Family Ruptures, Stress, and the Mental Health of the Next Generation: Comment

By Brett Matsumoto*

The empirical methodology used by Persson and Rossin-Slater (2016) to estimate the causal effect of in utero *exposure to stress contains a potentially significant flaw. They define the control group in a way that may bias their causal estimates and can lead to the finding of a significant relationship when there is none. In this note, I describe the source of the bias and suggest an alternative specification of the control group.*

In their paper "Family Ruptures, Stress, and the Mental Health of the Next Generation", Persson and Rossin-Slater attempt to estimate the causal effect of *in utero* exposure to stress on birth and later life outcomes. They do this by considering mothers who experience the loss of a close relative during pregnancy as the treatment group. The control group is mothers who experience this loss shortly after birth. Let c denote conception, b denote birth, and e_b denote expected date of birth ($e_b = c + 280$ days). The authors estimate the causal effect using the following equation (equation 5 in the paper):

$$Outcome = \beta_0 + \beta_1 \mathbf{1}[c \leq \ RelativeDeath \ < e_b] + \beta_2 \mathbf{X} + \epsilon, \tag{1}$$

where the estimation sample is limited to mothers who experience the relative death during pregnancy or within one year of the actual date of birth. The control group is defined through this sample restriction and includes those who experience the death of a relative between the *expected* date of birth and one year after the *actual* date of birth. The treatment group is defined based on the *expected* date of birth. The concern with defining the treatment group using the actual date of birth is that the actual date of birth may be correlated with the error term. On the one hand, there is the mechanical relationship that a relative death is more likely to occur during pregnancy the longer the pregnancy lasts. Also, there is the concern that the treatment defined using actual date of birth is endogenous since relative death can affect the length of the pregnancy.

The problem with this empirical specification is that defining the control group as experiencing a relative death within one year of the actual date of birth cre-

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ates spurious correlation that may bias the causal estimates. The next section describes this issue in greater detail and argues that the results presented in Persson and Rossin-Slater (2016) contain at least some amount of bias.[1]

I. Control Group Specification

The problem with the authors' specification of the control group is that the endpoint of the interval that defines the control group depends on the actual date of birth. Therefore, the length of the period that defines the control group differs depending on when a child is born relative to the expected date of birth. The probability that someone is control (versus out of the sample) depends on the length of the control group interval (e_b to $b + 365$). Since individuals who are born prematurely ($b < e_b$) have a shorter control group window, these individuals are less likely to be in the control group.

Figure 1. Comparing Treatment and Control Groups

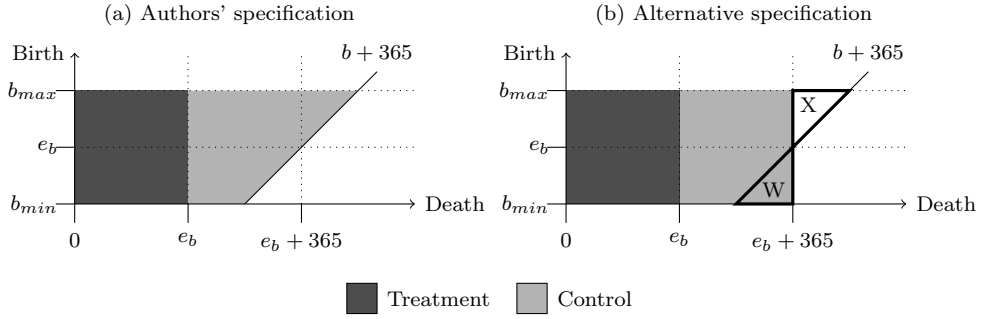

Figure 1 presents a graphical representation of this issue.[2] The timing of birth is on the y-axis and the timing of the relative death is on the x-axis. Conception occurs at time zero. All births occur in the interval $[b_{min}, b_{max}]$. Panel (a) is the specification used in Persson and Rossin-Slater (2016). Panel (b) represents an alternative specification of the control group (using one year from the *expected* date of birth to define the endpoint of the control group window). If relative death is independent of gestation length, then the expected gestation length conditional on being in the treatment group is e_b. The expected gestation length conditional on being in the control group under the alternative specification is also e_b. The difference between the specifications of the control groups are the areas X and W. The authors' specification of the control group includes the area X (who have longer than average gestation lengths) and excludes the area W (who have shorter than average gestation lengths). The net effect is that the control group will have

[1] Black, Devereux and Salvanes (2016) use a similar identification strategy but also include mother fixed effects. The control group is correctly specified in their paper.

[2] Figure 1 is for illustrative purposes only and is not drawn to scale.

an average gestation length that is greater than e_b, and the treatment effect will be negative even though it was assumed to be zero.

For any outcome correlated with actual gestation length, the average outcome of the control group will be skewed towards individuals with higher gestation lengths. The bias in the average outcome for the control group will bias the estimated treatment effect. Virtually all of the outcomes considered are correlated to some degree with actual gestation length (e.g., birth weight, health conditions at birth or shortly after, and potentially even later life outcomes).[3] The magnitude of the bias depends upon the degree of correlation between actual gestation length and the outcome. The bias will be largest for outcomes that are highly correlated with the actual gestation length (e.g., likelihood of premature birth). For outcomes that are not correlated with actual gestation length, however, the authors' specification will not bias the estimated causal effect. The direction of the bias is in the direction of the estimated results, so the authors likely overstate the true causal effect. The authors' empirical specification could even recover a significant causal effect when the true causal effect is zero.[4] Using the alternative specification of the control group would correct the bias.

REFERENCES

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[3]The correlation between the outcomes and actual gestation length is the motivation for the authors' empirical strategy of defining treatment based on the expected date of birth. If the outcome is not correlated with actual gestation length, then there would be no endogeneity problem from using the actual date of birth to assign treatment.

[4]In an earlier version of this comment, I use data on U.S. births and estimate a significant negative effect on birth outcomes for a randomly generated treatment when using the authors' specification for the control group. The results of this empirical exercise are available on request.