The Philosophy of Epidemiology

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CONTENTS

WELCOME

Welcome to what, as far as we know, is the first conference devoted specifically to the Philosophy of Epidemiology.

Epidemiology is attracting increasing philosophical attention, even though most philosophers know very little about epidemiology, and philosophy of epidemiology is not yet a part of regular philosophy of science curricula. Epidemiology rewards philosophical study for several reasons, but particularly because it is such a poor fit for standard philosophical pictures of science. These pictures tend to place emphasis on explanatory theories and experiment as central features of science, yet neither is central to epidemiology. This fact prompts a recasting of the entire realism debate in philosophy of science, and means that many well-known positions on the nature of science do not apply to epidemiology.

The purpose of this conference is to offer an opportunity to philosophers of science to engage with epidemiology, and to encourage epidemiologists, statisticians, lawyers, social scientists, and others with relevant interests to explore the philosophical aspects of the discipline further.

Epidemiology attracts philosophical attention because epidemiologists deal explicitly with conceptual questions to a greater extent than scientists in many other disciplines. Working epidemiologists devote time and energy to publishing papers on the nature of causation, methods of causal inference, and the nature and role of statistical significance testing, for example. Epidemiology also raises important questions about the relation between general (population) and singular (individual) causal claims, nowhere more clearly than in the context of litigation. Epidemiology is often central to litigation because it deals with phenomena whose underlying mechanisms are not well understood. Thus there are circumstances where epidemiology provides the only evidence available to prove or disprove a causal link between wrong and harm. However, epidemiologists deal in generalities, and litigants are individuals (or classes thereof). It is both a philosophical and a legal question how evidence for a general causal claim relates to the attempt to prove singular causal claims.

These are just a few of the topics awaiting the attention of scholars from various disciplines. We hope you find this short meeting stimulating.

Alex Broadbent, Jeremy Howick, Hennie Lotter

PROGRAMME

MONDAY 12 DECEMBER

8.45	**Welcome**
9	Alfredo Morabia (Professor of Epidemiology, Columbia and CUNY): 'David Hume and Bradford Hill: similar but different.'
10.15	**BREAK**
10.30	Gerald Oppenheimer. 'Problems of Causal Thinking in Epidemiology: A Case History'
11.15	Hanna van Loo. 'What co-morbidity tells us about diagnoses in psychiatry.'
12	David Frank. 'Modeling Chagas disease risk in Texas: Idealization and multiple models for use.'
12.45	**LUNCH**
2	John Worrall (Professor of Philosophy of Science, London School of Economics): 'Taking off the mask: a clear-eyed view of the methodological virtues of "blinding" in clinical trials.'
3.15	**BREAK**
3.45	Andrew Schroeder. 'Measuring Health and the Problem of Changing Populations.
4.30	Sridhar Venkatapuram. 'Reconciling group level analysis in epidemiology with justice claims of individuals.'
5.15	Jasper Littman. 'Infectious Disease Modelling and its Ethical Implications for Policy Making.'
6	End Day 1
6.45	**Drinks & dinner**

TUESDAY 13 DECEMBER

9	Richard Wright (Distinguished Professor of Law, Illinois Institute of Technology Chicago-Kent College of Law): 'Epidemiology and Epistemology: The Probable versus the Actual'
10.15	**BREAK**
10.30	Claire McIvor. 'Using epidemiological evidence to resolve questions of probabilistic causation in personal injury litigation.'
11.15	Sandy Steel. 'Probabilistic Liability for Causation.'
12.00	'Ted Schrecker. 'Knowledge, Power, and Standards of Proof in Epidemiology and Public Policy.
12.45	**LUNCH**
1.45	'Jeremy Howick. 'Why mechanisms rarely bridge the gap between randomized trials and "target" populations: a reply to Cartwright.
2.30	Alex Broadbent. 'Explanation and prediction in epidemiology.'
3.15	**BREAK**
3.30	Christopher Hitchcock (Professor of Philosophy, California Institute of Technology): 'Probabilistic Measures of Causal Strength'

