Essential Assessment

Lead Paint Injuries—Causation and Discovery

By Robert J. McCaffrey, Julie K. Lynch and William J. Greagan

The defense attorney must educate the court as to the issues of general and specific causation and the need for non-party discovery.

Untying the Gordian knot of lead exposure specific injury causation requires an examination of numerous medical, scientific, socioeconomic and environmental variables. This is critical since no "neurobehavioral signature" of lead has been reported to date. Specifically, there is no unique cognitive, behavioral or emotional sequelae that has been attributed to lead exposure. In fact the 2002 CDC guidelines note that elevated blood lead levels in children typically account for only one percent to three percent of the total variance in the neurodevelopmental measures under investigation. This article provides an overview of the causal assessment process that is an essential part of any forensic evaluation, especially in toxic tort litigation and the discovery issues related to it.

Differential Diagnosis vs. Causation Assessment

A clear understanding of the distinction between differential diagnosis and causation assessment is critical in the determination of causality and liability in children with histories of elevated blood lead levels. Differential diagnosis is defined by Stedman's Medical Dictionary as "the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings." Hensyl, W.R. (Ed.), *Stedman's Medical Dictionary* (25th edition), Baltimore, MD: Williams and Wilkin, p. 428 (1990). Differential diagnosis seeks to identify the internal disease or process that underlies the cause of the patient's clinical symptoms or clinical findings; it is not concerned directly with matters of causation nor can it be used alone to arrive at the issue of causation of a patient's condition. Causation assessment goes beyond the differential diagnostic process in that it is a search for the ultimate cause of a disease process or disorder that has been identi-

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fied by the differential diagnostic process. *See* Gots, R.E., The Distinct Roles of Differential Diagnosis and Causation Assessment, *For The Defense*, pp. 24–30 (July 2005). For example, a patient presents to an emergency department with leg pain and is found to have a fracture of the femur. The diagnosis of fractured femur does not address the cause of the fracture, which could have arisen from a car accident, a slip and fall, a physical altercation or any of a number of causes; this is the domain of causation assessment.

Chemical Exposure and Specific Health Effects

Any analysis of a causal association between chemical exposure and a specific health effect involves two levels of analysis. The first level is "general causation" in which one tries to determine whether a substance is capable of causing a specific adverse health effect in humans; the second level is "specific causation" in which one tries to determine whether exposure to a particular quantity of a specific substance over a particular time frame has caused a given adverse health effect in a particular person. Both levels of analysis must be firmly based on accepted scientific principles and methodology. General causation relies almost exclusively on the results of epidemiological studies. Factors to be considered include:

- The strength of the association;
- The consistency of the association;
- The specificity of the association;
- The temporality of the association;
- The biological gradient of the association (the dose-response relationship);
- The biological plausibility of the association;
- The coherence of the association; and
- Experimental support for the association.

Reference Manual on Scientific Evidence, Second Edition, pp. 374–79 (West 2000)

Once general causation has been established, the second level of analysis is specific causation; whether the chemical exposure is causally associated with the development of a specific disorder or group of disorders in a given person. This analysis involves the following factors:

- Evidence of the condition or disease;
- Epidemiologic evidence of general causation;
- Evidence of adequate exposure—that the individual received a dose necessary and causally demonstrated to produce the alleged disease, together with data sufficient to demonstrate consistency of timing from first exposure to the onset of the disease; and
- Evaluation of alternate causes—that is consideration and elimination of other more likely causes.

Reference Manual on Scientific Evidence, Second Edition, pp. 422–26 (West 2000).

It is this last factor of ruling out other more likely causes which requires discovery of extensive details of the child's family history, including but not limited to medical, educational, psychological/psychiatric, employment, and social histories of parents and non-party family members. Such family background information is a key element in a causal assessment. Access to this information is almost always met with an objection as to privilege; however, absent due consideration of these factors, the establishment of causality is tenuous at best.

