Research Article

Arvid Sjölander*

Sharp bounds for causal effects based on Ding and VanderWeele's sensitivity parameters

https://doi.org/10.1515/jci-2023-0019
received April 12, 2023; accepted February 19, 2024

Abstract: In a seminal article, Ding and VanderWeele proposed a method of constructing bounds for causal effects that has become widely recognized in causal inference. This method requires the analyst to provide guesses of certain "sensitivity parameters," loosely defined as the maximal strength of association that an unmeasured confounder may have with the exposure and with the outcome. Ding and VanderWeele stated that their bounds are sharp, but without defining this term. Using a common definition of sharpness, Sjölander A. A note on a sensitivity analysis for unmeasured confounding, and the related E-value. J Causal Inference. 2020;8(1):229–48 showed that Ding and VanderWeele's bounds are sharp in some regions of the sensitivity parameters, but are non-sharp in other regions. In this note, we follow up the work by Sjölander A. A note on a sensitivity analysis for unmeasured confounding, and the related E-value. J Causal Inference. 2020;8(1):229–48, by deriving bounds that are guaranteed to be sharp in all regions of Ding and VanderWeele's sensitivity parameters. We illustrate the discrepancy between Ding and VanderWeele's bounds and the sharp bounds with a real data example on vitamin D insufficiency and urine incontinence in pregnant women.

Keywords: bounds, causal inference, sensitivity analysis

MSC 2020: 62D20, 62J12

1 Introduction

Unmeasured confounding is an important obstacle when estimating causal effects from observational data. In the presence of unmeasured confounding, causal effects cannot be point-identified; however, it is often possible to construct bounds for them [1–5]. In a seminal article, Ding and VanderWeele (DV) [6] proposed a method of constructing such bounds that has become widely recognized in causal inference and related fields; as of 2023-04-11, DV's article [6] has more than 400 citations according to Google Scholar. Briefly, DV's method requires the analyst to provide guesses of certain "sensitivity parameters," loosely defined as the maximal strength of association that an unmeasured confounder may have with the exposure and with the outcome. Given these parameters, DV derived bounds for the causal risk ratio and the causal risk difference.

DV stated that their bounds are sharp, but without defining this term. In the causal inference literature, bounds are usually said to be sharp if all values inside the bounds are logically compatible with the observed data distribution and with any auxiliary information, such as the specified values of the sensitivity parameters [7–9]. Sjölander [10] showed that, under this definition, DV's bounds are sharp in some regions of the sensitivity parameters, but are non-sharp in other regions. He characterized certain regions where DV's bounds are

* **Corresponding author: Arvid Sjölander**, Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, University in Solna, Solna, Sweden, e-mail: arvid.sjolander@ki.se

guaranteed to be sharp, but he did neither prove that these are the only regions where the bounds are sharp, nor did he derive bounds that are narrower than DV's bounds in regions where the latter are non-sharp.

Given the wide recognition of DV's bounds, it is important to establish in which regions they are guaranteed to be sharp, in which regions they are guaranteed to be non-sharp, and to derive sharp bounds in the latter regions, given DV's sensitivity parameters. In this note, we accomplish these tasks. We start by laying down notation, definitions, and assumptions. Then, we briefly review DV's bounds. Then, we present bounds that are guaranteed to be sharp in all regions of DV's sensitivity parameters. Finally, we illustrate the discrepancy between DV's bounds and the sharp bounds with a real data example on vitamin D insufficiency and urine incontinence in pregnant women. Like DV, we focus on scenarios where both the exposure and the outcome are binary. However, whereas DV only derived bounds for the causal risk ratio and risk difference, our bounds are applicable to any measure of causal effect that can be written as a contrast between two counterfactual outcome probabilities.

2 Notation, definitions, and assumptions

DV developed their theory conditional on measured covariates, and we do the same. For brevity, we keep the conditioning on measured covariates implicit in all probability expressions below.

Let E and D denote the binary exposure and outcome, respectively. Let $p(E = e)$ denote the marginal probability of $E = e$, and let $p(D = d|E = e)$ denote the conditional probability of $D = d$, given $E = e$, for $(d, e) \in \{0, 1\}$. The exposure–outcome association is defined as some contrast between $p(D = 1|E = 1)$ and $p(D = 1|E = 0)$, for instance, the risk ratio $\text{RR}_{ED} = p(D = 1|E = 1)/p(D = 1|E = 0)$ or the risk difference $\text{RD}_{ED} = p(D = 1|E = 1) - p(D = 1|E = 0)$.