4.45	**CLOSE**

ABSTRACTS

CAUSATION AND EXPLANATION IN EPIDEMIOLOGY

Alex Broadbent

Epidemiologists and philosophers have devoted considerable attention to the task of obtaining causal knowledge, that is, to causal inference. They have devoted much less attention to the question of what we do with causal knowledge once we have it. Two uses are particularly salient: explanation and prediction. In this paper, I attempt to establish two claims. First, explanation acts as a guide to causal inference in epidemiology: a good answer to the general question, "What makes a good causal inference?" will mention explanation. In this I rely on Austin Bradford Hill. Second, I seek to identify a similarly general answer to the question, "What makes a good prediction?" Incredibly, philosophers have not yet attempted to answer this question; and epidemiological text books are also quiet on the topic. I introduce a concept of "robustness", which I argue is to be aimed for in the public health context. I propose that a prediction is robust when we can explain why the predicted outcome will occur rather than salient alternatives.

MODELLING CHAGAS DISEASE RISK IN TEXAS: IDEALIZATION AND MULTIPLE MODELS FOR USE

David Frank

This paper uses the example of modeling Chagas disease risk in Texas to explore some interconnections between the philosophical issues of scienti.c idealization (Wimsatt 2007, Weisberg 2007) and the use of science in society (Kitcher 2001, Cartwright 2006). The modeling consisted of constructing species distribution models for the vector Triatoma species, computing an incidence-based relative risk map based on known occurrences of the Chagas-causing parasite Trypanasoma cruzi, and combining these and other risk metrics (Sarkar et al. 2010). In the resulting paper we argued, following Hanford et al. (2007) and others, that the risk of Chagas in Texas - particularly south Texas - is signi.cant enough that Chagas should be declared reportable.

This paper offers some preliminary philosophical reflections on this modeling process. The example of modeling Chagas risk illustrates the role of "multiple-models idealization" in the epidemiology of vector borne diseases. The case study also shows that the permissibility of idealizations in multiple models depends upon the use-context. In this case, one important use of these models was to support the normative claim that Chagas should be declared reportable in Texas. This modest goal permitted significant idealizations, as well as omissions like not modeling reservoir species.

Causation, Risk and Epidemiological Evidence in Toxic Tort Litigation: Law's Coming of Age

Richard Goldberg – CANCELLED DUE TO UNFORESEEN CIRCUMSTANCES

This paper examines the recent cases involving the role of epidemiological evidence in assessing causation in toxic tort cases in the UK. In essence, it seeks to determine the extent to which the courts in the highlighted cases have been efficient and equitable in their interpretation and utilisation of epidemiological evidence from the perspective of both consumers and producers. The first section explores the difference between evidence of causation for purposes of science and for the law, and the difficulties in reconciling the standards of proof in law and science, including the controversial theory that causation can be proved on the balance of probabilities by reference to the doubling of risk of injury. In particular, these matters have come to recent attention in the context of the utilisation and value of epidemiological or statistical evidence alone in determining causation on a balance of probabilities, with discussion in the United Kingdom Supreme Court in Sienkiewicz v Greif. The distinction between association and causation and the difficulty in proving general and specific causation between a product and damage using epidemiological evidence is reviewed in the context of the controversial Scottish case of McTear v Imperial Tobacco Limited which is subject to criticism of the way that the epidemiological evidence was received by the trial judge. The problem of utilising statistics deriving from trends in general populations to prove causation in an individual case is highlighted in McTear, and a possible solution in the form of utilisation of the Bayes' Theorem is discussed.

