

*Stanley Sugarman, et al. v. Chauncey Liles, Jr.*, No. 80, September Term, 2017, Opinion by Adkins, J.

**EXPERT WITNESS TESTIMONY — MARYLAND RULE 5-702 — SUFFICIENT FACTUAL BASIS:** An expert witness in a lead paint case had a sufficient factual basis to testify that lead exposure can cause certain attention problems, namely, deficiencies in “auditory encoding of information” and “information processing speed.” The expert relied on studies recognizing a causal link between lead exposure and attention problems. The testimony did not suffer from an “analytical gap” like the testimony of the medical expert in *Rochkind v. Stevenson*, 454 Md. 277 (2017). Unlike *Rochkind*, the epidemiological studies the expert relied upon specifically set out a causal link between elevated blood lead levels (“BLL”) and attention problems.

**EXPERT WITNESS TESTIMONY — MARYLAND RULE 5-702 — MEDICAL CAUSATION:** In a lead paint case, a pediatrician designated as an expert witness could offer an opinion as to whether the plaintiff’s elevated BLLs caused a loss of 4 IQ points. The expert can opine about the loss of IQ points after considering the plaintiff’s elevated BLLs, the results of his neuropsychological evaluation, and a methodology developed in population studies. The expert’s testimony provided sufficient evidence for the jury to draw the inference that, more likely than not, the plaintiff’s elevated BLLs caused a measurable loss of IQ points.

**TORTS — NEGLIGENCE — DAMAGES — IMPAIRMENT OF EARNING CAPACITY — SUFFICIENCY:** In a personal injury case alleging cognitive deficits as a result of elevated BLLs, plaintiff provided sufficient evidence of loss of earning capacity. An expert witness in the field of vocational rehabilitation provided an opinion beyond mere speculation. The vocational rehabilitation counselor interviewed the plaintiff, conducted vocational testing, reviewed educational and medical records, and a neuropsychological evaluation. Testimony from the vocational rehabilitation expert coupled with an expert in the field of economics provided sufficient evidence for the jury to conclude that plaintiff had a reduced earning capacity because of his elevated BLLs.

Circuit Court for Baltimore City  
Case No.: 24-C-14-005808  
Argued: June 1, 2018

IN THE COURT OF APPEALS  
OF MARYLAND

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No. 80

September Term, 2017

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STANLEY SUGARMAN, et al.

v.

CHAUNCEY LILES, JR.

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Barbera, C.J.  
Greene  
Adkins  
McDonald  
Watts  
Hotten  
Getty,

JJ.

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Opinion by Adkins, J.  
Getty, J., concurs and dissents.

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Filed: July 31, 2018

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Suzanne C. Johnson, Clerk

This case presents yet another opportunity for clarification of when epidemiological studies relied upon by an expert provide a sufficient factual basis for the expert's testimony. Specifically, we shall consider whether an expert's opinion on causation, relying on epidemiological studies, suffers from the same "analytical gap" identified in *Rochkind v. Stevenson*, 454 Md. 277 (2017). We shall also assess whether an expert may offer an opinion on specific causation when relying on epidemiological data coupled with an individualized analysis of the plaintiff and his claimed injuries. Finally, we consider the sufficiency of proof required for a plaintiff to demonstrate a loss of earning capacity.

## **BACKGROUND**

Respondent, Chauncey Liles, Jr. sued Ivy Realty, Inc. and Stanley Sugarman (collectively "Sugarman") in the Circuit Court for Baltimore City. Liles alleged injury and damages caused by lead paint exposure at a residential property (the "Residence") owned by Sugarman. At trial, the parties stipulated that, due to Sugarman's negligence, Liles was exposed to deteriorating lead paint at the Residence. The parties further stipulated that the exposure caused Liles's elevated blood lead levels ("BLL"). The only remaining questions for the jury were whether the lead exposure caused Liles any injury, and if so, what damages he incurred.

### **Liles's Developmental History**

Liles was born in 1998. At the age of 2, Liles's BLL measured 11 mcg/dL. At the age of 3, his BLL was 10 mcg/dL. When he entered the fourth grade, Liles began to have educational difficulties. At the same time, Liles was experiencing grief and anxiety from the death of several family members. His educational records reflect that in fifth grade, as

a result of his educational difficulties, his teachers provided additional accommodations, including one-on-one support, individualized help on his math skills and test taking practice, additional response time in class, additional time to complete assignments, and repetition of directions. Through middle school, Liles earned grades in the 90s, 80s, and mid-to-high 70s.

After middle school, Liles attended Baltimore City College (“City College”), a selective high school located in Baltimore. He graduated in May 2016. In 2012, Liles broke the thumb on his dominant hand. In 2012 and 2013, his academic performance faltered. Initially, in a deposition, Liles attributed this dip in his grades to not being able to write with an injured hand. At trial though, he testified that this was just an excuse meant to cover for his faltering performance and difficulties paying attention. In 9th grade, his GPA was 2.38. In 10th grade, he had a GPA of 1.09. His GPA increased in 11th and 12th grade, when he earned GPAs of 2.57 and 3.36 respectively. He graduated 194th out of 301 students in his class.

Liles described his grades at City College as “terrible.” He attributed his performance to his inability to focus and claimed that he is easily distracted, has difficulty sitting still, and stated that he “just can’t grasp things as fast as other people do.” He claimed that these issues started when he was young and that “as the work got harder, [he] couldn’t get it.” Despite these difficulties in the classroom, Liles tested at or above grade level on the Maryland School Assessment and passed his High School Assessment without any additional support. Liles was accepted at West Virginia University and Bowie State

University, both of which are four-year schools. At trial, Liles testified that he intended to pursue a bachelor's degree from Bowie State.

Liles's mother graduated from high school and obtained some college education, but has no college degree. His father also graduated from high school but did not attend college and works as a truck driver.

### **Expert Testimony**

At trial, Liles called four expert witnesses: (1) Robert Kraft, Ph.D.; (2) Jacalyn Blackwell-White, MD; (3) Mark Lieberman; and (4) Michael Conte, Ph.D. We shall examine the testimony of each in turn.

#### ***Robert Kraft, Ph.D.***

Liles offered Dr. Robert Kraft as an expert in the field of psychology and neuropsychology. Dr. Kraft testified that he conducted a neuropsychological examination of Liles, which consisted of an interview as well as several tests administered to assess various aspects of intelligence including attention, memory, learning, and language. Specifically, Dr. Kraft administered an IQ test, the Wechsler Adult Intelligence Scale ("WAIS-IV"). Dr. Kraft testified that Liles had a full-scale IQ of 94, which placed him in the 34th percentile and in the normal range. Dr. Kraft also explained that an average range IQ does not necessitate a conclusion that there was no evidence of brain impairment.

In addition to this overall score, the IQ test revealed several sub-scores, which measured various aspects of intelligence. Dr. Kraft found a "statistically significant" discrepancy between some of Liles's sub-scores, which indicated that Liles had some form

of brain impairment. Liles received a sub-score of 86 on two indexes Dr. Kraft identified as most sensitive to attention and concentration.

Notably, according to Dr. Kraft, Liles scored 86 in both: (1) auditory encoding of information in the working memory; and (2) information processing speed. Dr. Kraft testified that these are both factors of attention. Dr. Kraft described auditory encoding in the following manner: “Any time an individual gives you a piece of information that you are expected to use in some way, you are encoding that information until you’ve completed the task, then you can forget about the information.” Regarding information processing speed, Dr. Kraft said:

Processing speed is your ability to independently focus and complete a task in a self-direct manner. So any time a student sits down to study, an adult, anyone sits down to read a book, any time a person sits down to complete a writing task—that requires information processing speed.

