# Integrating Quantitative and Qualitative Evidence using Evidential Pluralism

## Introductory Guide

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**Contents**

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

Evidential Pluralism (EP) is a framework for understanding how to integrate quantitative and qualitative evidence when evaluating whether an intervention works. EP yields evaluations informed by *all* relevant evidence, not just a small subset of quantitative studies.

EP does this by assessing 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 methods, which focus on mechanisms. However, while theory-based evaluation designs 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;
- where mechanistic considerations might undermine the results of experimental and observational studies (e.g., by identifying potential confounding variables that have not been adequately controlled for);
- to ensure that qualitative evidence informed by stakeholders is not marginalised.

# 2. Introducing Evidential Pluralism

Correlation is not causation. 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*), uncontrolled confounding variables, or various kinds of bias introduced by the study design. 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*. So, 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.

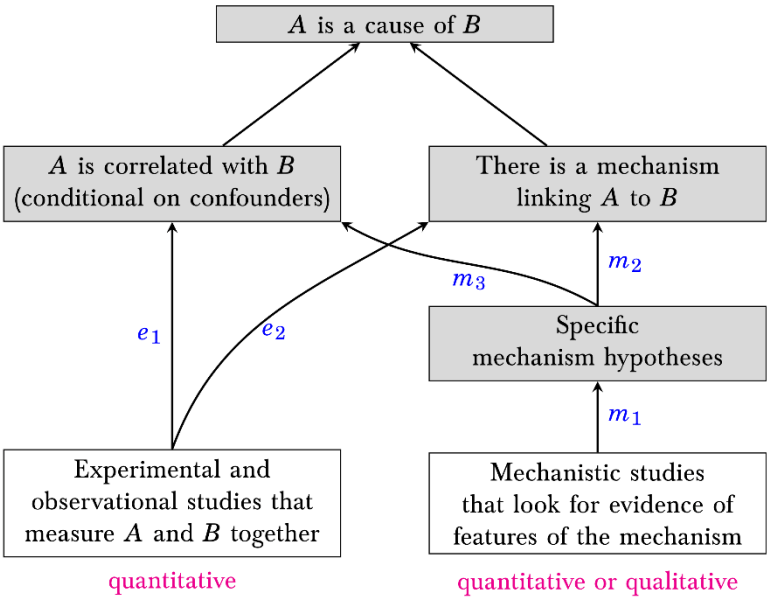



Figure 1: Evidential relationships for causal enquiry, according to Evidential Pluralism.

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 (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 uncontrolled 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; qualitative comparative analysis (QCA) might be used to test which features of a mechanism are key to its execution. 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.

- Quantitative and qualitative evidence will need to be scrutinised in order to assess specific mechanism hypotheses (channel $m_1$ of Fig. 1).
- 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 ($m_3$).

The EP approach to integrating these streams of evidence is summarised in Table 1 below. See section 7 below for an in-depth example that goes through the steps of Table 1.

*Table 1. The EP approach to evaluating the effectiveness of an intervention.*

| | Task | Main questions |
|---|---|---|
| 1. | Specify the causal claim. | What is the intervention, the outcome of interest, the population and the time-frame? |
| 2. | Specify the correlation claim. | What are the potential confounders? |
| 3. | Formulate specific mechanism hypotheses. | What are the key features of the mechanism by which the intervention is supposed to work? What the key features of any counteracting and enhancing mechanisms? |
| 4. | Search for and assess experimental and observational studies. | What are the search terms? What are the inclusion criteria for the studies? How high quality is each individual study? |
| 5. | Screen the need for a mechanistic evaluation. | What is the preliminary status of the correlation, general mechanistic and causal claims? Could evidence of specific mechanism hypotheses change these preliminary determinations? (If so, proceed.) |
| 6. | Search for mechanistic studies. | Which features of specific mechanism hypotheses have already been established or ruled out? Which review questions should be used to find studies relevant to remaining features? |
| 7. | Assess mechanistic studies. | How relevant are the population and variables of each study? How reliable are its methods? Does it implement these methods well? Are the results independently verified, consistent and robust? |
| 8. | Assess specific mechanism hypotheses. | What status do the mechanistic studies confer on each specific mechanism hypothesis? |
| 9. | Assess the correlation claim. | Do specific mechanism hypotheses modify the preliminary status conferred on the correlation claim by experimental and observational studies? |
| 10. | Assess the general mechanistic claim. | Have alternative explanations of the correlation, such as bias and confounding, been ruled out? How well confirmed are the features of the mechanism complex? Can it account for the magnitude of the observed correlation? |
| 11. | Assess the causal claim. | What is the minimum status of the correlation and general mechanistic claims? (This is the status of the causal claim.) |

