dangers of jaundice are accompanied by reassurance that jaundice is almost always harmless is currently commonplace. Reassurance is warranted but is perhaps more appropriately conveyed by emphasizing that effective treatment is available to prevent injury if jaundice is identified and care secured in a timely fashion. The parent guide to newborn jaundice safety of the Centers for Disease Control and Prevention effectively incorporates these bilirubin neurotoxicity concerns.[7]

It is important to recognize that adherence to birth hospital discharge instructions is not entirely a parental responsibility. Failure of compliance can be multifactorial. Improved parental instruction must be combined with efforts to reduce or eliminate community and institutional barriers that impede access to care, and providers must always act on parental jaundice concerns.

As the Nigerian experience suggests, we can all do better. ■

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Closing the Gap: Interventions to Ameliorate Inequities in Early Brain Development and School Performance in Poor Children

Poverty-related disparities in early brain development and school performance are a pervasive problem in our society, with potential life-long consequences. We know these disparities start early. By the child's first birthday and well before that, lower cognition and language can be documented in children of low socioeconomic status (SES) compared with their higher-SES peers.[1] There has been a growing desire to start early in infancy to intervene to ameliorate these differences. The 2014 American Academy of Pediatrics Policy Statement on literacy promotion in primary care pediatrics suggested that literacy promotion start at the first postpartum visit.[2] Reach Out and Read (ROR), which a large, widespread, early literacy intervention program, traditionally has provided counseling and books starting with the 6-month primary care visit; however, ROR reacted to the American Academy of Pediatrics Policy by providing counseling materials for pediatricians for earlier visits and suggested giving out books as early as the visit at 1 week of age.[3]

See related articles, p 72 and in *The Journal of Pediatrics: X* https://doi.org/10.1016/j.ympdx.2020.100020

In 2 studies published in this volume of *The Journal of Pediatrics* and in *The Journal of Pediatrics: X*, groups of investigators pondered this question from different points of view. Leung and Suskind looked at parental knowledge at the 1-week newborn visit and compared that with caregiving behaviors (responding to infant cues and fostering social–emotional and cognitive growth) at the 9-month visit.[4] They found that early parental knowledge partially mediated the strong impact of SES on these parenting behaviors at 9 months, especially fostering cognitive growth. Early knowledge did not mediate responsivity and promotion of social–emotional growth. As the authors point out, even the finding of significant mediation does not prove that increasing early parental knowledge per se (eg, with counseling, classes, or written material) will necessarily change parenting behaviors. It is likely that interventions that focus on improving parent–child relationships,

ROR Reach Out and Read
SES Socioeconomic status

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and empower parents by imparting skills and strategies, will be more impactful.

Guevara et al tested the new recommendations that literacy promotion such as ROR start earlier in infancy than 6 months.[5] They compared children randomly assigned to receive ROR starting at the 1-week visit (early) with those receiving ROR starting at 6 months (standard). Main outcomes were richness of home reading environments and language development measured when children were 6 months and 24 months of age. They found that whereas the home reading environment was significantly richer at 6 months for those children who received early ROR, that effect faded by 24 months and at both 6 and 24 months of age language scores were not different. The authors point out that their measure of infant language, designed to identify children with language delays, may not be sensitive enough to measure small differences of language development in infancy and early childhood.

How should we incorporate the results of these 2 studies in our efforts to reduce disparities in child development based on SES? Here are some important points:

You cannot start too early in childhood to intervene to decrease disparities based on poverty. These 2 studies provide somewhat-conflicting evidence regarding the necessity to begin interventions right after birth to improve the reading, cognitive, and parenting home environment. We know that environmental influences as early as the prenatal period (ie, maternal stress while the child is in utero) impact brain growth in the fetus and infant. It is likely, therefore, that the early environment the infant is exposed to after birth is likely to be important for language, cognitive, and social–emotional development. It is prudent for us to intervene as early as possible.

Pediatric primary care is a universal and cost-effective platform for interventions. Pediatric primary care is uniquely situated to provide almost universal access to children starting at birth through the first 5 years of life for interventions to improve parenting and early brain development. There is a total of 13-15 visits during this time period, which allows for a significant "dose" for interventions that are provided. Because of the expansion of public health insurance, almost all poor children are insured and able to have visits for primary care. The primary care setting also has the advantage of being low cost, because the parent is already at the visit, and space is available at no cost. Finally, the relationship of the family with the pediatrician can be leveraged to enhance the impact of the intervention, because it is here that expected counseling occurs in a trusted medical home. In addition to ROR, the Video Interaction Project is an evidence-based intervention provided within pediatric primary care at every preventive visit. It has been shown to enhance parenting and cognitive stimulation in the home, including verbal responsivity, reading, teaching, and availability of learning materials and lead to enhanced socioemotional development and better language and cognition in low-SES children.[6,7]

Income matters. There is substantial causal research showing that increased income in and of itself is associated with better early childhood outcomes in poor children. The National Academies of Sciences, Engineering, and Medicine report, *A Roadmap to Reducing Child Poverty*, documents the causal evidence for the importance of family income, as well as lays out proposals to reduce child poverty in one-half in 10 years.[8] These efforts need to be part of any program to reduce inequities in developmental and academic outcomes for poor children.

