A Comment on *Family Ruptures, Stress, and the Mental Health of the Next Generation*

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Abstract

Persson and Rossin-Slater (2016b) claim to be the first to credibly estimate causal effects of fetal stress exposure on mental health in later life. They emphasize that their analysis is the first to control for non-random exposure to a relative's death and non-random gestation length. In light of discoveries regarding prior literature, we find these claims to be exaggerated and misleading.

1 Introduction

Have Persson and Rossin-Slater (2016b) discovered a novel causal effect of *in utero* maternal stress from family ruptures on the later life and health outcomes of children? The authors claim two substantive contributions relative to the prior literature. First, that they use mothers who experienced a post-natal death as a control group to compare with treatment group mothers, defined as those who experienced a relative's death with a baby *in utero*. Second, that they instrument for actual gestation length with predicted gestation length. Persson and Rossin-Slater (2016b) claim that these two innovations enable them to recover–for the first time–the causal effect of family ruptures on later life outcomes. In this note we will demonstrate that both claims of novelty are false, and that Persson and Rossin-Slater's acceptance into the *American Economic Review*, after the relevant literature has been discovered, was potentially enabled by an editor who is Rossin-Slater's co-author.

Persson and Rossin-Slater (2016b) are *not* the first to use exposure to maternal bereavement *in utero* for identification, and they are not the first to document a causal link between fetal stress exposure and mental health.[1] In fact, a large literature–starting with Huttunen and Niskanen (1978)–uses the same control group as Persson and Rossin-Slater (2016b) to identify the effect of fetal stress exposure on mental health.[2] Much of the literature invokes the same

*This note sythesizes the critiques of several anonymous economists. Email: nicolas.bearbaki@gmail.com.

[1]In May 2016, after the paper was accepted at the *American Economic Review*, Persson and Rossin-Slater added two footnotes: footnote 7 and footnote 10. These footnotes purport to address additional literature not cited in the April 2016 draft of Persson and Rossin-Slater (2016a) which was the version that was accepted for publication. For further information, see retractionwatch.com/2016/05/26/economists-go-wild-over-overlooked-citations-in-preprint-on-prenatal-stress/. Despite these additions, Persson and Rossin-Slater (2016b) do not appear to have been subjected to a further round of refereeing as might have been expected following the revelation of several closely related contributions.

[2]It is important to realize that the public health literature on the topic has been growing steadily since the late 1970s. Class et al. (2011), who use the same dataset as Persson and Rossin-Slater (2016b) to address similar questions, review that literature.

argument as Persson and Rossin-Slater, by letting the effect of a relative's death vary with the timing of that death. For example, Abel *et al.* (2014) estimate models which allow the effect of bereavement to vary in categories ranging from preconception to well into childhood.[3] Using the reasoning in Persson and Rossin-Slater's (2016b) paper, we must conclude that these earlier papers had also recovered causal effects (whether or not that is explicitly claimed by the earlier authors in the same language used by economists).

Persson and Rossin-Slater's second claim to innovation is an instrument that turns out to be irrelevant to their estimates (as expressed in more detail by Matsumoto (2016), which we summarize in Section 3.)

2 Econometric Specification

Persson and Rossin-Slater's (2016b) empirical strategy is not novel–despite the authors' and the AER editor's claims. Huttunen and Niskanen (1978) had used the same control and treatment groups, and had also compared *in utero* exposure to post-natal exposure. The only major difference in empirical strategy is Persson and Rossin-Slater's IV method (described in detail in Section 3 below)–which does not affect the estimates.

Another earlier work, Abel *et al.* (2014), offers estimates that are not explicitly placed in the treatment-control framework, but from which we can read off a variety of causal effects. For example, Abel's Table 3 reports that any pre-natal exposure has an odds ratio of 1.29 for psychosis, relative to no-exposure, and 1.45 for post-natal exposure. The difference in odds ratios, or some transformation thereof, is an estimate of the same causal effects as in Persson and Rossin-Slater (2016b).

Although Persson and Rossin-Slater (2016b) claim to have made the first "causal" analysis, in fact Abel and other papers in the medical literature permit far more detailed "causal" analyses than Persson and Rossin-Slater, because the latter restrict their analysis to binary treatments. However, there are sound biological reasons for the effect to vary with the timing of the relative's death even *in utero* (as described in Class et al. (2014), which allows the effect to vary by month of pregnancy).

More formally, let d_1 indicate a relative's death *in utero* and d_2 denote a relative's death within 280 days after birth. Persson and Rossin-Slater (2016b) note correctly that a regression of some mental health outcome y on d_1 and observable controls does not recover a consistent estimate of the effect of exposure to a relative's death during pregnancy. Persson and Rossin-Slater leave the impression that the putatively "correlational" medical literature limits attention to this specification, but that is incorrect.

