

Bounding Causes of Effects with Mediators

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June 30, 2020

Abstract

Suppose X and Y are binary exposure and outcome variables, and we have full knowledge of the distribution of Y , given application of X . We are interested in assessing, for a case that was exposed and exhibited a positive outcome, whether the exposure caused the outcome. The relevant “probability of causation” is typically not identified by the distribution of Y given X , but bounds can be placed on it. Here we provide a full characterization of the bounds that can be achieved in the ideal case that X and Y are connected by a causal chain of complete mediators, and we know the probabilistic structure of the full chain. Our results are largely negative. We show that, even in these very favourable conditions, the gains from positive evidence on mediators is modest. In contrast, we show, the scope for improvement from learning about conditional causal effects is large.

Key words and phrases: Causal pathway; Causes of effects; Interval bounds; Mediator; Potential outcome; Probability of causation

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¶Research supported by the project GESTA of the Fondazione di Sardegna and Regione Autonoma di Sardegna

1 Introduction

Even the best possible evidence regarding the effects of a treatment on an outcome in a population is generally not enough to identify the probability that a positive outcome in an individual treated case was in fact caused by the treatment.

For instance, researchers conducting randomised controlled trials may determine that providing a medicine to school children increases the overall probability of good health from one third to two thirds. This information, no matter how precise, is not enough to answer the following question: Is Ann healthy because she took the medicine? It is not even enough to answer the question probabilistically. The reason is that, consistent with these results, it may be that the medicine makes a positive change for 2 out of 3 children, but a negative change for the remainder: in that case the medicine certainly helped Ann. But it might alternatively be that the medicine makes a positive change for 1 in 3 children but no change for the others. In that case the chances it helped Ann are just 1 in 2. For, of the children taking the medicine, two thirds are healthy. Half of these are healthy because of the medicine, whereas the other half would have been healthy anyway.

Put differently, the experimental data identifies the “effects of causes,” but we are interested in the reverse problem, of quantifying “causes of effects.” The causes of effects task of defining and assessing the *probability of causation* (Robins and Greenland, 1989) in an individual case has been considered by Tian and Pearl (2000); Dawid (2011); Yamamoto (2012); Pearl (2015); Dawid, Musio and Fienberg (2016); Murtas, Dawid and Musio (2017).¹ Note that this is distinct from the “reverse causal question” of Gelman and Imbens (2013), which is a collection of effects of causes questions

¹General procedures for deriving bounds on causal queries are given in Sachs, Gabriel and Sjölander (2020) though unfortunately these cannot be used for the problem considered here as our causal query is in general not linear, or, in their formulation, not a linear function of joint probabilities of response function variables.

aimed at ascertaining which causes have an effect on an outcome—the difference being that the estimand in this formulation does not condition on an observed value for the outcome.

A common approach to learning about causes of effects is to seek additional evidence along causal pathways. For example, researchers evaluating development programs specify “theories of change” and seek evidence for intermediate outcomes along a pathway linking treatment to outcomes (Ghate, 2018): Was the treatment received? Was the medicine ingested? Van Evera (1997) describes various tests that might be implemented using such ancillary evidence. A “smoking gun test” searches for evidence that, though unlikely to be found, would give great confidence in a claim if it were to be found; a “hoop test” test is a search for evidence that we expect to find, but which, if found to be absent, would provide compelling evidence against a proposition (as if the proposition were asked to jump through a hoop).

Seeing positive evidence at many points along a such a causal chain would appear to give confidence that the final outcome is indeed *due* to the conjectured cause. This is the core premise of “process tracing,” as deployed by qualitative political scientists (Collier, 2011), as well as of mixed methods research as used in development evaluation (White, 2009). In the most optimistic accounts it is assumed that, as one gets close enough to a process, by observing more and more links in a chain, the link between any two steps becomes less questionable and eventually the causal process reveals itself (Mahoney, 2012, p. 581).

Many empirical studies assume that outcomes are generated through relatively simple chains, interpreted causally. For example, Acemoglu, Johnson and Robinson (2001) propose that low early European settler mortality caused higher levels of settlement, which gave rise to institutions to protect property rights, which in turn gave rise to better economic performance. Or, African exposure to the slave trade

made groups less trusting of non co-ethnics, which weakened legal and political institutions, which in turn reduced economic growth (Nunn and Wantchekon, 2011). Such studies often seek to estimate average effects of treatments, or average effects of treatments for units that comply with an instrument's assignment. Yet there is also interest in using such general information to make inferences about individual cases: are Congo's poor development outcomes due to its colonial history, mediated by institutions? (Acemoglu, Johnson and Robinson (2001) point to the reinstatement of forced labor by Mobutu as historical evidence for this). Miguel, Satyanath and Sergenti (2004) examine whether rainfall deviations produce growth shocks that in turn increase conflict risks. But they also ask whether the conflict in Sierra Leone was plausibly due to weather shocks, mediated by economic shocks. These examples involve instrumental variables strategies with, assumptions of $Z \rightarrow X \rightarrow Y$ processes in which there is unobserved confounding between X and Y . Other studies focus on surrogacy relations in which there are still more optimistic assumptions regarding causal chains. In such studies there is interest in using an estimate of the effect of Z on X to understand the effect of Z on Y , under assumptions about the effects of X on Y (Athey et al., 2016). In such cases, the causes of effects question might be posed even before the observation of the long term outcome: if a child taking part in early childhood interventions were later to attend college, what are the chances that this would have been because of these interventions? Would knowledge of school performance help answer this question?

We here provide a comprehensive treatment of the scope for inferences of this form from knowledge of causal chains. We obtain a general formula for calculating bounds on the probability of causation, for an arbitrary pattern of data along chains of binary variables. We derive implications of this formula, and calculate the largest and smallest upper and lower bounds achievable from any causal chain consistent

with known relation between X and Y . We give special attention to what might appear to be the best possible conditions: those in which causal processes really do follow a simple causal chain, in which researchers have complete experimental evidence about the probabilistic relationship between any two consecutive nodes in the chain, in which the chain is arbitrarily long, in which the causal effect of each intermediate variable on its successor climbs to 1, and in which researchers observe outcomes consistent with positive effects at every point on the chain.

Even for this apparently promising case, our results are largely negative. Such information does indeed increase confidence that an outcome can be attributed to a cause and, for homogeneous chains at least, that the longer the chain the better. However, even under these ideal conditions our ability to narrow the bounds for the probability of causation is modest. In the example of attributing Ann’s health to good medicine, a homogeneous process with arbitrarily many positive intermediate steps observed might only tighten the bounds from $[.5, 1]$ to $[\.58, 1]$. Non-homogeneous processes can tighten the bounds more. For example, suppose Ann was prescribed the medicine and recovered. If we know that being prescribed the medicine is the only way in which Ann could have obtained and taken the medicine, and that taking the medicine helps anyone who would otherwise be sick, then with positive evidence on a single intermediate point on the causal chain—that Ann did indeed take the medicine—we can identify the probability that prescribing the medicine caused Ann’s recovery at $2/3$. (We are still short of 1, because it is possible that Ann would have recovered even without the medicine.) A process like this, in which we observe a “necessary condition for a sufficient condition”, provides the largest possible lower bound on the probability of causation available from any observations on any chain. At this point we have done the best possible and more data along the chain will not help.

Although achieving identification of the probability of causation at 1 is generally elusive, negative data can yield identification at 0, either in two steps from a heterogeneous process, or from alternating data along an infinite homogeneous chain. In this sense, information on mediators can support “hoop” tests but not “smoking gun” tests.

Existing results (Dawid, Murtas and Musio, 2016) have considered the case of a single unobserved mediator. We generalize this in two ways. First, we consider situations with chains of arbitrary length. Second, we calculate bounds for general data, that is, for situations in which the values of none, some or all the mediators are observed.