Neurodevelopmental Risk Factors

A child's developmental outcome is determined by many variables, which include genetic, social/environmental, and medical/biological factors. The strength of the relationship, or correlation, between these factors and children's cognitive and behavioral outcome varies. Some factors have a stronger relationship to the outcome variable and therefore make a more substantial contribution. There are a large number of factors known to have a deleterious effect on children's cognitive and behavioral development. Lead, a neurotoxin, is one such risk factor. The impact of elevated blood lead levels on children's development has received a great deal of research attention, particularly with regard to its neuropsychological consequences. The current Centers for Disease Control (CDC) guidelines for the medical management of children with elevated blood lead levels advise on-going monitoring of children with blood lead levels of 10μg/dL or higher (CDC, 2002) based upon scientific evidence that blood lead concentrations at this level *may* have a negative effect on children's cognitive and behavioral functioning. Since the CDC statement of 2002, there have been published reports of a negative relationship between blood lead levels *below* 10μg/dL and children's cognitive and behavioral functioning. *See* Canfield, R.L., *et al.*, Intellectual Impairment in Children with Blood Lead Concentrations Below 10μg per Deciliter, *New England Journal of Medicine, 348*, pp. 1517–26 (2003); Canfield, R.L., *et al.*, Impaired Neuropsychological Functioning in Lead-exposed Children, *Developmental Neuropsychology, 26*, 513–40 (2004); Chiodo, L.M., *et al.*, Blood Lead Levels and Specific Attention Effects in Young Children, *Neurotoxicology and Teratology, 29*, 538–46 (2007); Chiodo, L.M., *et al.*, Neurodevelopmental Effects of Postnatal Lead Exposure at Very Low Levels, *Neurotoxicology and Teratology, 26*, 359–71(2004); Coscia, J.M., *et al.*, Cognitive Development of Lead Exposed Children from Ages 6–15 years: An Application of Growth Curve Analysis, *Child Neuropsychology, 9*, 10–21 (2003); Wasserman, G.A., *et al.*, The Relationship Between Blood Lead, Bone Lead, and Child Intelligence, *Child Neuropsychology, 9*, 22–34 (2003).

Elevated blood lead levels have been reported to have an adverse impact on cognitive and behavioral functioning of children; however, no specific pattern or neurobehavioral signature has been identified. *See* Center for Disease Control and Prevention, *Managing Elevated Blood Lead Levels among Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention* (2002).

The impact of elevated blood lead levels on children's development has received a great deal of research attention, particularly with regard to its neuropsychological consequences.

The only consistent finding across studies is that the relationship between elevated blood lead levels and cognitive and/or behavioral outcome is small. For example, low to moderate elevations in blood lead levels have a small negative relationship with childhood intellectual functioning. The research to date, across multiple studies, has demonstrated that only three percent to seven percent of the variance in IQ scores of the children studied is due to a history of elevated blood lead levels. These findings indicate that of the children who were participants in these studies, fully 93 percent to 97 percent of their performance on intellectual testing was determined by other non-specified factors. Baghurst, P.A., *et al.*, Environmental Exposure to Lead and Children's Intelligence at Age Seven Years: The Port Pirie Cohort Study, *New England Journal of Medicine, 327*, 1279–84 (1992); Wasserman, G.A., *et al.*, The Relationship Between Blood Lead, Bone Lead, and Child Intelligence, *Child Neuropsychology, 9*, 22–34 (2003); Wasserman, G.A., *et al.*, Lead Exposure and Motor Functioning in 4½-Year-Old Children: The Yugoslavia Prospective Lead Study, *Journal of Pediatrics, 137*, 555–61 (2000); Wasserman, G.A., *et al.*, Lead Exposure and Intelligence in Seven-Year-Old children: The Yugoslavia Prospective Study, *Environmental Health Perspective, 105*, 956–62 (1997).

The home environment is strongly related to children's cognitive, intellectual and behavioral development.