Let $D(e)$ be the potential outcome [11,12] for a given subject, had the exposure been set to $E = e$ for that subject. Similarly, let $p\{D(e) = 1\}$ be the counterfactual probability of the outcome, had the exposure been set to $E = e$ for all subjects. The causal effect of the exposure on the outcome is defined as some contrast between $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$, for instance, the causal risk ratio $\text{CRR}_{ED} = p\{D(1) = 1\}/p\{D(0) = 1\}$ or the causal risk difference $\text{CRD}_{ED} = p\{D(1) = 1\} - p\{D(0) = 1\}$. We assume consistency

$$E = e \Rightarrow D(e) = D \tag{1}$$

and the existence of a set of (unmeasured) confounders U sufficient for confounding control. In terms of potential outcomes, we require conditional exchangeability, given U:

$$\{D(0), D(1)\} \perp E|U. \tag{2}$$

Under assumptions (1) and (2), the potential outcome probability $p\{D(e) = 1\}$ can be expressed as a function of $p(D, E, U)$:

$$\begin{aligned} p\{D(e) = 1\} &= E[p\{D(e) = 1|U\}] \\ &= E[p\{D(e) = 1|E = e, U\}] \\ &= E\{p(D = 1|E = e, U)\}, \end{aligned} \tag{3}$$

where the expectation is taken over the marginal distribution of U, the first equality follows from the law of total probability, the second from assumption (2), and the third from assumption (1).

3 DV's bounds

The bounds proposed by DV use two sensitivity parameters, informally defined as the maximal strength of association between E and U, and between U and D, respectively. Formally, the parameter RR_{UD} is defined as

$$\text{RR}_{UD} = \max_e \left\{ \frac{\max_u p(D = 1|E = e, U = u)}{\min_u p(D = 1|E = e, U = u)} \right\},$$

and the parameter RR_{EeU} is defined as

$$\mathrm{RR}_{EeU} = \max_u \left\{ \frac{p(U = u|E = e)}{p(U = u|E = 1 - e)} \right\}, \quad \text{for } e \in \{0, 1\}.$$

DV defined the bounding factor

$$\mathrm{BF}_e = \frac{\mathrm{RR}_{EeU} \times \mathrm{RR}_{UD}}{\mathrm{RR}_{EeU} + \mathrm{RR}_{UD} - 1}, \quad \text{for } e \in \{0, 1\}.$$

They showed that, given $\{\mathrm{BF}_0, \mathrm{BF}_1\}$, CRR_{ED} is bounded by

$$\mathrm{RR}_{ED}/\mathrm{BF}_1 \leq \mathrm{CRR}_{ED} \leq \mathrm{RR}_{ED}\mathrm{BF}_0 \tag{4}$$

and CRD_{ED} is bounded by

$$\begin{aligned} \mathrm{RD}_{ED} - \{p(E = 0)p(D = 1|E = 1)(1 - 1/\mathrm{BF}_1) + p(E = 1)p(D = 1|E = 0)(\mathrm{BF}_1 - 1)\} \\ \leq \ \mathrm{CRD}_{ED} \leq \\ \mathrm{RD}_{ED} + \{p(E = 1)p(D = 1|E = 0)(1 - 1/\mathrm{BF}_0) + p(E = 0)p(D = 1|E = 1)(\mathrm{BF}_0 - 1)\}. \end{aligned} \tag{5}$$

Sjölander [10] showed that DV's lower bounds are sharp if $\mathrm{BF}_1 \leq 1/p(D = 1|E = 0)$ and that DV's upper bounds are sharp if $\mathrm{BF}_0 \leq 1/p(D = 1|E = 1)$, but that DV's bounds are not necessarily sharp outside these regions. However, he did neither prove that these are the only regions where the bounds are sharp, nor did he derive bounds that are narrower than DV's bounds in regions where the latter are non-sharp.

4 Sharp bounds

Define

$$l_e = p(D = 1|E = e)\{p(E = e) + p(E = 1 - e)/\mathrm{BF}_e\}$$

and

$$u_e = p(D = 1|E = e)[p(E = e) + p(E = 1 - e)\min\{\mathrm{BF}_{(1-e)}, 1/p(D = 1|E = e)\}],$$

and consider the following bounds for $p\{D(e) = 1\}$:

$$l_e \leq p\{D(e) = 1\} \leq u_e. \tag{6}$$

In the Appendix, we show that the bounds in (6) have two important properties, which we summarize in a theorem.

Theorem 1. *Validness and simultaneous sharpness of the proposed bounds.*

- *The bounds* (l_e, u_e) *are valid, in the sense that the inequalities in* (6) *hold for all distributions* $p(D, E, U)$.
- *The bounds* (l_1, u_0) *are simultaneously sharp, in the sense that, for any specific distribution* $p^*(D, E)$ *and bias factor* BF_1^*, *there exists a distribution* $p(D, E, U)$ *such that* $p(D, E) = p^*(D, E)$, $\mathrm{BF}_1 = \mathrm{BF}_1^*$, $p\{D(1) = 1\} = l_1$, *and* $p\{D(0) = 1\} = u_0$.
- *The bounds* (l_0, u_1) *are simultaneously sharp, in the sense that, for any specific distribution* $p^*(D, E)$ *and bias factor* BF_0^*, *there exists a distribution* $p(D, E, U)$ *such that* $p(D, E) = p^*(D, E)$, $\mathrm{BF}_0 = \mathrm{BF}_0^*$, $p\{D(1) = 1\} = u_1$, *and* $p\{D(0) = 1\} = l_0$.

