Chapter 10
Conclusion: Common Themes and Directions for Future Research

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Common Themes

Across the research on mechanisms of impact and effects of neurotoxins, there is a consistent pattern of heterogeneity of effects for children exposed. For some toxins with in utero effects, including many medications prescribed to address maternal health (Chaps. 5 and 6), there is minimal research of sufficient methodological rigor to allow for firm conclusions to be drawn. This limited research is also the case for in utero effects of opiate and marijuana use, especially with regard to specific academic outcomes among children exposed (Chap. 4). This is contrasted by the relative wealth of information available on FASD (Chap. 2) as well as research related to stimulants (particularly nicotine and cocaine; Chap. 3). Despite considerations for smog and pollution (Chap. 9), pesticides (Chap. 7), and lead exposure (Chap. 8), it is difficult to describe trends and patterns in the literature regarding neurodevelopmental effects due to inconsistent results across studies.

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C. Riccio, J. Sullivan (eds.), *Pediatric Neurotoxicology*, Specialty Topics in Pediatric Neuropsychology, DOI 10.1007/978-3-319-32358-9_10

Moderating Factors

The inconsistency in research findings may be due to differences in specific substances, but also suggests the need to identify potential moderating factors that may help predict outcomes among individual children. These factors include timing of exposure and extent of exposure. For example, Loring et al. (Chap. 5) noted that exposure to valproate in the first trimester of pregnancy is associated with anatomical abnormalities, while exposure during the third trimester is associated with functional/behavioral deficits. Similar timing and extent of exposure effects have been demonstrated for alcohol and resulting severity of FASD (see Chap. 2), as well as lead (see Chap. 8) and air quality/pollution (see Chap. 9). With maternal use of recreational substances, the potential for exposure to multiple toxins at similar points in time adds yet an additional confound (see Chap. 4).

Additional risk and protective factors identified in the previous chapters are more systemic to the individual rather than the toxin. Genetic variations and differences in genetic vulnerability/predisposition may contribute to differences in manifestation. For example, the risk of a child developing FASD is influenced by the mother's ability to metabolize alcohol, which is genetically determined (Jones, 2011). As another example, effects of prenatal SSRI exposure on child behavior and mood may be influenced by genetic differences in a serotonin transporter gene (serotonin transporter promoter SLC6A4 genotype) within the exposed child (Oberlander et al., 2010; Chap. 6).

Socioeconomic status (SES) and resulting access (or lack of access) to resources and social supports, as well as exposure to (or lack thereof) an enriched environment also may impact outcome. For children with delayed or atypical developmental trajectories, early identification and early intervention efforts have been found to be critical. For example, among children with FASD, early identification is associated with significantly lower risk for adverse life experiences such as legal issues and substance abuse (Chap. 2). Further, early identification is necessary to prevent ongoing exposure to environmental neurotoxins (e.g., lead, pesticides, pollutants). As the child ages, parental involvement, parental psychopathology, parental substance use, maternal self-esteem, general nutrition, parent stress, and child abuse and neglect also can moderate outcome (Chaps. 2–4, and 8). Taken together, these moderating factors may explain the wide degree of variability in neuropsychological functioning among children who have been exposed to these toxins, such as the finding that effects on cognitive functioning may be long-lasting for some children, while others may "catch up" over time. Although it is difficult to account for all of these possible moderating factors when conducting a neuropsychological or psychoeducational evaluation or gathering developmental history information, this information will be important as part of the diagnostic process and for drawing inferences with regard to potential later difficulties.

Diagnostic Considerations

While there is variation in presentation, research suggests that neurotoxin exposure often results in referral for evaluation, often due to developmental delays or behavioral or academic concerns. Comprehensive assessment is necessary to identify individual strengths and weaknesses, and services must be individualized based on that profile of strengths and weaknesses. Knowing that a child has been exposed to a given toxin or toxins, and those functional systems likely affected, can help with tailoring the assessment and intervention to ensure that these areas are considered. For example, children with FASD, children who have a history of prenatal exposure to AEDs or stimulants such as nicotine or cocaine, and children with postnatal exposure to pesticides or lead often exhibit deficits in executive function. It would be important then for executive function to be assessed for children with those histories and monitored over time as classroom demands require more executive function skills. Conversely, children with learning disabilities, intellectual disabilities, and behavior disorders are at increased risk of abuse and neglect by parents (Sullivan & Knutson, 2000) in addition to bullying from peers (Blake, Lund, Zhou, Kwok, & Benz, 2012), so service providers should monitor closely for signs of maltreatment and bully victimization.