Lead exposure has also been reported to have a negative effect on other cognitive abilities including visuospatial skills, attention, reaction time, planning, problem-solving, reasoning, cognitive flexibility, and verbal memory. *See* Canfield, *et al.*, (2004); Chiodo *et al.*, (2007); Faust, D., & Brown, J., Moderately Elevated Blood Lead Levels: Effects on Neuropsychologic Functioning in Children, *Pediatrics, 80*, 623–29 (1987); Ris, M.D., *et al.*, Early Exposure to Lead and Neuropsychological Outcome in Adolescence, *Journal of the International Neuropsychological Society, 10*, 261–70 (2004); Wasserman, G.A., *et al.*, Yugoslavia Prospective Lead Study: Contribution of Prenatal and Postnatal Lead Exposure to Early Intelligence, *Neurotoxicology and Teratology, 22*, 811–18 (2000); Winneke, G., *et al.*, Brockhaus, A., Results from the European Multicenter Study on Lead Neurotoxicity in Children: Implications for Risk Assessment. *Neurotoxicity and Teratology, 12*, 553–59 (1990). The findings across these studies have not revealed a consistent pattern of cognitive deficits related to elevated blood lead levels, and the magnitude of these relationships is small. In addition, a small negative relationship between childhood elevated blood lead levels and various behavioral problems including overactive behavior, destructive behavior, and delinquent behavior has been reported. Chiodo, *et al.*, (2007); Ris, *et al.*, (2001); Silva, P.A., *et al.*, Blood Lead, Intelligence, Reading Attainment, and Behaviour in Eleven-year-old Children in Dunedin, New Zealand, *J Child Psychol Psychiatry*, 29,43–52 (1988); Wasserman, G.A., *et al.*, The Effect of Lead Exposure on Behavior Problems in Preschool Children, *American Journal of Public Health, 88*, 482–86 (1998). In these studies, a history of elevated blood lead levels has been found to explain approximately one percent to six percent of the variance in the specific behavior(s) under investigation.

In summary, the published literature indicates that elevated blood lead levels have a small negative contribution to the overall development of cognitive and behavioral problems among the groups of children studied. There are a multitude of variables in children's medical, family, social and environmental history that are known to have a far greater negative effect on cognitive and behavioral development than elevated blood lead levels (*See* Table 1). The 2002 CDC report *Managing Elevated Blood Lead Levels Among Young Children* contains the statement that "…an EBLL [elevated blood lead level] should be viewed as a risk factor for neurodevelopmental problems, not a diagnosis" (p. 82). Other risk factors must be given careful consideration when attempting to understand the contribution of a single risk factor to the results of a neuropsychological evaluation.

As outlined in Table 1, the list of known risk factors that have been demonstrated to impact negatively upon a child's biopsychosocial development is extensive. A family history of learning disorders, speech and language-related difficulties, attention deficit/hyperactivity disorder (ADHD), behavioral difficulties, and many psychological disorders (*e.g.*, depression, anxiety, conduct disorder, oppositional defiant disorder) are risk factors. These disorders tend to run in families, and the reason for this is that each has a sizeable genetic basis Barkley, R.A., *Attention-Deficit Hyperactivity Disorder (3rd ed.)*, New York: Guilford Press (2006); Goldstein, S., & Reynolds, C.R. (Eds.), *Handbook of Neurodevelopmental and Genetic Disorders in Adults*, New York: Guilford Press (2005). For example, genetic factors have been identified as the primary cause of ADHD, that is, most individuals with ADHD will have one or more biological relatives with the disorder. In fact, the 2002 CDC report indicates that current evidence *does not* support the conclusion that a history of elevated blood lead levels increases a child's risk for ADHD. Intelligence is also largely inherited with approximately 50 percent of the variance in childhood IQ scores due to genetic factors. Neisser, U., *et al.*, Intelligence: Knowns and Unknowns, *American Psychologist, 51*, 77–101 (1996). Therefore, a child may have genetically based risk factors which largely account for cognitive and behavioral deficiencies.