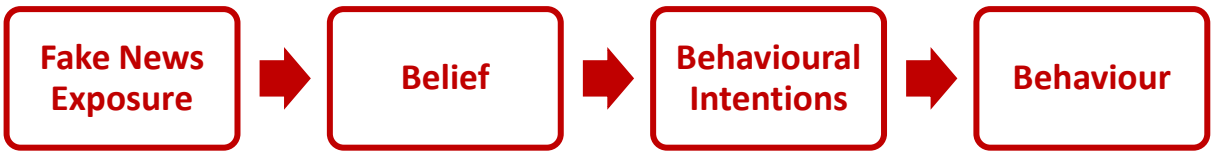
# 4. Example: interventions to tackle fake news

**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.[1] 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.[2]

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




Qualitative and quantitative studies provide evidence of this mechanism.[3] 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.[4] 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.[5]

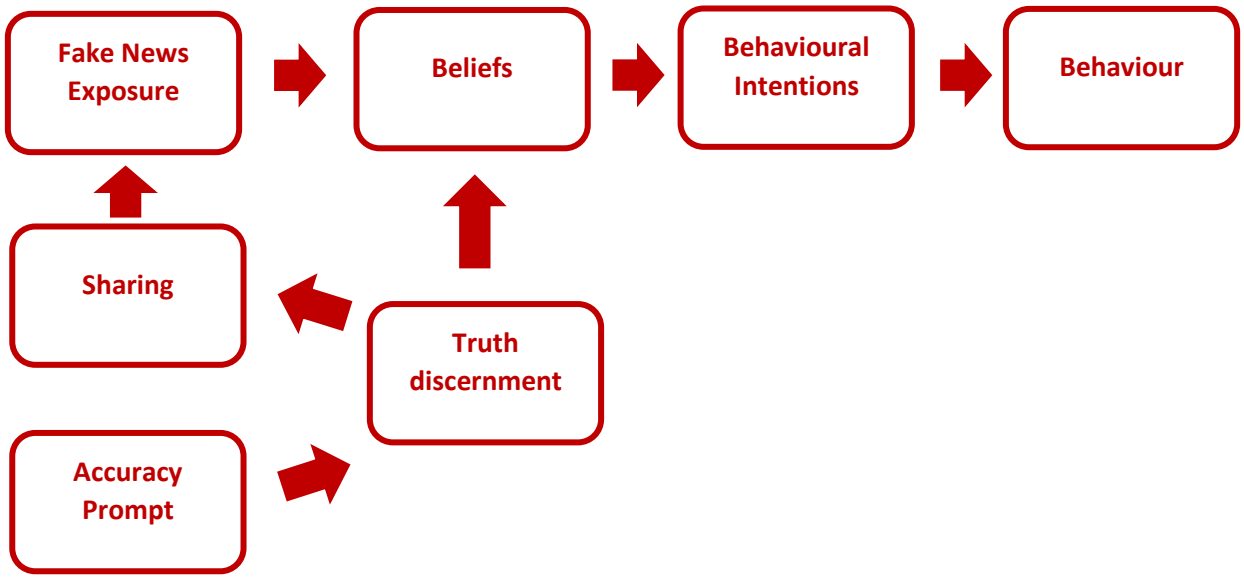
Thus, by combining quantitative evidence of correlation and quantitative and qualitative evidence of mechanisms, the detrimental effects of online fake news can be established.

**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

[1] 'Fake news' is here being used to cover all kinds of false and misleading information, including misinformation, disinformation and mal-information.
[2] Pierri et al. (2022).
[3] See, for example, Allington et al. (2021); Greene and Murphy (2021); Lockyer et al. (2021); Pennycook et al (2018); Roozenbeek et al. (2020)
[4] Lockyer et al. (2021).
[5] Greene and Murphy (2021).

truth discernment which (i) reduces belief in fake news[6] and (ii) reduces sharing of, and in turn exposure to, fake news.[7]




Evidence of mechanisms is also crucial to evaluating interventions. 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.[8] A large field experiment conducted on Twitter shows that accuracy prompts reduce the amount of false content shared online.[9] 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.[10]

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.