As indicated across chapters, neurotoxin exposure often results in deficits and possible diagnosis. For example, FASD and in utero exposure to stimulants are associated with high rates of externalizing issues such as attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD), which are often the reason for initial seeking of services (see Chaps. 2 and 3). In addition to deficits in intellectual functioning, FASD is associated with deficits in psychosocial functioning, motor skills, balance, language, executive function, vision, hearing, academic performance in math and reading, and internalizing issues such as depression, self-harm, and suicide. Valproate exposure is associated with lower cognitive ability and possible intellectual disability, as well as deficits in verbal skills, adaptive behaviors, social skills, memory, executive function, and increased risks of ADHD and ASD have been reported in the literature (see Chap. 5). Children exposed to cocaine in utero are more likely than their non-exposed peers to receive special education services and are at higher risk for problems with aggression, substance use, and language development (Chap. 3).

Similarly, recent studies suggest some of the neuropsychological domains impacted by pesticide exposure include motor skills, memory, and executive function (Chap. 7). Exposure to air pollution has been associated with deficits across a range of neuropsychological constructs and emotional/psychiatric symptoms, including overall intellectual functioning, motor skills, language, adaptive behavior, social functioning, and sensory abilities such as hearing and smell. There may be an association between early exposure to pollutants and symptoms of ADHD and ASD, and attending schools located in areas with high levels of air pollution has been associated with lower academic performance (in addition to increased absenteeism), even after controlling for sociodemographic variables (Chap. 9). Villarreal

and Castro (Chap. 8) reviewed the literature on lead exposure and performance on tests of intellectual functioning, academic achievement, and executive function. Lead exposure is related to externalizing behaviors with diagnoses of ADHD, ODD, and CD common, all of which are related to problems with behavioral and emotional regulation. Although it is less direct, prenatal exposure to opioids is linked to low birth weight and other medical issues (Chap. 4), which in turn are associated with a multitude of neuropsychological difficulties (Riccio, Sullivan, & Cohen, 2010). Thus, despite variations in impact, children exposed to neurotoxins are more likely to need support and possible special education services in school than non-exposed children.

Prevention and Intervention

Major components of preventive efforts include psychoeducational approaches and the policies developed as a result of research. For example, it is critical that parents and other family members, as well as officials responsible for schools, daycare centers, and other settings be informed of the dangers of pesticides so they can help prevent exposure (Chap. 7). Prevention efforts based on reducing lead (e.g., in gasoline, in paint) have been largely effective in reducing exposure, but also may generate a false sense of security leading people to believe that lead exposure is no longer an issue. The effects of air pollutants on respiratory and cardiopulmonary function have been well documented, and increased attention is now being paid to the impact of air pollution on neurodevelopment, as extremely small particles are able to cross the blood–brain barrier and enter the brain via respiration. As with pesticides and lead, exposure to pollutants can occur prenatally as well as throughout the lifespan, so exposure may be ongoing. For example, studies of cohorts of children in certain geographic areas have found that they are impacted by pollutants in the air related to nearby factories, smog, power plants, and traffic-related air pollution (see Chap. 9). Thus, parent training and education are necessary to prevent both in utero and postnatal exposure to neurotoxins. For potential effects in utero, folate supplementation as an intervention is recommended for women who are pregnant and using AEDs; however, the supplementation has not been shown to completely prevent the teratogenic risks associated with AEDs (see Chap. 5).