There are other variables, in addition to genetics, that increase a child's risk for poor cognitive, behavioral or psychological outcomes. Maternal use of drugs, alcohol, or tobacco during pregnancy places a child at risk for lower intellectual and cognitive functioning, behavioral difficulties, and developmental disorders. Jacobson, J.L., *et al.*, Relation of Maternal Age and Pattern of Pregnancy Drinking to Functionally Significant Cognitive Deficit in Infancy, *Alcoholism: Clinical and Experimental Research, 22*, 345–51 (1998); Howell, K.K., *et al.*, Prenatal Alcohol Exposure and Ability, Academic Achievement and School Functioning in Adolescence: A Longitudinal Follow-up, *Journal of Pediatric Psychology, 31*, 116–26 (2006); McCance-

Katz, E.F., The Consequences of Maternal Substance Abuse for the Child Exposed in Utero, *Psychosomatics, 32*, 268–74 (1991); Shankaran, S., *et al.*, Impact of Maternal Substance Use During Pregnancy on Childhood Outcome, *Seminars in Fetal & Neonatal Medicine, 12*, 143–50 (2007). Children born to mothers with a chronic medical illness during pregnancy (*e.g.*, thyroid dysfunction, hypertension, seizure disorder) are at increased risk for developmental deficiencies. Maternal infections during pregnancy may adversely impact the fetus' brain development. Bell, M.J., Infections and the Fetus, In M.L.Batshaw, *et al.*, (Eds.), *Children with Disabilities, 6th ed*. (pp. 71–82), Maryland: Paul H. Brookes Publishing Co. (2007); Haffner, W.H.J., Development Before Birth, In M.L.Batshaw, *et al.*, (Eds.), *Children with Disabilities, 6th ed*. (pp. 23–33), Maryland: Paul H. Brookes Publishing Co. (2007). Premature birth and low birth weight are also known development risk factors for lower intellectual/cognitive ability and school achievement. Picard, E.M., *et al.*, Prematurity and Low Birthweight, In K.O. Yeates, *et al.*, (Eds.), *Pediatric Neuropsychology: Research, Theory, and Practice* (pp. 237–51). New York: Guilford Press (2000). Maternal age at the time of the child's birth is another important risk factor. Pregnancies after the age of 35 have an increased risk of chromosomal disorders and pregnancy complications whereas pregnancy at a young age carries an increased risk of low cognitive ability to the child. Chapman, D.A., *et al.*, Early Risk Factors for Mental Retardation: Role of Maternal Age and Education, *American Journal of Mental Retardation, 107*, 46–59 (2000).

Socioeconomic status (SES) is another important neurodevelopmental risk factor. In his book James Garbarino noted that:

> …As the social environment becomes more toxic, it is the children—particularly the most vulnerable among them—who show the effects first and worst. And the children who will show the effects of social toxicity first and most dramatically are the ones who have accumulated the most developmental risk factors. These children already stand on the edge of life's abyss. Their risk factors are the stuff of talk shows and headlines and policy seminars: absent fathers, poverty and other economic pressures, racism, addiction, educational failures, poor physical health, family violence and adult emotional problems that impair parenting. Each of these factors multiplies the effects of any others that may be present, and so risk accumulates. The children's vulnerability to social toxicity increases. Social toxicity undermines their self-confidence and feelings of self-worth. It squanders opportunities for positive experiences that might give them strength. It erodes childhood itself.

Garbarino, J., *Raising Children in a Socially Toxic Environment*, San Francisco: Jossey-Bass Publishers (1995). Children from low socioeconomic backgrounds have higher mortality rates and are at increased risk for several morbidities of childhood including injury, chronic medical disorders, as well as the development of behavioral/emotional disorders. Chen, E., Boyce, W.T., & Matthews, K.A., Socioeconomic Difference in Children's Health: How and Why Do These Relationships Change with Age?, *Psychological Bulletin, 128*, 298–329 (2002). Moreover, there is substantive literature demonstrating that poverty in childhood impacts later adult functioning in several significant ways. It is clear that socioeconomic disparities among children impact health effects during childhood but also contribute to health-related outcomes among these children as adults.