Why Mechanisms Rarely Bridge the Gap between Randomized Trials and "Target" Populations: A Reply to Cartwright

Jeremy Howick

While there is a debate between mechanist philosophers of science and Evidence-Based Medicine (EBM) proponents over the use of mechanistic evidence as evidence for efficacy there is surprising agreement that knowledge of underlying mechanisms can help us implement the results of controlled studies. Meanwhile philosophers of science including La Caze and Bluhm argue that research into underlying mechanisms is required to implement the results of controlled studies. More recently, Cartwright can be interpreted as having made a coherent philosophical argument supporting the necessity of mechanisms for implementing the results of trials. We will argue that mechanisms as a bridge for applying trial results to other populations is far more problematic than many philosophers have hitherto presumed. We will begin by describing the problem of implementing the results of controlled studies (which is often misleadingly referred to as the 'problem of external validity') and briefly characterizing mechanisms and mechanistic reasoning. Next, we will outline the arguments used to support the view that mechanisms can help us generalize the

results of clinical trials to individuals. Then, we will describe the epistemological and ontological problems with using mechanisms to generalize. In brief, our understanding of underlying mechanisms is often (and likely to remain) insufficient to make any useful predictions, mechanistic knowledge is often produced in tightly controlled laboratory conditions that do not generalize, and more fundamentally the underlying mechanisms themselves may not always produce 'regular' relationships between interventions and outcomes. We conclude that much more empirical and theoretical research is required before bold claims about the usefulness of mechanisms to generalize the results of comparative studies can be accepted.

INFECTIOUS DISEASE MODELLING AND ITS ETHICAL IMPLICATIONS FOR POLICY

Jasper Littman

Infectious Disease Modelling is a crucial part of pandemic preparedness and regularly determines the policy setting and reaction to pandemic outbreaks. This paper will provide an overview and analysis of the ethical challenges that the use of models in pandemic preparedness pose. A model is essentially a mathematical tool that simulates socio-demographic structures to predict how a pathogen will spread. While models are widely used at both national and international levels, their use presents policy makers and epidemiologists with ethical challenges:

(i) Models are calculations of what will happen in a representative society, given a number of assumptions. These assumptions include estimates of transmission rates, lethality of a pathogen or incubation periods. During pandemics, where reliable information is scarce especially in the early stages, realistic estimation of these parameters is difficult.

(ii) Parameters may have to be adjusted over time, if conditions change or a pathogen undergoes mutation. This is problematic, since models will usually impact on the distribution of scarce resources such as vaccines or antivirals during pandemics. Consequently, the use of imprecise of changing parameters as basis for decision-making raises concerns with regard to distributive fairness.

(iii) Since policy decisions will be made on the basis of models, their predictions potentially impact on the life of many people. However, policy makers will usually treat models as 'black boxes' that generate output without fully appreciating their design. If the limitations of models are not clearly understood, this may create unrealistic expectations of what epidemiological models can provide, as made evident during the 2009 H1N1 pandemic, where initial assumptions about lethality and virulence proved too high. The paper will suggest that modelling is an important starting point for risk assessment but cannot serve as moral legitimisation for pandemic policy-making or to establish fair distribution of scarce resources.

What Co-Morbidity Tells Us About Diagnoses in Psychiatry

Hanna van Loo

Co-morbidity – the occurrence of two or more disorders in one individual – is one of the interests of epidemiologists in psychiatry. Regularly, high rates of co-morbidity are reported in psychiatry (Bijl 1998, Jacobi 2004, Kessler 2005). This means that the chances of finding two psychiatric disorders in one individual often exceed the expected probability based on disease occurrence of each separate disorder. Thus, for two disorders as major depressive disorder (MDD) and generalized anxiety disorder (GAD) the chances of finding both in one individual are strikingly high, viz. p(MDD^GAD) > p(MDD)p(GAD).

How should we interpret those high rates of co-morbidity? In particular, do those rates tell us something about the classification of psychiatric disorders? In current debates, different answers are given to this last question. Some see high rates of comorbidity as a result of a shared etiological background, and therefore as an argument in favor of a valid classification (Andrews 2009). Others, however, regard it as an indication that our definitions of psychiatric disorders are too complex (Kendell and Jablensky 2003). However, before a sensible answer can be given to the question what co-morbidity implies for the correctness of psychiatric diagnoses, some conceptual issues should be resolved. In this paper I want to address two conceptual issues obscuring the co-morbidity debate. First, I will discuss the specific properties (e.g. considerable symptom overlap and the absence of certain biological mechanisms) of mental disorders which make them – and the finding of co-morbidity – susceptible for arguments of circularity and arbitrariness. The second reason for obscurity concerns different uses of psychiatric diagnoses. Should the classification system serve primarily an epistemological goal or is its foremost aim guiding treatment decisions? A closer look to those issues will clarify what co-morbidity can tell us about diagnoses in psychiatry, and how we should interpret co-morbidity in epidemiological research more in general.