An important corollary of Theorem 1 is that one can obtain a sharp lower bound for any contrast between $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$ by contrasting the minimal value of $p\{D(1) = 1\}$ with the maximal value of $p\{D(0) = 1\}$, within the range in (6). Similarly, one can obtain a sharp upper bound for any contrast between $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$ by contrasting the maximal value of $p\{D(1) = 1\}$ with the minimal value of $p\{D(0) = 1\}$, within the range in (6). For instance, we obtain sharp bounds for the causal risk ratio as

$$\begin{aligned} \mathrm{RR}_{ED}/\frac{p(E=0)+p(E=1)\min\{\mathrm{BF}_1, 1/p(D=1|E=0)\}}{p(E=1)+p(E=0)/\mathrm{BF}_1} \\ \leq \ \mathrm{CRR}_{ED} \leq \\ \mathrm{RR}_{ED}\frac{p(E=1)+p(E=0)\min\{\mathrm{BF}_0, 1/p(D=1|E=1)\}}{p(E=0)+p(E=1)/\mathrm{BF}_0} \end{aligned} \tag{7}$$

and sharp bounds for the causal risk difference as

$$\begin{aligned} \mathrm{RD}_{ED} - (p(E=0)p(D=1|E=1)(1-1/\mathrm{BF}_1) + p(E=1)p(D=1|E=0)[\min\{\mathrm{BF}_1, 1/p(D=1|E=0)\}-1]) \\ \leq \ \mathrm{CRD}_{ED} \leq \\ \mathrm{RD}_{ED} + (p(E=1)p(D=1|E=0)(1-1/\mathrm{BF}_0) + p(E=0)p(D=1|E=1)[\min\{\mathrm{BF}_0, 1/p(D=1|E=1)\}-1]) \ . \end{aligned} \tag{8}$$

One can easily obtain sharp bounds for other contrasts as well, such as the odds ratio or odds difference.

DV's bounds in (4) and (5) coincide with the sharp bounds in (7) and (8), respectively, in the regions where Sjölander [10] proved that DV's bounds are sharp. Specifically, DV's lower bounds in (4) and (5) coincide with the sharp the lower bounds in (7) and (8) when $\min\{\mathrm{BF}_1, 1/p(D=1|E=0)\} = \mathrm{BF}_1$, and DV's upper bounds in (4) and (5) coincide with the sharp upper bounds in (7) and (8) when $\min\{\mathrm{BF}_0, 1/p(D=1|E=1)\} = \mathrm{BF}_0$. However, outside these regions, DV's bounds are wider than the sharp bounds.

The reason why DV's bounds are not generally sharp is that, by replacing the term $\min\{\mathrm{BF}_{(1-e)}, 1/p(D=1|E=e)\}$ in the upper bound u_e with $\mathrm{BF}_{(1-e)}$, as DV effectively did, one ignores a particular restriction $E\{p(D=1|E=e,U)|E=1-e\} \leq 1$ on the underlying distribution $p(D,E,U)$. In the Appendix, we show where this restriction enters in the derivation of the bounds. Ignoring this restriction will have consequences when $\mathrm{BF}_{(1-e)}$ is large, i.e., when there is a substantial degree of unmeasured confounding.

When BF_0 and BF_1 go to infinity, the bounds for $p\{D(e)=1\}$ in (6) converge to

$$p(D=1|E=e)p(E=e) \leq p\{D(e)=1\} \leq p(D=1|E=e)p(E=e) + p(E=1-e),$$

which were previously derived by Robins [1]. These bounds are assumption-free, in the sense that they are guaranteed to include the true value of $p\{D(e)=1\}$, irrespective of the values of $\{\mathrm{BF}_0, \mathrm{BF}_1\}$.

5 Illustration

Stafne et al. [13] carried out a cross-sectional study to estimate the causal effect of vitamin D insufficiency, defined as having low levels of circulating 25-hydroxvitamin D (25(OH)D), on the risk of urine incontinence in pregnant women. The study included 851 women in mid-pregnancy (gestational weeks 18–22), who were generally healthy and above 18 years of age. Levels of 25(OH)D were measured with blood samples, whereas urine incontinence was self-reported and categorized as stress incontinence or urge incontinence.