We proceed as follows. Section 2 introduces the set-up, and gives general formulae for bounding the probability of causation for a simple one-step process. In Section 3 we provide new results for cases in which all mediators are unobserved, all are observed, or just some are observed. Theorem 2 provides a general formula applicable to all cases. Then Theorem 3 details the maximum and minimum upper and lower bounds for all possible processes. In all cases these can be achieved by processes of at most two steps. In Section 4 we compare the extrema with the bounds obtained from homogeneous process, with bounds achievable when processes are known to be monotonic, and bounds obtainable from knowledge of covariates, which can be much tighter. We summarize our results, and consider some implications, in Section 5. Various technical details for the proofs in the paper are elaborated in appendices.

2 Preliminaries

Consider a binary treatment or exposure variable X , and binary outcome variable Y . We suppose we have access to experimental (or unconfounded observational) data

supplying values for $\Pr(Y = y \mid X \leftarrow x)$, where we use the notation $X \leftarrow x$ to denote a regime in which X is set to value x by external intervention.

Define

$$\begin{aligned}\tau &:= \Pr(Y = 1 \mid X \leftarrow 1) - \Pr(Y = 1 \mid X \leftarrow 0) \\ \rho &:= \Pr(Y = 1 \mid X \leftarrow 1) - \Pr(Y = 0 \mid X \leftarrow 0).\end{aligned}$$

Then τ is the *average causal effect* of X on Y , while $\rho = \Pr(Y = 1 \mid X \leftarrow 1) + \Pr(Y = 1 \mid X \leftarrow 0) - 1$ is an indicator of how common $Y = 1$ is.

The transition matrix from X to Y (where the row and column labels of any such matrix are implicitly 0 and 1 in that order) can be written:

$$P = P(\tau, \rho) := \begin{pmatrix} \frac{1}{2}(1 + \tau - \rho) & \frac{1}{2}(1 - \tau + \rho) \\ \frac{1}{2}(1 - \tau - \rho) & \frac{1}{2}(1 + \tau + \rho) \end{pmatrix}. \quad (1)$$

All entries of P must be non-negative: this holds if and only if

$$|\rho| + |\tau| \leq 1. \quad (2)$$

We have equality in (2) if and only if one of the entries of (1) is 1, in which case we term P *degenerate*. For $\tau \geq 0$, this will happen if either $\rho = 1 - \tau$, in which case $\Pr(Y = 1 \mid X = 1) = 1$ and $X = 1$ can be thought of as a sufficient condition for $Y = 1$; or $\rho = \tau - 1$, in which case $\Pr(Y = 1 \mid X = 0) = 0$, and $X = 1$ can be thought of as a necessary condition for $Y = 1$. Defining, for $\tau \geq 0$,

$$\sigma := \frac{\rho}{1 - \tau}, \quad (3)$$

we might thus regard $\sigma \in [-1, 1]$ as measuring the *relative sufficiency* of $X = 1$ for $Y = 1$.

2.1 Potential outcomes and causes of effects

While knowledge of the transition matrix P , and in particular the “average causal effect” τ , is directly relevant for studying “effects of causes,” it is not enough for analysing “causes of effects.” For this we need to introduce the pair of *potential outcomes*, $\mathbf{Y} = (Y_0, Y_1)$, where we conceive of Y_x as the value Y would take, if $X \leftarrow x$. We assume (“consistency”) that, even when X is not set by intervention, the outcome Y will be Y_X . We regard both Y_0 and Y_1 as existing simultaneously, even prior to setting the value of X , and as having a bivariate probability distribution.

We can now define the following events in terms of \mathbf{Y} (where \bar{x} denotes $1 - x$):

General causation $C^{(X,Y)} := “Y_1 \neq Y_0”$.

That is, changing the value of X will result in a change to the value of Y . We can also describe this as “ X affects Y .”

When the relevant variables (here X and Y) are clear from the context we will simplify the notation to C .

Specific causation $C_{xy}^{(X,Y)} := “Y_x = y, Y_{\bar{x}} = \bar{y}”$ (for $x, y = 0$ or 1).

That is, changing the value of X from x to \bar{x} would change the value of Y from y to \bar{y} . We can also describe this as “ $X = x$ causes $Y = y$.” When the relevant variables X and Y are clear from the context we will simplify the notation to C_{xy} .

We note that $C_{xy} = C_{\bar{x}\bar{y}}$.

Probability of Causation In cases of interest we will have observed $X = x, Y = y$, and want to know *the probability that X caused Y* , given this information. We denote this quantity by $\text{PC}_{xy}^{(X,Y)}$, or PC_{xy} when the relevant variables X and Y are clear from the context. Thus

$$\text{PC}_{xy} = \Pr(C \mid X = x, Y = y) = \Pr(C_{xy} \mid X = x, Y_x = y). \quad (4)$$

Note that, unlike for the definition of the average causal effect, the probability of causation conditions on a value for the outcome. Our PC_{11} is what [Pearl \(1999\)](#) terms the “probability of necessity”, PN, while our PC_{00} is his “probability of sufficiency”, PS.

2.2 Simple bounds

The joint distribution for \mathbf{Y} , while constrained by knowledge of the transition matrix P , is in general not fully determined by it. Rather, we can only deduce that it has the form of Table 1, where the marginal probabilities agree with (1) according to $\Pr(Y_x = y) = \Pr(Y = y \mid X \leftarrow x)$. However, the internal entries of Table 1 are not

	$Y_1 = 0$	$Y_1 = 1$	
$Y_0 = 0$	$\frac{1}{2}(1 - \rho - \xi)$	$\frac{1}{2}(\xi + \tau)$	$\frac{1}{2}(1 + \tau - \rho)$
$Y_0 = 1$	$\frac{1}{2}(\xi - \tau)$	$\frac{1}{2}(1 + \rho - \xi)$	$\frac{1}{2}(1 - \tau + \rho)$
	$\frac{1}{2}(1 - \tau - \rho)$	$\frac{1}{2}(1 + \tau + \rho)$	1

Table 1: $\Pr(Y_0 = y_0, Y_1 = y_1)$

determined by P , but have one degree of freedom, expressed by the “slack” quantity $\xi = \xi(P)$. We see that

$$\xi = \Pr(Y_0 = 0, Y_1 = 1) + \Pr(Y_0 = 1, Y_1 = 0) = \Pr(C), \quad (5)$$

the probability of general causation.

The only constraints on ξ are that all internal entries of Table 1 must be non-negative, which holds if and only if

$$|\tau| \leq \xi \leq 1 - |\rho|. \quad (6)$$

In particular ξ , and thus the bivariate distribution of (Y_0, Y_1) in Table 1, is uniquely determined by P if and only if P is degenerate.

We further note

$$\Pr(C_{00}) = \Pr(C_{11}) = \frac{1}{2}(\xi + \tau) \quad (7)$$

$$\Pr(C_{01}) = \Pr(C_{10}) = \frac{1}{2}(\xi - \tau) \quad (8)$$

whence, by (6),

$$\max\{0, \tau\} \leq \Pr(C_{00}) = \Pr(C_{11}) \leq \frac{1}{2}(1 + \tau - |\rho|) \quad (9)$$

$$\max\{0, -\tau\} \leq \Pr(C_{01}) = \Pr(C_{10}) \leq \frac{1}{2}(1 - \tau - |\rho|). \quad (10)$$

Throughout this article we shall assume *no confounding*, expressed mathematically as $X \perp\!\!\!\perp \mathbf{Y}$. Then

$$\begin{aligned} \text{PC}_{xy} &= \frac{\Pr(C_{xy})}{\Pr(Y_x = y)} \\ &= \frac{\Pr(C_{xy})}{\Pr(Y = y \mid X \leftarrow x)} \end{aligned}$$

which is thus subject to the interval bounds, given by (9) or (10), as appropriate, divided by the known entry $\Pr(Y = y \mid X \leftarrow x)$ of the transition matrix P .

