EVIDENCE AND CAUSALITY IN THE SCIENCES

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University of Kent |Centre for Reasoning

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ORGANISERS:

PHYLLIS ILLARI & FEDERICA RUSSO

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Abstracts – Plenary speakers

Atocha Aliseda – *Abduction and Evidence in Medical Diagnosis*

This talk concerns the relationship between evidence and hypotheses under the framework of abductive reasoning within the context of medical diagnosis in Neurology.

When a medical doctor is faced with her patient´s symptoms and signs, she has to resort –among many other things-- to her existing knowledge of pathologies, in order to choose the best fit as an *diagnostic syndromic hypothesis* explaining her patient's condition. In this case we say she is performing a straightforward *novelty abduction* in which the supporting evidence is found among those symptoms and signs the patient reports. In the case of Neurology, there are two additional diagnostic kinds complementing the syndromic one, namely *topographic* and *etiologic*. While the former aims at establishing a correlation between a physiological alteration and an anatomical location, the latter relies on evidence drawn from physicians knowledge and from a bibliographical review about the nosological entities in view.

As it turns out, the ways in which evidence is searched for as well as weighted in favour or against a certain hypothesis, is quite different in each of these diagnostic types. In this talk, I will analyze the dynamics of diagnostics through a clinical neurological case, and show how medical practice provides us with an excelent setting to highlight some of the challenges the relationship between evidence and hypotheses faces.

Iain Chalmers – *Trying to do more good than harm*

Why am I interested in research evidence about the effects of health care?

What do I assume about the contribution of research evidence to knowledge?

What kind of research evidence do I think should inform policy in the NHS and choices for my own health care?

Why is research evidence necessary but insufficient for informing choices in health care?

Mathias Frisch – *Physics and the Human Face of Causation*

Many contemporary philosophers of physics (and philosophers of science more generally) follow Bertrand Russell in arguing that there is no room for causal notions in physics. Causation, as James Woodward has put it, has a 'human face', which makes causal notions sit ill with fundamental theories of physics. In this talk I examine a range of anti-causal arguments and show that the portrait that the neo-Russellians paint of causation is the face of scientific representations much more generally. Causal notions, I argue, play no less of an important role in physics than they do in other sciences.

David Lagnado – *Stories & Statistics: what can the Sally Clark case tell us about the psychology of evidential reasoning*?

Sally Clark was convicted of murdering her two children, but was eventually released after a lengthy legal process. This case has had substantial repercussions in the legal domain. In this talk I argue that the case also highlights many issues in the psychology of evidential reasoning. These include: the key role of causal reasoning; issues of witness reliability; the interpretation of

probabilistic evidence; the role of stories and the attribution of blame. I will discuss how these issues can be elucidated within a general framework for evidential reasoning based on causal models.

Sandra Mitchell – *Evidence of Complex Causes*

Complex causal structures pose special problems for standard methods of eliciting causal knowledge from experiment and observation. In this talk I will outline several such problems stemming from robustness and the role of "context" in the dynamic stabilization characteristic of causation in complex biological systems.

Abstracts – Contributed speakers

Ramona Bongelli, Carla Canestrari, Ilaria Riccioni, Andrzej Zuczkowski, Cinzia Buldorini, Ricardo Pietrobon - *Evidentiality and Epistemicity in a Corpus of Scientific Biomedical Papers from the British Medical Journal*

In the present study the conference topic is approached from the viewpoint of linguistic communication in scientific writing. The communication of evidence is related to what in linguistic literature is called evidentiality (linguistic markers that reveal the source of information communicated by a speaker/writer, namely how s/he gains access to that information) and epistemicity (linguistic markers that reveal speaker/writer's certainty or uncertainty about the communicated piece of information). According to the results of our previous studies on written and oral corpora, evidentiality and epistemicity seem to be two sides of the same coin: the diverse and numerous evidential and epistemic markers can be led back to two main macro-markers, each of them has two faces, one evidential and the other epistemic: I know / I am certain; I believe / I am uncertain. The aim of the present study was to analyze from both a qualitative and quantitative perspective 1) the evidential and epistemic markers in a biomedical corpus (80 full text papers from the British Medical Journal randomly sampled from a 168-year period 1840-2007); 2) those sentences which express evidence and causality among events. Examples will be given of evidence and causality communicated either as certain or as uncertain.