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[6] 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).

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

[8] 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).

[9] Pennycook et al. (2021); Pennycook and Rand (2022).

[10] Lin, Pennycook and Rand (2023).

# 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.[11]

A *Monographs* working group evaluates a range of evidence in order to determine whether an exposure is a cause of cancer.[12] 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 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.[13] 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.[14]

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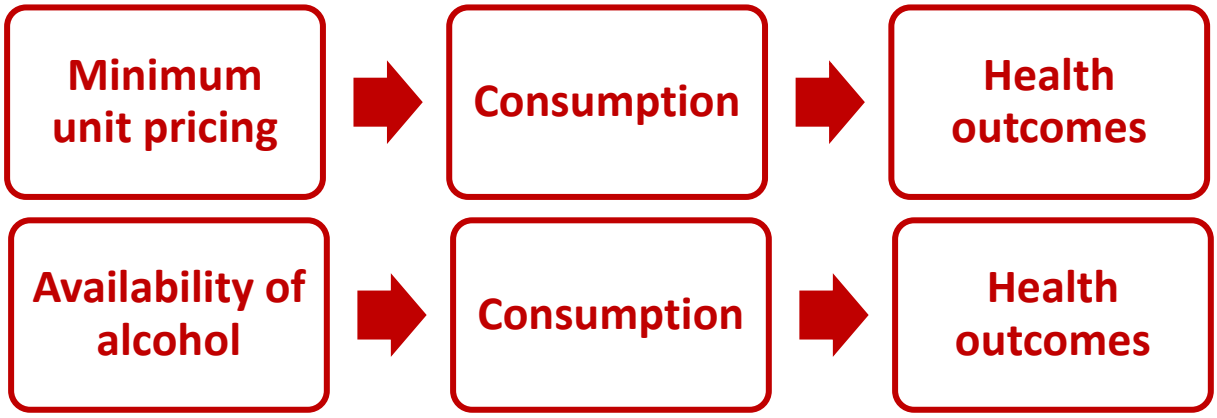
[11] IARC (2010).
[12] IARC (2019).
[13] See, for example, Wyper et al. (2023).
[14] See, for example, PHS (2023) pp. 60-77.




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.[15] 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.[16] 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 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;[17] and a reduction in a number of health issues, particularly depressive disorders and preventable long-term conditions, and their subsequent cost on the NHS.[18]

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;[19] 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;[20] and proposals for trials in Scotland and Ireland are currently under consideration.[21]

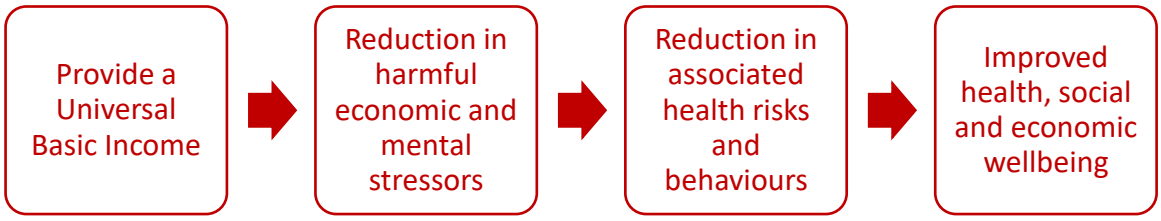
However, these micro-trials have limitations: they are very small, with current trials only including between 30 and 500 participants; they are localised to specific areas, such as Jarrow and East

[15] PHS (2023), pp. 72-74.
[16] See, for example, Holmes et al. (2022).
[17] See, for example, Lowrey, 2018; Reed et al, 2023.
[18] See, for example, Gibson et al, 2020; Johnson et al, 2023.
[19] See Drakeford, 2022.
[20] See Ali Hussen, 2023.
[21] See Redmond et al, 2022.

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 evidence[22].