This analysis delivers the following lower and upper bounds (superscript “ s ” for “simple”):

$$L_{00}^s := \frac{\max\{0, \tau\}}{\Pr(Y = 0 \mid X \leftarrow 0)} \leq \text{PC}_{00} \leq \frac{\frac{1}{2}(\tau + 1 - |\rho|)}{\Pr(Y = 0 \mid X \leftarrow 0)} =: U_{00}^s \quad (11)$$

$$L_{10}^s := \frac{\max\{0, -\tau\}}{\Pr(Y = 0 \mid X \leftarrow 1)} \leq \text{PC}_{10} \leq \frac{\frac{1}{2}(1 - |\rho| - \tau)}{\Pr(Y = 0 \mid X \leftarrow 1)} =: U_{10}^s \quad (12)$$

$$L_{01}^s := \frac{\max\{0, -\tau\}}{\Pr(Y = 1 \mid X \leftarrow 0)} \leq \text{PC}_{01} \leq \frac{\frac{1}{2}(1 - |\rho| - \tau)}{\Pr(Y = 1 \mid X \leftarrow 0)} =: U_{01}^s \quad (13)$$

$$L_{11}^s := \frac{\max\{0, \tau\}}{\Pr(Y = 1 \mid X \leftarrow 1)} \leq \text{PC}_{11} \leq \frac{\frac{1}{2}(\tau + 1 - |\rho|)}{\Pr(Y = 1 \mid X \leftarrow 1)} =: U_{11}^s. \quad (14)$$

In the absence of additional information, the above bounds constitute the best available inference regarding the probability of causation.

Specifically, when $\tau \geq 0$, on defining

$$\gamma := \frac{1 - \tau - |\rho|}{1 - \tau + |\rho|} = \frac{1 - |\sigma|}{1 + |\sigma|} \quad (15)$$

$$\delta := \frac{1 + \tau - |\rho|}{1 + \tau + |\rho|} \quad (16)$$

we have the upper bounds given in Table 2.

	$\rho \geq 0$	$\rho < 0$
U_{00}^s	1	δ
U_{01}^s	γ	1
U_{10}^s	1	γ
U_{11}^s	δ	1

Table 2: U_{xy}^s denotes the upper bound on the probability that $X = x$ caused $Y = y$ in a one-step process.

A particular interest is in cases where $\tau > 0$ (so the overall effect of X and Y is positive) and we observe positive outcomes, $X = 1, Y = 1$. In this case we omit the

subscript 11. We have

$$\text{PC} = \frac{\xi + \tau}{2 \Pr(Y = 1 \mid X \leftarrow 1)}, \quad (17)$$

and interval bounds given by

$$L^s = \frac{2\tau}{1 + \tau + \rho} \leq \text{PC} \leq U^s = \begin{cases} \delta & (\rho \geq 0) \\ 1 & (\rho < 0). \end{cases} \quad (18)$$

This result agrees with [Tian and Pearl \(2000\)](#) and [Dawid \(2011\)](#).

PC is identified (*i.e.*, the interval in (18) reduces to a single point) if and only if $|\rho| = 1 - \tau$, which holds when P is degenerate with either the lower left or upper right element of P being 0. In the former case $\text{PC} = \tau$, while in the latter case $\text{PC} = 1$.

More generally, we have $L^s = \tau / \Pr(Y = 1 \mid X \leftarrow 1) \geq \tau$, and so $\text{PC} \geq \tau$.

3 Bounds from mediation

We now suppose that, in addition to X and Y , we can gather data on one or more binary mediator variables M_1, \dots, M_{n-1} . We also define $M_0 \equiv X$ and $M_n \equiv Y$. We are interested in assessing the probability that $X = x$ caused $Y = y$ for a new case where we have information on the values of some or all of the mediators M_1, \dots, M_{n-1} .

We assume that the data are based on experiments, or in any case are such as to allow us to determine the one-step interventional probabilities $\Pr(M_{i+1} = m_{i+1} \mid M_i \leftarrow m_i)$, $i = 0, \dots, n-1$. We shall here confine attention to the case of a *complete mediation sequence*, where

$$\Pr(M_{i+1} = m_{i+1} \mid M_j \leftarrow m_j, j = 0, \dots, i) = \Pr(M_{i+1} = m_{i+1} \mid M_i \leftarrow m_i), \quad (i = 0, \dots, n-1).$$

We shall further suppose that, for any new case considered, there is no confounding at every step, so that

$$\Pr(M_{i+1} = m_{i+1} \mid M_j = m_j, j = 0, \dots, i) = \Pr(M_{i+1} = m_{i+1} \mid M_i \leftarrow m_i), \quad (i = 0, \dots, n-1).$$

In this case the sequence of observations ($X \equiv M_0, \dots, M_n \equiv Y$) on a new case will form a (generally non-stationary) Markov chain. This is an empirically testable consequence of our assumptions, assumptions which would therefore be falsified if the Markov property is found to fail (although those assumptions are not guaranteed to be valid when it is found to hold.)

Let the transition matrix from M_{i-1} to M_i be $P_i = P(\tau_i, \rho_i)$, and the overall transition matrix from X to Y be $P = P(\tau, \rho)$. We shall write

$$P = P_1 \mid P_2 \dots \mid P_n \tag{19}$$

to indicate that we are assuming the above mediation sequence, and refer to (19) as a *decomposition* of the matrix P . In particular we then have $P = P^{(n)} := \prod_{i=1}^n P_i$.

We can readily show by induction that

$$\tau = \tau^{(n)} := \prod_{i=1}^n \tau_i \tag{20}$$

$$\rho = \rho^{(n)} := \sum_{i=1}^n \left(\rho_i \prod_{j=i+1}^n \tau_j \right). \tag{21}$$

In particular, for the case $n = 2$, (21) becomes

$$\rho = \rho_1 \tau_2 + \rho_2. \tag{22}$$

On account of (20) we have the following result:

Lemma 1 *The average causal effect of X on Y is the product of the successive average causal effects of each variable in the sequence on the following one.*

Again, to conduct causes of effects analysis, we introduce, for $i \geq 1$, bivariate variables

$$\mathbf{M}_i := (M_{i0}, M_{i1})$$

where M_{im} denotes the potential value of M_i under $M_{i-1} \leftarrow m$, supposed unaffected by values of previous M 's. We further assume that the variable \mathbf{M}_i is common to all the various worlds, whether actual or counterfactual, under consideration. The actually realised values (M_i) satisfy $M_i = M_{i,M_{i-1}}$.

As the expression of our “no confounding” assumptions, we impose mutual independence between $X, \mathbf{M}_1, \dots, \mathbf{M}_n$.

Lemma 2 $C^{(X,Y)} = \bigcap_{i=0}^{n-1} C^{(M_i, M_{i+1})}$. *That is to say, $M_0 \equiv X$ affects $M_n \equiv Y$ if and only if each M_i affects the next.*

Proof. Suppose first that each variable affects the next. Then changing the value of X will change that of M_1 , which in turn will change that of M_2 , and so on until the value of Y is changed, so showing that X affects Y . Conversely, if, for some $j < n$, M_j does not affect M_{j+1} , then, whether or not M_j has been changed, the value of M_{j+1} will be unchanged, whence so too will that of M_{j+2} , and so on until the value of Y is unchanged, whence X does not affect Y . \square

Corollary 1

(i). $\Pr(C^{(X,Y)}) = \prod_{i=1}^n \Pr(C^{(M_{i-1}, M_1)})$

(ii). $\xi(P) = \prod_{i=1}^n \xi(P_i)$

(iii). Given knowledge of the decomposition (19), the constraints on $\xi = \xi(P)$ are now:

$$|\tau| \leq \xi \leq \prod_{i=1}^n (1 - |\rho_i|). \quad (23)$$

Proof.

