

Response to Invited Commentary

Parker et al. Respond to “Preeclampsia Risk After Induced Abortion”

Samantha E. Parker*, Mika Gissler, Cande V. Ananth, and Martha M. Werler

* Correspondence to Dr. Samantha E. Parker, Department of Epidemiology, Boston University School of Public Health, 715 Albany Street, Boston, MA 02118 (e-mail: separker@bu.edu).

Initially submitted June 25, 2015; accepted for publication June 29, 2015.

We appreciate the comments by Basso (1) regarding our study of induced abortion and the risk of preeclampsia (2). Basso raised concerns that our estimated decrease in risk of preeclampsia in women with a prior induced abortion compared with those without induced abortion might represent an overall effect of gravidity because the comparison group was largely comprised of women with no prior pregnancy. This concern does have implications for understanding the respective roles of previous induced abortion, spontaneous abortion, and other pregnancy outcomes in the development of preeclampsia. It should be noted that in these data, the association between history of spontaneous abortion and preeclampsia was null, and indeed the crude odds ratio for comparison of induced abortion relative to spontaneous abortion for preeclampsia resulted in the same point estimate of 0.9, as did the comparison that we presented. A secondary aim of our study was to consider a possible role of endometrial injury in the development of preeclampsia by separately examining surgical and medical induced abortions. The issue of gravidity should remain less of a concern here because both point estimates would be equally affected by a bias and could still be compared to each other.

Concerns regarding the role of confounding as an alternative explanation, particularly confounding due to smoking status and fecundity, were also raised. We observed no material confounding by smoking during pregnancy. Nevertheless, data on smoking during pregnancy were ascertained through maternal self-report, and although excellent agreement between these data in the birth register and data from questionnaires and medical records was reported in a small validation study, the possibility of misclassification cannot be overlooked (3). Information on fecundity was unavailable. Residual and unmeasured confounding do pose threats to the validity of our findings and remain a limitation of this research.

We appreciate Basso's recognition that our study provides additional support for an association between induced abortion and a reduced risk of preeclampsia, although the mechanism through which this operates remains unclear. Ultimately, one of the major strengths of our study was the utilization of a nation-wide register of induced abortions in Finland, arguably

one of the most valid and complete data sources of its type. Although data on spontaneous abortions were likely incomplete, most notably with regard to early losses, the information is expected to be of a quality similar to that of existing registries and cohort studies that collected data on spontaneous abortion through a combination of medical records and self-report. Studies in preconception cohorts should offer improved collection of these data. Given the considerable strengths and improvements over previously published work on this topic, we speculate our study may be “as good as it gets” for now. Along with Basso, we look forward to studies with more detailed information on fecundity and early spontaneous abortions to better understand this association.

ACKNOWLEDGMENTS

Author affiliations: Department of Epidemiology, School of Public Health, Boston University, Boston, Massachusetts (Samantha E. Parker, Martha M. Werler); National Institute of Health and Welfare, Helsinki, Finland (Mika Gissler); Nordic School of Public Health, Gothenburg, Sweden (Mika Gissler); Department of Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University, New York, New York (Cande V. Ananth); and Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, New York (Cande V. Ananth).

This work was supported by the National Institutes of Health (grant T32 HD052458 to S.E.P.).

Conflict of interest: none declared.

REFERENCES

1. Basso O. Invited commentary: induced abortion and risk of preeclampsia in a subsequent pregnancy. *Am J Epidemiol.* 2015; 182(8):670–672.

2. Parker SE, Gissler M, Ananth CV, et al. Induced abortions and the risk of preeclampsia among nulliparous women. *Am J Epidemiol.* 2015;182(8):663–669.
3. Jaakkola N, Jaakkola MS, Gissler M, et al. Smoking during pregnancy in Finland: determinants and trends, 1987–1997. *Am J Public Health.* 2001;91(2):284–286.