When Dr. Kraft tested Liles’s information processing speed a second time, Liles received a scale score of 76, which placed him in the 5th percentile. Liles also performed in the “borderline” range on a test related to “simple and complex visual motor tracking.” This, Dr. Kraft testified, was “consistent with his processing speed difficulty and difficulty shifting attention.” Dr. Kraft opined that Liles’s lower scores on these tests of attention were statistically significant and attributable to brain impairment.

The lower scores on auditory encoding and information processing speed were the only impairments Dr. Kraft identified. On cross-examination, when asked about how these attention impairments related to Liles’s academic performance, Dr. Kraft said, “[h]e could, in fact, have these deficits but still [have] B’s all the way through [school]. These deficits

are mild, and they may not always show up in every school year.” He further stated, that “[m]y opinion . . . is that he may not show any impairments in his school performance at all based on these deficits, if he’s able to adapt and accommodate in some way for them.”

***Jacalyn Blackwell-White, MD***

Dr. Blackwell-White was accepted as an expert in the fields of pediatrics and childhood lead poisoning. She testified that lead is a neurotoxin that “causes damage to the central nervous system.” She explained that lead blocks the transmission of neurotransmitters to the brain, which impedes “learning pathways.” Lead exposure during the prenatal stage through at least age six or seven can cause damage to the brain because it disrupts the learning pathways during critical developmental periods. Dr. Blackwell-White testified that this means that “attention is going to be affected.” She also explained that the Center for Disease Control “issue[s] standards and protocols for diagnosis and treatment of children who have elevated blood lead levels . . .” and has set the current reference level at 5 mcg/dL. The reference level was once higher, but later lowered due to medical studies showing that BLLs under 10 mcg/dL are harmful to children.

Dr. Blackwell-White did not examine Liles, but instead reviewed his medical records, Dr. Kraft’s report, and other associated records. She opined that, “within a reasonable degree of medical certainty,” Liles “was exposed to sustained toxic blood lead levels at an early age. . . . [and] [h]is documented period of toxicity was at least 12 months.” Liles “incurred brain impairment as a result of his early lead toxicity. . . .” This impairment included, but was not limited to, a loss of cognitive function. Dr. Blackwell-White also opined that Liles lost 4 IQ points “as a result of early lead toxicity.”

Dr. Blackwell-White relied extensively on the United States Environmental Protection Agency’s Integrated Science Assessment for Lead (“EPA-ISA”).<sup>1</sup> She explained that the EPA-ISA found causal relationships between lead exposure and attention problems in children, as well as issues with hyperactivity and impulse control. Dr. Blackwell-White relied on other studies, chiefly one by Dr. Bruce Lanphear, (“Lanphear Study”),<sup>2</sup> showing a causal relationship between lead exposure in childhood and the loss of IQ points. We shall examine the conclusions of both the EPA-ISA and the Lanphear Study in more detail *infra*. Dr. Blackwell-White offered her opinion, within a reasonable degree of medical certainty that, Liles “suffered brain damage as a result of his early lead exposure.” She also testified that the cognitive deficits Dr. Kraft described were caused by Liles’s early lead exposure. She explained that injuries suffered from lead exposure are permanent.

When asked on cross-examination whether the EPA-ISA set forth a causal relationship between elevated BLLs and the specific attention deficits Dr. Kraft identified, Dr. Blackwell-White answered that the EPA-ISA speaks only to attention “generally.” She concluded that the attention problems identified by Dr. Kraft fell under the umbrella term “attention” as used in the EPA-ISA.

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<sup>1</sup> U.S. Env’tl. Protection Agency, Integrated Science Assessment for Lead, EPA/600/R-10/075F (June 2013), <https://perma.cc/5ZRA-W54H>.

<sup>2</sup> Bruce P. Lanphear et al., *Low-Level Environmental Lead Exposure and Children’s Intellectual Function: An International Pooled Analysis*, 113(7) Env’tl. Health Persps. 894 (July 2005).



***Mark Lieberman***

The trial court accepted Mark Lieberman as an expert in vocational rehabilitation counseling. Lieberman explained that a vocational rehabilitation expert assesses how an individual's physical, cognitive, or psychiatric disability will affect the individual's ability to learn new skills and function in the job market. In evaluating Liles, Lieberman met with him, reviewed Dr. Kraft's report, and Liles's medical and educational records. With this information he then applied the RAPEL methodology to evaluate Liles's employment capacity. Lieberman explained that each letter in "RAPEL" stands for a separate stage of the vocational rehabilitation analysis. We have paraphrased his description below:

- **R = Rehabilitation plan.** This assesses the responsibilities of the rehabilitation counselor, the client time frames, and the expenses of rehabilitation.<sup>3</sup>
- **A = Access to labor market.** What type of job can the client acquire?
- **P = Placeability.** Who will hire the client and why?
- **E = Earning capacity.** How much can the client earn in his current condition and how much could they have earned prior to the disability?
- **L = Labor force participation.** What is known about persons with the specific type of limitations that the client presents?

Relying on the report from Dr. Kraft, Lieberman concluded that Liles suffered from major cognitive problems, including problems with attention. He recognized that Liles has difficulty with auditory encoding and information processing speed. He explained that these issues "affect[] your ability to learn when you're sitting, listening to a lecture." He

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<sup>3</sup> Mark Lieberman explained that, in a forensic setting, the rehabilitation counselor typically meets with the client only once, so no formal rehabilitation plan is created. Lieberman did not create a rehabilitation plan for Liles.

said this was “[b]ecause you [need] to hear the information, quickly process it based on other stuff you know, and put everything together to understand the new concept that’s being presented.”

Lieberman also administered the Career Ability Placement Survey (“CAPS”), which he described as an “eight[-]part test,” for “mechanical reasoning, spatial relations, verbal reasoning, . . . math, . . . grammar, [and] word knowledge.” Liles scored below average on some aspects of the test, particularly reading and math. Although Lieberman explained that Liles had the skills of a high school graduate, he anticipated that difficulties would arise for Liles once he started college. Lieberman stated that Liles “has the IQ potential and basic academics to be . . . at least an Associate[’s] degree graduate.” He also opined that Liles would be unable to attain that degree:

It’s his ability to function in the college setting as the work gets more difficult. It’s my expectation, I see [Liles] as going to college, being able to pass some classes, but eventually hitting that brick wall—the point where he’s not going to be able to get the Associate[’s] degree. . . . And what we see here is that [Liles is] going to get up to the level of an individual with a high school diploma and some college, but won’t eventually reach the earnings of an individual with at least an Associate[’s] degree.

***Michael Conte, Ph.D.***

Liles next offered Dr. Michael Conte as an expert in economics. Dr. Conte explained the concept of loss of earning capacity, which he described as the difference between “how much you probably would have earned absent a certain impediment or insult versus how much you’re likely to earn now.” To understand an individual’s prospects in the labor market, Dr. Conte typically relies on an evaluation from a vocational

rehabilitation expert. Here, he relied on Lieberman's conclusions regarding Liles's likely outcomes without his disability, and his likely outcome with the disability. In addition to reviewing Lieberman's report, Dr. Conte spoke with Lieberman regarding what Liles could have accomplished without his current deficits.