(i) By the assumed mutual independence of the (M_i) .

(ii) By (5).

(iii) By (ii), (6) for each P_i , and (20).

□

On account of (i) we have:

Corollary 2 *For any decomposition, the probability that X affects Y is the product of the probabilities that each variable in the sequence from X to Y affects the next in the sequence.*

On comparing (23) with (6), we see that detailed knowledge of the mediation process has not changed the lower bound for ξ . However, the upper bound is typically reduced:

Theorem 1 *The upper bound that results from knowledge of the decomposition of P is no greater than the upper bound that results from P alone. It will be strictly less if for some $i > 1$, P_i is non-degenerate and $\rho_{i-1} \neq 0$.*

Proof. We compare the upper bound of (23) with that of (6).

Consider first the case $n = 2$. Then

$$\begin{aligned} |\rho| &= |\rho_1\tau_2 + \rho_2| \quad \text{by (22)} \\ &\leq |\rho_1||\tau_2| + |\rho_2| \end{aligned} \tag{24}$$

$$\leq |\rho_1|(1 - |\rho_2|) + |\rho_2| \quad \text{by (2)}. \tag{25}$$

It follows that

$$(1 - |\rho_1|)(1 - |\rho_2|) \leq 1 - |\rho|. \tag{26}$$

Moreover, we shall have strict inequality in (25), and hence also in (26), if P_2 is non-degenerate and $\rho_1 \neq 0$, since these together imply $|\rho_1|(1 - |\rho_2|) < |\rho_1||\tau_2|$.

Noting that if $(1 - |\rho_1|)(1 - |\rho_2|) = 1 - |\rho|$ then $\rho_2 \neq 0$ implies $\rho \neq 0$, the result for general n follows by induction.

□

We note that the above condition for strict inequality in (26), while sufficient, is not necessary. For example, in the case $n = 2$ it will also hold if $\rho_1\tau_2$ and ρ_2 have different signs, since then we would have strict inequality in (24).

It follows from (23) and (26) that collapsing two mediators into a single one can only increase the upper bound for ξ :

Corollary 3 *Consider two decompositions $P = P_1 | P_2 \dots | P_n$ and $P = P_1 | \dots | P_i | Q | P_{i+2} | \dots | P_n$, where $Q = P_i P_{i+1}$. Then the upper bound for ξ for the former does not exceed that for the latter.*

3.1 Unobserved mediators

Suppose first that, for the new case, we have observed $X = x, Y = y$, but the values of the mediators are not observed. Even in this case, as shown for the two-term decomposition in Dawid, Murtas and Musio (2016), knowledge of the decomposition (19) of P can alter the bounds for PC.

Indeed, in this case (4) still applies, where $\Pr(C_{xy})$ is given by (7) or (8) as appropriate, but now with ξ subject to the revised bounds of (23). In each case the lower bound is unaffected, but, by Theorem 1, the upper bound is reduced.

This analysis delivers the following revised bounds (superscript “ \emptyset ” for “not observed”):

$$L_{00}^{\emptyset} := L_{00}^s = \frac{\max\{0, \tau\}}{\Pr(Y = 0 \mid X \leftarrow 0)} \leq \text{PC}_{00} \leq \frac{\tau + \prod_{i=1}^n (1 - |\rho_i|)}{2 \Pr(Y = 0 \mid X \leftarrow 0)} =: U_{00}^{\emptyset} \quad (27)$$

$$L_{10}^{\emptyset} := L_{10}^s = \frac{\max\{0, -\tau\}}{\Pr(Y = 0 \mid X \leftarrow 1)} \leq \text{PC}_{10} \leq \frac{\prod_{i=1}^n (1 - |\rho_i|) - \tau}{2 \Pr(Y = 0 \mid X \leftarrow 1)} =: U_{10}^{\emptyset} \quad (28)$$

$$L_{01}^{\emptyset} := L_{01}^s = \frac{\max\{0, -\tau\}}{\Pr(Y = 1 \mid X \leftarrow 0)} \leq \text{PC}_{01} \leq \frac{\prod_{i=1}^n (1 - |\rho_i|) - \tau}{2 \Pr(Y = 1 \mid X \leftarrow 0)} =: U_{01}^{\emptyset} \quad (29)$$

$$L_{11}^{\emptyset} := L_{11}^s = \frac{\max\{0, \tau\}}{\Pr(Y = 1 \mid X \leftarrow 1)} \leq \text{PC}_{11} \leq \frac{\tau + \prod_{i=1}^n (1 - |\rho_i|)}{2 \Pr(Y = 1 \mid X \leftarrow 1)} =: U_{11}^{\emptyset} \quad (30)$$

Note in particular, for the case $\tau > 0$, where we observe $X = 1, Y = 1$ (but the values of mediators are not observed), we have revised bounds

$$L^{\emptyset} := \frac{2\tau}{1 + \tau + \rho} \leq \text{PC} \leq \frac{\tau + \prod_{i=1}^n (1 - |\rho_i|)}{1 + \tau + \rho} =: U^{\emptyset}. \quad (31)$$

For $n = 2$ this agrees with the analysis of Dawid, Murtas and Musio (2016).

3.2 Bounds when some or all mediators are observed

Now suppose that, in addition to $X = x, Y = y$, we also observe data on k mediators ($0 \leq k \leq n - 1$) for the new case. In particular we observe $M_{i_r} = m_{i_r}$, for $0 < i_1 < \dots < i_r \dots < i_k < n$. For notational simplicity we write \widetilde{M}_r for M_{i_r} , \widetilde{m}_r for m_{i_r} . We also identify $\widetilde{M}_0 \equiv X$ and $\widetilde{M}_{k+1} \equiv Y$ (so $\widetilde{m}_0 = x, \widetilde{m}_{k+1} = y$).

The relevant probability of causation is now

$$\widetilde{\text{PC}}_{xy} := \Pr \left(C \mid \widetilde{M}_r = \widetilde{m}_r, i = 0, \dots, k + 1 \right).$$

Note that in contrast to the difference between (27)–(30) on the one hand and (11)–(14) on the other hand, which relate to the same quantity PC_{xy} but express different conclusions about it (since based on different external evidence), $\widetilde{\text{PC}}_{xy}$ is a genuinely different quantity from PC_{xy} , as it conditions on different information about the case in question.

Theorem 2 *Given observations on $X, \widetilde{M}_1, \dots, \widetilde{M}_k, Y$, the probability that X caused Y is given by the product of the probabilities that each observed term in the sequence caused the next observed term:*

$$\widetilde{\text{PC}}_{xy} = \prod_{r=0}^k \text{PC}_{\widetilde{m}_r, \widetilde{m}_{r+1}}^{(\widetilde{M}_r, \widetilde{M}_{r+1})}.$$

Proof. From Lemma 2 we have

$$C = \bigcap_{r=0}^k C^{(\widetilde{M}_r, \widetilde{M}_{r+1})},$$

whence, using the “no-confounding” independence properties,

$$\begin{aligned}
\widetilde{\text{PC}}_{xy} &= \prod_{r=0}^k \Pr \left(C^{(\widetilde{M}_r, \widetilde{M}_{r+1})} \mid \widetilde{M}_r = \widetilde{m}_r, \widetilde{M}_{r+1} = \widetilde{m}_{r+1} \right) \\
&= \prod_{r=0}^k \text{PC}_{\widetilde{m}_r \widetilde{m}_{r+1}}^{(\widetilde{M}_r, \widetilde{M}_{r+1})}.
\end{aligned} \tag{32}$$

□

Now since we have the decomposition information about the mediators (if any) occurring between $\widetilde{M}_r \equiv M_{i_r}$ and $\widetilde{M}_{r+1} \equiv M_{i_{r+1}}$, but not their values for the new case, the bounds on any factor in (32) will, *mutatis mutandis*, have the form of the relevant expressions for L_{xy}^\varnothing and U_{xy}^\varnothing , as displayed in (27)–(30). Then the overall lower [resp., upper] bound on $\widetilde{\text{PC}}_{xy}$ will be the product of these lower [resp., upper] bounds, across all terms. This procedure supplies a complete recipe for determining the appropriate bounds on $\widetilde{\text{PC}}_{xy}$ in the knowledge of the full decomposition of P and the values of the observed mediators for the new case.