Nancy Cartwright – *If you aren't doing arguments, you aren't doing evidence*

This paper proposes an argument theory of evidence. Evidence is not a natural kind. So our account of what makes for a good theory should be responsive to what needs the theory addresses. The argument theory is designed for use where high standards of both rigor and explicitness are demanded, as in many scientific endeavours and in contemporary evidence-based policy.

The central tenets of the argument theory are:

- If you aren't doing arguments, you aren't doing evidence.
- So…Evidence = facts recorded in good arguments.

I also insist:

- It's not evidence unless there's evidence it's evidence.

The paper will defend this theory from obvious philosophical objections and argue its superiority over alternatives that define evidence in terms of probabilistic relations or explanatory relevance. I will illustrate with examples from evidence-based policy's claims about evidence for causal effectiveness conclusions. The argument theory rules out as good evidence much that is highly touted in evidence-based policy and that, I shall argue, is all to the good.

A.Philip Dawid and Monica Musio – *From Statistical Evidence to Evidence of Causality*

Science is largely concerned with understanding "effects of causes" (EoC), and Law with understanding "causes of effects" (CoE). EoC can be addressed using experimental design and statistical analysis, but it is less clear how to apply statistical evidence to CoE questions. While some form of counterfactual reasoning, such as Rubin's potential outcome approach, appears unavoidable, this can yield "answers" that are dependent on arbitrary and untestable assumptions. We must recognise that a CoE question simply might not have a well-determined answer. It may nevertheless sometimes be possible to use statistical data to set bounds on the answer, and to narrow these by taking account of scientific knowledge about the system in question. Further care is required when identifying the relevant "counterfactual contrast", which may be a matter of policy as much as of science. Defining the question is as non-trivial a task as finding a way to answer it. The presentation will illustrate some of these philosophical issues with technical elaborations and data analyses.

Benjamin Hawkins and Justin Parkhurst – *Evidence and health policy: The conceptual, political and institutional dynamics of evidence informed policy making*

Claims about the causes of social problem or health inequalities (and the evidence marshalled to support these claims) are key justifications of policy. Whilst there is no shortage of voices arguing for evidence informed policy is, there is little understanding about what constitutes evidence, and what it actually means to use evidence. The role of evidence in policy making, the institutions which govern evidence use and the pathways through which evidence enters into practice remain poorly understood. The GRIP Health Project aims to address the gaps in our understanding of the evidence to policy process. The current paper begins this process by investigating the underlying theoretical and conceptual issues which arise from the project. It asks which conception of evidence is practically useful for guiding policy? Which institutions, rules, norms and practices govern the use of evidence? What criteria do we apply in defining evidence informed policy? Addressing these questions, we argue, can help to create a rigorous approach to the study of evidence and policy, while providing practical guidance to planners and policy makers on how to establish institutional responses that can promote a more effective and democratic use of evidence in policy decision making.

Dieneke Hubbeling – Predicting effect of interventions in psychiatry

In medical practice one has to predict the results of possible interventions and randomized controlled trials are considered essential for doing this. However, randomized controlled trials are sometimes not possible, and tend to have not enough statistical power to detect rare effects. Furthermore, even if randomized controlled trials are available, extrapolation of the findings to other settings can be difficult because information about the underlying causal structure is often not available. Cartwright advocated the use of 'capacities' – that is fixed causal contributions – in predicting the effects of interventions. However, in psychiatry, it is often not possible to determine what fixed causal contributions are and one can only establish 'approximate capacities', i.e. a certain response in a certain percentage of patients, because fixed causal contributions are unknown.

However, using 'approximate capacities' does imply a different way of evaluating health services, especially combined interventions. In health service research, if different studies, randomized controlled trials or other designs, have given different outcomes, the best way to investigate the effectiveness of a particular way of service organization is using capacities. One has to study the effects of certain elements of the complex intervention, which have been tested before in other settings, that is investigating 'approximate capacities'. One should check whether the separate elements do form a part of the complex intervention in practice and whether they have the same effect as in other studies and if not, why not. This increases the possibility of extrapolation and sometimes also our knowledge about 'approximate' capacities. One can also use 'approximate capacities' when randomized controlled trials are not possible, but especially for rare effects or for developing completely new treatments one needs to do something different as well.