Using Epidemiological Evidence to Resolve Questions of Probabilistic Causation in Personal Injury Litigation

Claire McIvor

The recent decision of the UK Supreme Court in Sienkiewicz v Greif [2011] UKSC 11 contains a seriously misinformed account both of Epidemiology as a discipline and of its potential relevance to questions of probabilistic causation in tort law. By correcting the misconceptions and misunderstandings set out in the decision, this paper aims to demonstrate the benefits of using Epidemiologists as expert witnesses in certain categories of personal injury litigation, most notably claims for negligent diagnoses and claims in respect of negligent exposures to carcinogenic substances. The main body of the paper will present the House of Lords decision in Gregg v Scott [2005] UKHL 2 as a classic example of the errors made by the UK courts when assessing issues of probabilistic causation. It will identify problems both with the nature and source of the statistical evidence relied upon in that case and also with the manner in which the statistical data was applied to the legal

principles of factual causation. It will then turn to an epidemiological account (produced specifically for the purposes of this paper) of the personalised statistical chances of Mr Gregg avoiding the harmful outcome in the absence of Dr Scott's negligent misdiagnosis and demonstrate how the problems with the Gregg decision could have been easily addressed through the use of a reputable epidemiological expert witness.

Problems of Causal Thinking in Epidemiology: A Case History

Gerald Oppenheimer

Epidemiology has focused on the conceptual issue of causality because its demonstration is problematic. This paper would use the history of cardiovascular disease (CVD) to explore the epidemiological struggle with causation. Along with cigarette smoking and lung cancer, CVD research (like the Framingham study, 1947-present) was central to the development of modern, black box epidemiology. Unlike lung cancer, which quickly became the story of one robust causal variable, not unlike the germ theory, CVD was from the beginning seen as—and was demonstrated to be--a disease produced by multiple risk factors. (The term, "risk factor" was crystallized and popularized by CVD epidemiology.) These factors, unlike cigarette smoking, were of relatively small effect size, and were neither necessary nor sufficient to explain CVD morbidity. Epidemiologists' subsequent attempts to translate statistical association into causation in this instance, as is true of putative environmental and occupational carcinogens for example, proved difficult and contentious. Nevertheless, given the impact of CVD on population morbidity and mortality, the pressure for public health action and clinical response was enormous. This raised a number of questions: Under what circumstances were public health and clinical interventions justified by statistical association alone? How could one justify applying associations and causal arguments based on the study of populations to individuals (e.g. clinical patients)? Should chronic diseases like CVD and lung cancer be conceptualized as problems of individuals or integral to communities, and what were the implications for causal thinking and social/legal policy? Does epidemiology differ from other sciences in that the determination and acceptance of causality is socially negotiated by experts and by popular consensus?

Knowledge, Power, and Standards of Proof in Epidemiology and Public Policy

Ted Schrecker

The choice of a standard of proof (how much evidence is enough) is familiar from the different standards applied in civil and criminal courts in many common law countries. I demonstrate the importance of this issue in epidemiology and public policy with two examples, drawn from more than 20 years of professional experience in policy analysis and academia (see e.g. Schrecker, 2001; Schrecker et al., 2001).