Again consider the special case with $\tau > 0$, $X = Y = 1$. On account of (20) we can, after possibly switching the labels 0 and 1 for some of the M_i 's, take $\tau_i > 0$, all i . We assume henceforth that this is the case. The above procedure then delivers lower bound 0 unless $\widetilde{m}_i = \widetilde{m}_{i-1}$, all i , so that $m_i = 1$, all i . In that case we obtain lower bound (with superscript + for “positive mediators”):

$$\begin{aligned}
L^+ &:= \frac{\tau}{\prod_{r=0}^k \Pr \left(\widetilde{M}_{r+1} = 1 \mid \widetilde{M}_r = 1 \right)} \\
&= \frac{\tau}{\Pr \left(Y = 1, \widetilde{M}_r = \widetilde{m}_r, r = 2, \dots, k \mid X = 1 \right)}.
\end{aligned} \tag{33}$$

It is easy to see that this lower bound can only increase if we introduce further

observed mediators. It follows that the smallest lower bound occurs when there are no observed mediators, when it reduces to $L_{\underline{=}}^{\emptyset} L^s$ as in (31) and (18); while the largest lower bound occurs when all mediators are observed (all taking value 1)—that is to say, there is positive evidence for every link in the mediation chain.

In the remainder of this paper we shall give special attention to this case, and write simply $\widetilde{\text{PC}}$ for $\widetilde{\text{PC}}_{11}$, *etc.* The bounds for $\widetilde{\text{PC}}$ are then:

$$L^+ := \prod_{i=1}^n \left(\frac{2\tau_i}{1 + \tau_i + \rho_i} \right) \leq \widetilde{\text{PC}} \leq \prod_{i=1}^n \left(\frac{1 + \tau_i - |\rho_i|}{1 + \tau_i + \rho_i} \right) =: U^+. \quad (34)$$

The following result follows directly from the above considerations:

Lemma 3 *The lower bound L^+ of (34) is at least as large as the lower bound L^s of (18).*

However, it will follow from Theorem 3 below that U^+ can be smaller or larger than U^s .

3.3 Largest and smallest upper and lower bounds

Equation (32) provides a general formula for calculating bounds on the probability of causation for any pattern of data observed on mediating variables (including no data). We now use this result to assess the largest and smallest possible upper bounds from observation of possible values on mediating variables.

Consider an arbitrary decomposition of P :

$$P = P_1 | P_2 | \dots | P_n, \quad (35)$$

with $P = P(\tau, \rho)$, $P_i = P(\tau_i, \rho_i)$. We restrict attention to the case $\tau > 0$ and assume that variables are labeled so that each $\tau_i > 0$.

We investigate the smallest and largest achievable values for $L^\emptyset, U^\emptyset, L^+, U^+, L^-, U^-$ (superscript $-$ for some negative evidence) and show that in each case these are achievable by decompositions involving at most one mediator.

Theorem 3 Consider transition matrix $P = P(\tau, \rho)$ from X to Y with $\tau > 0$ and $|\rho| < 1 - \tau$. The largest and smallest upper and lower bounds on the probability that $X = 1$ caused $Y = 1$ from any complete mediation process for (a) the case with mediators unobserved (b) the case with positive outcomes on all mediators observed and (c) cases that include some negative evidence on the mediators, are as given in Table 3. These can all be achieved by decompositions of length 1 or 2.

		No evidence	Positive evidence	Some negative evidence
Largest	Upper	$\overline{U^\emptyset} = \frac{1+\tau- \rho }{1+\tau+\rho}$	$\overline{U^+} = \min\{1, 1 - \rho\}$	$\overline{U^-} = 1$
	Lower	$\underline{L^\emptyset} = \frac{2\tau}{1+\tau+\rho}$	$\underline{L^+} = \frac{1+\tau-\rho}{2}$	$\underline{L^-} = 0$
Smallest	Upper	$\underline{U^\emptyset} = \frac{2\tau}{1+\tau+\rho}$ (*)	$\underline{U^+} = \frac{2\tau}{1+\tau+\rho}$ (*)	$\underline{U^-} = 0$ (*)
	Lower	$\underline{L^\emptyset} = \frac{2\tau}{1+\tau+\rho}$	$\underline{L^+} = \frac{2\tau}{1+\tau+\rho}$	$\underline{L^-} = 0$

Table 3: Largest and smallest achievable upper and lower bounds from decompositions of any length, given no mediators observed (L^\emptyset, U^\emptyset), positive evidence observed for all mediators (L^+, U^+), or when some negative evidence is observed (L^-, U^-). (*) Indicates that PC can be identified.

Proof. See Appendix A. □

The largest upper bound with mediators unobserved, $\overline{U^\emptyset}$, can be achieved without any mediators. Since unobserved mediators do not alter the lower bound we have $\underline{L^\emptyset} = \underline{L^\emptyset} = L^s$. In addition we have $\underline{U^\emptyset} = L^s$, which is achievable, for example, from

the following decomposition:

$$P = \left(\begin{array}{cc} \frac{2\tau}{1+\tau+\rho} & \frac{1-\tau+\rho}{1+\tau+\rho} \\ 0 & 1 \end{array} \right) \left| \left(\begin{array}{cc} 1 & 0 \\ \frac{1-\tau-\rho}{2} & \frac{1+\tau+\rho}{2} \end{array} \right). \quad (36)$$

Note that with this decomposition PC is identified *via* two degenerate transition matrices: $X = 1$ is a sufficient condition for $M = 1$, while $M = 1$ is a necessary condition for $Y = 1$.

The smallest upper and lower bounds available when mediators are observed agree with the simple lower bound. Positive evidence cannot reduce the lower bound, but it can reduce the upper bound to the lower bound, at which point $\widetilde{\text{PC}}$ is identified. This can be achieved by the same decomposition given in (36).

The largest upper bound with positive evidence on mediators, $\overline{U^+}$, can exceed the simple upper bound when $\rho > 0$. It is achieved by the following two-term decomposition, involving a single mediator:

$$P = \left(\begin{array}{cc} \frac{1-\rho+\tau}{2(1-\rho)} & \frac{1-\rho-\tau}{2(1-\rho)} \\ \frac{1-\rho-\tau}{2(1-\rho)} & \frac{1-\rho+\tau}{2(1-\rho)} \end{array} \right) \left| \left(\begin{array}{cc} 1-\rho & \rho \\ 0 & 1 \end{array} \right). \quad (37)$$

The lower bound can be raised with positive information on mediators, and takes its largest value with the following degenerate two-term decomposition $P = P_1 | P_2$, involving a single mediator:

$$P = \left(\begin{array}{cc} 1 & 0 \\ \frac{1-\tau-\rho}{1+\tau-\rho} & \frac{2\tau}{1+\tau-\rho} \end{array} \right) \left| \left(\begin{array}{cc} \frac{1+\tau-\rho}{2} & \frac{1-\tau+\rho}{2} \\ 0 & 1 \end{array} \right). \quad (38)$$

With this decomposition $\widetilde{\text{PC}}$ is identified *via* two degenerate transition matrices: in this case $X = 1$ is a necessary condition for $M = 1$, while $M = 1$ is a sufficient

condition for $Y = 1$. The largest lower bound with positive evidence from this decomposition is $\frac{1+\tau-\rho}{2}$ which can fall far short of 1, implying that in general mediators cannot provide “smoking gun” evidence that $X = 1$ caused $Y = 1$. A benchmark of 50%—a balance of probabilities—is sometimes used (for example in civil legal proceedings) as the standard of proof. This result shows that this standard cannot be met by any information on mediators if $\tau < \rho$, or equivalently, if $\Pr(Y = 1|X = 0) > 0.5$.