Ciprian Jeler – *Causal partitioning and causal status in multi-level natural selection*

The two main statistical techniques for measuring the contributions of individual and group selection to evolutionary outcomes (the Price and the contextual approach, endorsed by Elliott Sober and Samir Okasha, respectively) are shown here to imply a trade-off between evidence and causality. General conditions for distinguishing between two processes, as well as a hypothetical case designed for this purpose show that supporters of the Price approach are forced either to deprive natural selection of its causal "powers", or to conceive group and individual selection as being essentially interconnected *qua* processes (and not merely interconnected with regards to their end result or their non-selective supervenience-base). On the other hand, as an analysis of "soft selection" shows, supporters of the contextual approach are led either to a statisticalist (non-causal) view of selection or to a view that would conflate – at the most basic definitional level – individual and group selection. Paradoxically, both approaches can either continue to claim offering correct statistical decompositions of evolutionary processes while giving up all claims about the causality of selection, or continue to claim that they offer correct – yet essentially incomplete – causal decompositions while giving up all claims about the possibility itself of statistically decomposing multi-level evolutionary processes.

Mike Joffe – *Evidence and causation in biology and economics*

Confidence in a causal relationship increases greatly when evidence of different types is mutually reinforcing. This is particularly true of evidence on both difference making and mechanism: they are complementary – both are ultimately necessary, although in the early stages of a hypothesis only one may be available. This is because a causal relationship is one that has a mechanism that by its operation makes a difference. This paper puts forward an ontic view of causation, corresponding to the epistemic view of Russo & Williamson based on an agent's rational beliefs given the available evidence, but focusing instead on the ontic question, what is the real-world source of this evidence? I apply this perspective in various branches of biology. Epidemiological studies provide evidence on difference-making, but epidemiologists have a strong sense of the complementary role of mechanism, and increasingly incorporate biomarkers into their research. Physiology is primarily mechanistic, but the mechanisms that are uncovered have the function of making a difference. Physiological systems,

such as homeostatic "devices", are composed of links; each link has both mechanism and difference-making, and in addition there is system-level difference-making (for homeostasis this is stability). Other branches of biology have a parallel structure, e.g. predator-prey systems (fluctuating populations due to negative feedback with delay), and evolutionary arms races (positive feedback). Finally, I briefly apply this analysis to economic theory: the different types of systems correspond to different types of market, that have radically different properties – a phenomenon that appears to have escaped economic theorists, despite the clear evidence that their price series behave in correspondingly radically different ways.

Roger Kerry – Ontology of causation in health science and evidence-based practice.

I argue that for a philosophy of evidence-based practice (EBP) to be complete, ontological attention towards causation is needed. Causation is a firm part of clinical decision–making in particular instances. The clinical decisions made represent something about the agent's understanding of causation and its scientific derivation. I will use causation in the context of interventions, i.e. what intervention causes health change. Thus, in an EBP framework, how causal claims are made scientifically relate directly to single-instances of clinical decision making. I claim that attention to epistemological matters of causal claims alone restricts a philosophy of EBP, and that ontological attention may help our understanding of the complexities associated with causation in health science and EBP. Evidential hierarchies expose how health science considers the nature of causation. Commonly, causation is thought to be something to do with controlled comparative studies. There are numerous problems associated with this as it is apparent that causal claims are made in the absence of such methods. Thus health science does not have a coherent view of its own ontology of causation. Alternative ontologies may be indicated.