Persson and Rossin-Slater proceed by estimating OLS models of the form

$$y = \beta_0 + \beta_1 d_1 + X'\delta + u$$

in the subpopulation for which either d_1 or d_2 has occurred. They argue that, "intuitively, our empirical strategy exploits a discontinuity around the threshold of 280 days after conception, and assigns a child to intrauterine stress exposure if the relatives death occurred before this date."

Persson and Rossin-Slater are mistaken. This is not a standard regression discontinuity design in any sense. An estimate of β_1 from the specification above should asymptotically lead

[3] Abel et al. (2014) also stratify by cause of relative's death, which is another of Persson and Rossin-Slater's (2016b) minor claims of innovation.

to the same estimate of $\theta_1 - \theta_2$ from the specification

$$y = \theta_0 + \theta_1 d_1 + \theta_2 d_2 + X'\gamma + e$$

estimated over the entire population. Both models use the regression-adjusted for X difference in means across the pre- and post-partum outcomes in the dependent variable to identify the effect of exposure *in utero*. The argument is essentially that θ_1 and θ_2 are biased but by the same magnitude, so the difference $\theta_1 - \theta_2$ is an unbiased estimate of the effect of a relative's death *in utero* relative to post-partum.

Note that Abel et al.'s (2014) odds ratios, suitably transformed, can also serve as an estimate of both β_1 and $\theta_1 - \theta_2$. In essence, Huttunen and Niskanen (1978) and Abel et al. (2014) both use the same approach to identify the "causal" effect as Persson and Rossin-Slater (2016b). Thus Persson and Rossin-Slater's first claim to novelty is unwarranted.

3 Endogeneity in the Medical Literature

Can Persson and Rossin-Slater claim an original contribution to the literature based on their introduction of an instrumental variable? In this section, we highlight concerns regarding the IV method used in Persson and Rossin-Slater (2016b).[4] In order to make a claim to an original contribution to the literature, Persson and Rossin-Slater argue that date of birth is endogenous, and that consequently the prior research results in the medical literature (for example, Huttunen and Niskanen (1978) and Class et al. (2011)) are not "causal". To address the supposed endogeneity problem, Persson and Rossin-Slater (2016b) instrument date of birth with the expected delivery date.

Persson and Rossin-Slater present in their Appendix D the estimation results of a two-stage least squares regression (Table D1). They report a first stage R^2 of 0.97, and they mention that the instrument (relative death before expected birth date) is different from the actual exposure variable (relative death before actual birth date) for only about 1 percent of the individuals in our data (p. D-25).

What this suggests is that the endogeneity they are supposedly correcting for is not an important issue. Because of the high degree of similarity between the potentially endogenous variable and the instrument, they should get almost the same result from the naive comparison using actual birth date–just as Huttunen and Niskanen (1978) did. While Persson and Rossin-Slater dismiss these previous scholars' findings as merely "correlational", and their own findings as "causal", they fail to demonstrate that their own estimates are different from those earlier findings.

In fact, the opposite is likely to be true: Persson and Rossin-Slater's instrumental variable is the same as their "endogenous" variable for 99% of their data. In other words, using the same assumptions that make their instrumental variable design valid, the simple OLS estimate is unlikely to be biased.

Persson and Rossin-Slater's IV offers no improvement over the approach used in the medical literature.[5] Thus their second claim to an original contribution to the literature is also unwarranted.

[4] Matsumoto (2016) discusses these issues in greater depth.

[5] In addition, it is unclear whether Persson and Rossin-Slater's instrument is truly exogenous. The expected delivery date is calculated based on the gestational age of the baby at birth (conception date equals birth date minus gestational age, while expected delivery date equals conception date plus 280 days). However, the gestational age is itself an estimate based on the last menstrual cycle or measurements taken from a prenatal ultrasound. The prenatal ultrasound is the preferred method for estimating gestational age and is used if it gives a significantly

4 Discussion

Persson and Rossin-Slater's (2016b) paper incorrectly dismisses the previous literature and misrepresents their own paper's claims to novelty. We recognize that scholars may occasionally fail to locate previous literature, or fail to cite those who have gone before them. What is concerning in this case is that even after Persson and Rossin-Slater were made aware of their oversight of earlier literature, they have still refused to honestly situate their work in the context of the larger literature. Instead, they incorrectly demean the work of previous scholars as merely "correlational", and falsely claim novelty for their own work that it does not deserve.