Stafne et al. [13] carried out various analyses, using different cutoffs for 25(OH)D levels in their exposure definition, different combinations of stress/urge incontinence in their outcome definition, and adjusting for different sets of potential confounders. Here, we focus on one of these analyses, in which they defined the exposure ($E=1$) as 25(OH)$D < 50$ nmol/l, the outcome ($D=1$) as either stress or urge incontinence, and did not adjust for any confounders.

Table 1 shows the crude data under these exposure and outcome definitions. Based on these data, we have that $p(E=1)=0.27$, $p(D=1|E=0)=0.38$, and $p(D=1|E=1)=0.49$. The risk ratio and risk difference are

Table 1: Data from Stafne et al. [13] on vitamin D insufficiency (E) and urine incontinence (D)

	$E=0$	$E=1$
$D=0$	382	118
$D=1$	239	112

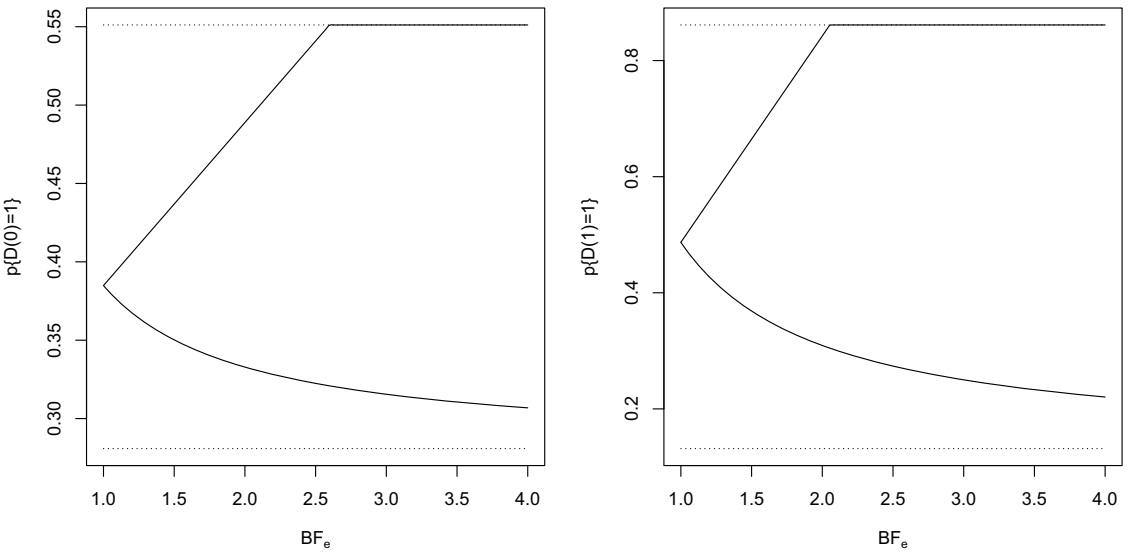

Figure 1: Sharp bounds (solid lines) and the assumption-free bounds (dotted lines) for $p\{D(0) = 1\}$ (left panel) and $p\{D(1) = 1\}$ (right panel), for the data in Table 1.

equal to 1.27 and 0.10, respectively, and a χ^2-test gives a p-value equal to 0.01. Hence, there is strong evidence for a statistical association between vitamin D insufficiency and urine incontinence.

Figure 1 shows the sharp bounds (solid lines) and the assumption-free bounds (dotted lines) for $p\{D(0) = 1\}$ (left panel) and $p\{D(1) = 1\}$ (right panel) as functions of BF_e, assuming that $\mathrm{BF}_0 = \mathrm{BF}_1$.

