

Early Media Exposure and Autism Spectrum Disorder Heat and Light

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The rising prevalence of autism spectrum disorder (ASD) has given pause to many epidemiologists and spawned aggressive attempts to search for an explanation. Whatever the underlying genomics of autism are, they cannot explain the increase because they have remained largely unchanged while the incidence



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has steadily risen. Hence it seems logical to think of environmental perturbations that have occurred concomitant with this rise that might plausibly be linked to it and would lend themselves to modification or remediation. Notably, the rise in ASD incidence began in earnest in 1996.¹ What else began in the early 1990s? Infant television viewing. In 1970, the mean age at which children began to watch television was 4 years; by 2006 it was 4 months.² Ecological associations are always fraught and problematic, but there are compelling theoretical reasons to believe that a potential causal linkage may exist between excessive early exposure to media and developmental outcomes in children, many of which are enumerated by Heffler et al³ in this issue of *JAMA Pediatrics* as motivating their study.

I think of media's potential effects on child development as playing out via 2 pathways. The first is the direct one: how does *what* a child watches—or the actual experience of watching—affect their development? This pathway is mediated by the content viewed. In the case of infants, who do not cognitively process what they watch, the direct pathway relies primarily on the formal features of the programs—the pacing and the edits—which for infant shows are unusually rapid and abrupt.⁴ The second pathway is the indirect one and is mediated largely by displacement. Simply put, there are only so many hours in a day—young children are only awake for 10 to 12 of them—and time spent with media comes at the expense of time spent in other activities: reading, physical play, social interactions, etc. From this perspective, even harmless or arguably educational content can have untoward effects if it crowds out other critical activities that drive children's cognitive, social, and emotional development.

As a general editorial principle, *JAMA Pediatrics* looks less favorably on studies that use nonexperimental designs when experimental ones are feasible. Such is not the case here. It is neither practical nor ethical to randomize children to prolonged or no early media exposure and assess their developmental outcomes years later. Accordingly, Heffler and colleagues³ tested early environmental exposure and ASD traits and diagnoses using observational data collected as part of the now defunct National Children's Study (NCS). Notably, the NCS was conceived specifically with the intention of assessing how environmental exposures might affect child development and

health. It is most unfortunate, then, that for this key and dramatically changed environmental exposure, media, the measurement procedure was not sufficient. It was flawed because it was not collected with sufficient granularity to assess direct effects.⁵ After 1 year of age, we know how much media children in the NCS watched, but not *what* they watched, and it was flawed because the data were collected initially too late (at 1 year of age, when most children are already regularly watching), hampering the ability to assess indirect effects. Diminished interactions with caregivers because of time spent with media likely exert greater effects before 1 year of age. Furthermore, with respect to these indirect effects, the models in the report may be overspecified. Play and media are included simultaneously in regression models, which is appropriate for assessing direct effects but may overcontrol for indirect effects because diminished "face time" may be in the causal pathway. Finally, the relatively limited sample size available for these analyses may have reduced the power to detect effects, particularly with respect to diagnoses of ASD rather than symptoms of it. However, it also may be that the effects of increased media or decreased play on ASD are seen primarily at the higher-functioning end of the spectrum, which is where most of the increase in prevalence has occurred. In a larger sense, the findings suggest that there may be multiple pathways to developing ASD, some genetically predetermined, others largely environmental, and still others that rely on an admixture of both.

What can we deduce from the study, given these irresolvable limitations based on constraints imposed by the existing data, and more important, what are its implications? Science is an iterative process. The findings are provocative and clearly motivate additional study. Unfortunately, this would require a new large, better-specified longitudinal study. The Healthy Brain and Child Development Study funded by several affiliates of the National Institutes of Health is modeled on the hugely successful (thus far) ABCD (Adolescent Brain Cognitive Development) study and is preparing to launch. With the correct sample size and data collection, that study may shed much-needed light on these findings and the heat this question has generated. In the meantime, parents may rightly ask what they are to do now, given that waiting for answers 5 to 7 years before acting is hardly helpful to those with young children at present. The short answer is to follow Hippocrates' mantra to first do no harm. The American Academy of Pediatrics Media and Young Minds statement recommends no digital media before 18 months of age, given the absence of demonstrable benefits and the theoretical and limited empirical suggestion of harm.⁶ This study adds additional credence to those recommendations.

ARTICLE INFORMATION

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