Integrating Quantitative and Qualitative Evidence using Evidential Pluralism

Introductory Guide

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1. Evidential Pluralism: What it is and when to use it

Evidential Pluralism (EP) provides a framework for understanding how to integrate quantitative and qualitative evidence when evaluating whether a policy or programme causes the desired outcome. The purpose of EP is to make use of all relevant evidence to answer the question: 'does the intervention work?'

EP does this by scrutinising mechanistic evidence alongside experimental and observational studies. Providing evidence of key features of the proposed mechanism of action of the intervention can raise confidence in effectiveness. On the other hand, if these features are found to be absent, or if key features of counteracting mechanisms are found to be present, this can undermine confidence in effectiveness. So, evidence of mechanisms can be very informative. EP can help us to evaluate this evidence when assessing whether an intervention works.

EP can be thought of as a framework for integrating standard methods for systematically reviewing experimental and observational studies with theory-based evaluation methods for assessing mechanistic evidence. However, while theory-based evaluation methods such as realist evaluation usually seek to ask *how* an intervention works, EP focusses on *whether* the intervention works.

EP is particularly appropriate:

- where experimental and observational studies on their own fail to establish or rule out causation;
- to ensure that qualitative evidence informed by stakeholders is not marginalised.

EP may not be appropriate when:

- the effectiveness of the intervention is already established or ruled out;
- the human and financial resources required to evaluate the full evidence base are not available.

2. Introducing Evidential Pluralism

Correlation is not causation. This remains the case even if we consider correlation conditional on potential confounding variables. Why? Because a correlation between intervention variable *A* and outcome variable *B* (controlling for potential confounders) could be attributable to any one of a large number of possible explanations—of which causation is just one. For example, a correlation might be due to reverse causation (i.e., *B* causing *A*); unforeseeable confounding variables; various kinds of bias introduced by the study design; fishing for correlations; sheer chance; insufficient sample size; or some connection between *A* and *B* other than causation (e.g., a logical connection). If the correlation really is attributable to *A* being a cause of *B* then there must be some mechanism of action by which *A* produces *B*. Hence, in order to establish causation one needs to establish not only correlation but also the existence of an underlying mechanism that can account for the extent of the observed correlation. This observation motivates Evidential Pluralism, which is depicted in Fig. 1.

¹ See Shan and Williamson (2023) for an introduction.

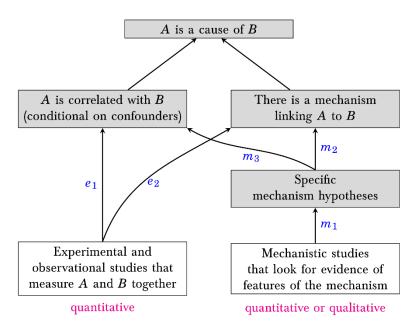


Figure 1: Evidential relationships for causal enquiry, according to Evidential Pluralism.

A purely quantitative approach to evaluation seeks to establish causation just by means of experimental and observational studies that repeatedly measure A and B together with potential confounders. Here, randomised controlled trials (RCTs) are particularly informative because they not only provide an estimate of the extent of the correlation (thereby confirming correlation via channel e_1 in Fig. 1), but they also indirectly confirm the existence of an underlying mechanism (channel e_2) because they lower the probability that the correlation is attributable to unforeseeable confounders.

But the presence of an appropriate mechanism can be tested more directly by hypothesising specific features of the mechanism (m_2) and then exploring whether or not these features are actually present (m_1). Such features include key mediating variables, or entities and activities involved in the mechanism, or features of the way in which these entities and activities are organised. A study that looks for such features is classified as a 'mechanistic study'.

A mechanistic study might use quantitative methods. For example, to test the hypothesis that some variable *C* mediates a mechanism between *A* and *B*, one might carry out an RCT that looks for a correlation between *A* and *C* and another quantitative study that looks for a correlation between *C* and *B*. A mechanistic study might, alternatively, use qualitative methods. For example, qualitative responses from interviews might be used to isolate features of the ways in which *A* and *B* are connected in the lives of individual stakeholders. Alternatively, a mechanistic study might use mixed methods: e.g., triangulation from quantitative and qualitative methods to establish the presence of some key feature of interest.

3. Integrating quantitative and qualitative evidence using Evidential Pluralism

EP can provide guidance on where and how to combine quantitative and qualitative evidence.

As we have just seen, a mixture of quantitative and qualitative evidence will need to be scrutinised in order to assess specific mechanism hypotheses (channel m_1 of Fig. 1). This is one point at which quantitative and qualitative evidence is combined.