Education and training programs targeting parents should also include training for physicians (e.g., obstetricians, gynecologists) in how to work effectively with mothers to eliminate exposure to harmful substances during pregnancy. Most doctors will advise against the use of alcohol or recreational drugs if pregnancy is anticipated or once the pregnancy has been confirmed (e.g., Carson et al., 2010; Centers for Disease Control and Prevention, National Center on Birth Defects and Developmental Disabilities, 2005). Since AEDs are used to control a legitimate medical issue (as opposed to used recreationally), their use presents an interesting dilemma. Sudden discontinuation of AEDs during pregnancy can be harmful to both mother and child, so professionals must work closely with the mother and the

mother's physician when making decisions about continuing versus discontinuing treatment. Ideally, the physician will work to find the smallest effective dose to treat the symptoms of epilepsy while also minimizing risk to the fetus (see Chap. 5). Similarly, Power et al. (Chap. 6) addressed the use of antidepressants, antipsychotics, and lithium during pregnancy. Use of these medications during pregnancy is becoming more common with the prevalence of mental health issues among women who are pregnant. As with the continuation of AEDs, there is a difficult balance between the importance of treating mental illness among women who are pregnant and preventing harm to the developing fetus. For example, many physicians may recommend discontinuing the use of SSRIs during pregnancy due to potential teratogenic effects, but untreated maternal depression can also be harmful to the developing child (Hermansen & Melinder, 2015). Ideally, these issues would be articulated before pregnancy or early in the pregnancy.

Early Identification and Early Intervention

Early identification and early intervention make a significant contribution to reducing the effects of many risk factors (e.g., Kilgus, Riley-Tillman, Chafouleas, Christ, & Welsh, 2014; Oberklaid, Baird, Blair, Melhuish, & Hall, 2013; Stormont, Reinke, Herman, & Lembke, 2012). For example, research consistently supports the importance of early identification of children at risk for reading difficulties as a means of preventing more serious reading problems (Catts, Nielsen, Bridges, Liu, & Bontempo, 2015). Similarly, early intervention to address behavioral problems is a means of preventing more problematic behaviors (Greenwood, Kratochwill, & Clements, 2008; Stormont et al., 2012). Research suggests that children below 2 years of age receive fewer services than older children and children of high need parents are less likely to use the services available to them through the Individual Family Service Plan (Block, Rosenberg, Kellar-Guenther, Robinson, & Goetze, 2015). While family demographics have not consistently been found to be a factor in early intervention services, a necessary first step is that parents be referred to early intervention services. As the referral source for infants and toddlers is most often the pediatrician, it is important for medical personnel to be aware of prenatal and postnatal exposure, as well as the possible signs and symptoms of delayed development. It is critical for these professionals to identify the need for close follow-up, especially among children with additional biological risk factors (e.g., low birth weight). At the same time, the heterogeneity with which children exposed to neurotoxins often present points to the need for appropriate assessment methods that can identify these children with adequate reliability, sensitivity, and specificity.

Analogous to newborn screening for various medical conditions, some preschool programs and many school districts use brief universal screeners for academic and/or behavioral status in order to provide supportive services from a preventive perspective. The use of screeners for emerging and developmental problems is still controversial, in part because of the dynamic nature and individual variation in child

development particularly prior to age 3, the limited focus of the screener used, or the length of the more comprehensive approaches (Stormont, Herman, Reinke, King, & Owens, 2015). The use of universal screeners in relation to potential academic problems, usually reading, has been supported in the literature, with provision of intervention services to those identified as at-risk (e.g., O'Connor & Jenkins, 1999). With regard to pollution, Johnson et al. (Chap. 9) speak to the need for screening protocols to identify exposed children, so continued exposure can be mitigated and interventions can be initiated. Similar protocols seem appropriate for other environmental toxins with risk for continued exposure, such as pesticides and lead. In conjunction with universal screening, the need to monitor progress and rate of development in response to intervention is important in determining next steps for support and the appropriateness of formal diagnosis.

Once difficulties have been identified, parent education will be necessary to teach parents how to manage their children's behavior and to advocate for services at school. Chapter 2 describes studies on the effectiveness of several parent education programs (e.g., CHOICES, Step-by-Step, Families Moving Forward), which may serve as useful models for similar program development. Similarly, pediatricians, psychologists, and education professionals (e.g., school counselors, school nurses) should receive continuing education to recognize children who may have been exposed to toxins, as well as methods to maximize positive outcomes for the child, rather than on blaming the child or parents for exposure-related deficits. Services may be provided in general education as a component of early intervention and response to intervention (Gresham, 2014). With continued difficulties, children with neurodevelopmental deficits may be eligible for special education services under the Individuals with Disabilities Education Act (IDEA; 2004). When deficits are apparent, but do not significantly impair educational functioning, support services (e.g., accommodations and modifications) may be provided through 504 services (Section 504 of the Rehabilitation Act of, 1973).