Samantha Kleinberg – *Quantifying the Impact of Rare Causes*

A key barrier to applying causal inference methods to large-scale data, such as from ICUs, is that due to the frequency and volume of data collection, many important events will seem rare. An ICU patient monitored at a granularity of 5-seconds over a week will have 120,960 measurements – for each variable – yet, each observation is not equally important. Rather, many systems function in a steady state for a period of time before an event that triggers a shift to a new state. This vast amount of data must be mined in an automated way to find such changes, but causal inference methods usually take a probabilistic approach and it cannot be assumed that the relevant probabilities can be correctly estimated when dealing with rare events. However, data mining methods detect when rare events (such as credit card fraud) occur, but cannot identify how they affect the rest of system. This paper introduces an approach for evaluating the significance of a rare event for the expected value of a continuous-valued effect (that has other non-rare causes) using the connection between type-and token-level causality and testing whether the rare event explains a deviation from usual behavior.

Jaakko Kuorikoski – Mechanism-based extrapolation: the case of neuroscience of addiction

According to the conception of mechanism-based extrapolation, the inductive gap between evidence of a causal relation in one population and a causal hypothesis in another can be partially abridged by focusing on key similarities between the mechanisms mediating the causal effect in the source and target populations. We investigate the issue of the viability of mechanistic extrapolation in general, and the new formal tools for extrapolative causal reasoning in particular, in the context of a specific case of medication-based treatment of different forms of addiction: a proposal to treat problem gamblers with naltrexone, an opioid receptor antagonist used to treat alcoholics and opiate addicts. We assess the premise according to which the dopamine system is the relevant core mechanism on which the extrapolation should be based. Addiction also requires the presence of a set of social factors (or lack of thereof) in order to be stabilized as a problematic behavioural pattern, and this obviously complicates the extrapolation. We also explore whether the formal framework of selection diagrams helps to make the mechanistic premises grounding the extrapolation explicit and whether it can be used to estimate probabilities for the transportability of the causal effect even in a case in which there are significant differences between the source and target mechanism bases due to the relevance of social factors.

Adame La Caze – *When randomized trials?*

Success in suitably conducted randomized trials provide an evidential standard that plays a central role in the regulation of new medicines. Randomized trials are seen to confirm the effects of an intervention. Many would like to extend the use of randomized trial evidence as a confirmatory standard to social science and policy. The limits of randomized studies are increasingly well recognised, and a range of specific problems have been discussed extensively. What is missing in the literature, and the focus of this paper, is a more general account of when randomized trials provide an appropriate standard for testing the effects of interventions, medical or otherwise. This paper aims to clarify when, and for which questions, randomized trials are best-suited.

Catherine Laurent – *Evidence-aware policies, causality and plurality of science*

The results of research on evidence-based or evidence-aware decision (EBD) are seldom linked to those on the plurality of science. The heterogeneity of research programmes existing within disciplines is therefore rarely taken into account. Yet, in practice, by construction each theory has significant blind spots, and research programmes with contradictory theoretical underpinnings can yield complementary results for decision making. Ignoring this plurality can lead to over-simplified representation of the problems encountered in practice, out of phase with the complex causal structures facing practitioners. First, evidence, whatever its level of proof, is always incomplete knowledge of the piece of the world that is analyses. Second, scientific approaches produce various kind of evidence (presence, causality, efficacy, toxicity…) that ought to be recombined if it is to serve to guide action and/or evaluate its effects. When these limits are not clearly indicated, the solutions devised on the basis of such simplified representations may not correspond to the objectives, and may even produce serious adverse effects. The communication, based on two case studies, will

examine this issue of adverse effects of inappropriate use of evidence for practice.

Margaret MacDougall – *Assessing the integrity of clinical data: when is causality too good to be true?*

Evidence, as viewed through the lens of statistical significance, is not always as it appears! In the investigation of clinical research findings arising from statistical analyses, a fundamental initial step for the emerging fraud detective is to retrieve the source data for cross-examination with the study data. Recognizing that source data are not always forthcoming and that, realistically speaking, the investigator may be uninitiated in fraud detection and investigation, this talk will highlight some key methodological procedures for providing a sounder evidence base for withdrawing from a study on grounds of integrity. The promotion of patient safety is paramount. However, there is a broader rationale for disseminating these ideas. This includes empowering researchers to optimize their personal integrity, make informed choices regarding membership of future research collaborations and successfully voice their concerns to journal editors, particularly where a conflict of interests can render such dialogues particularly difficult. Recommendations will be supported by topical case studies, such as that of the Mendel-Fisher Controversy and practical steps involving data exploration, testing of baseline data and application of Benford's Law. While this talk has a clinical focus, the advice provided is transferrable to a wide range of multidisciplinary research settings outside of Medicine.

