

Perinatal Exposure in Later Psychological and Behavioral Disabilities

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The thalidomide tragedy of the late 1950s heightened understanding of the importance of a healthy pregnancy for later development. Before that time, few were aware of the extent to which prenatal development could be adversely affected by chemicals ingested by the mother. There were a handful of researchers who dabbled in the field of teratology and understood that the womb was not impregnable to environmental insult. These

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researchers postulated that exposure of the developing fetus to noxious environmental events (e.g., toxins such as alcohol or tobacco, prenatal infection) would cause adverse physical and developmental outcomes. However, the consensus was that the placenta filtered harmful substances and protected the developing fetus from outside insult. After the thalidomide tragedy, research on prenatal and perinatal development accelerated.

After decades of research on profound outcomes that are readily observable at birth, researchers have begun focusing on prenatal and perinatal exposures that may result in outcomes that remain clinically silent until later in development. Factors such as prenatal exposure to influenza, being born with low birth weight, and gestational exposure to smoking and stress have been investigated for their relationship to adverse cognitive (e.g., borderline cognitive ability), learning, psychological (e.g., schizophrenia), and behavioral (e.g., attentional deficits) outcomes (Dombrowski, Martin, & Huttunen, 2003; Fried, Watkinson, & Gray, 1992, 2003; Martin, Noyes, Wisenbaker, & Huttunen, 1999; Mednick, Machon, & Huttunen, 1988; Shenkin, Starr, & Deary, 2004). However, we contend that environmental events that may perturb development during the prenatal and perinatal period are still poorly understood (Cordero, 2003), and their influence on the development of the child, particularly on pathological trajectories, has been underestimated.

The purpose of this miniseries is to introduce the field of school psychology to prenatal and perinatal exposures that may result in later psychological and behavioral disability. These outcomes often remain clinically silent until later in development, such as school entry, when the cognitive, behavioral, and language tasks become more complex and demanding. To our knowledge, this is the first time that a codified special issue on this topic has appeared in the school psychology literature. We have endeavored to make this issue multidisciplinary and have been fortunate to attract leading figures from the fields of psychiatry, public health, neuropsychology, and school psychology. Appointing this Special Issue is a commentary (Lollar & Cordero, 2007) from Dr. Jose Cordero, U.S. Assistant Surgeon General and Director of the Center for Birth Defects and Developmental Disabilities at the Centers for Disease Control and Prevention. He is joined by colleague Dr. Donald Lollar, who himself was former Chief of the Disability and Health Branch in the National Center for Environmental Health at the Centers for Disease Control and Prevention. Presently, Dr. Lollar is a Senior Scientist and Acting Director of Extramural Research at the National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention. Their contribution represents the first time that such public health luminaries have contributed to the field of school psychology and marks recognition of

the topic's value and relevance to the common interests of public health, medicine, and school psychology.

Each article discusses the implications for school psychology prevention, assessment, and intervention. Consistent with any progressive model of physical or psychological health, the general consensus of each article is that prevention is essential, and early assessment is clearly desirable to understand etiology and inform intervention. Thus, this Special Issue represents a beginning step toward more fully exploring and conceptualizing the relationship between prenatal and perinatal exposures and later educational and behavioral disability. Further research on this topic is necessary to explicate fully these complex and dynamic relationships.

This Special Issue provides a representative cross section of research on prenatal and perinatal factors. The first article written by Davis and Dean provides insight into the numerous prenatal and perinatal factors that might contribute to later outcomes. These researchers use a statistical concept called a relative risk ratio. This statistic is widely used in medical and epidemiological research because of its easy interpretability, but it might be less familiar to psychologists.

The second article in this Special Issue (Dombrowski, Noonan, & Martin, 2007) discusses one of the more prevalent pre/perinatal abnormality—low birth weight. The low birth weight literature is voluminous, reporting an array of adverse developmental outcomes. Dombrowski et al. (2007) provide data on the relationship between African American low birth weight/preterm birth and cognitive outcomes. There is a noted paucity of low birth weight/preterm birth outcomes research in homogenous African American populations, despite the nearly twofold increased prevalence of prematurity in the African American population compared to the Caucasian population. This piece, therefore, responds to a largely unaddressed void in the low birth weight/preterm birth literature.

For the third article, Polizzi, Martin, and Dombrowski contribute one of the more unique empirical articles in this Special Issue. These authors provide evidence for a season of birth effect in regard to emotional disturbance classification. Upon initial reaction, some might misapprehend the nature of season of birth research, concluding a linkage to astrology. Such association would be clearly spurious, because this line of research has been studied for decades in the medical and epidemiological literature. In fact, Polizzi et al. (2007) hypothesize that the etiological mechanism is either developmental (i.e., maturational) or neurodevelopmental, reminding us once again of Shakespeare's famous quotation: "It is not in the stars, dear Brutus, but within ourselves."

In the fourth article, Penner and Brown utilize a rarely available data set that contains individuals with serologically confirmed gestational rubella infection to discern whether gestational infection is associated with

precursors of later psychopathology. In 1941, Gregg was among the first to investigate the linkage between gestational rubella exposure and later ophthalmological defects. More recent research, including that of Brown, Penner, and colleagues, has discussed rubella and influenza in relation to more subtle psychological and behavioral outcomes, as well as psychopathology such as schizophrenia and depressive disorders.

Anhalt, Telzrow, & Brown (2007) discuss gestational stress exposure in relation to later outcomes. Of all gestational exposure studies in the Special Issue, the relationship between maternal stress during pregnancy and developmental outcomes represents one of the more recent foci of researchers (Martin et al., 1999). However, Plato recognized nearly 24 centuries ago (400 BCE) the potential role of stress during the pregnancy time period:

Special watch should be kept over our pregnant women during the year of their pregnancy to guard the expectant mother against the experiences of frequent and violent pleasures or pains and insure her cultivation of a gracious, bright and serene spirit (<http://www.teratology.org>).