To reach a conclusion on earnings, Dr. Conte factored in assumptions including sex, educational attainment, and work-life expectancy. In Liles's case, Dr. Conte assumed that:

Liles suffers from a cognitive disability, and . . . on the basis of Lieberman's opinions, that he would not most likely have been able to compete in the labor market as an Associate's degree holder, absent the severity of his current deficits. [A]t this point in time however, he manages to proceed in the course of obtaining additional educational credits, that when he decides to enter the competitive labor market, he will most likely be able to compete at the level of a high school graduate with some college.

Dr. Conte then offered an opinion as to Liles's lost earning capacity. He described this as the difference between the career earnings of someone with the educational attainment of an Associate's degree, \$3,456,127 (Liles's likely earnings without any deficits) and the career earnings of someone with the educational attainment of a high school diploma and some college, \$1,757,320 (Liles's likely earnings with deficits). This difference, \$1,698,808,<sup>4</sup> represents the sustained loss of earnings resulting from Liles's injuries.

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<sup>4</sup> Dr. Conte made a minor, and ultimately insignificant, error in his calculations. He testified that the difference was \$1,698,808. We checked Dr. Conte's arithmetic and discovered that he was off by one dollar—the difference should be \$1,698,807.

Sugarman also called expert witnesses: (1) David Nelson, MD; (2) Scott Beveridge, Ph.D.; (3) Jack Spector, MD;<sup>5</sup> and (4) Jerome Paige, Ph.D.<sup>6</sup>

*David Nelson, MD*

Dr. David Nelson was accepted as an expert in pediatrics, epidemiology, and childhood lead exposure. Like Dr. Blackwell-White, Dr. Nelson did not examine Liles but reviewed his medical records, education records, and relied upon Dr. Kraft's neuropsychological testing. Dr. Nelson testified that, based on his review of the epidemiological literature, he would not expect a BLL of 10 mcg/dL or even 15 mcg/dL to be significant in terms of causing brain impairment. Rather, he opined that he would expect a BLL of 20 mcg/dL or above to have a significant effect. Dr. Nelson also testified that Liles's educational records did not indicate any difficulty due to cognitive impairment. Instead, Dr. Nelson attributed Liles's educational difficulties to severe anxiety from the loss of several family members while in the fifth grade and Liles's broken thumb in high school. Dr. Nelson offered his expert opinion that, within a reasonable degree of medical

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<sup>5</sup> Dr. Spector was offered and accepted as an expert in the field of clinical neuropsychology. Dr. Spector offered testimony regarding his own testing of Liles and an interpretation of the testing conducted by Dr. Kraft. Dr. Spector did not offer an opinion on the cause of Liles's alleged cognitive defects. Neither of the parties rely upon or dispute Dr. Spector's testimony in this appeal.

<sup>6</sup> Dr. Paige was offered and accepted as an expert in the field of economics. Dr. Paige's testimony refuted Dr. Conte's conclusions. Specifically, Dr. Paige speculated that Dr. Conte made incorrect calculations regarding Liles's likely fringe benefits and his work-life expectancy. In this appeal though, Sugarman only disputes Dr. Conte's testimony on the basis that he relied upon improper assumptions Lieberman proffered. The issues Dr. Paige addressed regarding calculation of fringe benefits and work-life expectancy are not raised in this appeal.

certainty, Liles's lead exposure did not cause him any issues at all, that Liles does not suffer any cognitive damage from any cause, and that even if Liles had never been exposed to lead, he would not be any better off than he is today.

*Scott Beveridge, Ph.D.*

Dr. Scott Beveridge was offered as an expert in forensic vocational rehabilitation counseling. Dr. Beveridge reviewed Liles's medical records, his educational records, and the reports from Dr. Kraft, Dr. Blackwell-White, and Lieberman. He also met with Liles just before his 18th birthday. Dr. Beveridge administered an IQ test that revealed a full-scale IQ of 96. Dr. Beveridge identified this IQ as in the "average range."

Like Lieberman, Dr. Beveridge was asked to perform a vocational evaluation "to determine if there was any impact on [Liles's] ability to work and maintain employment." Dr. Beveridge employed the PEEDS-RAPEL method of analysis, a method that differs from the RAPEL method employed by Lieberman. Dr. Beveridge testified that the PEEDS-RAPEL method also accounts for parental history and educational attainment when considering the likely outcome of an evaluated individual. Based on this evaluation, Dr. Beveridge testified that Liles has the capacity to complete an Associate's degree. Dr. Beveridge explained that Liles had expressed an interest in going into the medical field and that he could likely pursue further education in nursing after obtaining an Associate's degree. He further opined that Liles could work as a nurse throughout his normal life, and that his educational and vocational capacity was the same as it was before his lead exposure.

## Motions for Judgment

At the conclusion of Liles's case in chief, Sugarman moved for judgment. Sugarman argued that Liles had not proven that his elevated lead levels caused any injury, and that he had not proven the existence of any damages beyond mere speculation. Regarding causation, Sugarman pointed to Dr. Blackwell-White's testimony and argued that her opinion rested solely on epidemiological literature "without any identification of measurement for the individual to support her opinions that there has been cognitive loss caused by lead exposure." Sugarman also argued that Liles had not sufficiently proven that, but for his cognitive difficulties, he would have obtained a college degree. The trial court concluded that there was enough evidence for the issues to go to the jury and denied Sugarman's motion. At the conclusion of all the evidence, Sugarman again moved for judgment and raised the same arguments. Once again, the trial court deemed the evidence sufficient for the jury's consideration.

The jury returned a verdict for Liles in the amount of \$1,302,610 (\$600,000 in non-economic damages and \$702,610 in economic damages). Final judgment was entered in the amount of \$1,277,610 after a reduction consistent with the statutory cap on non-economic damages.

Sugarman filed a timely appeal and argued that Dr. Blackwell-White did not have a sufficient factual basis for her opinions regarding causation. Sugarman also contended that Liles had not sufficiently proven that his claimed injuries had resulted in any damages. In a reported opinion, the Court of Special Appeals affirmed the final judgment of the trial court. *See Sugarman v. Liles*, 234 Md. App. 442 (2017).

We granted Sugarman’s petition for writ of certiorari to answer the following questions:<sup>7</sup>

1. Did Dr. Blackwell-White’s causation opinion have a sufficient factual basis to establish a causal relationship between lead exposure and cognitive defects identified in Liles or his IQ loss?
2. Was there sufficient evidence for the trial court to submit the case to the jury on the issue of whether Liles’s lead exposure resulted in damages?

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<sup>7</sup> We have rephrased the questions presented for brevity and clarity. The questions presented in the petition for writ of certiorari were:

1. Mr. Liles’s medical expert opined that lead exposure caused cognitive deficits in two distinct areas measured on neuropsychological evaluation. But she conceded that the epidemiological data she relied upon did not show a causal association between lead exposure and either metric. Did the CSA err when it held that her opinion had a sufficient factual basis?
2. There was no evidence that Mr. Liles has any IQ deficit due to any cause. Yet, his medical expert relied solely on epidemiological evidence of a causal association showing that lead exposure can cause IQ loss (general causation) to assume that IQ loss occurs in every individual exposed (specific causation). Did the CSA err when it held that the expert had a sufficient basis to opine that lead exposure caused Mr. Liles to suffer IQ loss?
3. The damages experts’ opinions that Mr. Liles – a Baltimore City College high school graduate accepted to two four-year colleges and enrolled in one – has incurred millions of dollars in loss of earning capacity were based on assumptions not supported by the evidence, and failed to employ any accepted methodology to determine likely pre-injury capacity. Did the CSA err when it concluded that Petitioner’s arguments regarding the insufficient factual and methodological basis underlying the experts’ opinions went to the weight, not sufficiency of the evidence?