Since children with FASD are a more researched population as compared to children exposed to some of the other neurotoxins covered in this volume, it is not surprising that there are empirically evaluated prevention and intervention programs such as CHOICES, MILE, and other programs targeting academic, social, and adaptive behavioral variables. At the same time, there is still a long way to go in terms of investigating the generalizability and acceptability of these interventions when used with children with FASDs. With the exception of FASD, the research literature provides very few psychosocial or psychoeducational interventions with sufficient empirical support to be considered evidence-based, and this is especially true for school-based interventions. As a result, practitioners are left to apply construct-specific interventions (see Riccio et al., 2010, chapter 18) without empirically supported guidelines. It is important to note that although problems with attention, memory, executive function, motor skills, and so on may look similar across children exposed to different neurotoxins, the differing etiological, neurological, and environmental factors may lead to differential efficacy of interventions. Commonly used interventions for these deficits may need to be modified based on the child's level of cognitive functioning, which is often impacted by in utero

exposure to alcohol or other substances. To be sure, there are excellent resources (e.g., Clay, 2004) that provide very helpful strategies for integrating children with medical issues into the school system. Finally, at least some individuals who experience exposure to neurotoxins will likely need long-term care, even into adulthood, due to impact on cognitive functioning and adaptive behaviors.

Limitations to Existing Research

Power et al. (Chap. 6) describe some of the difficulties with conducting research in pediatric neurotoxicology, including limitations to using the ideal experimental design, difficulty controlling for many variables, lack of random selection, and inability to control dosage or amount of exposure in relation to research on psychotropic medications. These research design challenges apply to all of the neurotoxin research reviewed in this volume. Although many chapters report a dose–response relationship, such that neuropsychological deficits are more pronounced with higher levels of exposure to neurotoxins, it is difficult to control or measure reliably and consistently dose response; the exposure is not likely a single instance, but variable over time. This applies to in utero exposure as well as exposure to environmental toxins postnatally. Further, research is also limited as the same child may be exposed to multiple toxins. For example, the research on the use of opiates, marijuana, and stimulants indicated that many studies involved women who reported using multiple substances during pregnancy (Chaps. 3 and 4). Similarly, a child exposed to pesticides in utero may experience continued exposure postnatally, which makes it difficult to determine when the greater impact occurred.

There also has been minimal control for other moderating factors in many studies. For instance, many studies examining differences due to maternal AED use in children's cognitive ability did not control for differences in maternal cognition. Maternal use of psychotropic medications adds a layer of complexity because it is extremely difficult to determine which developmental outcomes should be attributed to in utero medication exposure versus what should be attributed to the genetic predisposition to depression, anxiety, and other forms of psychopathology.

Research findings on developmental outcomes are extremely inconsistent, with some studies finding differences compared to control children, other studies finding differences that decrease over time (i.e., exposed children's functioning becomes more "normal" over time), and still other studies finding no differences. In addition to the moderating factors discussed earlier, some of these inconsistencies are likely due to a lack of uniform neuropsychological, academic, and psychosocial measures used across studies (Chap. 3). Further, most of the research focuses on young children. For example, studies of maternal psychotropic use have not followed children past 6 years of age so there is no indication of long-term effects (see Chap. 4). This is a significant gap in the literature, as many outcomes associated with exposure may become more conspicuous and more of a hindrance as children face the increased behavioral and academic demands of the school setting.

Professionals also lack a firm knowledge base regarding prognostic variables or factors that may impact or predict outcomes among exposed children. Although there have been advances in screening for school-age children, and for preschool, this screening does not occur until the child enters the school system. As yet, there is no practical, yet effective screener that could be used by pediatricians at well-child visits that might trigger referral and early identification. Finally, there has been very little research examining impact on specific academic skills or on school-based interventions or accommodations for children exposed to most of the toxins discussed in this volume.