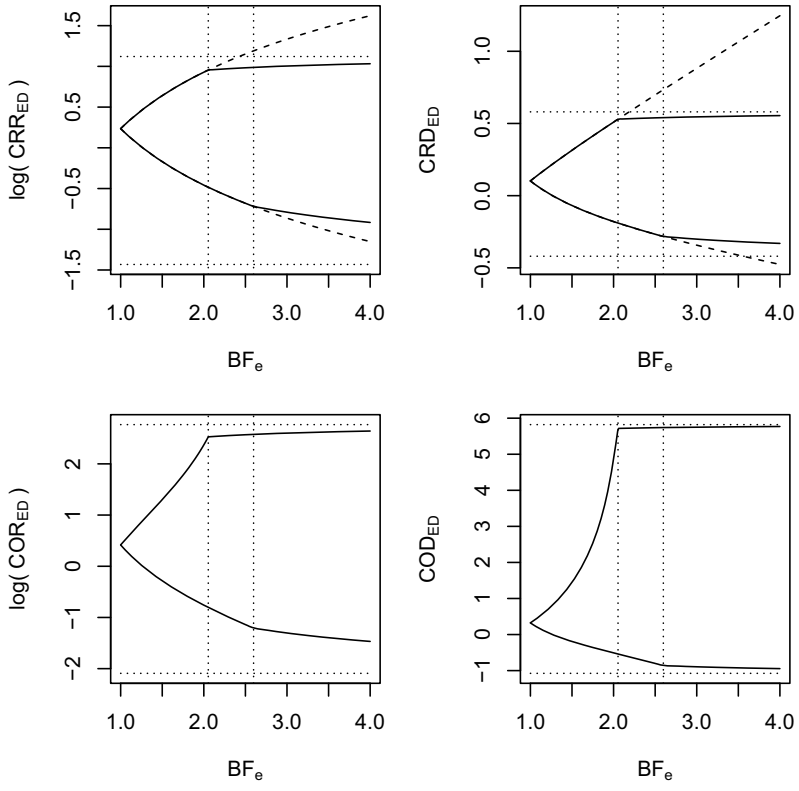

Figure 2: Sharp bounds (solid lines), DV's bounds (dashed lines), and the assumption-free bounds (dotted lines) for the causal log risk ratio (top-left panel), causal risk difference (top-right panel), causal log odds ratio (bottom-left panel), and causal odds difference (bottom-right panel), for the data in Table 1.

Figure 2 shows the bounds for several contrasts between $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$. The top panels of Figure 2 show the sharp bounds (solid lines) and DV's bounds (dashed lines) for the causal log risk ratio (top-left panel) and causal risk difference (top-right panel) as functions of BF_e, assuming that $BF_0 = BF_1$. The vertical dotted lines in Figure 2 indicate the values $BF_e = 1/p(D = 1|E = 0)$ and $BF_e = 1/p(D = 1|E = 1)$. Up to these points, DV's lower and upper bounds agree with the sharp lower and upper bounds, respectively, but are wider than the sharp bounds thereafter. The horizontal dotted lines indicate the assumption-free lower and upper bounds. The sharp bounds are confined within the assumption-free bounds, whereas DV's bounds exceed the assumption-free bounds for large values of BF_e. Furthermore, for large values of BF_e, DV's bounds exceed the logical limits −1 and 1 for the causal risk difference.

The bottom panels of Figure 2 show the sharp bounds (solid lines) for the causal log odds ratio (bottom-left panel) and causal odds difference, together with the corresponding assumption-free bounds (dotted lines). We note that DV did not derive any bounds for these parameters based on the bounding factors $\{BF_0, BF_1\}$. However, the sharp bounds for these, or any other contrasts between $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$, are easily obtained from the bounds in (6).

6 Discussion

In this note, we have derived sharp bounds for causal effects based on DV's sensitivity parameters. We have shown that the bounds previously derived by DV are equal to the sharp bounds in the regions where Sjölander [10] proved that DV's bounds are sharp, but that DV's bounds are wider than the sharp bounds outside these regions.

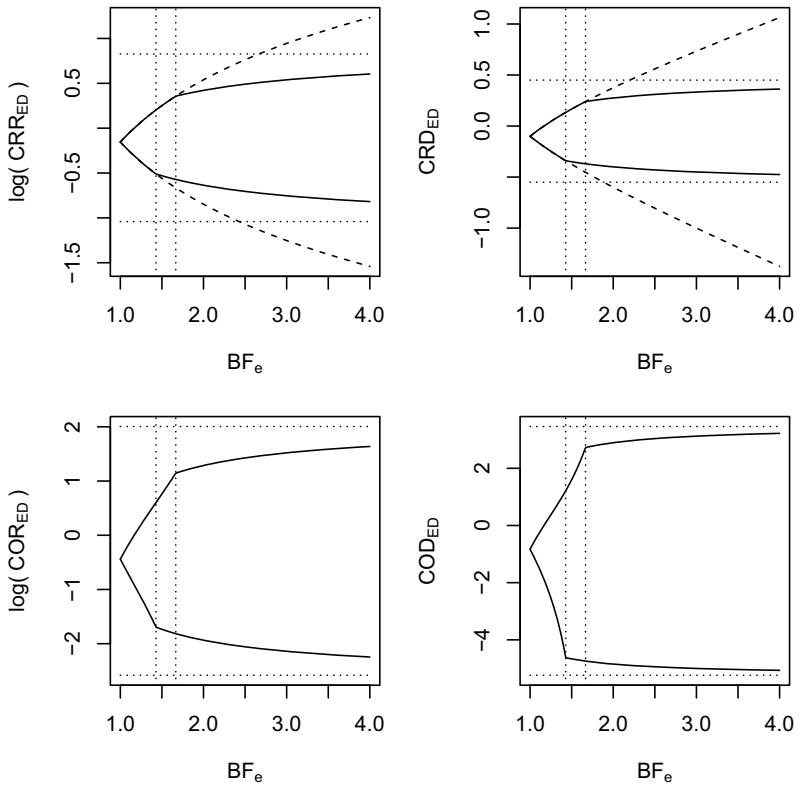

Figure 3: Sharp bounds (solid lines), DV's bounds (dashed lines), and the assumption-free bounds (dotted lines) for the causal log risk ratio (top-left panel), causal risk difference (top-right panel), causal log odds ratio (bottom-left panel), and causal odds difference (bottom-right panel), for $p(E = 1) = 0.5$, $p(D = 1|E = 0) = 0.6$, and $p(D = 1|E = 1) = 0.7$.