## DISCUSSION

Sugarman moved for judgment on two occasions, at the end of the presentation of Liles’s case in chief, and at the conclusion of the evidence. The trial court denied both motions and the case was ultimately submitted to the jury. “A judge must grant a civil defendant’s motion for judgment as a matter of law if the plaintiff failed to present evidence that could persuade the jury of the elements of the tort **by a preponderance of the evidence.**” *Darcars Motors of Silver Spring, Inc. v. Borzym*, 379 Md. 249, 270 (2004) (emphasis in original). *See also District of Columbia v. Singleton*, 425 Md. 398, 406–07 (2012) (“Where the defendant, in a jury trial for negligence, argues that plaintiffs’ evidence is insufficient to create a triable issue, the court determines whether an inference of negligence is permissible; that is, whether the evidence demonstrates that it is more probable than not that the defendant was negligent.”). The court considers the evidence and reasonable inferences drawn from the evidence in the light most favorable to the non-moving party. *Singleton*, 425 Md. at 407. We review the trial court’s decision to grant or deny a motion for judgment in a civil case without deference. *Id.* at 406.

### General Causation And Attention Decrements

The Court of Special Appeals held that Liles had produced sufficient evidence that his lead exposure caused injury—specifically his deficits in the areas of auditory encoding and processing speed—to survive a motion for judgment. *Sugarman*, 234 Md. App. at 469. It relied on our analysis set forth in *Rochkind v. Stevenson*, 454 Md. at 288–91, wherein we observed that epidemiological evidence showing a causal link between exposure and injury is usually sufficient for general causation. Liles’s experts—Dr. Kraft and Dr. Blackwell-



White—both testified that Liles’s specific deficits were measures, or factors, of attention. Because the studies in the EPA-ISA found a causal link between lead exposure and attention decrements, the intermediate appellate court reasoned that there was no analytical gap. *Sugarman*, 234 Md. App. at 469. Thus, there was a sufficient factual basis for Dr. Blackwell-White’s opinion and the Circuit Court correctly determined that the weight and credibility of the parties’ dueling experts was a matter for the jury to resolve. *Id.*

Sugarman urges us to reverse the intermediate appellate court, arguing that Liles did not satisfy his burden to produce legally sufficient evidence because Dr. Blackwell-White’s testimony lacked a sufficient factual basis under Md. Rule 5-702. Sugarman maintains that the opinion suffers the same analytical gap present in *Rochkind* because epidemiological literature does not establish a causal relationship between lead and Liles’s specific deficits. Therefore, Sugarman reasons, Dr. Blackwell-White’s opinion does not permit a conclusion of general causation and is ultimately speculative. Sugarman forecasts dire consequences if we affirm the Court of Special Appeals, such as medical experts offering increasingly abstract testimony in the absence of scientific evidence or research.

Liles maintains that Dr. Blackwell-White’s testimony, referencing numerous medical studies in addition to the EPA-ISA, satisfied Liles’s burden of proof and that the Circuit Court properly denied Sugarman’s motion for judgment. Liles also points to *Rochkind*, emphasizing that this Court observed that the EPA-ISA identifies a causal relationship between lead exposure and “general attention deficits.” Liles emphasizes that the EPA-ISA does not examine the effects of lead exposure in a “hyper-specific, granular manner[,]” but rather, the studies linking lead exposure to attention decrements use

“attention” as an “umbrella term.” This, Liles concludes, is a sufficient basis for Dr. Blackwell-White’s testimony, and there is no analytical gap.

### *Maryland Rule 5-702*

We first address the “sufficient factual basis” required by Md. Rule 5-702, which permits expert testimony “in the form of an opinion or otherwise,” if the trial court determines “that the testimony will assist the trier of fact to understand the evidence or determine a fact in issue.” *Id.* To make that assessment, the trial court evaluates: “(1) whether the witness is qualified as an expert by knowledge, skill, experience, training, or education[;] (2) the appropriateness of the expert testimony on that particular subject[;] and (3) whether a sufficient factual basis exists to support the expert testimony.” *Id.* The proponent of the expert testimony carries the burden of demonstrating that these requirements are satisfied. *Rochkind*, 454 Md. at 286.

A “sufficient factual basis” requires both “an adequate supply of data and a reliable methodology.” *Id.*; *see also Roy v. Dackman*, 445 Md. 23, 42–43 (2015). As we explained in *Rochkind*, “[t]o constitute ‘more than mere speculation or conjecture,’ the expert’s opinion must be based on facts sufficient to ‘indicate the use of reliable principles and methodology in support of the expert’s conclusions.’” 454 Md. at 286 (quoting *Exxon Mobil Corp. v. Ford*, 433 Md. 426, 478 (2013)). Materials relied upon by an expert need not be admissible provided that they are of the kind reasonably relied upon by experts in the particular field to form opinions or inferences on the subject. *See* Md. Rule 5-703(a).

To satisfy the requirement of a reliable methodology, “an expert opinion must provide a sound reasoning process for inducing its conclusion from the factual data and

must have an adequate theory or rational explanation of how the factual data led to the expert's conclusion." *Exxon Mobil Corp. v. Ford*, 433 Md. 426, 481 (2013). Expert opinions must be able to assist a trier of fact. To satisfy that requirement, the trier of fact must be able to evaluate the reasoning underlying the opinion. *Ross v. Housing Auth. of Balt. City*, 430 Md. 648, 663 (2013). Conclusory or *ipse dixit* assertions are not helpful—an expert “must be able to articulate a reliable methodology for how she reached her conclusion.” *Rochkind*, 454 Md. at 287.

Before delving into Dr. Blackwell-White's factual basis for her testimony, it is helpful to illuminate some principles of causation in cases in which an injury is alleged based on exposure to substances.

### ***Causation***

General causation addresses whether a particular substance can cause the kind of injury suffered by the plaintiff. *See, e.g., id.* at 291–92; *Blackwell v. Wyeth*, 408 Md. 575, 600 (2009). Specific causation addresses whether the substance actually caused the plaintiff's injury. *See, e.g., Aventis Pasteur, Inc. v. Skevofilax*, 396 Md. 405, 442 (2007); *Wilhelm v. State Traffic Safety Comm'n*, 230 Md. 91, 99–100 (1962); *see also Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005) (“General causation is whether a substance is capable of causing a particular injury or condition in the general population and specific causation is whether a substance caused a particular individual's injury.”).

In *Ross*, 430 Md. at 668, we explained a common pattern of specific causation in lead-paint exposure cases:

The theory of causation . . . can be conceived as a series of links: (1) the link between the defendant’s property and the plaintiff’s exposure to lead; (2) the link between specific exposure to lead and the elevated blood lead levels[;] and (3) the link between those blood lead levels and the injuries allegedly suffered by the plaintiff.

(footnote omitted).<sup>8</sup> The third link encompasses both general and specific causation—whether lead can generally cause certain injuries, and whether that exposure did cause Liles’s injuries.

Here, the parties’ stipulations have satisfied the first and second links. Liles lived at the property owned and managed by the Petitioners and the parties stipulated that “due to Ivy Realty’s negligence[,] deteriorated paint at the [Residence] substantially contributed to Mr. Liles’s [BLL] . . . measured by two BLLs of 11 mcg/dL and 10 mcg/dL.” Thus, we concern ourselves with the third link.