Addressing the impact of racism and bias. Many poor children are likely to be children of color: African American, Latinx, or Native American. We know that racism is experienced as an adverse childhood experience by these children and leads to negative consequences for their physical and mental health as well as their academic success. There is both structural racism and interpersonal bias that is experienced by these children. Combatting racism and bias in all their forms is another essential part of our attempts to reduce inequities of outcomes of poor children.[9]

No one intervention will be strong enough by itself. Finally, we know that each of the interventions we have to help poor young children is not strong enough by itself to create equity between them and children living in greater-income and educational households. Neither interventions in primary care, nor high-quality preschool, nor income supports, nor addressing racism and bias will be enough as individual interventions. We need to combine them if we are going to make significant progress in closing the gap between the chances poor children have for a successful life compared with their greater-income peers. Among other important endeavors, we need to perform research in scalability of interventions and determine the best way to implement multiple programs simultaneously. We must make this a national priority because there is no time to spare! ■

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Never Exposed to Nicotine - But Still Impacting Executive Function

It would be reasonable for the busy reader's initial response to the report[1] in the current volume of *The Journal*, "Impact of Early Low-Level Tobacco Smoke Exposure on Executive Function at Age 8 Years" to be an exasperated sigh. "Haven't we known about the early developmental consequences of smoking since the 1964 Surgeon General's Report?"[2]

See related article, p 174

Like birth weight and prematurity, neurobehavioral function can be influenced by a multitude of factors. Executive function is a broad term frequently used by neuropsychologists and behavioral neurologists to refer to higher order cognitive processes, including attention, working memory, judgement, planning, cognitive flexibility, decision making, and impulse control. Executive function is a component of attention deficit hyperactivity disorder and other psychiatric conditions. Just because maternal genetics contributes to executive (ie, frontal lobe) function, does not negate the adverse, and dose-dependent, potential of teratogens.[3] This prospective cohort of 239 children, 87% of whom had mothers who self-reported as nonsmokers during pregnancy, included repeated measurements, 3 prenatal and 4 postnatal, of the nicotine metabolite cotinine. The dependent measure selected, the parental rating form of the Behavioral Rating Inventory of Executive Function (BRIEF), has excellent reliability and validity.[4] The finding that higher cotinine levels, both prenatally and postnatally, correlated with more problems with executive function in these children, particularly in the initiate domain, is an important extension of prior investigations.[5,6] Perhaps most interesting, however, was that this pattern was present even in children whose mother did not report smoking. This finding has public health significance and contributes to our understanding of the under-reporting of exposure to, and risks associated with, second-hand smoke.

This study reinforces that prenatal and postnatal exposure to nicotine, even in small quantities or by second-hand contact in nonsmokers, can have measurable effects on neurobehavioral outcomes. The finding that 89% of children had detectable cotinine, despite 76% reporting no exposure, is equally alarming. It should strengthen our resolve to dig deeper into the possible exposures that are incurred by pregnant mothers and young children, and it should encourage us to educate mothers about these effects, both during pregnancy and during early childhood.

This report did not assess the role of vaping nicotine and studies will be necessary to determine if it causes similar effects. Although this work may be challenging owing to the wide variety and rapidly changing vaping products, as well as dual use with cigarettes, cotinine measurement may provide a valuable approach to begin to side-step these concerns. An elevation of one-half of an SD as was observed here may have appreciable consequences at a population level.[1] An advantage of using standardized instruments like the BRIEF is that the findings can be expressed in terms of clinically significant abnormalities in executive function which differ by 1.5 or more SDs or raw scores of greater than 65.[5,6] A prior report from Germany determined that self-reported maternal smoking resulted in an OR of 3.8 for clinically significant executive function problems in the inhibit domain.[6] Perhaps not coincidentally, stimulant use from 2006 to 2016 in the US doubled, which may be driven by increasing rates of attention deficit hyperactivity disorder in females and adults.[7] The Medicaid spending in 2015 alone for amphetamine, methylphenidate, and lisdexamfetamine approached $2 billion.[7]

Another future direction will be to take advantage of the teacher and self-report versions of the BRIEF and determine the generality and persistence of these results.[1,4,6,8] Similarly, characterization of the potential neurodevelopmental risks of prenatal exposure to nicotine replacement therapies is also needed. Real-world executive function as measured by the BRIEF is sensitive to a wide variety of prenatal and perinatal exposures.[9-12] A more comprehensive appreciation of the risks of nicotine and second-hand smoke exposure may

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