For the case with some negative evidence on the mediators the lower bound is always 0. The smallest upper bound is also 0, which can be achieved by the decomposition (38) above, with the single mediator observed at 0 (the key feature of this decomposition is that $Y = 1$ can not be caused by $M = 0$). In this case $\widetilde{\text{PC}}$ is identified at 0, showing that it is possible for negative data on mediators to provide “hoop” evidence that $X = 1$ did not cause $Y = 1$. The highest upper bound when there is some negative evidence, $U^- = 1$, can be achieved by a two-step decomposition $P(\tau, \rho) = P(\tau_1, \rho_1) | P(\tau_2, \rho_2)$, with the mediator taking value 0. For $\rho \leq 0$ this occurs with the decomposition with parameters:

$$\tau_1 = \frac{2\tau}{1 + \tau + \rho} \quad \rho_1 = 0 \quad \tau_2 = \frac{1 + \tau + \rho}{2} \quad \rho_2 = \rho. \quad (39)$$

For $\rho \geq 0$ it occurs with decomposition parametrized by:

$$\tau_1 = \frac{\tau(1 + \rho + \tau)}{2(\tau + \rho)} \quad \rho_1 = \frac{\rho(1 + \rho + \tau)}{2(\tau + \rho)} \quad \tau_2 = \frac{2(\tau + \rho)}{1 + \tau + \rho} \quad \rho_2 = 0. \quad (40)$$

4 Comparisons

Although knowledge of mediators can narrow bounds, the scope for learning from knowledge of mediation processes—and the specific values taken on by mediators—is often small. In particular, although negative evidence can yield low upper bounds, providing confidence that an outcome was *not* due to a putative cause, positive evidence generally does not raise lower bounds substantially.

To put these claims in context, we compare the extrema on bounds in Theorem 3 with bounds that can be achieved from homogeneous processes, from knowledge of monotonicity, and from covariate information.

Homogeneous processes. First we consider bounds for a special case: long homogeneous processes—that is, cases in which we have a potentially unlimited sequence of variables directly mediating between X and Y , with one-step transition matrices that are identical at each step (and having positive average causal effect).

The extrema on the bounds given in Theorem 3 are generally achieved through 2-step processes in which transition matrix P_1 is different from transition matrix P_2 . The bounds from long homogeneous processes can be no better than those described in Theorem 3, but how different are they?

Table 4 shows the upper and lower bounds achievable with homogeneous processes of unbounded length, for three cases: in which there are no data on the values of the mediators; in which all mediators are observed and positive ($M_t \equiv 1$); and in which values alternate 1010... For further details, see Appendix B.

We see that, for $\rho \neq 0$, with alternating evidence identification can be achieved in the limit, at 0. In other cases, however, identification is not achieved. In particular the lower bound with positive evidence can fall far short of the highest possible lower

	No evidence	Positive evidence	Alternating evidence
Upper	$U_{\infty}^{\emptyset} = \frac{\tau + \tau^{ \sigma }}{1 + \tau + \rho}$	$U_{\infty}^{+} = \min \{1, \tau^{\sigma}\}$	$U_{\infty}^{-} = \begin{cases} 0 & \text{if } \rho \neq 0 \\ 1 & \text{if } \rho = 0 \end{cases}$
Lower	$L_{\infty}^{\emptyset} = \frac{2\tau}{1 + \tau + \rho}$	$L_{\infty}^{+} = \tau^{\frac{1}{2}(1 + \sigma)}$	$L_{\infty}^{-} = 0$

Table 4: Upper and lower bounds from homogeneous decompositions of length $n \rightarrow \infty$, given no mediators observed, positive evidence observed for all mediators, and alternating evidence.

bound, especially when $|\rho|$ and τ are small. For example, if $\rho = 0$ then $\overline{L}^{+} - L_{\infty}^{+} = \frac{1}{2}(1 - \sqrt{\tau})^2$.

Monotonicity. Suppose that we knew that there are no cases for which the exposure would prevent the outcome, *i.e.*, such that $Y_0 = 1, Y_1 = 0$. We note that since monotonicity is an attribute of the typically unidentifiable joint distribution of (Y_0, Y_1) , it is not easy to justify without additional knowledge. One case where this is possible is when we know of the existence of a mediation process with decomposition (36).

From Table 1 we have that monotonicity implies $\xi = \tau$, that is, ξ is identified at its lower limit. In turn this implies that PC, given by (17), is identified at its lower limit, $L^s = 2\tau/(1 + \tau + \rho)$. In this case, knowledge of the value of mediators does nothing to raise the lower bound.

Observed covariate. Suppose that, in addition to X and Y , we can observe a binary covariate C , which can affect the dependence of Y on X . Let $\pi = \Pr(C = 1)$, and let P_i be the transition matrix from X to Y , conditional on $C = i$; for consistency with the known $P = P(\tau, \rho)$ we must have $P = \pi P_1 + (1 - \pi)P_0$.

In particular, it could then be the case that $\pi = (1 + \tau - \rho)/2$, and

$$P_1 = \begin{pmatrix} 1 & 0 \\ \frac{1-\tau-\rho}{2} & \frac{1+\tau+\rho}{2} \end{pmatrix} \quad P_0 = \begin{pmatrix} 0 & 1 \\ \frac{1-\tau-\rho}{2} & \frac{1+\tau+\rho}{2} \end{pmatrix}.$$

In this case knowledge that an individual with $X = Y = 1$ also has $C = 1$ is enough to identify PC at 1.

Unobserved covariate. As shown in Dawid (2011), knowledge of covariates can improve bounds, even if their values are not observed for the case at hand. In particular, this can let us identify PC at the upper bound, $U^s = \min\{1, \frac{1+\tau-\rho}{1+\tau+\rho}\}$. For this to be possible, however, the average treatment effect must be negative for some value of C . Thus suppose $\pi = \frac{1+\tau+\rho}{2}$, and the conditional transition matrices are:

For $\rho < 0$,

$$P_1 = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \quad P_0 = \begin{pmatrix} \frac{-2\rho}{1-\tau-\rho} & \frac{1+\tau-\rho}{1-\tau-\rho} \\ 1 & 0 \end{pmatrix}.$$

For $\rho \geq 0$,

$$P_1 = \begin{pmatrix} \frac{1+\tau-\rho}{1+\tau+\rho} & \frac{2\rho}{1+\tau+\rho} \\ 0 & 1 \end{pmatrix} \quad P_0 = \begin{pmatrix} 0 & 1 \\ 1 & 0 \end{pmatrix}.$$

In either case, knowledge that $X = Y = 1$ is sufficient to infer that $C = 1$. This identifies the probability of causation: PC = 1 for $\rho < 0$, PC = $\frac{1+\tau-\rho}{1+\tau+\rho}$ for $\rho \geq 0$. In both cases we hit the upper bound.