“[B]oth general and specific causation testimony are subject to [Md.] Rule 5-702 . . . .” *Rochkind*, 454 Md. at 287 n.4. If an expert’s general causation opinion fails for the lack of a sufficient factual basis, and the specific causation opinion is based on the general causation opinion, then logically, the specific causation opinion also fails. *Id.* The first question we must resolve is whether Dr. Blackwell-White had an adequate factual basis for her testimony that lead exposure can cause attention deficits, including deficits in auditory encoding and processing speed.

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<sup>8</sup> This causation theory is not the only one available to plaintiffs, of course. In *Ross v. Housing Auth. of Balt. City*, 430 Md. 648, 668 n.20 (2013), we observed that there may be other ways a plaintiff could demonstrate that lead exposure was a substantial factor in causing the plaintiff’s harm.

In *Rochkind*, we considered whether an expert had a sufficient factual basis to offer general causation testimony that lead exposure can cause Attention Deficit Hyperactivity Disorder (“ADHD”). *Id.* at 281–88. There, the plaintiff’s expert, Dr. Hall-Carrington, testified that epidemiological studies collected in the EPA-ISA demonstrated that children with blood lead levels similar to those of the plaintiff could have attention problems, learning disabilities, or ADHD. *Id.* at 288. Dr. Hall-Carrington agreed that the EPA-ISA did not establish a causal link between ADHD and lead exposure, but that the studies listed the symptoms of ADHD. Based on this information, she concluded that lead exposure caused the plaintiff’s ADHD. *Id.*

At the time *Rochkind* was decided, we had not yet determined “the extent to which epidemiological studies can support expert testimony on causation.” *Id.* We examined other jurisdictions’ precedent addressing the use of epidemiological studies, particularly *General Elec. Co. v. Joiner*, 522 U.S. 136 (1997). There, the United States Supreme Court agreed with a district court that the epidemiological studies the plaintiff’s experts relied on did not provide a sufficient basis to offer an opinion that exposure to polychlorinated biphenyls (“PCBs”) caused lung cancer. *Id.* at 145–46. Although some of the studies the expert relied on showed statistically significant increases in lung cancer, the studies either did not refer to PCBs, or did not account for exposure to other known carcinogens. *Id.* The Supreme Court explained that although experts “commonly extrapolate from existing data[,]” courts are not required to “admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert.” *Id.* (italics in original). “A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.”

*Id.* In short, the studies upon which an expert relies must provide factual support for conclusions regarding causation. *Id.* at 146–47.

Applying *Joiner* to *Rochkind*, we observed that the studies in the EPA-ISA “finding a causal relationship between lead exposure and attention deficits and hyperactivity” did not support a causal relationship between lead exposure and ADHD. 424 Md. at 290–91. Although the EPA-ISA contained studies that examined the relationship, it was one of association, rather than causation.<sup>9</sup> *Id.* at 291–92. Thus, although there is evidence of a causal relationship between lead and some symptoms associated with ADHD, “these lead-caused behaviors do not necessarily indicate that an individual has ADHD because these behaviors are also symptoms of a variety of other learning disorders and learning disabilities.” *Id.* at 290.

Indeed, the EPA-ISA cautioned that research showing an association between lead exposure and ADHD had been critiqued for failing to account for potential confounding<sup>10</sup>

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<sup>9</sup> We discuss these terms in greater detail, *infra*.

<sup>10</sup> Confounding is an important concept in assessing causation from studies finding an association.

Confounding occurs when another causal factor (the confounder) confuses the relationship between the agent of interest and the outcome of interest. . . . [O]ne instance of confounding is when a confounder is both a risk factor for the disease and a factor associated with the exposure of interest. . . . When researchers find an association between an agent and a disease, it is critical to determine whether the association is causal or the result of confounding.

Michael D. Green, Michal Freedman & Leon Gordis, *Reference Guide on Epidemiology*, in *Reference Manual on Scientific Evidence* 549, 591 (3d ed. 2011).

factors such as familial history, parenting, and socio-economic status. *Id.* at 292. Thus, Dr. Hall-Carrington’s testimony suffered from an analytical gap because it “overstated the known effects of lead exposure.” *Id.* at 291. Her testimony was not admissible under Md. Rule 5-702 because it lacked “epidemiological studies—or other reliable evidence—demonstrating a causal link between lead exposure and ADHD . . . .” *Id.* at 294. Even if an expert believes wholeheartedly that the link exists, that opinion requires a scientific basis. Without it, that opinion is *ipse dixit*. *Id.* (citing *Palmer v. Asarco, Inc.*, 510 F. Supp. 2d 519, 531 (N.D. Okla. 2007)).

Mindful of our recent decision in *Rochkind* and its discussion of the “analytical gap,” we next examine the basis for Dr. Blackwell-White’s opinion. We begin with a discussion of some principles of epidemiology, as well as an explanation of the EPA-ISA’s methodology and findings.

### ***Epidemiology And The EPA-ISA***

“Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations.” Michael D. Green, Michal Freedman & Leon Gordis, *Reference Guide on Epidemiology*, in *Reference Manual on Scientific Evidence* 549, 551 (3d ed. 2011). Epidemiological research can lend support to the question of general causation. *Id.* at 552. Such studies have been used routinely in litigation. “Where the study properly accounts for potential confounding factors and concludes that exposure to the agent is what increases the probability of contracting the disease, the study has demonstrated *general* causation—that exposure to the agent ‘is capable of causing the illness in the general population.’” *In re Silicone Gel Breast*

*Implants Prods. Liab. Litig.*, 318 F. Supp. 2d 879, 893 (C.D. Cal. 2004) (emphasis in original) (quoting *In re Hanford Nuclear Reservation Litig.*, 292 F.3d 1124, 1134 (9th Cir. 2002)); see also *Norris*, 397 F.3d at 882; *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1158 (E.D. Wash. 2009); Green et al., *supra*, at 551–52 & n.2.

The primary basis for much of Dr. Blackwell-White’s testimony was the EPA-ISA. It is necessary to first explain precisely **what** the EPA-ISA is. It is an integrated science assessment that reviewed, synthesized, and evaluated other scientific studies and research. U.S. Env’tl. Protection Agency, Integrated Science Assessment for Lead, EPA/600/R-10/075F, at xliv (June 2013), <https://perma.cc/5ZRA-W54H>. It included peer-reviewed studies, reviewed reports, and EPA analyses using publicly available data. *Id.* at xlv. Studies were selected “based on the general scientific quality of the study, and consideration of the extent to which the study is informative and policy relevant.” *Id.* at xlvi. The EPA-ISA scrutinized the evidence, received peer input, and ultimately evaluated the weight of that evidence to reach conclusions about causal determinations or the lack thereof. *Id.* at 1, Fig. III.

“Cause” is defined as “an agent that brings about an effect or result.” *Id.* at li. “Association” is “the statistical relationship among variables; alone, however, it is insufficient proof of a causal relationship between an exposure and a health outcome.” *Id.* Epidemiological studies primarily assess whether associations exist, as well as their relative strength, between an agent and a disease. *Berry v. CSX Transp., Inc.*, 709 So. 2d 552, 567 (Fla. Dist. Ct. App. 1998). Epidemiologists may or may not infer that causal relationships exist from studies demonstrating associations. *Id.* at 567–68. Finding causation from associations also requires identifying and controlling for potential confounding factors to



rule out alternative explanations for an association. EPA-ISA, *supra*, at liv–lv. *See also King v. Burlington N. Santa Fe Ry. Co.*, 762 N.W.2d 24, 35–36 (Neb. 2009).