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 NHS[23] and their affordability is rationalised within reasonable budgets:[24] 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.[2526] 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.[27] 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:




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 health[28] and economic benefits[29] of UBI, there is a clear need for more confidence in the effectiveness of UBI as a policy intervention, as well as greater

[22] See Hoynes and Rothstein (2019) for further information on the limitations of these micro-trials.
[23] Johnson et al, 2023.
[24] Reed et al, 2023.
[25] Johnson et al, 2021.
[26] 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).
[27] See, for example, Fitzpatrick, 2022; Jaimovich et al, 2022.
[28] See, for example, Ruckert et al, 2017; Johnson et al, 2023.
[29] See, for example, Srnicek & Williams, 2015; Harrop & Tait, 2017; Lowrey, 2018.

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.

# 7. Example: Covid-19 face mask mandates

A narrow focus on experimental studies, especially RCTs, resulted in controversy and uncertainty concerning the effectiveness of public health interventions to reduce the spread of Covid-19, including legal requirements to wear a face mask in public. A Cochrane Systematic Review, which included only RCTs, found face masks make little or no difference to the spread of Covid-19.[30] Various limitations of the review and of the studies included have, however, been highlighted. Taking account of a broader range of evidence is crucial to understanding the effectiveness of face masks.[31] This case study therefore provides a good example of the need for and benefits of an EP evaluation. We sketch such an evaluation here, following the steps set out in Table 1.

1. Specify the causal claim:

The causal claim is that a legal requirement to wear a face mask in public reduces the prevalence of symptomatic Covid-19 infections and thereby reduces the number of hospitalizations and deaths.

The population of interest is the global population. An evaluation of a specific law on reducing the prevalence of symptomatic Covid-19 infections will focus on the population of the jurisdiction within which the specific law applies. However, here we are concerned with the effectiveness of legal requirements to wear face masks at a more general level.

The legal requirements of interest are those that require a cloth face mask to be worn in public, especially when indoors or when outside and maintaining social distancing is difficult. Exemptions include relevant health conditions that make wearing a face mask problematic or difficult.

2. Specify the correlation claim:

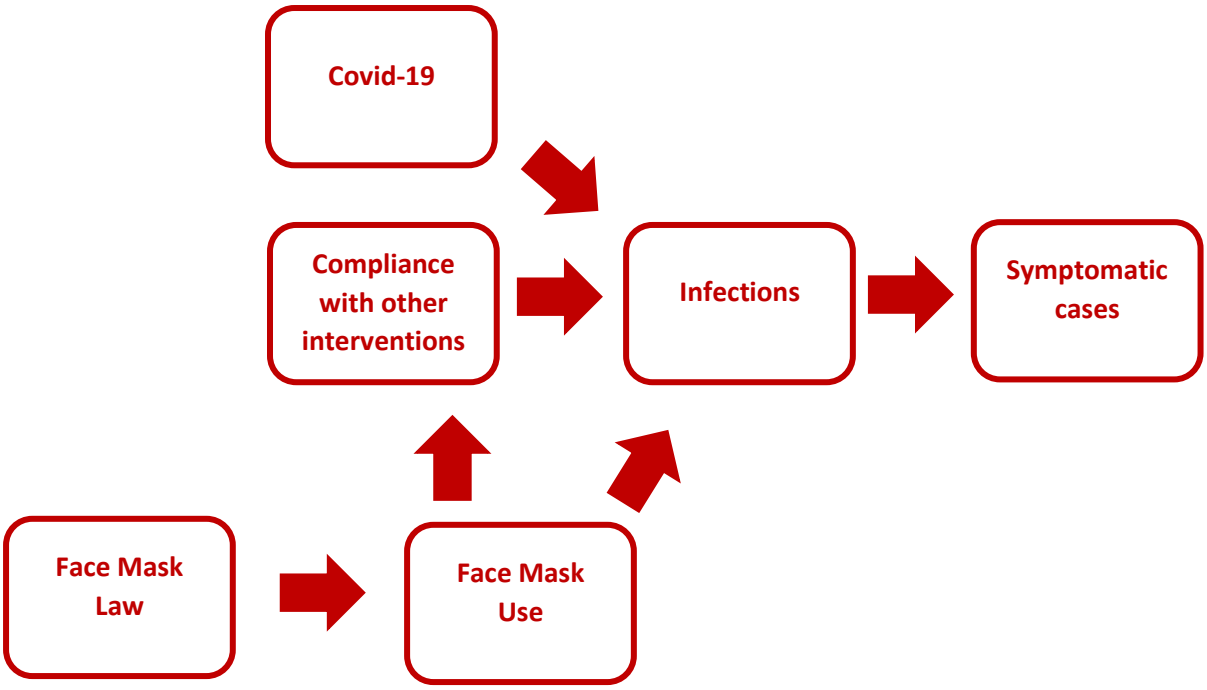
The correlation claim is that a legal requirement to wear face masks in public is negatively correlated with symptomatic Covid-19 infections, conditional on potential confounders. Potential confounders include Covid-19 test rates, other public health measures such as social distancing, underlying health conditions, individual mobility, population density, socio-economic factors, age, gender, temperature and humidity.