Figure 1 compares the bounds obtained, under various assumptions, for a range of values of τ and ρ . It illustrates how, in general, lower bounds rise with τ and fall with ρ . For homogeneous processes the lower bounds improve on the simple bounds, although the gain from unlimited steps is not a striking improvement on that for

just two steps. The gains from non-homogeneous decompositions can be substantial. The best lower bounds achievable from knowledge of covariates are higher than lower bounds achieved from any knowledge of mediators.

5 Conclusion

We provide a general formula for calculating bounds on the probability of causation for complete mediation processes involving binary variable of arbitrary length and with arbitrary data patterns. In addition we characterize the largest and smallest achievable bounds obtainable from any data. Knowledge of these bounds is useful for assessing when there can be gains from learning about processes in a population and from gains from learning about the values of mediators for cases.

Our analysis focuses on ideal cases in which there is a very simple known causal structure. We show, however, that even in these ideal conditions, access to even unlimited data on mediators has only a modest, and asymmetric, impact on inferences. Knowledge of mediation processes, and of positive values for some mediators in a particular case, can raise the lower bound on the probability of causation, thus providing some evidence against a skeptic who doubts that the outcome in the case can be attributed to the putative cause. Moreover this information can be enough to achieve identification. However, the gains are generally modest and may not be sufficient to convince a skeptic. For instance, if most outcomes are positive for untreated units, then it follows from our results that there is no evidence on mediators for a treated unit with positive outcomes that can raise the lower bound on the probability that the outcome was due to the treatment above 50%. More generally identification at 1 is not possible. In contrast, for some processes, observing negative evidence on a single mediator can effectively convince a skeptic that the outcome is *not* due to the

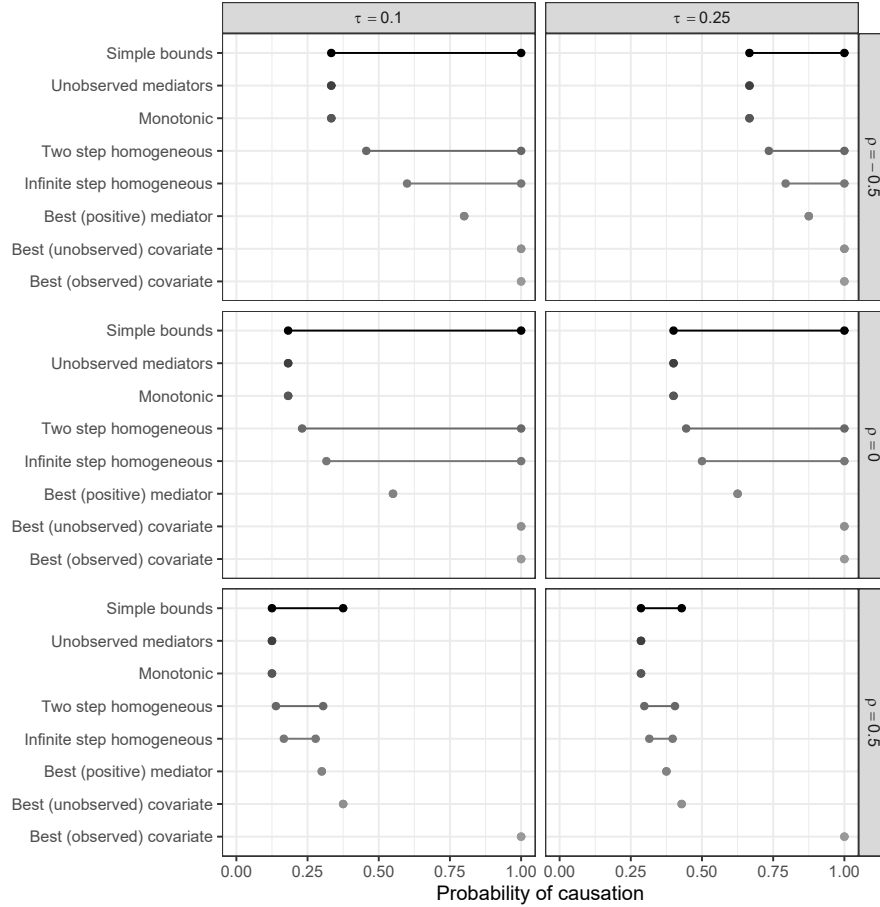


Figure 1: Comparison of bounds on PC. Simple bounds are derived from the distribution of Y given X and are given by (18). Tightest bounds from unobserved mediators are given by the decomposition in (36). Monotonicity implies the same bounds. Bounds from a homogeneous two-step decomposition and positive evidence can be calculated from Theorem 3. Infinite-step bounds, assuming positive evidence observed at every step from a homogeneous process, are given in Table 4. Best two-step bounds show the highest lower bound achievable from information on mediation shown in Table 3 and can be achieved with positive evidence for the decomposition of (38). Greatest lower bounds given information on an unobserved and observed binary covariate are as described in § 4.

exposure.

These general results have implications for when gathering further intermediate data on particular cases can be useful. We see, for instance, starkly contrasting implications for a process in which X is a necessary condition for a sufficient condition for Y and a process in which X is a sufficient condition for a necessary condition for Y . In the first case, positive evidence on mediators yields the largest possible upper bound, and identifies the probability of causation. For example, if it is known that the effect of delivering a deworming medicine passes uniquely through ingestion, and ingestion is sufficient for effective deworming, then evidence of ingestion raises the lower bound and identifies the probability of causation. In contrast for a process in which X is a sufficient condition for a necessary condition for Y , we already enjoy identification and there is no gain from gathering data on the mediator. For instance if ingesting medicine is a sufficient condition for good health, and good health is a necessary condition for good school performance, then observing ingestion and good school performance is sufficient to achieve identification. There are no additional gains from measuring health, since good health is already implied by good performance.

Finally, these results also have implications for the potential gains from research agendas that seek to learn about mediation processes (as for example in the designs described in [Imai, Keele and Tingley \(2010\)](#)) compared to the potential gains from learning about effect heterogeneity (as for example is done in factorial designs, ([Fisher, 1992](#))). The scope for gains from knowledge of mediation processes are typically weaker than potential gains from knowledge of conditions under which interventions are more or less effective. While of course the actual gains from knowledge of mediators and covariates depends on underlying causal relations, by providing extrema on bounds, the results we provide can inform the choice of experimental design.

Online Appendix

Appendix A Proof of Theorem 2

A.1 Mediators unobserved

Lower bounds: L^\varnothing is unchanged by knowledge of the mediation process alone and

$$\text{so } \underline{L^\varnothing} = \overline{L^\varnothing} = L^s.$$

Smallest upper bound: From (31) we can see that for a degenerate two-term decomposition with $|\rho_1| = 1 - \tau_1$ and $|\rho_2| = 1 - \tau_2$, $U^\varnothing = L^\varnothing = L^s$. In this case PC is identified.

Largest upper bound: It follows from Corollary 3 and (17) that this is achieved when there are no mediators, and so $\overline{U^\varnothing} = U^s$.

A.2 Positive data observed at every step

We now consider the case where mediators are observed. Then, for the decomposition (35),

$$\begin{aligned} P &= P_1 \times P_2 \times \dots \times P_n \\ \widetilde{\text{PC}} &= \prod_{i=1}^n \text{PC}_i \\ L^+ &= \prod_{i=1}^n L_i^s \\ U^+ &= \prod_{i=1}^n U_i^s \end{aligned}$$

Smallest lower bound

It follows from Lemma 3 that the smallest achievable lower bound is

$$L^s = 2\tau/(1 + \tau + \rho),$$

which does not require any mediators.

Smallest upper bound

Trivially we must have $U^+ \geq L^+ \geq L^s$.

Note now that the decomposition (36) identifies $\widetilde{PC} = L^s$, whence in particular $U^+ = L^s$, the smallest possible value.