To synthesize studies demonstrating associations, the EPA-ISA applied a variant of the Hill Factors,<sup>11</sup> a generally accepted method used in epidemiology,<sup>12</sup> to determine causation. These factors include:

- Consistency of the observed association;<sup>13</sup>
- Coherence;<sup>14</sup>
- Biological plausibility;<sup>15</sup>
- Biological gradient (exposure-response relationship);<sup>16</sup>
- Strength of the observed association;<sup>17</sup>

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<sup>11</sup> The Hill Factors, also known as the Hill Criteria, are criteria identified by the epidemiologist Sir Austin Bradford Hill in a 1965 article. *See Harris v. CSX Transp., Inc.*, 753 S.E.2d 275, 289 (W. Va. 2013); Green et al., *supra*, at 600 & n.148; EPA-ISA, *supra*, at lviii & n.1.

<sup>12</sup> *See Harris*, 753 S.E.2d at 280–90 (discussing acceptance of the Hill Factors).

<sup>13</sup> This criterion examines “whether a similar association may be found in a variety of different situations.” *Harris*, 753 S.E.2d at 290; *see also* EPA-ISA, *supra*, at lix.

<sup>14</sup> Causation inferences may be strengthened by support from multiple lines of evidence and other relevant knowledge. This may refer to coherence across both multiple scientific fields and multiple study designs. *See* EPA-ISA, *supra*, at lix; *Harris*, 753 S.E.2d at 292–93.

<sup>15</sup> This factor supports causation by demonstrating that biological facts support the conclusion. *Harris*, 753 S.E.2d at 292; EPA-ISA, *supra*, at lix; Green et al., *supra*, at 604–05.

<sup>16</sup> “The biological gradient factor seeks to show or determine whether increased exposure to a chemical agent increases the incidence of the disease.” *Harris*, 753 S.E.2d at 292; EPA-ISA, *supra*, at lix (“A well-characterized exposure-response relationship . . . strongly suggests cause and effect, especially when such relationships are also observed for duration of exposure . . . .”); *see also* Green et al., *supra*, at 603.

<sup>17</sup> Demonstrating a strong association between an agent and a disease is more likely to indicate that a causal relationship exists. The stronger the relationship between the two

- Experimental evidence;<sup>18</sup>
- Temporal relationship of the observed association;<sup>19</sup>
- Specificity of the observed association;<sup>20</sup> and
- Analogy.<sup>21</sup>

*Id.* at lix.

When the EPA-ISA reaches a conclusion about a causal determination for a particular health effect from exposure to an agent, it means that:

Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (i.e., doses or exposures generally within one to two orders of magnitude of current levels). That is, **the pollutant has been shown to result in health effects in studies in which chance, bias, and confounding could be ruled out with reasonable confidence.** For example: a) controlled human exposure studies that demonstrate consistent effects; or b) observational studies that cannot be explained by plausible alternatives or are supported by other lines of evidence (e.g., animal studies or mode of action information). **Evidence includes multiple high-quality studies.**

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variables, the less likely that it is due to chance or confounding. *Harris*, 753 S.E.2d at 290; *see also* EPA-ISA, *supra*, at lix; Green et al., *supra*, at 602.

<sup>18</sup> “Strong evidence for causality can be provided through ‘natural experiments’ when a change in exposure is found to result in a change in occurrence or frequency of health or welfare effects.” EPA-ISA, *supra*, at lix; *see also Harris*, 753 S.E.2d at 293.

<sup>19</sup> This factor examines whether the exposure preceded the disease. *Harris*, 753 S.E.2d at 291–92; EPA-ISA, *supra*, at lix; *see also* Green et al., *supra*, at 601–02.

<sup>20</sup> This factor seeks to show that an effect has a particular cause. EPA-ISA, *supra*, at lix; *Harris*, 753 S.E.2d at 291; Green et al., *supra*, at 605–06.

<sup>21</sup> This factor examines information on analogous relationships and agent structure, as well as information on the mode of action for a particular agent. EPA-ISA, *supra*, at lix. Simply put, it examines “whether an accepted phenomenon in one area can be applied to another area.” *Harris*, 753 S.E.2d at 292.

*Id.* at lxii (emphasis added).<sup>22</sup> In short, the EPA-ISA used a widely accepted method to reach conclusions about causation, and neither party has questioned the accuracy of those conclusions.

The EPA-ISA examined studies addressing the effects of lead exposure on “externalizing behaviors such as attention, impulsivity, hyperactivity, destructive behavior, and truancy[,]” which, it observed, had not been examined as extensively as the effect of lead on cognitive function. *Id.* at 4–150. It identified three “major domains of externalizing behavior disorders,” which include attention deficits. *Id.* The EPA-ISA was careful to emphasize that clinically diagnosed disorders like ADHD are “considered to have a strong familial component,” and studies reporting associations between BLLs and ADHD must account for confounding factors. *Id.* at 4-151. “Within the attention deficit hyperactivity disorder domain of externalizing behaviors, studies of Pb<sup>[23]</sup> exposure have focused

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<sup>22</sup> In *Rochkind v. Stevenson*, 454 Md. 277, 293 (2017), we rejected Dr. Hall-Carrington’s testimony because the data she relied upon for her opinion that lead exposure caused the plaintiff’s ADHD did not support that underlying conclusion. It is worth mentioning that Dr. Hall-Carrington did not extrapolate the conclusion about causation from the data herself—rather, she relied on the EPA-ISA’s assessments. *Id.* at 283. Trained and qualified epidemiologists relying on a wide pool of studies demonstrating strong associations have testified to causal associations based on generally accepted principles of epidemiology applied to a body of reliable research. *See, e.g., Harris v. CSX Transp., Inc.*, 753 S.E.2d 275 (W. Va. 2013). Our focus today is **not** on whether a qualified epidemiologist testifying as an expert witness can appropriately synthesize research demonstrating associations and offer an opinion as to causation. *See, e.g., Blackwell v. Wyeth*, 408 Md. 575 (2009). Rather, it is whether an expert in pediatrics and childhood lead poisoning has an adequate basis, from such a synthesis conducted by another reputable organization using generally accepted methods, to offer opinions on general causation.

<sup>23</sup> Pb refers to lead, abbreviated from the Latin *plumbum*. This abbreviation is the one that appears on the periodic table of elements, and it appears throughout the EPA-ISA.

primarily on attention, impulsivity, and hyperactivity.” *Id.* at 4-153. The EPA-ISA examined the evidence for each outcome individually. *Id.*

Of most relevance to this case is the survey of studies of attention in children. The EPA-ISA defined attention as “the ability to maintain a consistent focus on an activity or relevant stimuli and can be assessed by examining sustained attention, concentration[,] or distractibility.” *Id.* It then reviewed the studies that found associations between blood lead levels and attention decrements. *Id.* at 4-153–54. While some studies included examinations of attention and hyperactivity, others focused solely on attention. *See id.* at 4-156–63.

In summarizing its findings, the EPA-ISA evaluated attention, impulsivity, and hyperactivity together because they fell within the same relevant domain of externalizing behaviors. *Id.* at 4-289. The EPA-ISA found a “causal relationship between Pb exposure and attention decrements, impulsivity, and hyperactivity in children” that was “supported by multiple lines of evidence . . . .” *Id.* This relationship was “further supported by the consideration for several potential confounding factors in prospective studies.” *Id.* at 4-291. These findings were based on the consistency of the available evidence, biological plausibility, and underlying modes of action. *Id.* at 4-294.