[30] Jefferson et al., (2023).
[31] Greenhalgh et al., (2022); Howard et al., (2020).

3. Formulate specific mechanism hypotheses:

A plausible mechanism hypothesis is that a legal requirement to wear a face mask in public increases the use of face masks which in turn reduces the prevalence of Covid-19 which reduces the prevalence of symptomatic covid infections and thereby the number of hospitalizations and deaths.

A plausible hypothesised counteracting mechanism is that a legal requirement to wear a face mask in public will decrease compliance with other public health interventions, such as social distancing. This in turn would result in an increase in the number of symptomatic infections compared to the number that would have occurred if the legal requirement to wear a face mask had not been introduced.




4. Search for and assess experimental and observational studies:

An event study conducted in the U.S. that covered all 50 states found mask mandates are associated with a 14% decrease in Covid-19 cases, conditional on several potential confounders including Covid-19 test rates, other public health interventions, individual mobility and weather.[32]

An observational study of matched cohorts from 412 U.S. counties found mask mandates are associated with decreases in Covid-19 cases ranging from 11% to 25 % during a six-week post intervention period, conditional on potential confounders.[33]

[32] Adjodah et al., (2021).
[33] Huang et al., (2022).

Analysis of data covering 188 nations found that mask mandates are consistently associated with lower Covid-19 infection rates in the short term and early adoption of mask mandates is consistently associated with lower infection rates in the longer term, conditional on potential confounders.[34]

### 5. Screen the need for a mechanistic evaluation:

Taken together, the quantitative studies detect a robust correlation across contexts. It is therefore plausible to conclude that the correlation claim is established and that confidence in the general mechanistic claim is increased.

Although each of the quantitative studies controls for several confounders, some residual confounding may persist. The causal claim is therefore only provisionally established. A mechanistic evaluation is necessary because it could increase confidence in the causal claim.

### 6. Search for mechanistic studies:

The mechanism hypothesis is that a legal requirement to wear a face mask will increase face mask use and thereby decrease the prevalence of Covid-19 symptomatic infections, hospitalizations and deaths.

The first part of the mechanism hypothesis is that a legal requirement to wear face masks will increase face mask use. Analysis of survey data found that mask wearing increased up to 23.4% after the implementation of State mandates in Hawaii, Iowa, North Dakota and New Hampshire.[35] Betsch et al. conducted cross-sectional surveys with 6973 German participants. They found that the implementation of a public mask mandate steeply increased mask wearing. They also found that sufficient compliance would unlikely be achieved with a voluntary mask policy.[36] MacIntyre et al. conducted cross sectional surveys in Sydney and Melbourne (Australia), London (UK) and Phoenix and New York (USA). They found mask mandates are a predictor of mask wearing.[37]

The second part of the mechanism hypothesis is that face mask use will reduce the spread of Covid and thereby reduce symptomatic infections, hospitalizations and deaths. A recently updated Cochrane review found face masks make little or no difference to the spread of Covid-19.[38] In contrast, multivariate analysis of 196 countries found that duration of mask wearing is negatively associated with Covid-19 mortality and that cultural norms and policies supporting public face mask wearing are associated with lower per-capita coronavirus mortality.[39] Model simulations found widespread use of face masks effectively reduces community transmission and decreases hospitalizations and deaths.[40] Experimental studies provide support for the efficacy of face masks in reducing transmission by supporting aerosol transmission as a key mode of transmission.[41]

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[34] An et al., (2021).
[35] Adjodah et al., (2021).
[36] Betsch et al. (2020).
[37] MacIntyre et al., (2021).
[38] Jefferson et al., (2023).
[39] Leffler et al., (2020).
[40] Eikenberry et al., (2020).
[41] Van Doremalen et al., (2020); Leung et al., (2020).