The sharp bounds are clearly of strong theoretical interest, but may also have important practical relevance, since they may sometimes be substantially narrower than both DV's bounds and the assumption free bounds. As an example, Figure 3 shows the same bounds as Figure 2, for $p(E = 1) = 0.5$, $p(D = 1|E = 0) = 0.6$, and $p(D = 1|E = 1) = 0.7$.

A reader may wonder if our developments have any implications for the *E*-value. The answer is no. To see this, note that, for an observed risk ratio larger than 1, the *E*-value is defined as the common value of the sensitivity parameters such that the lower bound for the causal risk ratio is equal to 1. Suppose that DV's lower bound for the causal risk ratio was not sharp when being equal to 1. If so, then the *E*-value would be conservative in the sense that, even if the sensitivity parameters were as large as the *E*-value, the observed association could still not be explained away by unmeasured confounding. However, Sjölander [10] showed that DV's lower bound for the causal risk ratio is always sharp when it is equal to 1; hence, the observed association can, indeed, be explained away by unmeasured confounding if the sensitivity parameters are as large as the *E*-value.

Given the wide recognition of DV's work, we hope that this note will make an important contribution to the literature on sensitivity analysis and bounds for causal effects in the presence of unmeasured confounding.

Funding information: The author gratefully acknowledges funding from the Swedish Research Council, grant number 2020-01188.

Author contribution: The author confirms the sole responsibility for the conception of the study, presented results and manuscript preparation.

Conflict of interest: The author has no conflict of interest.

References

[1] Robins JM. The analysis of randomized and non-randomized AIDS treatment trials using a new approach to causal inference in longitudinal studies. In: Sechrest L, Freeman H, Mulley A, editors. Health service research methodology: a focus on AIDS. US Public Health Service, National Center for Health Services Research; 1989. p. 113–59.
[2] Balke A, Pearl J. Bounds on treatment effects from studies with imperfect compliance. J Amer Stat Assoc. 1997;92(439):1171–6.
[3] Zhang JL, Rubin DB. Estimation of causal effects via principal stratification when some outcomes are truncated by death. J Educat Behav Stat. 2003;28(4):353–68.
[4] Cai Z, Kuroki M, Pearl J, Tian J. Bounds on direct effects in the presence of confounded intermediate variables. Biometrics. 2008;64(3):695–701.
[5] Sjölander A. Bounds on natural direct effects in the presence of confounded intermediate variables. Stat Med. 2009;28(4):558–71.
[6] Ding P, VanderWeele TJ. Sensitivity analysis without assumptions. Epidemiology. 2016;27(3):368–77.
[7] Tian J, Pearl J. Probabilities of causation: Bounds and identification. Ann Math Artif Intell. 2000;28(1-4):287–313.
[8] Imai K. Sharp bounds on the causal effects in randomized experiments with truncation-by-death. Stat Probability Lett. 2008;78(2):144–9.
[9] Huber M, Mellace G. Sharp bounds on causal effects under sample selection. Oxford Bulletin Econ Stat. 2015;77(1):129–51.
[10] Sjölander A. A note on a sensitivity analysis for unmeasured confounding, and the related *E*-value. J Causal Inference. 2020;8(1):229–48.
[11] Rubin DB. Estimating causal effects of treatments in randomized and nonrandomized studies. J Educat Psychol. 1974;66(5):688–701.
[12] Pearl J. Causality: models, reasoning, and inference. 2nd ed. New York: Cambridge University Press; 2009.
[13] Stafne SN, Mørkved S, Gustafsson MK, Syversen U, Stunes AK, Salvesen KÅ, et al. Vitamin D and stress urinary incontinence in pregnancy: a cross-sectional study. Int J Obstetr Gynaecol. 2020;127(13):1704–11.

