

# Authors' response to: Cohort effects explain the increase in autism diagnosis: an identifiability problem of the age-period-cohort model

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Hansen & Parner<sup>1</sup> conclude that when applying age-period-cohort analyses, the assumptions that we make about model constraints can have a pivotaleffect on the results obtained. They demonstrate this using data from our study<sup>2</sup> of autism diagnoses in California from children born from 1992 to 2003. We concur; all age-period-cohort models have identification problems, and thus the model alone cannot guide us. We used a classical constraint-based method, but even modern methods such as the intrinsic estimator<sup>3</sup> and hierarchical age-period-cohort<sup>4</sup> approaches have been under fire for potential bias due to unverifiable assumptions about underlying data structure.<sup>5,6</sup> Thus, whereas Hansen & Parner are correct that one could constrain the model in such a way as to change the interpretation of the results, this is certainly not an issue unique to our data or our analytical approach.

Hansen & Parner show that one could generate a model in which autism diagnoses trends are explained by a period-effect rather than a cohort-effect, if one assumes a linear negative slope between ages 8 and 12 years. However, it is unlikely that autism diagnoses have a linear negative relationship with age over this developmental time period<sup>7</sup> and thus the validity of using such a constraint is dubious. Further, a period-effect in autism diagnoses would imply that all children, regardless of age, experience the same relative increase in autism diagnoses across time. Autism diagnoses most frequently occur between the ages of 3 and 6 years,<sup>8</sup> however, and few diagnoses are made after age 9 years. Although a period-effect would not change the underlying distribution of relative differences in diagnoses across age (those aged 3–6 years more likely to be diagnosed than other groups), we should see an increase in diagnoses across all age groups. Given that there is limited evidence for such an increase, the plausibility of a period-effect explaining the increase in autism diagnoses is weak, even in the presence of a model that suggests it.

We approached the issue of model building and inference for our study in several steps. First, as Hansen & Parner note, we used the graphical displays of the data to guide model building. Second, we corroborated the robustness of the model using various analytical strategies with different underlying assumptions. Finally, we used our knowledge of the epidemiology of autism and the graphical data

to guide the interpretation of the model. This last step is critical to successful inference in age-period-cohort studies.

Identification and validity issues in age-period-cohort analyses will continue to be an important discussion. Perhaps equally important, however, is not getting mired in the intractability of the identification problem in age-period-cohort models, given that one can always tweak assumptions to generate different sets of results. Using age-period-cohort analyses as a guide, with substantive knowledge of the field and a priori hypotheses about the structure of time trends, is critical in producing age-period-cohort research that informs our understanding of the aetiology of illness.

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