Sara Matera – *Evidence and causality in climate change debate: do skeptics bear the burden of proof?*

Human responsibility in climate change is now clearly shown by climate models, which simulate Earth system and its evolution through mathematical description of the evolution of each subsystem (like atmosphere, cryosphere, hydrosphere, lithosphere, biosphere, etc.). They are not able to predict actual temperatures without accounting for anthropogenic forcings, but, being climate a highly complex system, this "reductionist" approach can't be conclusive. Other approaches, based on statistical data analysis (Granges causality tests) and artificial intelligence techniques (simulation through neural networks), show that human influence on climate is causing the actual change in climate. So, the question is: do the so called "skeptics" actually bear the burden of proof? If so, they have to provide a model, be it statistical rather than mechanistic or based on neural networks, which is able to reconstruct the observed anomalies in temperature taking into account only natural causes.

Patrick McGivern – *Evidence and inter-level inference*

In this paper, I try to clarify the nature of inter-level evidential relations, drawing on examples from epidemiology. Inter-level relations are particularly important in epidemiology, both because epidemiological phenomena can be naturally characterized at a variety of levels (for instance, in terms of individual health or in terms of population health) and because it is often difficult to collect data at the level of primary interest. As a result, epidemiologists have long been concerned with the validity of inferences from one level to another. Here, I focus on two characteristics of inter-level inference in epidemiology. First, unlike discussions in many other areas of science, the concern in epidemiology is rarely

with inter-theoretic relations. To the extent that theories are involved at all, there is no obvious concern with reducibility, derivability, or even with the discovery of underlying mechanisms. Instead, the concern seems to be with the more basic task of establishing the presence of a phenomenon that can be the target of an explanation, such as a genuine causal relation or a lawful correlation. Second, epidemiological studies are often explicitly multi-level in nature, involving evidence from a variety of levels. In these cases, we can be concerned with the strength of evidence at different levels, and we can also be concerned with the evidence that a particular selection of levels is appropriate for a particular phenomenon. I consider several ways in which this second kind of evidence can be established, and how these relate to the question of realism about levels more generally in science.

Chris Miller – *Causation in personal injury law: the case for a probabilistic approach*

This paper argues for a wider acceptance of a probabilistic approach to causation in the law. This acceptance would help to remove much of the incoherence which has come to afflict the English law of negligence. This incoherence has spread to other common law jurisdictions (notably those of the United States, Canada and Australia). The argument opposes the contention that 'naked statistics' can play no role in establishing causation in personal injury cases. My argument is controversial but it is based on three unremarkable grounds:

1. with its acceptance (albeit in certain carefully delineated circumstances) of liability for a negligently increased risk which has eventuated, the common law has already embraced a probabilistic conception of causation; 2. English common law already employs a probabilistic (frequentist) approach to identifying coincidences; and 3. in its use of the 'balance of probabilities' as the standard of proof in civil cases, the common law has long had a probabilistic (subjective) concept at its core.

A wider acceptance of a probabilistic perspective on causation would entail no major challenge to any fundamental principle of the common law and it could assist the basic aims of tort, viz. deterrence and corrective justice.

Sean Muller – *External validity, causal interaction and randomised trials*

Randomized control trials are becoming increasingly popular in social science, particularly because of their ostensible value in developing `evidence-based policy'. I look to build on existing criticisms of such claims, in philosophy and econometrics, focusing on the implicit assumption of various kinds of external validity. Contributions by Mackie and Cartwright are reformulated in an econometric context to show how very strong ontological assumptions are required to justify the kind of external validity that randomized programme evaluations – as they are typically conducted - need to inform policy. To emphasise the practical import of these arguments, I analyse a particular example: attempts to assess the effect of school class size changes on educational outcomes. This case is chosen both for its suitability in illustrating the problem in question and because it has been held-up by proponents of randomized evaluations as an example of successful extrapolation. I conclude by arguing that plausible, rather than convenient, assumptions about underlying causal structures in the domain of social science imply failure of external validity as the

norm rather than the exception; this in turn suggests greater modesty in claims made in favour of, or using, the randomized evaluation method.