Appendix

A Proof of Theorem 1

A.1 Proof of validness

From (3), we have that $p\{D(e) = 1\} = E\{p(D = 1|E = e, U)\}$. Using the law of total probability, we further have that

$$\begin{aligned} E\{p(D = 1|E = e, U)\} &= \sum_{e' \in \{0,1\}} E\{p(D = 1|E = e, U)|E = e'\}p(E = e') \\ &= p(D = 1|E = e)p(E = e) + E\{p(D = 1|E = e, U)|E = 1 - e\}p(E = 1 - e) \\ &= p(D = 1|E = e)\{p(E = e) + \text{CRR}_{EeD}p(E = 1 - e)\}, \end{aligned}$$

where we have defined

$$\text{CRR}_{EeD} = \frac{E\{p(D = 1|E = e, U)|E = 1 - e\}}{p(D = 1|E = e)}.$$

Our parameters CRR_{E0D} and CRR_{E1D} correspond to the parameters CRR^{+}_{ED} and $1/\text{CRR}^{-}_{ED}$, defined in Section 2.2 of DV's eAppendix. In that section of eAppendix, DV showed that

$$1/\text{CRR}_{E1D} \leq \frac{\text{RR}_{E1U} \times \text{RR}_{1UD}}{\text{RR}_{E1U} + \text{RR}_{1UD} - 1}$$

and

$$\text{CRR}_{E0D} \leq \frac{\text{RR}_{E1U} \times \text{RR}_{0UD}}{\text{RR}_{E1U} + \text{RR}_{0UD} - 1},$$

where

$$\text{RR}_{eUD} = \frac{\max_u p(D = 1|E = e, U = u)}{\min_u p(D = 1|E = e, U = u)}.$$

Since $\text{RR}_{eUD} \leq \text{RR}_{UD}$ and $xy/(x + y - 1)$ is monotonically increasing in y, it follows that $1/\text{CRR}_{E1D} \leq \text{BF}_1$ and $\text{CRR}_{E0D} \leq \text{BF}_1$, and by symmetry also that $1/\text{CRR}_{E0D} \leq \text{BF}_0$ and $\text{CRR}_{E1D} \leq \text{BF}_0$. In short, we have that

$$1/\text{BF}_e \leq \text{CRR}_{EeD} \leq \text{BF}_{(1-e)}.$$

However, we also have that

$$E\{p(D = 1|E = e, U)|E = 1 - e\} \leq 1,$$

so that

$$1/\text{BF}_e \leq \text{CRR}_{EeD} \leq \min\{\text{BF}_{(1-e)}, 1/p(D = 1|E = e)\},$$

which gives the bounds in (6).

A.2 Proof of sharpness

We prove that (l_1, u_0) are simultaneously sharp. That (l_0, u_1) are simultaneously sharp follows by symmetry. Thus, we prove that it is possible to find a distribution $p(D, E, U)$ that is consistent with any given $\{p^*(D, E), \text{BF}^*_1\}$, and is such that $p\{D(1) = 1\} = l_1$ and $p\{D(0) = 1\} = u_0$. We only consider the case $\text{BF}^*_1 > 1/p^*(D = 1|E = 0)$ since sharpness was proven by Sjölander [10] for the opposite case. We construct the distribution $p(D, E, U)$ in the following steps:

(1) Let $p(E) = p^*(E)$.
(2) Let U be binary, with

$$p(U = 1|E = 1) = 1,$$
$$p(U = 1|E = 0) = 1/x,$$

where x is an arbitrary number such that $x \geq \mathrm{BF}_1^*$. We have that $0 < p(U = 1|E = e) \leq 1$ for $e \in \{0, 1\}$. We also have that $p(U = 1|E = 1)/p(U = 1|E = 0) = x > 1$ and $p(U = 0|E = 1)/p(U = 0|E = 0) = 0$, so that $\mathrm{RR}_{E1U} = p(U = 1|E = 1)/p(U = 1|E = 0) = x$.
(3) Let

$$\begin{aligned}
p(D = 1|E = 0, U = 0) &= \frac{p^*(D = 1|E = 0) - 1/x}{1 - 1/x},\\
p(D = 1|E = 0, U = 1) &= 1,\\
p(D = 1|E = 1, U = 0) &= p^*(D = 1|E = 1)\frac{1/\mathrm{BF}_1^* - 1/x}{1 - 1/x},\\
p(D = 1|E = 1, U = 1) &= p^*(D = 1|E = 1).
\end{aligned}$$

We have that $0 \leq p(D = 1|E = e, U = u) \leq 1$ for $(e, u) \in \{0, 1\}$. We further have that

$$\begin{aligned}
p(D = 1|E = 0) &= p(D = 1|E = 0, U = 1)p(U = 1|E = 0) + p(D = 1|E = 0, U = 0)p(U = 0|E = 0)\\
&= 1 \times 1/x + \frac{p^*(D = 1|E = 0) - 1/x}{1 - 1/x} \times (1 - 1/x)\\
&= p^*(D = 1|E = 0)
\end{aligned}$$

and

$$\begin{aligned}
p(D = 1|E = 1) &= p(D = 1|E = 1, U = 1)p(U = 1|E = 1) + p(D = 1|E = 1, U = 0)p(U = 0|E = 1)\\
&= p^*(D = 1|E = 1) \times 1 + p^*(D = 1|E = 1)\frac{1/\mathrm{BF}_1^* - 1/x}{1 - 1/x} \times 0\\
&= p^*(D = 1|E = 1).
\end{aligned}$$