A second point of integration arises when ascertaining whether there is a mechanism linking A and B. Here, one needs to consider how plausible it is that some suitable mechanism exists, given quantitative evidence from experimental and observational studies (channel e_2) and the quantitative and qualitative evidence from mechanistic studies (m_2).

A third point of integration arises when assessing correlation. Although quantitative studies are likely to be most informative here (a_1), mechanism hypotheses can suggest potential confounders that may not have been adequately controlled for, decreasing confidence that A and B are correlated conditional on potential confounders (m_3). Alternatively, if the mechanism is well established and sufficiently simple, the features of the mechanism may increase confidence in a correlation (m_3).

Shan and Williamson (2023, Chapters 3 and 4) and Parkkinen et al. (2018) provide detailed guidance on how to integrate evidence at these three points, in order to arrive at an overall evaluation of effectiveness.

4. Example: interventions to tackle fake news

The Problem

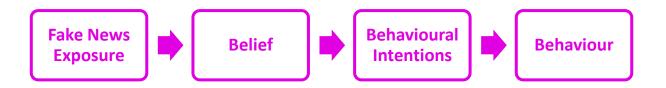
It is often assumed that online fake news has a detrimental impact on behaviour, but is this really the case? According to EP, to establish that online fake news affects behaviour, one needs to establish the existence of both a correlation and a mechanism.² This requires combining quantitative and qualitative evidence.

Quantitative studies provide evidence of correlation. For example, there is evidence of a correlation between the amount of misinformation shared and a decrease in daily Covid-19 vaccination rates, conditional on potential confounders.³

A plausible mechanism hypothesis connecting fake news and problematic behaviour is:

² 'Fake news' is here being used to cover all kinds of false and misleading information, including misinformation, disinformation and mal-information.

³ Pierri et al. (2022).

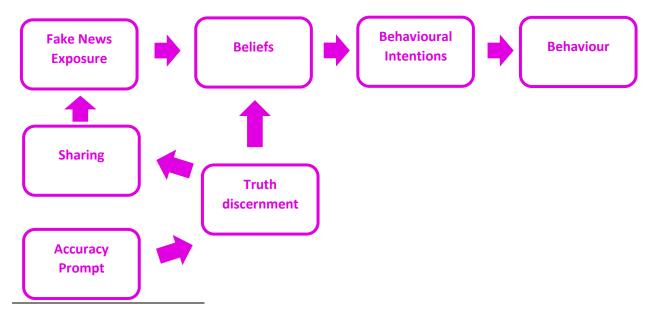


Qualitative and quantitative studies provide evidence of this mechanism.⁴ For example, qualitative surveys identify volume of information, repeated exposure, using family and friends as sources of information and emotive content as drivers of belief in fake news.⁵ An Experimental study found exposure to misinformation is associated with small but significant changes in behavioural intentions and that behavioural intentions are associated with belief.⁶

Thus, by combining quantitative evidence of correlation and quantitative and qualitative evidence of mechanisms, the detrimental effects of online fake news can be established.

Interventions

The above mechanism hypothesis and associated evidence can be used to identify possible points of intervention. For example, accuracy prompts aim to reduce the impact of fake news by improving truth discernment which (i) reduces belief in fake news⁷ and (ii) reduces sharing of, and in turn exposure to, fake news.⁸



⁴ See, for example, Allington et al. (2021); Greene and Murphy (2021); Lockyer et al. (2021); Pennycook et al (2018); Roozenbeek et al. (2020)

⁵ Lockyer et al. (2021).

⁶ Greene and Murphy (2021).

⁷ There is evidence that belief in fake news is driven by failing to engage in reflective reasoning and instead relying on automatic, intuitive thinking. See, for example, Bago et al. (2020); Pennycook and Rand (2019); Pennycook and Rand (2021).

⁸ There is evidence that repeated exposure increases belief in fake news. See, for example, Pennycook et al. (2018).

Quantitative and qualitative studies provide evidence of mechanisms to support the effectiveness of accuracy prompt interventions. For example, survey studies show that accuracy prompts increase truth discernment and reduce the amount of false content people intend to share online. A large field experiment conducted on Twitter shows that accuracy prompts reduce the amount of false content shared online. Computational modelling provides evidence that accuracy prompts increase truth discernment and improve quality of content shared by focusing attention on accuracy rather than increasing the amount of deliberation.