Largest lower bound

Lemma 4

$$\frac{2\tau}{1 + \tau + \rho} \leq \frac{1 + \tau - \rho}{2}.$$

Proof. This holds since

$$(1 + \tau + \rho)(1 + \tau - \rho) - 4\tau = (1 - \tau)^2 - \rho^2 \geq 0.$$

□

Lemma 5 *Let $P = P_1 \times P_2$. Then*

$$\frac{1 + \tau_1 - \rho_1}{2} \times \frac{1 + \tau_2 - \rho_2}{2} \leq \frac{1 + \tau - \rho}{2}.$$

Proof. Follows from matrix multiplication, on noting that each term is the leading entry of its associated transition matrix. \square

Corollary 4 *Let $P = P_1 \times \dots \times P_n$. Then*

$$\prod_{i=1}^n \frac{1 + \tau_i - \rho_i}{2} \leq \frac{1 + \tau - \rho}{2}.$$

From (34), Lemma 4 and Corollary 4 we deduce:

Corollary 5 *Let $P = P_1 | \dots | P_n$. Then $L^+ \leq (1 + \tau - \rho)/2$.*

However the value $L^+ = (1 + \tau - \rho)/2$ can be achieved, specifically for the degenerate two-term decomposition (38), so this is indeed the largest lower bound. And in this case we have identification: $\widetilde{PC} = (1 + \tau - \rho)/2$.

We note that, since $\rho \leq 1 - \tau$, the largest lower bound, $(1 + \tau - \rho)/2$, can not exceed the simple upper bound $U^s = (1 + \tau - \rho)/(1 + \tau + \rho)$. Thus any lower bound must lie in the simple interval $[L^s, U^s]$.

Largest upper bound

Lemma 6

$$\min \left\{ 1, \frac{1 + \tau - \rho}{1 + \tau + \rho} \right\} \leq \min\{1, 1 - \rho\}.$$

Proof. Trivial if $\rho \leq 0$. Otherwise follows from $(1 - \rho)(1 + \tau + \rho) - (1 + \tau - \rho) = \rho(1 - \tau - \rho) \geq 0$. \square

Lemma 7 *Let $P = P_1 \times P_2$. Then*

$$\min\{1, 1 - \rho_1\} \times \min\{1, 1 - \rho_2\} \leq \min\{1, 1 - \rho\}.$$

Proof. Trivial if both ρ_1 and ρ_2 (and hence, by (22) and the fact that $\tau_2 > 0$, also ρ) are negative.

If $\rho_1 \leq 0$, $\rho_2 \geq 0$, we have to show $\rho_2 \geq \rho$. This follows from (22). Similarly if $\rho_1 \geq 0$, $\rho_2 \leq 0$ (using $\tau_2 \leq 1$).

Finally, if $\rho_1 > 0$, $\rho_2 > 0$ (and so also $\rho > 0$), the result follows from (26). \square

Corollary 6 *Let $P = P_1 \times \dots \times P_n$. Then*

$$\prod_{i=1}^n \min\{1, 1 - \rho_i\} \leq \min\{1, 1 - \rho\}.$$

From Lemma 6 and (34) we deduce:

Corollary 7 *For decomposition $P = P_1 | \dots | P_n$, $U^+ \leq \min\{1, 1 - \rho\}$.*

However the value $UB = \min\{1, 1 - \rho\}$ can be achieved. If $\rho \leq 0$, no mediators are required. If $\rho > 0$, the value $U^+ = 1 - \rho$ is achieved by the two-term decomposition (37). By Lemma 6, this largest upper bound $1 - \rho$ is at least as large as the simple upper bound U^s of (18).

Since we know that $L^+ \leq U^s$, we cannot have identification of \widetilde{PC} in this case unless these inequalities become equalities, which only holds when $\rho = 1 - \tau$.

In fact for the decomposition (37) we have $L^+ = \tau$.

A.3 Negative data observed at some steps

The lower bounds at 0 are immediate from Equation (32). It is easy to verify that the lowest upper bound at 0 is achievable by the decomposition (37), and the highest upper bound at 1 is achievable from the decompositions in (39) and (40). Since these bounds are at 0 and 1 they are the extreme values obtainable from any process involving some negative data.

Appendix B Homogeneous transitions

To examine homogeneous cases with unboundedly many mediators we consider bounds when processes are decomposed into n homogeneous processes and then take n to infinity.

We consider cases with a constant one-step transition matrix, $P_i = P' = P(\tau', \rho')$ for all i . We assume $\tau' > 0$, and we define σ' , γ' , δ' in terms of τ' and ρ' in parallel to (3), (15) and (16).

In this case, by (20) and (21), we have

$$\tau = (\tau')^n \tag{A.1}$$

$$\rho = \rho' \times \frac{1 - (\tau')^n}{1 - \tau'} = \rho' \times \frac{1 - \tau}{1 - \tau'}. \tag{A.2}$$

In particular, we note that relative sufficiency is preserved at each intermediate step: $\sigma' = \rho'/(1 - \tau') = \rho/(1 - \tau) = \sigma$. It follows that $\gamma' = \gamma$.

We have

$$\tau' = \tau^{1/n} \tag{A.3}$$

$$\rho' = \rho \times \frac{1 - \tau^{1/n}}{1 - \tau}. \tag{A.4}$$

Note that, for large n , τ' must be close to 1 and ρ' close to 0, with the same sign as ρ .

We assume $X = Y = 1$, and consider three cases: one with no data on mediators, one with all data positive, and a third evidence pattern in which values of 1 and 0 alternate at every step (with a necessary adjustment at the last two steps if n is odd).

We are interested in assessing the lower and upper bounds for a homogeneous decomposition of length n , which for data type j ($j = \emptyset, +, -$) we will denote by L_n^j and U_n^j .

For the first two cases, inserting (A.3) and (A.4) into (31) and (34) yields the following bounds:

$$L_n^\emptyset = \frac{2\tau}{1 + \tau + \rho} \quad (\text{A.5})$$

$$U_n^\emptyset = \frac{\tau + (1 - |\rho'|)^n}{1 + \tau + \rho} \quad (\text{A.6})$$

$$L_n^+ = \tau \left(\frac{2}{1 + \tau' + \rho'} \right)^n \quad (\text{A.7})$$

$$U_n^+ = \begin{cases} (\delta')^n & (\rho \geq 0) \\ 1 & (\rho < 0). \end{cases} \quad (\text{A.8})$$

The limiting values of these expressions for $n \rightarrow \infty$, as displayed in Table 4, were confirmed using *Mathematica* (Wolfram Research, Inc., 2018).

For the third case, with alternating evidence, we have:

$$L_n^- = 0 \quad (\text{A.9})$$

$$U_n^- = \begin{cases} \gamma^{(n-1)/2} \delta' & (n \text{ odd}) \\ \gamma^{n/2} & (n \text{ even}). \end{cases} \quad (\text{A.10})$$

The lower bound of 0 here follows from the presence of a 0 in the sequence (see § 3.2). For the upper bound, we have from the expressions in Table 2 (and recalling that $\gamma = \gamma'$) that the upper bound of a 1 to 0 transition followed by a 0 to 1 transition is γ . The alternating pattern repeats such pairs of transitions $n/2$ or $(n-1)/2$ times depending on whether n is even or odd. For the case with n odd, we take the final three values as 011 ($\rho > 0$) or 001 ($\rho < 0$), yielding an additional term δ' . It can be shown that, for large enough n , this sequence has the smallest upper bound, (A.10), attainable by any sequence of values.

For $\rho \neq 0$ we have $\gamma < 1$, so the limiting upper bound for alternating data, as $n \rightarrow \infty$, is 0. If $\rho = 0$, then $\gamma = \delta' = 1$ and the upper bound is 1 (as indeed it is for any sequence of values).

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