Badawi and colleagues (p 85) report on a positive association between newborn encephalopathy (NE) in term neonates and the risk of autism spectrum disorder (ASD). The study they conducted has several strengths worth mentioning that include the population-based nature of their sample, the systematic ascertainment of cases of NE in a well-defined population, the selection of a proper control series, and a comprehensive independent assessment of case status at follow-up.

The authors report a rate of ASDs of 5%, which represents a six-fold increase in the risk of ASDs in newborns with encephalopathy as compared with study controls and with known population estimates from Australia and other countries. Although based on a relatively small sample of cases, the increase in risk was statistically significant and, furthermore, being able to compare their case series to both controls and newborns with NE but without autism, the authors could assess the specificity of risk factors and documented that episodes of antepartum bleeding, trauma, or infection during pregnancy were mediating this risk association.

The findings are important for two reasons. First, they point towards insults occurring during gestation in the fetus as important mechanisms leading to neurodevelopmental disorders, and in particular, to ASDs. This is consistent with previous studies of obstetric and prenatal complications in autism which identified increased rates of adverse events during pregnancy (such as bleeding during the second trimester) and other prenatal events. It is also noteworthy that the environmental risk factors which have been associated thus far to ASDs consist of prenatal exposures to particular teratogens that include thalidomide, valproic acid, or misoprostol. Although these exposures or adverse events might explain only a relatively small proportion of the overall population risk to autism, they are important pointers towards possible biological mechanisms underlying susceptibility to autism and indicate that fetus maldevelopment is a likely pathway to autism, even though the onset of first symptoms as recognized by parents might only occur in the second or third year of life.

Unfortunately, and as discussed by Badawi and colleagues, it is not possible from their study and the present state of knowledge to differentiate in the causal pathways to ASD the respective contribution of genetic factors and of early prenatal environmental insults, and to evaluate their interactions. The findings, however, draw attention to the immediate need to detect deviant development at an early age in vulnerable infants.

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