intervention, whilst also not including elements of the intervention being tested that could impact relevant outcome measures. It would be very difficult, for instance, to placebo control an exercise intervention, as is argued by Maddocks et al. (2016: Problematic placebos in physical therapy trials, *JECP* 22(4), 598–602). How would one make a participant think they were exercising, without impacting any of the outcome measures that exercise may influence? Potential solutions to the difficulties of adequately placebo controlling are also difficult to employ in SES:

- Dose response trials need large sample sizes and effective placebo controls to provide good evidence.
- Active control trials assume the active control being tested against already has established efficacy, which is difficult to show without already having a suitable placebo against which to test it first.

Finally, it is very difficult to sufficiently blind sport and exercise trials. Even if a participant does not know whether they are receiving an intervention or a placebo, it will often be easy for someone conducting or administering an intervention to guess. For instance, a masseuse will know they are giving a fake massage, and a coach will know if they are administering a sham training plan. This can lead to changes in application of interventions or placebos, and interpretation of results, which can affect outcome measures.

Where SES RCTs do not well fulfil these requirements, outcomes observed cannot be readily attributed to interventions or exposures under investigation. For instance, chance and hunches about what trial group one is in may be the true explanation of observed outcomes, not what is being tested. Accordingly, RCTs not fulfilling these requirements provide low quality evidence. In turn, whatever one thinks is needed in order to establish a causal claim, RCTs in SES will often be unable to provide strong evidence for it because observed outcomes cannot be readily attributed to interventions under investigation.

Evidential Pluralism both helps us to explain why RCTs failing to meet these requirements also fail to establish causal claims, and also helps to provide us with practical solutions to the problem of justifying practice.

Through the lens of Evidential Pluralism: we may observe a correlation in an RCT, but until we can establish that a mechanism exists to explain that the intervention caused it, we cannot attribute it to the intervention under investigation. As RCTs do not provide evidence for a mechanism by providing details of mechanisms, to provide good evidence for a mechanism they must be sufficiently rigorous to rule out other explanations. As RCTs in SES often fail to meet the requirements needed to rule out alternate explanations for observed outcomes, even where they indicate a correlation may exist, their failure to rule in a mechanism means they fail to provide strong evidence of causality.

As this is the case, EBP seems to be wrong in privileging evidence from RCTs. Practice could be better informed where we had evidence that also provided strong justification that mechanisms exist. As such, EBP ought to assess RCTs and mechanistic studies together when assessing causal claims and justifying practice.

William Levack-Payne
Department of Philosophy and Centre for Reasoning
University of Kent

## Evidential Pluralism in the Social Sciences

Evidential Pluralism is a normative thesis concerning the epistemology of causation. It was first proposed by Russo and Williamson (2007: Interpreting causality in the health sciences, *ISPS* 21(2), 157–170), and further developed recently by Shan and Williamson (2021: Applying Evidential Pluralism to the social sciences, *EJPS* 11(4), 1-27). In a nutshell, Evidential Pluralism consists of two normative claims:

> (1) in order to establish a causal claim, one normally needs to have both evidence of correlation and evidence of mechanisms;
>
> (2) when assessing a causal claim, one ought to consider both association studies and mechanistic studies, where available.

Evidential Pluralism was originally introduced in the context of the health sciences and has been fruitfully applied to the biomedical sciences. However, the application of Evidential Pluralism to the social sciences has been controversial. For example, Weber (2009: How probabilistic causation can account for the use of mechanistic evidence, *ISPS* 23(3), 277–295) contends that Evidential Pluralism is 'correct' in the context of the social sciences, while Reiss (2009: Causation in the Social Sciences: Evidence, Inference, and Purpose, *PoSS* 39(1), 20–40) is sceptical of the application of Evidential Pluralism to the social sciences.

A major concern arises from the standard way of conceiving of the methodology of the social sciences, which focusses on a division between the quantitative tradition and qualitative tradition. For example, in sociology, quantitative researchers focus on statistical models and analyses and usually neglect the need to develop sociological models that mirror social mechanisms. In contrast, social theorists are mainly concerned with their concepts and theoretical frameworks and pay insufficient attention to the significance of quantitative findings. In political science, there has also been a methodological divide between the quantitative research approach and qualitative research approach. Such a methodological parallel pervades causal inquiry. When talking about causal analysis, social scientists tend to focus on looking for one type of evidence. For example, it is not unusual for political scientists to make within-case causal inferences by employing process-tracing methods to identify a mechanism. In other words, it seems to many that political scientists do not need any evidence of correlation when they establish single-case causal claims (e.g., the causes of the Russian Revolution). In a similar vein, Claveau (2012: The Russo-Williamson theses in the social sciences: Causal inference drawing on two types of evidence, *SHPSC* 43(4), 806–813) argues that economists can establish causal claims without evidence of correlation.

However, this is not quite right. As Shan and Williamson (2021: Applying Evidential Pluralism to the social sciences, *EJPS* 11(4), 1-27) have argued, those seeming counterexamples are not genuine counterexamples: process-tracing studies in political science typically assume some established correlations, while in Claveau's case, the relevant causal claims are

not established due to a lack of evidence of mechanisms and of correlation. Good social science research does accord well with the basic idea of Evidential Pluralism: social scientists tend to take both association studies and mechanistic studies into consideration when they assess causal claims.