THEORETICAL RATIONALE

Most child development etiological research has focused attention on genetic factors, social/cultural explanations, and characteristics of the caregiver/child relationship (Mash & Barkley, 2003). However, we contend that the prenatal and perinatal environment represents another pathway of psychopathology. Increasingly, it has been found to be associated with psychological, behavioral, and learning disabilities (Cordero, 2003; Dombrowski et al., 2003, 2005). However, environmental events that may perturb development during the prenatal and perinatal period are still poorly understood (Cordero, 2003), and their influence on the development of the child, particularly on pathological trajectories, has been underestimated. We base this argument on five points.

First, the development of the child is most easily disturbed by adverse events that occur during the period of most rapid development. In particular, insults to the central nervous system (CNS) have their greatest impact upon cell populations, structures, and processes that are in a period of rapid development at the time of the insult. This principle is a restatement of the Dobbing hypothesis outlined some 30 years ago (Dobbing & Sands, 1979). For instance, during the second trimester and to a lesser extent during the third trimester of fetal development, the structure of the brain undergoes very rapid development. Cells proliferate, migrate, and organize themselves at an astonishing rate. During some portions of the second and third trimesters, it is estimated that 250,000 neurons are being generated

per minute (LeDoux, 2002). Also, axon and dendritic growth and myelination begin during the later stages of fetal development, although these processes continue well into childhood and perhaps adolescence.

Aylward (1997) and others (e.g., Nowakowski & Hayes, 1999) posit that brain development is particularly vulnerable to insult during the prenatal period, even more so than during infancy and early childhood because insults during the fetal period result in damage to the developing architecture of the brain. During infancy and later in childhood, the structures are in place but functions have not become crystallized. Thus, if one region of the CNS is damaged during childhood in a normally maturing brain, another region might assume this function.

Second, there are higher levels of CNS compromise at the time of birth than has been recognized. Estimates by neuroscientists indicate that about 25% of conceptions are affected by developmental central nervous system disturbance (Aylward, 1997). A portion of these disturbances results in fetal and infant death. Of those infants that survive, the risk of pathological outcomes is substantial. There is good reason to believe that the rate of CNS disturbance in infants that appear healthy at birth is higher than expected. The most highly developed research on birth defects is focused on those factors that produce physical anomalies (structural defects) that can be observed at birth (e.g., structural defects of the face in Down's syndrome). Most of these defects are the result of some problem that occurs during the first three months of pregnancy (see Web site of the Centers for Disease Control and Prevention, <http://www.cdc.gov/ncbddd/bd>). This literature is substantial and rich in detail (Moore & Persaud, 1993; Norman & Armstrong, 1998). However, insults to the developing brain that occur during the later stages of fetal development (trimester 2 and 3) are much less likely to be accompanied by readily observable physical anomalies. These less obvious CNS perturbations result in so called "sleepier effects," effects on behavior that may not appear until the child begins to engage in higher-level cognitive functions (e.g., speech, reading, social cognition). Often, these problems do not become obvious until the first few years of schooling.

Third, there is overwhelming evidence that a large portion of prenatal and perinatal disturbance to fetal development is environmental and not attributable directly to genetic anomalies. The well-known negative effects of infections (e.g., toxoplasmosis, cytomegalovirus, rubella), maternally ingested drugs (e.g., alcohol, dioxins), extremely premature birth, and hypoxia (e.g., oxygen deprivation) in the perinatal period illustrate such environmental perturbations.

Fourth, there is a high level of comorbidity among psychopathologies. A disturbance during prenatal development may be responsible for central nervous system anomalies that manifest themselves as multiple psychopa-

thologies rather than as a single disorder. We conclude, as do other researchers, that the pervasiveness of comorbidity suggests similar processes or malfunctions are involved in the array of seemingly heterogeneous symptoms (Angold, Costello, & Erban, 1999). The mechanism for a heterogeneous set of symptoms from a common neuro-architectural anomaly is not difficult to understand. For example, a maternal viral infection might cross the placental barrier infecting the fetus. The resulting damage to the developing CNS would depend on the severity of infection, the immunological response to the infection, the particular regions of the CNS that were most exposed to the infection, and those regions that were most rapidly developing at that time. Thus, the same viral infection might produce profound mental retardation in one child and mild learning problems in another. The virus might alternatively produce visual-motor integration problems or language problems, depending on the location most affected. If the activity of the virus results in malformations of the structures involved in production of neurotransmitters, then altered emotional and social behavior might result.

The final reason that prenatal and perinatal factors are being underestimated as risk factors for psychological and behavioral problems of children is that the status of the CNS at the time of birth sets parameters for the influence of the other factors that affect the child throughout development. That is, prenatal and perinatal factors interact with genetic anomalies, less than optimal caretaker behavior, exposure to toxins during childhood, and social stress (e.g., because of poverty) in an additive fashion to further increase risks of psychopathology.

CONCLUSION

For all these reasons, we believe this Special Issue is timely and important. It is our firm hope that the articles presented here will spur the imagination of the school psychology community and will increase interest in continued study and research into prenatal and perinatal factors in child development, and particularly developmental psychopathology, as it applies to the practice of school psychology. Cordero and Lollar place a charge to the field of school psychology to not only increase understanding of these issues but also increase collaboration with other practitioners in public health and medicine. The school psychology community would be well served to heed this charge from two distinguished individuals who offer a broader perspective on children's health.

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