Directions for Future Research in Pediatric Neurotoxicology

The general themes presented above point to important directions for future research. First, the field is in need of more research on the developmental trajectory and prognosis of children exposed to neurotoxins, so that we can begin to understand what these children look like as they transition to adolescence and young adulthood. As is the case for children with other neurodevelopmental and genetic disorders, children exposed to neurotoxins may exhibit changes in how their neuropsychological strengths and weaknesses are manifest over time. Most of the extant research has focused on short-term outcomes. More frequent and longer-term monitoring will be necessary to identify these developmental changes, and to more clearly understand the long-term impact of exposure. Similarly, some exposed children may appear to be typically developing upon initial assessment, with more subtle deficits becoming more conspicuous over time; these deficits may be missed without long-term follow-up.

Long-term studies must assess potential moderating factors beyond dose–response data, as these may account for differences in neuropsychological outcomes. The inconsistent findings reported throughout this volume may be partially explained by these moderating factors, and these variables must be studied more carefully and deliberately to better understand their relative influence on children's outcomes. Along with moderating factors, any intervention programs need to be accounted for in the long-term trajectory. At this time, there has been surprisingly little research on the efficacy of school-based intervention programs or accommodations for children exposed to neurotoxins even short-term. What is needed are resources for children exposed to the toxins covered in this book, to address such questions as: How well do interventions targeting certain constructs (e.g., memory, social skills) work with children who have been exposed to neurotoxins, and How do these interventions need to be adapted for exposed children with deficits in executive function and adaptive behaviors? Answering these questions will require developing programs (or modifying existing programs) specifically for children exposed to neurotoxins, and then evaluating their efficacy with this specific population. In addition to empirical studies with well-defined groups of exposed children, there is a need to develop and empirically investigate (a) parent education curricula for prevention and intervention efforts and (b) continuing education programs for professionals who work with children and families.

Current research suggests a lack of specificity in terms of means for early identification of resulting problems, common deficits, or outcomes. That is, exposure to a certain neurotoxin tends to result in deficits across several areas, and exposure to multiple neurotoxins may result in deficits in the same domain (e.g., deficits in executive function are described in multiple chapters in this volume, as are externalizing behaviors such as hyperactivity, impulsivity, aggression, and conduct problems). This lack of specificity suggests that certain domains of function may have a general vulnerability to the effects of neurotoxin exposure, but more research will be needed to evaluate this impression. It also may be the case that in many studies of children exposed to toxins, a narrow range of constructs was assessed, and more comprehensive assessment may reveal more widespread deficits across a range of constructs.

Lastly, without more research on many of these neurotoxins and how they impact child development, it is difficult to establish appropriate and reasonable prevention programs. For example, without more research on the use of AEDs, psychotropic medications, or other medications for maternal health, how can physicians adequately advise pregnant women? Professionals need empirically grounded guidelines to help them navigate the risk/benefit ratio associated with these substances when used by women who are pregnant and who need the medications to control symptoms of various disorders while minimizing risk to the developing fetus.

Conclusion

With advances in medicine, technology, and knowledge of vulnerability factors, more children who have been exposed to neurotoxins are being identified for intervention and are surviving at higher rates. At the same time, there are environmental changes and increases in the use of medication to treat maternal health that may be identified as having deleterious effects in the future. Given the increased survival rates, along with the movement for inclusive education, educational and health care professionals need to work collaboratively with these children to maximize their outcomes in public school settings. Thus, it is important to understand the academic and psychosocial outcomes associated with exposure to various neurotoxins.

This book reviews a great deal of data based on research with *groups* of infants and children. While this information is helpful in making predictions about psychosocial and academic outcomes, and in developing prevention/intervention programs, the most critical question from the perspective of the *individual* child is how programs can remediate areas of weakness while capitalizing on areas of strength. Clearly, this requires comprehensive assessment and developmental surveillance to monitor changes in functioning over time, similar to the frequent monitoring required for children who experience a traumatic brain injury (Riccio & Reynolds, 1999; Wetherington & Hooper, 2006). Finally, given the vulnerability of the developing central nervous system to toxins, practitioners are urged to consider neurotoxin exposure when gathering developmental history information from children and parents.

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