Authors are always grateful to those who spend time and effort commenting on papers; in this case, we are exceptionally grateful that many of the leading figures whose work has advanced our understanding of autism so profoundly gave so freely of their thoughts. Our response will be brief and oriented towards the future more than the past.

The goal we all share is to understand the increased prevalence of autism and the first, and most important, step is to build integrated data structures that allow one to decompose the increase in prevalence into its constituent elements—those parts that arise from diagnostic and social dynamics, environmental changes, genetics and so on. All of these causal mechanisms are real; and all of them play some role in driving increased prevalence. The major scientific and population health questions at hand are: first, ‘How big a role each of the factors play?’ and secondly, ‘What can be done?’.

When scholars and observers ask ‘Has there been a real increase in incidence and if so, why?’¹ and ‘whether there has been a true increase over time in the incidence of autism’;² they are really trying to understand if there are new causes of autism that do not arise from ascertainment because if there are new causes then we can do something about them.

We would all like to understand what causes autism. At the moment our progress is stymied for a number of reasons. One reason is that, to date, prior studies of risk factors associated with autism have pooled observations over successive birth cohorts. Pooling observations across successive birth cohorts, as we show in other work,³ can lead to miss-estimation of risk. This article attacks another source of heterogeneity—processes that yield diagnosis of autism. It will be hard to make progress until we develop strategies for chasing heterogeneity—spatial, temporal and diagnostic—down.

Why are diagnostic dynamics important to understand? It is not that hard for us to imagine that there is an environmental cause of autism, the cause is distributed unevenly across space (i.e. it is not a universal treatment) and we do not know what it is. If those who achieved their diagnosis through accretion on the mental retardation (MR) pathway as a consequence of changes in diagnostic criteria are spatially clustered where the toxin is absent and we compared the incidence of autism across all communities, we would fail to see the toxin! Without identifying those that achieved an autism diagnosis through accretion, we would have absolutely no idea that an environmental factor played a role in causing autism.

If the world were this simple, everyone but scientists—who would have little to do—would be happier. But because the world is not so simple, the first steps of scientists have to be small. We can never get to cause if we do not tackle heterogeneity first. This article takes early steps in that direction by estimating the contribution to the autism caseload that arises from diagnostic accretion on the MR pathway. That contribution is significant, but it leaves 75% of the cases still unexplained. Our expectation is that we can do a better job by explaining the remaining 75% if we set the predicates up with more care. This less elegantly captures one of the central points raised by Dr Fombonne in his comment with which we are in complete agreement.

Understanding diagnostic dynamics is important at all locations on the severity distribution. There is no reason to privilege one location over another.

Dr Rutter concludes his comment by noting that: ‘the greater need is for hypothesis-testing focused research on possible causal mechanisms that could lead to changes in incidence’. We completely agree. The idea behind this article was ‘let us do that testing on data that work’. And one way to get data that work is to sweep away heterogeneity, so we can really identify cause.

Charman et al. provide fascinating confirmatory results to those offered in our article, and we are very pleased that our contribution helped in bringing these results to light, however indirectly. Different data structures allow for different insights. We cannot achieve the fine-granularity of the results
described by Charman et al., but we can observe the morphological patterns in diagnoses over time in a large representative delivery system. The morphological patterns we describe are consistent with those arising from the detailed analyses reported by Charman et al. As Charman et al. note, getting the right answers involves complementary data structures and analysis strategies. Special issues, such as this, provide a framework for such complementarities to develop.

In other work, we have focused intensely on the individual-level and community-level factors that yield a diagnosis of autism. This is not the place to review these results, but of greatest interest to us has been how community-level factors—especially those related to the socio-economic status (SES) composition of neighbourhoods—play different roles at different moments in time (K Marissa and P Bearman, 2009b, submitted for publication).

Echoing the comments of Dr Rutter, Charman et al. ask: ‘Has the prevalence of autism/autism spectrum disorder (ASD) increased?’ and call for a research design that helps us know the answer to this question. Our article provides an answer to this question, and that answer is ‘yes’. However, we do not know how much of the increased prevalence arises from the ASD side of the spectrum, for those individuals who are functioning at the highest. This article tackles only the other side. And we agree with Dr Hertz-Picciotto that the task is already daunting.

Dr Hertz-Picciotto finds our results plausible, and in fact they echo those reported in her own analyses. She also finds our methods suspect. Not surprisingly, we disagree that our methods are suspect. We took great care to test the robustness of our model in a variety of ways. The results were always similar, increasing our confidence in our methods. Our goal is to understand the dynamics of diagnostic accretion. We include MR severity in our model as a control variable because MR severity changes over time, and because it makes no sense to think that the probability of accreting an autism diagnosis is independent of severity—just as it makes no sense to think that diagnostic accretion for autism would be independent of MR aetiology. This comment aside, as Dr Hertz-Picciotto notes, this issue is absent in our supplementary analysis that reports complementary findings.

Hertz-Picciotto is right that our Figure 2 reports cumulative probability of change. Each year that passes generates a new risk for diagnosis; those who missed in the year before are at risk, usually but not always at higher risk, subsequently. That is history and that is what we are modelling. Thirdly, if the world turned upside down and the probability of a diagnostic change from autism to MR was greater than the baseline rate of change from MR to autism, our netting out strategy would yield a ‘negative probability’. Of course, there are no such things as negative probabilities; but in this substantive context—that would be precisely ‘right’—people with autism would flow into the MR population. The world is not upside down, and in our data we observe only a handful of persons moving from autism to MR over our entire observation period.

Dr Hertz-Picciotto notes that we assume—with empirical support—that 8% of the MR cases also have autism. We test and present in the article the impact of this assumption on our results and show (within reasonable estimates) the magnitude of the effect. Calculating the changing caseload based on MR cases (which does not rely on this assumption) reveals roughly the same impacts of changes in diagnostic criteria (26 vs 31%). Finally, Dr Hertz-Picciotto is right that our model presumes that changes in criteria are expressed in diagnostic behaviour in the year when the criteria are changed. We observe some evidence of a lag, but our sense is that in a system as well developed as in California new criteria—which are often the products of intense public debate and discussion—are well known in advance and diffuse rapidly. Future work can test this with more refined measures and methods.

Authors often have the sense that no one reads their papers—even those who subsequently cite them. We are honoured that four distinguished scholars took time to read and think critically about our article. As we do not agree on all the details, we do agree on the main scientific problems that need to be solved. And we agree on a central mechanism for solving those issues—collaboration across diverse communities of scholars deploying diverse analytic strategies and data structures. This special issue contributes to the collaborative activity that promises the opportunity for new solutions and visions to improve population health.

Conflict of interest: None declared.

References

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