We further have that

$$\begin{aligned}
\frac{p(D = 1|E = 1, U = 1)}{p(D = 1|E = 1, U = 0)} &= \frac{1 - 1/x}{1/\mathrm{BF}_1^* - 1/x}\\
&> \frac{p(D = 1|E = 0, U = 1)}{p(D = 1|E = 0, U = 0)} = \frac{1 - 1/x}{p^*(D = 1|E = 0) - 1/x}\\
&\geq 1,
\end{aligned}$$

so that

$$\begin{aligned}
\mathrm{RR}_{UD} &= \frac{p(D = 1|E = 1, U = 1)}{p(D = 1|E = 1, U = 0)}\\
&= \frac{1 - 1/x}{1/\mathrm{BF}_1^* - 1/x}\\
&= \frac{1 - 1/\mathrm{RR}_{E1U}}{1/\mathrm{BF}_1^* - 1/\mathrm{RR}_{E1U}}\\
&= \frac{\mathrm{BF}_1^*(\mathrm{RR}_{E1U} - 1)}{\mathrm{RR}_{E1U} - \mathrm{BF}_1^*}.
\end{aligned}$$

We now have that

$$\begin{aligned}\mathrm{BF}_1 &= \frac{\mathrm{RR}_{E1U} \times \mathrm{RR}_{UD}}{\mathrm{RR}_{E1U} + \mathrm{RR}_{UD} - 1} \\ &= \frac{\mathrm{BF}_1^*(\mathrm{RR}_{E1U} - 1)\mathrm{RR}_{E1U}}{\mathrm{RR}_{E1U}(\mathrm{RR}_{E1U} - \mathrm{BF}_1^*) + \mathrm{BF}_1^*(\mathrm{RR}_{E1U} - 1) - (\mathrm{RR}_{E1U} - \mathrm{BF}_1^*)} \\ &= \mathrm{BF}_1^*.\end{aligned}$$

Finally, we have that

$$\begin{aligned}E\{p(D = 1|E = 1, U)|E = 0\} &= p(D = 1|E = 1, U = 1)p(U = 1|E = 0) + p(D = 1|E = 1, U = 0)p(U = 0|E = 0) \\ &= p^*(D = 1|E = 1) \times 1/x + p^*(D = 1|E = 1)\frac{1/\mathrm{BF}_1^* - 1/x}{1 - 1/x} \times (1 - 1/x) \\ &= p^*(D = 1|E = 1)/\mathrm{BF}_1^* \\ &= p(D = 1|E = 1)/\mathrm{BF}_1\end{aligned}$$

and

$$\begin{aligned}E\{p(D = 1|E = 0, U)|E = 1\} &= p(D = 1|E = 0, U = 1)p(U = 1|E = 1) + p(D = 1|E = 0, U = 0)p(U = 0|E = 1) \\ &= 1 \times 1 + \frac{p^*(D = 1|E = 0) - 1/x}{1 - 1/x} \times 0 \\ &= 1,\end{aligned}$$

so that

$$\begin{aligned}p\{D(1) = 1\} &= E\{p(D = 1|E = 1, U)\} \\ &= p(D = 1|E = 1)p(E = 1) + E\{p(D = 1|E = 1, U)|E = 0\}p(E = 0) \\ &= p(D = 1|E = 1)\{p(E = 1) + p(E = 0)/\mathrm{BF}_1\} = l_1\end{aligned}$$

and

$$\begin{aligned}p\{D(0) = 1\} &= E\{p(D = 1|E = 0, U)\} \\ &= p(D = 1|E = 0)p(E = 0) + E\{p(D = 1|E = 0, U)|E = 1\}p(E = 1) \\ &= p(D = 1|E = 0)\{p(E = 0) + p(E = 1)/p(D = 1|E = 0)\} = u_0.\end{aligned}$$

We end the proof with a technical remark. For the distribution, we have constructed $p(U = 0|E = 1) = 0$ so that, strictly speaking, $p(D = 1|E = 1, U = 0)$ is undefined. To overcome this technical obstacle, one can modify the proof and let $p(U = 0|E = 1) = \varepsilon$ approach 0 in such a way that $\{p(D, E), \mathrm{BF}_1\}$ converges to $\{p^*(D, E), \mathrm{BF}_1^*\}$ and $p\{D(1) = 1\}$ and $p\{D(0) = 1\}$ converge to the lower and upper bounds.