Barbara Osimani – Hunting side effects and explaining them: should we reverse evidence hierarchies upside down?

In the last decade a series of papers written mainly by epidemiologists have developed the view that evidence on pharmaceutical harm and benefit should be evaluated according to different criteria. In particular, Vandenbroucke (2008) endorses the idea that evidence hierarchies should be reversed upside down when the problem is not to test a causal connection for an intended effect but to discover an unintended effect. Vandenbroouke presents several arguments in support of such a proposal. One point is methodological, and concerns the idea that selection bias is less likely to affect observational studies with respect to adverse reactions, because unintended effects, qua unintended, are not known by the drug prescriber, who cannot take them into consideration and thus bias treatment allocation. Another point draws on epistemological considerations and regards the distinction between the context of discovery and the context of evaluation. I flesh out Vandenbroucke's intuitions in Bayesian terms and argue that rather than a reversal of hierarchies the point at issue is the lexicographic implementation rule which they more or less explicitly endorse. I will illustrate this point through a case study: the recent debate on the causal association between paracetamol and asthma.

Wolfgang Pietsch – *The structure of causal evidence in deterministic settings*

Most modern approaches to causality spell out causal evidence in terms of correlational data between the parameters characterizing the considered system, sometimes supplemented with information on how these correlations change under interventions. I argue that in deterministic contexts, causal evidence has a different structure, which ideally is of the following form: pairs of observations that differ in only one C of all circumstances that are potentially relevant to a phenomenon P. Two cases must be distinguished: (i) First, the change in C has no effect on P and one infers that C is causally irrelevant to P. (ii) Second, the change in C prevents P from happening and one infers that C is causally relevant to P, i.e. it is at least a partial cause. I will point out the major differences between such a deterministic compared with probabilistic approaches and will show how this type of evidence fits well with various accounts of causality, especially counterfactual and interventionist accounts. My claims will be illustrated using examples from experimental physics and in particular the engineering sciences, i.e. disciplines in which the knowledge and control of the boundary conditions is generally good enough to warrant a deterministic approach.

Elena Popa – *Causality, Evidence and Intervention in Conceptual Development*

I will be concerned with how causation and evidence are used in cognitive scientists' investigation of causal reasoning in young children. My analysis will focus on causation as involving manipulability and intervention (specifically, Woodward's account), a version of which, I will argue, is used by cognitive scientists in attributing causal reasoning to children. There seems to be a convergence of Woodward's view on causation using Bayesian causal networks, with the experiments showing that children use the Bayesian inference, as

authors investigating conceptual development, such as Gopnik, argue (their main claim being that young children are capable of causal reasoning and they do so, in part, through updating their beliefs upon being presented with new evidence). This suggests a further connection between causation as manipulability and a Bayesian picture of evidence, intertwined in research on causal reasoning. A further important point, on which I am going to rely, is that children's intuitive theories share many fundamental features with full blown scientific theories (the theory-theory of conceptual development). From this point, one could argue for the importance of what counts as causal reasoning in children in order to better understand how causality operates in scientific theories.

Katie Steele – *Crime, Punishment, and 'Specific' Evidence*

Various real and imagined criminal law cases provoke the intuition that there is something wanting with statistical evidence in the court- room, i.e. the appeal to trends associated with a large collective whose members are similar to the candidate in question. These cases are referred to as the 'proof paradoxes' (see Redmayne 2008). A prominent position, in response to the proof paradoxes, is that legal verdicts should not simply be a matter of probability of guilt; evidence for guilt must also be 'specific' to the accused, rather than 'general'/statistical. Judith Jarvis Thomson, in particular, argues that the problem with general evidence is that the beliefs it generates do not vary across telling counterfactuals, i.e. the beliefs are not properly sensitive to the truth. Here we rebut Thomson's proposal, arguing that specific and general evidence should be seen as complementary rather than contrastive; the proof paradoxes too can be construed as yielding beliefs that are properly sensitive to the truth. Furthermore, it is unlikely that this specific/general distinction underpins the proof-paradox in- tuitions. We suggest an alternative 'error theory' for the intuitions—an unnecessary concern for weight of evidence, over and above strength of evidence for guilt.