Although the EPA-ISA evaluated the evidence relating to these effects collectively, it does **not** set forth clinical criteria for assessing whether a particular individual has been affected by lead. The EPA-ISA examined studies concerning attention, impulsivity, and hyperactivity to reach conclusions about causation due to lead exposure. But the EPA-ISA’s conclusion that lead can cause these three externalizing effects does not mean that

the absence of one or two of these effects necessarily leads to the conclusion that general causation fails. After all, some of the studies discussed in the EPA-ISA focused exclusively on attention, and the EPA-ISA examined attention separately from the other effects.<sup>24</sup> The EPA-ISA is an integrated assessment that broadly examined various health outcomes present in multiple studies to reach a generalized conclusion about the causal relationship between lead exposure and attention decrements. Sugarman is correct that the EPA-ISA does not specifically identify types of attention decrements found nor does it mention processing speed or auditory encoding.

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<sup>24</sup> Sugarman contends that Dr. Blackwell-White has an “even wider analytical gap” because she “conceded that Mr. Liles exhibited no signs of impulsivity or hyperactivity, and was found to have no deficits on other measures of attention . . . .” Sugarman appears to be mixing specific causation arguments with general causation arguments—essentially that the absence of a particular injury or symptom in Liles means that general causation fails. Sugarman also points to Dr. Blackwell-White’s testimony regarding the EPA’s determination of a causal relationship with attention decrements, impulsivity, and hyperactivity to support their contention that all three traits must manifest to satisfy the causal relationship.

First, that the EPA-ISA found causal relationships with three traits does not lead to a logical conclusion that all three **must** be present in an individual exposed to lead. After all, the EPA-ISA analyzes those traits separately. Second, Dr. Blackwell-White’s testimony described the EPA-ISA’s conclusions, and she discussed attention separately from hyperactivity or impulsivity.

Sugarman points to the chart from the EPA-ISA that Liles submitted into evidence that described health outcomes of “[c]lear evidence of attention decrements, impulsivity and hyperactivity (assessed using objective neuropsychological tests and parent and teacher ratings) in children 7-17 years and young adults ages 19-20 years.” This table, *see* EPA-ISA, *supra*, at lxxxiii, is identified as a **summary** of the causal determinations for the relationship between lead exposure and various outcomes and cross-references to other portions of the EPA-ISA. Further, the table refers to other studies. Although the chart refers to animal toxicology studies that demonstrated impulsivity and impaired response inhibition, as Sugarman’s counsel pointed out during oral argument, other portions of the EPA-ISA refer to animal studies that examined attention. *See id.*, *supra*, at 4-191.

But Sugarman ignores Dr. Blackwell-White’s medical expertise with respect to attention deficits. The question we must resolve is whether Dr. Blackwell-White had a sufficient factual basis to opine that auditory encoding and processing speed are within the realm of attention decrements caused by lead contemplated in the EPA-ISA, or whether her testimony suffered from the same analytical gap present in *Rochkind*.

### ***The Analytical Gap***

As we explained in *Savage v. State*, 455 Md. 138, 163 (2017), the “hallmark” of the analytical gap is “the failure by the expert witness to bridge the gap between his or her opinion and the empirical foundation on which the opinion was derived.” In *Rochkind*, we concluded that an analytical gap existed because the EPA-ISA, which supplied the factual basis for Dr. Hall-Carrington’s opinion testimony, although finding a causal relationship between lead exposure and attention deficits and hyperactivity, did not go so far as to conclude that lead exposure caused ADHD. 454 Md. at 290. We had discussed the analytical gap even before *Rochkind* in *Blackwell*, 408 Md. at 606–08. There, we concluded that an expert’s opinion did not bridge the gap because, although the data he used was reliable, the expert’s extrapolation from that data was defective. The basis of his opinion, including causation and methodology, were not generally accepted as reliable in the expert’s particular scientific field. *Id.* at 608–10.

Similarly, in *Giant Food, Inc. v. Booker*, 152 Md. App. 166, 178 (2003), the Court of Special Appeals considered whether a plaintiff presented sufficient evidence that exposure to Freon caused his asthma. The plaintiff’s expert in pulmonary medicine offered an opinion on causation. *Id.* at 185. The basis for the opinion was an examination of the

plaintiff, his medical history dating from the accident, and documented exposure to Freon. *Id.* at 184–87. The Court concluded that the expert lacked a sufficient factual basis under Md. Rule 5-702 and that his methodology was “woefully inadequate” because the expert did not identify any literature supporting the causation theory. *Id.* at 189.

Cases from other jurisdictions provide further examples of an analytical gap. *See, e.g., Bland v. Verizon Wireless, LLC*, 538 F.3d 893, 897–98 (8th Cir. 2008) (analytical gap existed because expert lacked data about level of exposure to Freon that risks asthma as well as degree and concentration of exposure); *Ruggiero v. Warner-Lambert Co.*, 424 F.3d 249, 253–55 (2d Cir. 2005) (no basis for expert opinion on causation because of inadequate support from research and the differential diagnosis did not bolster general causation opinion); *United States v. Mamah*, 332 F.3d 475, 478 (7th Cir. 2003) (expert testimony defective because of absence of empirical link between research and opinion); *Mitchell v. Gencorp, Inc.*, 165 F.3d 778, 779 (10th Cir. 1999) (analytical gap existed where expert witnesses provided no scientific evidence setting forth causal link between chemicals and the plaintiff’s specific type of leukemia); *Rockman v. Union Carbide Corp.*, 266 F. Supp. 3d 839, 847 (D. Md. 2017) (experts “conflated data on *pleural* mesothelioma and *amphibole* asbestos with data on *peritoneal* mesothelioma and *chrysotile* asbestos”) (emphasis in original); *see also Goeb v. Tharaldson*, 615 N.W.2d 800, 816 (Minn. 2000) (expert made significant leap in causation opinion because he did not review pre- or post-exposure medical records, relied only on self-reported information, and applied his own tests not in use or subject to peer review).

On the other hand, *King*, 762 N.W.2d at 44, illustrates how an expert who relies on peer-reviewed medical literature can “appropriately support a general causation opinion” without falling into the analytical gap. There, the expert cited to evidence including “human data studies, animal studies, and toxicology studies[]” to support his conclusion that exposure to diesel exhaust can cause a certain type of cancer. *Id.* at 49. The expert was required to “consult the relevant scientific literature and draw a conclusion.” *Id.* His testimony, although imperfect, did not require studies showing definite conclusions of a causal relationship because the expert was qualified to interpret and extrapolate from the relevant studies. *Id.*

To bridge the analytical gap, an expert’s testimony must have a sufficient factual foundation. *Savage*, 455 Md. at 163; *Rochkind*, 454 Md. at 294; *Blackwell*, 408 Md. at 606–08; *Joiner*, 522 U.S. at 146. It is permissible for an expert to reasonably extrapolate from existing data provided that a sufficient factual basis for that opinion exists. But when the only connection between opinion testimony and the data is the expert’s assertion, without more, such testimony cannot support general causation. *Rochkind*, 454 Md. at 293–94. Dr. Blackwell-White was permitted to extrapolate from the data in offering her opinion regarding causation, provided a sufficient foundation of fact existed.