The first involves the demand for epidemiological evidence as a basis for controlling toxic substances such as potential carcinogens in the workplace and the general environment. Uncritical application of a standard of proof that minimizes false positives remains routine in epidemiology, and often in the use of science in the regulatory process, even though: "In its extreme, the approach of limiting false positives requires positive evidence of 'dead bodies' before acting" (Page, 1978). Thus, epidemiological study designs themselves and purportedly science-based policy incorporate similarly powerful normative biases against intervention. The central value judgment about the relative consequences of being wrong in different kinds of ways (Jellinek, 1981; Maienschein, Collins & Strouse, 1996; McGarity, 1979) is seldom explicitly examined, with the exceptions – such as the debate on the US Occupational Safety and Health Administration's cancer policy during the Carter administration – underscoring its importance. European debates about the precautionary principle would appear to constitute another exception, but most discussions of the principle fail to recognize that precaution can take various forms, depending on the outcomes one wishes to avoid.

The second, considerably more recent example (and my primary focus in the paper) involves the sufficiency of evidence to support policies and interventions that address the social determinants of health (Commission on Social Determinants of Health, 2008). This example is more complicated, because multi-stage causal pathways and mechanisms of action are often involved; epidemiology may not clearly connect relevant variables with health outcomes, at least without long periods of study that – as with prospective studies of the relation between environmental agents and diseases with long latency periods - raise the ethical question of how long is too long to wait for 'proof'. A further layer of complexity is introduced by such choices as whether to focus on upstream or proximal influences: for example, should we attribute the mortality crisis in the former Soviet Union to increased alcohol consumption, or to the stresses associated with economic collapse and the disintegration of many forms of social provision? A plausible causal story can be told in each case but here I argue, using as an example the body of evidence related to the physiological effects of stress as they accumulate over the life course, that "micro-epidemiology" (Venkatapuram & Marmot, 2009) focused on individual behaviour without attention to context is scientifically incomplete in at least some situations. In others, the choice represents a "methodological value judgment" (Shrader-Frechette & McCoy, 1993) with respect to which epidemiologists and other scientists, qua scientists, have no special competence. Again, it resembles the choice of a standard of proof in regulating workplace carcinogens.

Nothing is especially new about these observations, although they remain frustratingly foreign to the quotidian work of most epidemiologists and public health policy researchers and practitioners. Apart from insisting on standards of proof as a core question for public health ethics, the major original contribution of this paper is to draw explicit connections among knowledge, economic prerogatives and power. Framing the choice of a standard of proof as a scientific or technical issue can be used as a covert strategy of resisting substantial redistribution of resources to subaltern populations, without being explicit

about this objective. (Conversely, the Commission on Social Determinants of Health was clear on the need for such redistribution, identifying it as one of three overarching recommendations for action.) It is therefore necessary not only to reframe academic debates about 'the evidence' in ways that incorporate explicit attention to standards of proof, but also to conduct comparative case study research on how such issues are dealt with in policy contests relevant to social determinants of health. The economic crisis of 2008 and subsequent austerity measures, which have generally been promoted with scant consideration of long-term consequences for population health, highlight the urgency of that research.

Measuring Health and the Problem of Changing Populations

Andrew Schroeder

It is usually said that summary measures of population health, such as the disability-adjusted life year (DALY), can be calculated from either of two perspectives: an incidence perspective (based on incidence rates and the duration of health problems) or a prevalence perspective (based on prevalence rates). I show that this is true only for a particular class of measurements, those that are linked to time intervals, and that the "two" options actually have a number of forms, which it is important to distinguish. These options are typically all treated as being candidate ways of calculating population health in time interval T. However, I show that the different methods are not alternate ways of measuring the same thing; each method results in the measurement of a very different property or quantity. I show that even if we grant a number of questionable assumptions -- enough to yield the conclusion that non-time interval-linked DALYs do measure population health -- it remains true that none of the time interval-linked calculation methods is capable of measuring population health in T. I show that this is a consequence of the fact that populations change in membership: as people are born/die, they enter/leave populations. There is therefore no clear answer to the question of whose health to measure, in order to measure population health over an interval. I suggest that the best way of dealing with this problem is to abandon the search for a measure of population health in T, and instead to seek an index of population health in T. I show that one of the original calculation methods, usually thought to suffer from certain conceptual defects, may plausibly fill this role.

