relating to the important disease problems of today.

Karl Popper’s contribution to science—and now specifically to epidemiology by the good offices of Dr. Buck—has been to stress that data and the methodology of their collection represent the ordeal through which hypotheses must pass before being admitted to the status of theory, and that it is theory and not data that is the functional element in science. Epidemiology, like any other science, therefore depends not on its data and not on the elaboration of its methodology but on the thought that goes into erecting the hypotheses whose surviving fraction will eventually constitute the central understanding which alone can justify a science.

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