The hypothesised counter-acting mechanism is that a legal requirement to wear face masks increases Covid-19 symptomatic cases by decreasing compliance with other public health interventions such as social distancing. Surveys found mask wearing is positively correlated with complying with other public health interventions, such as social distancing.[42]

By combining a range of quantitative and qualitative studies, evidence for each section of the mechanism hypothesis can be provided.

## 7. Assess mechanistic studies:

The studies relating to the first part of the mechanism hypothesis all support the effectiveness of a legal requirement in increasing face mask wearing. The studies provide evidence across contexts and control for a number of confounders. However, the studies rely on self-reported survey data and therefore there is a risk of recall bias and response bias in the results. Furthermore, although the studies cover a range of geographical regions, they are not fully representative of the global population.

Turning to the second part of the mechanism hypothesis, the Cochrane review included only RCTs and, as the authors acknowledge, the observed lack of effectiveness could be due to study design flaws. Furthermore, most of the studies included in the review evaluated masks in terms of how well they protect the user rather than how well they reduce community transmission and therefore focus on a different outcome of interest to the outcome of interest here. Given these limitations, the results of the systematic review should be taken as inconclusive rather than negative in relation to the effectiveness of face masks in reducing the spread of Covid-19.

Taking account of a broader range of evidence enables a more accurate assessment of the effectiveness of face masks. The other studies considered here provide support for the effectiveness of face masks in reducing community transmission and thereby reducing symptomatic cases, hospitalizations and deaths. Taking account of a broader range of evidence, Greenhalgh et al. conclude that airborne aerosol transmission 'is likely to be dominant'.[43] A narrative literature review concluded that face masks are effective at reducing community transmission when compliance is high.[44]

The survey results undermine the counteracting mechanism hypothesis. The surveys provide evidence across contexts and control for confounders but there remains a risk of bias in the survey results.

## 8. Assess the specific mechanism hypothesis:

The studies considered here provide support for the first part of the mechanism hypothesis. Some risk of bias remains, however, and the results are not representative of the global population. Therefore, the first part of the mechanism hypothesis is provisionally established.

The evidence considered here provides strong support for the second part of the mechanism hypothesis. The limitations of the systematic review mean it is inconclusive and does not provide

[42] Betsch et al., (2020); MacIntyre et al., (2021).
[43] Greenhalgh et al., (2021).
[44] Howard et al., (2021).

high-quality evidence against the second part of the mechanism hypothesis. Taken together, the rest of the evidence considered here provides strong support for the effectiveness of face masks, particularly when widespread adoption of face masks is achieved. Therefore, the second part of the mechanism hypothesis is established.

The survey results provide evidence against the counter-acting mechanism. Furthermore, given the extent of the net benefit of a legal requirement to wear face masks found across multiple studies, it is evident that the benefit is not negated, even if slightly reduced. The counteracting mechanism hypothesis is, therefore, provisionally ruled out.

Although the second part of the mechanism hypothesis is established, the first part is only provisionally established. The status of the mechanism hypothesis is that of its weakest part. Therefore, on the basis of the quantitative and qualitative evidence considered here, the mechanism hypothesis is provisionally established overall.

9. Assess the correlation claim:

The correlation claim is established. The quantitative and qualitative evidence considered provides strong evidence of both correlation and mechanisms. The evidence of mechanisms increases confidence in the correlation claim to such an extent that it is unlikely that further evidence would overturn it.

10. Assess the general mechanism claim:

The evidence of mechanisms provisionally establishes the specific mechanism hypothesis. However, the strength of the quantitative evidence of correlation further increases confidence in the general mechanistic claim. Given the mutually supporting evidence of correlation and mechanism, the general mechanistic claim is established.

11. Assess the causal claim:

On the basis of the quantitative and qualitative evidence considered here, both the correlation claim and the general mechanistic claim are established. Therefore, the causal claim is established.

# 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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