Children from lower SES perform more poorly than those from higher SES in several domains including intelligence, language functioning, and overall academic achievement. Bradley, R.H., & Corwyn, R.F., Socioeconomic Status and Child Development, *Annual Review of Psychology, 53*, 371–99 (2002); Duncan, G.J., *et al.*, Economic Deprivation and Early Childhood Development, *Child Development, 65*, 296–318 (1994); Lipina, S.J., & Colombo, J.A., *Poverty and Brain Development During Childhood: An Approach from Cognitive Psychology to Neuroscience*, Washington, D.C., American Psychological Association (2009); Najman, J.M., *et al.*, The Impact of Episodic and Chronic Poverty on Child Cognitive Development, *Journal of Pediatrics, 154*, 284–89 (2008). Moreover, compared to children from high SES, those from lower SES are more likely to do poorly in school (*i.e.*, the dropout rate is higher, as is the percentage enrolled in special education programs). Underlying these differences are significant differences in neuropsychological functioning between children from lower SES versus those from higher SES. Specifically, children from low SES demonstrate poorer performance on tests of executive functioning, attention, language and memory compared to children from middle and high SES. Farah, M.J., *et al.*, Childhood Poverty: Specific Associations with Neurocognitive Development, *Brain Research, 1110*, 166–74 (2006); Mezzacappa, E., Alerting, Orienting, and Executive Attention: Developmental Properties and Sociodemographic Correlates in an Epidemiological Sample of Young, Urban Children, *Child Development, 75*, 1373–86 (2004); Noble, K.G., *et al.*, Neurocognitive Correlates of Socioeconomic Status in Kindergarten Children, *Developmental Science, 8*, 74–87 (2005). In addition, electrophysiological studies have reported differences in the neural processes of children from low SES compared to children from high SES. D'Angiulli, A., *et al.*, Children's Event-related Potentials of Auditory Selective Attention Vary with Their Socioeconomic Status, *Neuropsychology, 22*, 293–300 (2008). One study identified a reduction in the prefrontal cortex functioning of low SES children similar to that of individuals who have sustained brain damage to the prefrontal cortex. Kishiyama, M.M., *et al.*, Socioeconomic Disparities Affect Prefrontal Function in Children, *Journal of Cognitive Neuroscience, 21*, 1106–15 (2008).

The adverse effect of low SES on children's development is due to the presence of social-environmental factors and medical risk factors that are known to influence brain development. There is a higher prevalence of poor prenatal care, prenatal substance use, low birth weight, iron deficiency, poor nutrition, and inadequate medical care in low SES children and all of these factors are negatively related to cognitive and behavioral development. In addition, children from low SES backgrounds often are born to mothers who are younger in age and have lower educational attainment, and both of these factors have been associated with lower cognitive development. Chapman, D.A., *et al.*, Early Risk Factors for Mental Retardation: Role of Maternal Age and Education, *American Journal of Mental Retardation, 107*, 46–59 (2002).

Further, the chronic stressors of living within a disadvantaged social setting can adversely effect both brain development and the child's overall functioning. The longer that a child is exposed to poverty and the accompanying stress, the greater the risk for poor academic achievement. Evans, G.W. & Schamberg, M.A., Childhood Poverty, Chronic Stress, and Adult Working Memory, *Proceedings of the National Academy Sciences, 106*, 6545–49 (2009).

> **The current functioning** of any child must be interpreted within the context of the child's *entire* history if causation is to be established.