A clear case is the study of socioeconomic status and health status. Social scientists have noticed that there is a strong association between socioeconomic status and health status. For example, lower socioeconomic status is associated with the 14 major causes of death in the International Classification of Diseases. In addition, lower socioeconomic status is shown to be associated with lower life expectancy, higher overall mortality rates, and higher rates of infant and perinatal mortality. However, it is debatable whether socioeconomic status is a cause of health status. Sceptics typically argue that socioeconomic status is a placeholder variable for real causes of diseases that have not yet been identified.

Even for some sociologists who argue for the causal relationship between socioeconomic status and health, a strong and pervasive association between socioeconomic status and health merely provides a description of the social pattern of disease. It is widely accepted that in order to establish the causal claim that socioeconomic status is a cause of disease, one has to establish the existence of some mechanisms as well as a correlation. As Link and Phelan (1995: Social Conditions As Fundamental Causes of Disease, *JHSB* Extra, 80–94) suggest, it is necessary to identify 'the direction of causation between social conditions and health and the mechanisms that explain observed associations' for the purpose of 'establishing a causal role for social factors'.

With their collaborators, Phelan and Link (2010: Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications, *JHSB* Sup, 28–40) have identified a variety of mechanisms linking socioeconomic status to health status. It is shown that persons of higher socioeconomic status possess a wide range of resources, including money, knowledge, power and beneficial social connections, which shape health-enhancing behaviours (such as getting flu jabs, eating fruits and vegetables, and exercising regularly) and access to broad contexts that are associated with risk and protective factors of health. For example, those who have lower status jobs more commonly have job strain (i.e., a combination of high job demands and low decision latitude), which is associated with coronary heart disease; people with lower socioeconomic status are more likely to smoke and be overweight, which lead to various health problems; and people with lower socioeconomic status experience greater residential crowding and noise, which is linked to poorer long-term memory and to reading deficits.

The debate over socioeconomic status and health thus illustrates that sociologists take both correlation and mechanism into account when they try to establish or assess a causal claim. The proponents of the theory of fundamental causes maintain that socioeconomic status is a fundamental cause of health status on the grounds that both the correlation and the mechanisms are established, while opponents challenge the causal claim by questioning the mechanism hypotheses. That both sides of the debate focus on evidence of correlation and evidence of mechanisms shows that Evidential Pluralism captures the structure of causal analysis in the social sciences.

Yafeng Shan
Department of Philosophy and Centre for Reasoning
University of Kent

## Evidential Pluralism in Social Policy

In 2007, Russo and Williamson put forward a form of evidential pluralism that argues, among other things, that a causal claim can be established only if it can be established that there is a difference-making relationship between the cause and the effect, and that there is a mechanism linking the cause and the effect that is responsible for that difference-making relationship (Russo and Williamson 2007: Interpreting causality in the health sciences, *ISPS* 21(2), 157–170). The applicability of Evidential Pluralism to biomedical research and health policies has provoked a lot of debate (see for instance Clarke et al. 2014: Mechanisms and the Evidence Hierarchy, *Topoi* 33, 339–360; Williamson 2019: Establishing Causal Claims in Medicine, *ISPS* 32(1), 33–61; Parkkinen et al. 2018: Evaluating Evidence of Mechanisms in Medicine, *Springer* 33, 339–360). In particular, Parkkinen et al. developed methods for evaluating mechanistic studies alongside association studies in medicine. In the social policy domain, however, the debate is yet to be rigorously shaped.

If we look at the UK What Works Centres (WWCs) and similar evidence-based policy centres that support government to develop policy, programmes and services, it is evident that the main approach to explore 'what works' is the use of difference-making studies, in particular randomised controlled trials. A closer look at these centres, however, can lead to an interesting observation: evidence of difference-making relationships is frequently combined with evidence of mechanisms, but different terminologies and a lack of methodological discussion make it difficult to recognise its use.

Let's consider, for instance, a typical evaluation of an evidence-based intervention. Such an evaluation is very likely to include a robust RCT, often called an 'impact evaluation', that helps to collect evidence of the difference-making relationship between the intervention and the outcome(s) of interest. An assumption often made by UK What Works Centres and clearing houses is that robust RCTs can support causal claims by ruling out the risk of confounding, therefore leading to the conclusion that the difference-making relationship between the cause and the effect is due to the presence of a mechanism linking them. In other words, if we consider Figure 1, RCTs would support causal claims via routes $C_1$ and $C_2$, and evidence directly supporting a difference-making relationship would also indirectly support the presence of a mechanism.

In social policy, as in the social sciences, the use of RCTs has been challenged (Morris et al. 2016: The importance of specifying and studying causal mechanisms in school-based randomised controlled trials: lessons from two studies of cross-age peer tutoring, *Educational Research and Evaluation* 22, 339–360), and the debate has often been framed around a call for evaluation of logic models, or process evaluations.