Combining quantitative and qualitative evidence provides strong evidence of mechanisms to support the effectiveness of accuracy prompt interventions. This evidence can be understood to provide indirect evidence of correlation via channel m_3 in Fig. 1 above. Quantitative evidence of a correlation between accuracy prompts and behaviour, such as improved Covid-19 vaccine uptake, would further support the effectiveness of accuracy prompts.

5. Example: interventions to reduce consumption of alcohol

The problem

Does alcohol consumption cause cancer? EP captures the evidence appraisal procedures employed by the International Agency for Research on Cancer (IARC) to answer questions such as this. The IARC *Monographs* programme evaluates the strength of the evidence in order to determine whether a particular preventable exposure can be classified as a possible, probable, or established cause of cancer in humans. IARC determined that consumption of alcoholic beverages does indeed cause cancer.¹²

A *Monographs* working group evaluates a range of evidence in order to determine whether an exposure is a cause of cancer.¹³ One subgroup evaluated evidence of cancer in human epidemiological studies relating to alcohol consumption. These quantitative studies provided strong evidence of the existence of a correlation between alcohol consumption and cancer in humans. But such studies alone are often insufficient to establish that the exposure is a cause of cancer, due to the risk of confounding and bias. Another subgroup therefore evaluates the evidence from mechanistic studies in order to determine whether there is a mechanism linking exposure to cancer. These studies can be quantitative or qualitative. There was substantial mechanistic evidence that alcohol causes cancer of the oesophagus, for example.

Interventions

Establishing that alcohol consumption is a cause of cancer and other diseases has led to a number of interventions aimed at reducing alcohol intake. One such intervention is the introduction of

⁹ Arechar et al. (2022); Brashier et al. (2020); Epstein et al. (2021); Fazio (2020); Pennycook et al. (2020); Pennycook et al. (2021); Pennycook and Rand (2022).

¹⁰ Pennycook et al. (2021); Pennycook and Rand (2022).

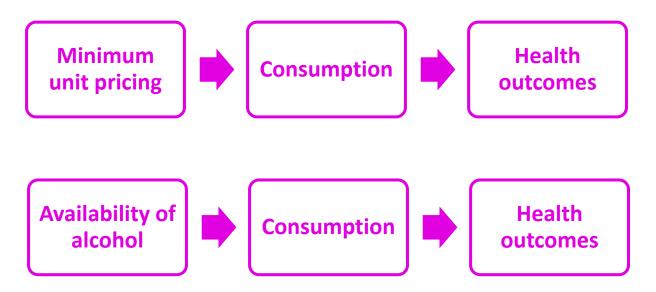
¹¹ Lin, Pennycook and Rand (2023).

¹² IARC (2010).

¹³ IARC (2019).

minimum unit pricing, that is, a legal minimum price per unit of alcohol. EP can help to assess the effectiveness of such an intervention on different populations.

Scotland introduced minimum unit pricing in 2018, but there is currently no minimum unit pricing in England. Quantitative studies comparing England and Scotland have shown a population-level correlation between the introduction of minimum unit pricing and a reduction in deaths and hospitalisations. One possible explanation of this correlation appeals to the familiar economic mechanism by which an increase in price leads to a fall in demand and therefore consumption of alcohol. However, there are also alternative mechanism hypotheses. For example, the Scottish population in which minimum unit pricing was introduced may have had an unrelated, simultaneous drop in the availability of alcohol relative to the English control population; it may be a drop in the availability of alcohol, rather than the minimum unit pricing, that is causing most of the reduction in deaths and hospitalisations. 15



Qualitative and quantitative studies can help to decide between the two competing mechanism hypotheses. For example, quantitative studies help to disconfirm the availability hypothesis by providing evidence of a similar level of availability of alcohol in Scotland and England. And qualitative or mixed methods studies, for example, studies including structured interviews, help to determine whether it is the affordability or the availability of alcohol that is causing the reduced consumption. In this way, integrating quantitative and qualitative evidence can help to establish the effectiveness of a proposed intervention.

6. Example: Universal Basic Income

EP can help to identify gaps in the evidence base, as in the example of Universal Basic Income (UBI). Where the quantitative evidence gives some inconclusive and low-quality support to the claim that

¹⁴ See, for example, Wyper et al. (2023).

¹⁵ See, for example, PHS (2023) pp. 60-77.

¹⁶ PHS (2023), pp. 72-74.

¹⁷ See, for example, Holmes et al. (2022).

UBI is correlated with health and economic benefits, EP suggests that there is a need for additional mechanistic evidence.