The home environment is strongly related to children's cognitive, intellectual and behavioral development. A home environment in which caretakers are nurturing and responsive to the child's needs and that includes cognitively stimulating experiences, can improve the developmental outcome of a child who is at risk for intellectual, cognitive or behavioral deficiency due to genetic, medical or other environmental factors. Those home environments characterized by poor parenting practices, low parental responsiveness, and minimal cognitive stimulation increase children's risk for poor cognitive, behavioral, and academic outcomes. Further, home settings in which children are maltreated and/or witness domestic violence have a negative effect on children's overall functioning. Lewis, M. & Feiring, C. (Eds.), *Families, Risk, and Competence*, New Jersey: Lawrence Erlbaum Associates Publishers (1998); Litrownik, A.J., *et al.*, Exposure to Family Violence in Young At-risk Children: A Longitudinal Look at the Effects of Victimization and Witnessed Physical and Psychological Aggression, *Journal of Family Violence, 18*, 59–73(2003); Love, J.M., *et al.*, Child Care Quality Matters: How Conclusions May Vary with Context, *Child Development, 74*, 1021–33 (2003); NICHD Early Child Care Research Network, Predicting Individual Differences in Attention, Memory, and Planning in First Graders from Experiences at Home, Child Care and School, *Developmental Psychology, 41*, 99–114 (2005); Veltman, H.W.M., & Browne, K.D., Three Decades of Child Maltreatment Research: Implications for the School Years, *Trauma, Violence & Abuse, 2*, 215–39 (2001).

The majority of children with elevated blood lead levels are from disadvantaged backgrounds and are therefore exposed to a host of developmental risk factors, in addition to lead. Many of these other risk factors have a stronger relationship to children's developmental outcome than has been identified for lead. This is well described in a 2002 CDC report that indicates that blood lead levels play a smaller role in children's neurodevelopmental outcome than such factors as parental intelligence and socioeconomic status, which have a much larger impact:

> Increased exposure to lead frequently occurs in the context of other factors that also place a child at increased neurodevelopmental risk (*e.g.*, poverty, single-parent household, teen-age mother, child abuse, poor nutrition). From this perspective, lead represents an additional "hit," adding to the child's cumulative neurodevelopmental risk. In multivariate statistical models, children's BLLs [blood lead levels] tend to account for a relatively small amount of the variance in their neurodevelopmental status…Other factors, particularly social class and parental intelligence, typically account for much larger percentages of outcome variance.

Managing Elevated Blood Lead Levels Among Children, p. 83.

Causal Assessment

Consideration of risk factors entails obtaining information about family members. It is routine clinical practice for neuropsychologists to obtain such information, as it is for other health professionals. Ask yourself this question: "When was the last time that healthcare professionals did *not* request that I provide her/him with a complete and detailed family history covering medical conditions, psychological conditions, alcohol/drug abuse, cause of death for deceased relatives, etc.?" This is routine clinical practice for all healthcare professionals, including neuropsychologists, because family history provides important and relevant information that is an essential aid in the overall assessment process. In evaluating a child with a history of elevated blood lead levels who has an IQ of 71, would it be relevant to know that their unaffected biological sibling's IQ was 71, the mother's IQ from her educational records was 71, and the father's IQ from his educational records was described as being in the borderline range (*i.e.*, IQ's between 70 and 79)? The point is that without a context within which to evaluate a child with a history of elevated blood lead levels, it is not possible to determine the child's expected IQ absent the history of elevated blood lead levels. The current functioning of any child must be interpreted within the context of the child's *entire* history if causation is to be established.

Privileged vs. Confidential Information

There is a difference between what is privileged and what is confidential. Privileged information, unless waived, is not discoverable. Confidential information, if relevant, is discoverable but may be subject to limitations on its dissemination. Since evidentiary privileges impede the search for truth, they are disfavored and construed narrowly. *Trammel v. US*, 445 U.S. 40 (1980); *Catone v. The Honorable Robert E. Miles*, 215 Ariz. 446; 160 P.3d 1204 (2007). They are an exception to the scope and purpose of discovery, which seeks the production of all evidence material and necessary in the prosecution or defense of an action. The words "material and necessary" are to be interpreted liberally to require the disclosure of any facts bearing on the controversy which will assist preparation for trial by sharpening the issues and reducing the delay and prolixity. The test is one of usefulness and reason and to permit discovery if there is any possibility that the information is sought in good faith for possible use as evidence-in-chief or in rebuttal or for cross-examination. *Allen v. Crowell-Collier Publishing Co.*, 21 N.Y.2d 403, 235 N.E.2d 430 (1968). Or, put another way: reasonably calculated to lead to the discovery of admissible evidence. Fed. R. Civ. P. 26(b)(1).