This point is particularly disturbing because Persson and Rossin-Slater's claims to novelty are publicly supported by Hilary Hoynes, the co-editor at the *American Economic Review* (*AER*) in charge of the paper. Even more concerning is that Hoynes is a recent coauthor with Maya Rossin-Slater.[6,7] This situation violates the editorial policy of the *AER*—to limit conflicts of interest, the editorial policy does not allow an editor to be in charge of their recent co-author's paper.

Given the hierarchical nature of economics, a single publication in the *American Economic Review* is enough to build a reputation as a leading researcher. It is no surprise that the impression that the top publications are sometimes handed out carelessly to friends and relations is disturbing to many. It is also no surprise that few are willing to publicly criticize those who control access to the leading journals in the discipline.

The culture of honest economic scholarship is threatened because the AER referees were not asked to re-assess the paper's contribution despite the new information that we have brought to light in other venues. This is why we found it important to produce this note and help correctly position this paper in the literature.

Appendix: A History of Events

A brief timeline of the events that transpired which motivated this note is as follows:

1. In an earlier accepted version of their *AER* paper, Persson and Rossin-Slater (2016a) failed to cite the health literature relating maternal stress to health outcomes of children, and instead falsely claimed a novel contribution.

2. When this came to light, instead of acknowledging the existing literature, Persson and Rossin-Slater (2016b) added footnotes which significantly misrepresented the content of said literature, and once again falsely claimed a novel contribution for themselves.

3. The revised paper was apparently not sent back for a new round of refereeing, and the changes were instead approved only at the sole discretion of the assigned co-editor who may be exposed to conflict of interest.

different answer from the estimate using the last menstrual cycle. If pregnant individuals happen to miss early prenatal appointments because say a close relative dies, then the estimate of gestational age is affected and the estimated date of birth is not exogenous.

[6]See retractionwatch.com/2016/05/26/economists-go-wild-over-overlooked-citations-in-preprint-on-prenatal-stress/. In particular, "Hoynes confirmed to us that Persson and Rossin-Slater had contacted her to ask if it was acceptable to revise the paper to include the Class et al. paper, a request which she granted and described as not unusual. Until a manuscript has been published, she wrote, she accepts such changes."

[7]See, for instance, sites.google.com/a/umich.edu/baileymj/research-and-publications.

4. A group of anonymous economists worked together to produce this note to clarify our position on the matter. We do not know one another's identities. We will not reveal our identities due to concerns of retaliation from editors at AER and other members of their networks.

References

[1] Abel, K. M., H. P. Heuvelman, L. Jorgensen, C. Magnusson, S. Wicks, E. Susser, J. Hallkvist, and C. Dalman. "Severe bereavement stress during the prenatal and childhood periods and risk of psychosis in later life." *BMJ: British Medical Journal* 348 (2014): f7679. doi: 10.1136/bmj.f7679.

[2] Black, Sandra E., Paul J. Devereux, and Kjell G. Salvanes. "Does Grief Transfer across Generations? Bereavements during Pregnan cy and Child Outcomes." *American Economic Journal: Applied Economics* 8, no. 1 (2016): 193-223.

[3] Class, Quetzal A., Paul Lichtenstein, Niklas Lngstrm, and Brian M. D'Onofrio. "Timing of prenatal maternal exposure to severe life events and adverse pregnancy outcomes: a population study of 2.6 million pregnancies." *Psychosomatic Medicine* 73, no. 3 (2011): 234-241.

[4] Currie, Janet, and Maya Rossin-Slater. "Weathering the storm: Hurricanes and birth outcomes." *Journal of Health Economics* 32, no. 3 (2013): 487-503.

[5] Huttunen, Matti O., and Pekka Niskanen. "Prenatal loss of father and psychiatric disorders." *Archives of General Psychiatry* 35, no. 4 (1978): 429-431.

[6] Matsumoto, Brett. "Comment on the Identification Strategy in "Family Ruptures, Stress, and the Mental Health of the Next Generation." *Working paper*, June 2016. Available at: https://www.dropbox.com/s/8t7k906ze2mfkk7/comment.pdf?dl=0.

[7] Persson, Petra, and Maya Rossin-Slater. "Family ruptures, stress, and the mental health of the next generation." Pre-print, April 2016 (2016a). *Forthcoming* in the *American Economic Review*. Available at: archive.org.

[8] Persson, Petra, and Maya Rossin-Slater. "Family ruptures, stress, and the mental health of the next generation." NBER Working Paper No. 22229, May 2016 (2016b). *Forthcoming* in the *American Economic Review*.