UBI is purported to have a range of effects, including: a reduction in economic inequality and precarity;¹⁸ and a reduction in a number of health issues, particularly depressive disorders and preventable long-term conditions, and their subsequent cost on the NHS.¹⁹

A number of small-scale trials have been piloted to assess the effectiveness of UBI in the UK: starting in 2022, a pilot study will test the effectiveness of a monthly £1600 stipend for the wellbeing of 500 people leaving care over 2 years;²⁰ in England a trial in two areas in the North and South of the country are planned for 2023, in which 30 people will also be given £1600 a month for 2 years;²¹ and proposals for trials in Scotland and Ireland are currently under consideration.²²

However, these micro-trials have limitations: they are very small, with current trials only including between 30 and 500 participants; they are localised to specific areas, such as Jarrow and East Finchley, and to specific demographics, such as Welsh young people leaving care; and while they contain control groups, trials can't be properly randomised or double-blinded, as participants know if they're receiving the stipend. Thus, they are far from ideal and their findings are inconclusive on their own. There is therefore a clear gap in the evidence base, that can be addressed in two ways: by conducting larger, higher quality experimental trials, and/or by seeking mechanistic evidence²³.

Large and more diverse trials are costly and hard to justify in the absence of provisional evidence of effectiveness. The economic benefits of UBI are modelled by the NHS²⁴ and their affordability is rationalised within reasonable budgets:²⁵ but expanding UBI trials to a sufficient size, scope, and design would be a hugely expensive and risky undertaking.

Rather than investing heavily in scaling up trials, a more cost-effective approach would be to integrate mechanistic evidence with the existing quantitative evidence.²⁶²⁷ At the current moment in time, however, there is limited mechanistic evidence for both the positive and negative effect of UBI on participation in the labour market, limited mechanistic modelling of how UBI could be funded and its impact on national budgets, and a limited mechanistic understanding of how UBI reduces low-income related stress and health issues.²⁸ There is a clear gap in the evidence base in this regard, and more research is needed to fully explore the hypothesised mechanism underpinning UBI as a health and economic intervention, which can be summarised as follows:

¹⁸ See, for example, Lowrey, 2018; Reed et al, 2023.

¹⁹ See, for example, Gibson et al, 2020; Johnson et al, 2023.

²⁰ See Drakeford, 2022.

²¹ See Ali Hussen, 2023.

²² See Redmond et al, 2022.

²³ See Hoynes and Rothstein (2019) for further information on the limitations of these micro-trials.

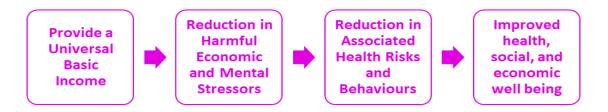
²⁴ Johnson et al, 2023.

²⁵ Reed et al, 2023.

²⁶ Johnson et al, 2021.

²⁷ This has been noted by Hoynes and Rothstein (2019), who argue that: "Unfortunately, the planned and ongoing pilots are not well suited to answer these questions [of effectiveness]. Experimentation aimed at identifying parameters and mechanisms... would be more useful than evaluations of small UBI pilots" (p. 24, my own bracketing).

²⁸ See, for example, Fitzpatrick, 2022; Jaimovich et al, 2022.



By investigating this proposed mechanism hypothesis through a range of quantitative and qualitative mechanistic studies that provide an understanding of how UBI reduces low-income stress and health issues, policy makers will be in a better position to evaluate whether UBI works. An analysis of mechanistic evidence would also be better grounds for deciding whether larger UBI trials are justified.

Given the public interest in the proposed health²⁹ and economic benefits³⁰ of UBI, there is a clear need for more confidence in the effectiveness of UBI as a policy intervention, as well as greater accountability and understanding. While larger and more diverse trials are desirable, obtaining mechanistic evidence can be more cost-effective and less risky, and EP provides a practical method for integrating quantitative and qualitative mechanistic evidence in the evaluation of UBI.

Further Resources

Introductory material on Evidential Pluralism can be found at: https://blogs.kent.ac.uk/jonw/ep/

EP also provides an account of how to assess the external validity of an intervention. For more information, see Section 4 and Chapter 3 of:

Shan, Y. and Williamson, J. (2023). *Evidential Pluralism in the Social Sciences*. Routledge, Abingdon. Open-access at https://www.taylorfrancis.com/books/oa-mono/10.4324/9781003143000/

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²⁹ See, for example, Ruckert et al, 2017; Johnson et al, 2023.

³⁰ See, for example, Srnicek & Williams, 2015; Harrop & Tait, 2017; Lowrey, 2018.

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