Table 1

Medical History

Prenatal/perinatal factors

Maternal nutrition
Maternal abnormal weight gain
Maternal drug/alcohol use
Maternal smoking
Perinatal infections
- Toxoplasmosis
- HIV
- Syphilis
- Rubella
- Cytomegalovirus
- Herpes simplex viruses
- Group B Streptococcus (GBS)
- Lymphocytic choriomeningitis virus (LCMV)

Maternal seizure disorder
Maternal hyptertension/preeclampsia
Maternal diabetes
Maternal thyroid dysfunction
Maternal age

Birth complications

Fetal hypoxia
Maternal infections (*e.g.*, Group B streptococcal (GBS), Herpes Simplex viruses)
Preterm birth
Low birth weight (< 2500 grams)
Very low birth weight (< 1500 grams)
Extremely low birth weight (< 1000 grams)

Other medical factors

Failure to thrive
High fevers
Repeated ear infections/otitis media
Seizure disorder
Cerebral palsy
Poor nutrition
Underweight
Iron deficiency
Cerebral concussion/head injury
Chromosomal disorders
Environmental toxins (*e.g.*, lead, mercury)

Family Medical/Psychological History

Substance abuse
Mental Illness (*e.g.*, depression, anxiety, psychosis, schizophrenia)
Seizure disorder
Neurodegenerative illness
Family history of neurodevelopmental disorders
- Attention-Deficit/Hyperactivity Disorder
- Learning disorders (*e.g.*, reading disorder, mathematics disorder)
- Intellectual disability
- Communication disorders (*e.g.*, Phonological/Articulation Disorder)
- Motor Skills Disorder
- Oppositional Defiant Disorder
- Conduct Disorder
- Pervasive developmental disorders (*e.g.*, Asperger Syndrome, Autism)

Socioeconomic Status/Poverty

Persistent poverty
Living conditions
- Poor home maintenance
- Community violence/stressors associated with low income urban neighborhood

Repeated negative life events
Employment/labor uncertainty
Reduced intellectual/social stimulation
Reduced educational resources
Attitude/expectations of parents
Attitude/expectations of teachers
Family size
Single parent
Parental education
Persistent economic deprivation

Other Risk Factors

Low parental education
Poor parenting/discipline
Domestic violence
Parental mental illness
Parental substance abuse
Abuse/neglect
Emotional climate of family

At common law, there was no doctor-patient privilege. *Whalen v. Roe*, 429 U.S. 589, 602 n. 28 (1977). The privilege is a creature of statute, first enacted in New York in 1828 to protect doctor-patient communication based on the belief that fear of embarrassment or disgrace flowing from disclosure of communications made to a physician would deter people from seeking medical help in securing adequate diagnosis and treatment. *Williams v. Roosevelt Hospital*, 66 N.Y.2d 391; 488 N.E.2d 94 (1985). This privilege has come to include protection of medical records from disclosure. *Id.* Such information is only discoverable if the individual waives the privilege expressly by providing authorizations, or by discussing the medical communication with third-parties for reasons other than medical care. It may be impliedly waived by placing one's medical condition in issue by commencing a lawsuit to recover damages for the condition. *See*, 21 ALR3d 912. Once waived, the communication is no longer subject to the privilege. It is forever waived. *McKinney v. The Grand Street, P.P. & F.R. Co.*, 104 N.Y. 352, 10 N.E. 544 (1887); *State v. Mincey*, 141 Ariz. 425, 687 P.2d 1180 (1984); *Cerro Gordo Charity v. Firemans Fund American Life Ins. Co.*, 810 F.2d 1471 (8th Cir. 1987). Any privilege is lost when the information is disclosed to and considered by a testing expert. *Palmer v. Asarco, Inc.*, 225 FRD 258 (N.D. Okla. 2004).