Probabilistic Liability for Causation

Sandy Steel

In response to the increased availability of statistical evidence in civil litigation, some American legal academics began, in the 1970s, to argue for probabilistic or proportional liability in the law of tort (where evidential difficulty over 'natural' causation arises most frequently) . There are some important differences in the formulations of this doctrine, but the gist of the idea is to allow the claimant to obtain damages in proportion to the

probability that the defendant caused her harm: a 35% probability of causation, 35% of the damages suffered are awarded.

A related, but different idea, often mixed with the first, is that the law ought to protect a person's chance (the probability of a good outcome) of avoiding an injury; if the claimant cannot prove that the defendant has caused her cancer, for example, she may still be able to show that the defendant has deprived her of a quantifiable chance of avoiding the cancer. Damages should be available which reflect that destroyed chance.

Various normative underpinnings were (and have since been) offered for these proposals: (1) that causation is itself inherently probabilistic and so the law should reflect this; (2) that losing a chance of avoiding an injury is something valuable and deserves protection; (3) that probabilistic liability avoids the arbitrariness and starkness of awarding the claimant everything or nothing; (4) that wrongdoers otherwise profit from the uncertainty over causation in such a way that they are less deterred from wrongful conduct.

In this paper, I argue against (1) and (2), not on the grounds that either of those propositions is false, but only that their truth will not justify either of the doctrines. I argue against (3) on the basis that it misunderstands the nature of the standard of proof in civil cases. I suggest that the validity of (4) depends upon the type of epidemiological or, more broadly, statistical evidence available. Finally, I suggest that even if (4) can be made out, the law (and normatively justified law) values truth too highly to allow probabilistic liability.

Reconciling Group Level Analysis in Epidemiology with Justice Claims of Individuals

Sridhar Venkatapuram

Social epidemiology has profoundly affected Anglo-American political philosophy and bioethics over the past decade. Along with undermining a number of assumptions central to the architecture of theorizing about social justice and bioethics, social epidemiology has raised a number of conceptual challenges regarding the rights and duties of different agents, particularly of individuals and groups. This paper sets out the conceptual problem facing liberal political philosophers regarding whether groups have moral status in light of 'population health' phenomena illuminated by social epidemiology. It then explores the classic bio-medical 'black box' epidemiology, social epidemiology's 'chinese boxes' epidemiology, and recent discussions on supervenience/methodological individualism in the social sciences with aim of identifying a plausible path for those committed to moral individualism.

In liberalism, the most dominant tradition, the ultimate moral unit is the individual. Various conceptions of liberal social justice are evaluated according to what aspects of the individual are considered morally relevant, and how they are addressed. Because health is instrumentally important to pursuing life plans and partly constitutes well-being, health is

almost universally recognized as being a morally relevant feature of individuals in theorizing about justice. However, in contrast to the engagement with disciplines such as moral psychology or economics, political philosophers have had surprisingly little engagement with epidemiology, the science of the causation and distribution of impairments and premature mortality ('ill-health'). As political philosophers increasingly engage with epidemiology in order to allocate moral rights and responsibilities of individuals in relation to the causes, distribution, consequences, persistence, and mitigation of ill-health, they face the profound problem of the lack of a clear chain or model of causation.

Compounding this long-standing problem in epidemiology is the recent growth in research findings on the social causes of ill-health and its distribution across social groups. Much of social epidemiology's insights come from analysis at the population or group level. The research has expanded understanding about both the social causes of ill-health as well as about their social distribution. However, social epidemiologists cannot predict which individual in which social group will next experience ill-health. Nor can they retrospectively identify a clear chain of causation from the individual back to the social factors. The research can only assert that in historical data, there is a statistical correlation between certain social phenomenon and the causation and distribution of ill-health in individuals belonging to various social groups.

While social epidemiologists may be satisfied with advocating for interventions that will prevent ill-health in social groups, for individuals to make claims regarding social determinants of ill-health, or for philosophers to link claims regarding social determinants of ill-health to individual moral agents requires filling in the conceptual causation gap between the social and the individual. As a step towards that this paper brings together discussions on causal models in epidemiology and discussions on methodological individualism and supervenience in the social sciences.