Mauricio Suárez – *The contextual character of causal evidence*

I argue that evidence for causal claims is contextual. The same causal claim may be warranted by the same piece of evidence in one context but not another. I argue for this thesis in particular with respect to the manipulability theory of causality defended by Woodward (2003), which takes evidence for causal claims to be provided by tests of putative causal relations between variables by means of interventions on the putative causal variable. Nevertheless I believe the claim that causal evidence is contextual may be generalised to other theories of causality, and at the end of the paper I indicate briefly how such generalization may go for both counterfactual theories, and process theories of causation. The relevant generalization rides on the back of the conceptual connections between interventions and counterfactuals, in the first case, and interventions and explanatory laws, in the second. However, there are ways to inject the relevant context-relativity directly into both counterfactuals and process accounts independently of any conceptual links to intervention, and I sketch briefly how this may be done.

Andrew Turner – *How should evidence-based medicine's 'hierarchies of evidence' be interpreted?*

The relationship between the EBM philosophy of evidence and EBM's hierarchies of evidence is not clear. Some philosophers have claimed that hierarchies of evidence offer the best insight into EBM's philosophical details. I first describe three well known problems with interpreting hierarchies 'categorically', and then describe two solutions to these problems. One suggested by Adam La Caze and a second suggested by both John Worrall and Jeremy Howick. I go on to describe the later solution in more detail, and then focus in particular on the way in which Worrall and Howick's view supports a 'heuristic' interptetation of evidence hierarchies. Despite its advantages however I argue that, while Worrall and Howick may provide a convincing account of what counts as good evidence, the heuristic interpretation that such an account seems to suggest is less convincing. I argue that there are at least two problems for such an interpretation: one epistemological, one sociological.

John A. Coster van Voorhout – *What causes legal evidence and what does legal evidence cause?*

If lawyers are looking for the *material truth* they might seek evidence 'making' the justified thought something really (factually) happened or is going to happen. Legal evidence is caused in a 'natural way' but -strangely enough- in a straitjacket of legitimacy. The human factor plays a role. In the end it is all about the attribution of responsibility to somebody; one human being doing something to another one. Not unusual in legal framework is to have two conditions considered. The first one being the *condicio sine qua non* (natural law) and the second one *the reasonableness of attribution of a specific effect to the agent's behavior* (law's law). Correctly accepted legal evidence may (or may not) cause legal proof of tort or of the indictment. This is all about the qualifications of the evidence (proof). Elements of quality are here: relevance, credibility (reliability, accuracy) and probative value. Now as far as the last quality is concerned the lawyer may use *Bayesian theory* or the so called *likelihood ratio.*

AHRC Symposium – How should the evidence hierarchy be developed?

Rationale

Evidence-based medicine is a relatively recent technique for supporting clinical decisions by the 'conscientious, explicit, and judicious use of current best evidence' (Sackett et al. 1996. BMJ. 312: 71). This 'best evidence' usually has a very specific meaning: the best evidence available to support decision-making in medicine is that arising from clinical trials, where treatments are tested on large numbers of patients. On the other hand, evidence of mechanisms – usually characterized as knowledge gained from experimental investigations in the laboratory – is held to be of low quality by the EBM practitioner.

However, recent work in the philosophy of causality has suggested that this hierarchical interpretation of evidence is problematic. Decisions about treatment make a difference to the health of individuals. Therefore it is of utmost importance to develop a concept of evidence that maximizes the available sources of evidence (trials, results of lab experiments) and minimizes the risks of errors in various medical decisions.

In this symposium, we aim to investigate the relationship between evidence-based medicine, evidence of mechanisms, and causality from a number of different theoretical and practical perspectives including philosophy of causality, philosophy and history of medicine, and medical practice.

Panellists:

Mauricio Barreto, Brendan Clarke, Jeremy Howick, Mike Kelly, Elsejin Kingma, Jacob Stegenga, Kurt Straif

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