In lead paint litigation, this privilege frequently serves as the basis for objections to inquiries relating to the mother's health history and the health history of family members and of the infant plaintiff's siblings. This is a misunderstanding of the privilege. The privilege does not protect medical information; it protects medical communication. It protects only the physician-patient exchange itself, not the underlying facts of the patient's medical condition or history. The witness does not have to answer the question "What did you and your doctor discuss about your medical history?" but she is required to answer "What is your medical condition and history?" In *Williams v. Roosevelt Hospital*, the mother of a brain damaged child alleging malpractice during the child's birth could be questioned about the physical condition and congenital abnormalities of her other children; the medications she took; and facts relating to a prior abortion.

If relevance can be demonstrated, the mother can be compelled to submit to an I.Q. test. *Andon v. 302–304 Mott Street Assoc.*, 94 N.Y.2d 740, 731 N.E.2d 589 (2000). This is not the case in federal court where Fed. R. Civ. P. 35 only permits examinations of parties and the court lacks the inherent power to order a non-party (or one suing only in their representative capacity) to submit to a physical or psychological examination. *Caban v. 600 E. 21st St. Co.*, 200 FRD 176 (EDNY 2001).

Other types of records, such as school records, are not privileged and therefore discovery as to the school records of siblings is available if relevancy can be established. Remember that "relevance" in the context of discovery, as compared to evidentiary relevance at trial, is a lower standard requiring only that it be reasonably calculated to lead to the discovery of admissible evidence. Because school records may contain otherwise privileged medical information courts may provide for in camera inspection of the academic records to redact the privileged medical information. *Bunch v. Artz*, 71 Va. Cir. 358; 2006 Va. Cir. Lexis 252 (2006).

Other methods of limiting dissemination of otherwise confidential, material and relevant information include protective orders limiting disclosure to those who need to know for purposes of the litigation; sealing the record; and clearing the courtroom for portions of the trial where the confidential information is discussed. *See Catone*, 160 P.3d 1204; *Bunch*, 71 Va. Cir. 358 (2006).

Review of the literature on neurodevelopmental risk factors provides clear evidence of the relevance of family history and the importance of a thorough review and consideration prior to rendering an opinion within a reasonable degree of professional certainty.

In seeking discovery from non-parties the defense attorney should submit the affidavit of a qualified expert educating the court as to the difference between diagnosis and causation. The child is diagnosed with a history of elevated blood lead levels and also has cognitive deficits. The causation assessment is the process by which an expert attempts to determine whether the history of elevated blood lead levels is the cause of the cognitive deficit. The expert must then explain the other alternate causes for the deficit, and the need to evaluate them in the context of this individual plaintiff, to demonstrate that discovery from the non-party family member is material and relevant evidence to the issue of specific causation. It must be stipulated by the scientific literature and the references must be supplied to the court. The attorney must educate the court as to the scope and limitation of the medical privilege; the legal relevance of the confidential information and suggest methods such as protective orders in balancing the need for discovery and the desire for privacy.

Conclusion

As stated in *Adams v. Rizzo*, 13 Misc. 3d 1235A; 831 N.Y.S.2d 351 (N. Y. Sup. 2006):

> Untangling the Gordian knot of lead exposure specific injury causation presents a complex and wide reaching examination of numerous medical, scientific, socioeconomic and environmental variables. This examination is not subject to conclusory, superficial or artificial restriction on the scope of information that may be necessary for careful and just resolution by a jury.

By default, the defense attorney bears the burden of educating the court as to the issues of general and specific causation and the need for non-party discovery. These issues are not only important for the defense expert but also to challenge the basis for the conclusions of the plaintiff's expert. How can a plaintiff's expert provide an opinion regarding causation, within a reasonable degree of professional certainty, without conducting a causation assessment? If the expert has not considered and ruled out more likely causes of the deficit through an analysis of the child's family medical, social and educational history, then the expert's methodology is subject to attack.

Like Alexander the Great, plaintiffs and their experts want to slice thru the intricacies of the causation knot and simply say that lead equals injury. Litigation is supposed to be a search for the truth and the defense needs to help the court unravel the knot rather than hack it to shreds. FD