Sugarman contends that the core failing in Dr. Blackwell-White’s opinion, and hence, her failure to bridge the analytical gap, was her assessment that auditory encoding and processing speed are within the realm of attention decrements identified in the EPA-ISA.

**[Defense Counsel]:** . . . [A]re you able to say that the attention association the EPA is speaking of is the same as the very



specific attention that Dr. Kraft is referring to when he specifies, I believe, auditory encoding and processing speed?

**[Dr. Blackwell-White]:** Not only the EPA, but a good much of the lead literature involving children speaks to attention. They don't parse it out as to what kind of attention. They are using the umbrella term "attention." And so it is my opinion that the deficits that Dr. Kraft found are part of that umbrella term "attention."

Dr. Blackwell-White testified that the EPA-ISA and other research on the effects of lead exposure found a causal relationship between lead and various problems in children, including attention, impulsivity, and hyperactivity. She explained that problems with attention were "one of the earliest effects that lead was thought to have on children." She also described the way lead affects brain development by disrupting neurotransmission, which she testified could affect attention. Both Dr. Kraft and Dr. Blackwell-White testified that deficits in auditory encoding and processing speed are "factors of attention." They also described how those deficits were related to attention. Although the literature does not mention these particular deficits, both experts testified that they were within the realm of general attention deficits, and the literature does state that general attention deficits can result from lead exposure.<sup>25</sup> See *Rochkind*, 454 Md. at 290–91.

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<sup>25</sup> Sugarman contends that Dr. Blackwell-White's opinion was flawed because she did not review the entire body of literature herself, and she lacked the expertise to discern from epidemiological studies whether those studies found causation. Maryland Rule 5-702 requires that an expert have a "sufficient factual basis" to support testimony, which we have determined to mean that the expert had an adequate supply of data and applied a reliable methodology to that data. *Rochkind*, 454 Md. at 286. An integrated assessment such as the EPA-ISA comprises thousands of studies. Experts are not required to have read every possible piece of literature on their topic. Rather, there must be sufficient facts underlying the opinion such that the opinion is "more than mere speculation or conjecture." *Exxon Mobil Corp. v. Ford*, 433 Md. 426, 478 (2013).

*Rochkind* is distinct from this case. There, the expert lacked “epidemiological studies—or other reliable evidence—demonstrating a causal link between lead exposure and ADHD” but nonetheless offered a causation opinion. *Id.* at 294. A causal relationship between lead exposure and **some** symptoms of ADHD did not warrant testimony linking the two. The symptoms, we observed, were present in a “variety of other disorders and learning disabilities.” *Id.* at 290. ADHD, however, has precise diagnostic criteria and requires ruling out other behavioral disorders. *Id.* at 291. Further, the EPA-ISA emphasized other potential confounding factors that undermined Dr. Hall-Carrington’s opinion. *Id.* at 292. The EPA-ISA could not support her opinion because the studies discussed therein “only reveal **an association** between lead exposure and ADHD.” *Id.* at 291 (emphasis in original). Her opinion lacked an adequate factual basis because the source did not logically support her conclusion. *Id.* at 294–95.

Here, by contrast, the EPA-ISA identified a causal relationship between attention decrements and exposure to lead. Unlike in *Rochkind*, none of the experts opined that Liles has a diagnosable learning disability or behavioral disorder. Dr. Blackwell-White explained lead’s impact on a developing brain, including the way it affects attention. She offered the opinion that Liles suffered from the kind of generalized attention deficits the EPA-ISA identified as being caused by lead exposure. Because Dr. Blackwell-White’s opinion testimony does not suffer the same defects as were present in *Rochkind*, we hold that she had a sufficient factual basis to offer an opinion regarding general causation.

We next address Sugarman’s challenge to Dr. Blackwell-White’s specific causation testimony that, as a result of his elevated BLLs, Liles lost 4 IQ points.

### Specific Causation And IQ Loss

The Court of Special Appeals concluded that Maryland cases have accepted the Lanphear Study as a “basis for calculating IQ loss for plaintiffs in lead paint cases.” *Sugarman*, 234 Md. App. at 469 (citing *Levitas v. Christian*, 454 Md. 233, 247–48 (2017); *Rochkind v. Stevenson*, 229 Md. App. 422, 469 (2016), *rev’d on other grounds*, 454 Md. 277 (2017)). The Court reasoned that Dr. Blackwell-White had a sufficient factual basis because she based her opinion regarding Liles’s IQ loss on the Lanphear Study. *Id.* at 470. Her testimony, the intermediate appellate court said, satisfied Liles’s “burden of providing evidence, which the jury could accept or reject, that Mr. Liles’s lead exposure caused cognitive damage.” *Id.*

Sugarman does not question that epidemiological data has found a causal relationship between lead exposure and IQ loss in population studies. Thus, general causation—that the agent can cause the kind of injury the plaintiff suffers from—is met. *See Norris*, 397 F.3d at 991. Rather, Sugarman insists that specific causation is lacking—namely, there is insufficient evidence to find that Liles’s exposure to lead actually caused a loss of 4 IQ points. *See id.* Sugarman asserts that “there is no question whether or how the expert can reliably use the Lanphear Study to quantify loss once it is determined that IQ loss did occur.” Thus, Sugarman reasons, an expert may use the Lanphear Study methodology to calculate IQ loss, provided that there is expert evidence that can prove the loss. Here, Sugarman maintains, Dr. Blackwell-White’s testimony was not sufficient because she assumed IQ loss without determining whether an injury occurred.

Liles disagrees, maintaining that Dr. Blackwell-White had a “sound basis in the medical literature”—the EPA-ISA and the Lanphear Study—to offer an opinion that Liles lost IQ points. He claims that Sugarman advances a “novel[] and unsubstantiated” argument—that a “plaintiff must first prove that he sustained an IQ loss before the Lanphear Study can be utilized to calculate that loss.” Liles maintains that he has shown an injury—“cognitive deficits and IQ loss[.]” He avers that merely because his IQ is in the average range does not mean that he has not lost IQ points due to lead exposure.

The Lanphear Study is a pooled analysis that examined the relationship between children’s performance on IQ tests and BLLs less than 10 mcg/dL in multiple cohort studies around the world. See Bruce P. Lanphear et al., *Low-Level Environmental Lead Exposure and Children’s Intellectual Function: An International Pooled Analysis*, 113(7) *Envtl. Health Persps.* 894, 894–95 (July 2005). It found that “children with certain average lifetime blood levels lost a specific number of IQ points.” *Levitas v. Christian*, 454 Md. 233, 240 (2017); see also Lanphear, *supra*, at 896–97.

Cases from this Court, as well as the Court of Special Appeals, have addressed the use of epidemiological studies, as well as the Lanphear Study, in expert medical testimony about a lead-exposed plaintiff’s IQ loss. The parties dispute how this precedent applies.

#### ***Maryland Cases Addressing IQ Loss In Lead Cases***

Sugarman contends that, contrary to the Court of Special Appeals’ conclusion, Maryland cases have not squarely addressed the question of whether an expert may apply

a methodology and conclusions set forth in an epidemiological study to an individual plaintiff.

In *Richwind Joint Venture 4 v. Brunson*, 96 Md. App. 330, 336–38 (1993), *aff'd in part, rev'd in part by Richwind Joint Venture 4 v. Brunson*, 335 Md. 661 (1994),<sup>26</sup> an expert, Dr. Chisholm, explained that research showed that BLL levels were linked with approximate average IQ losses in children. *Id.* at 336–37. From these studies, and the plaintiffs' BLLs, he testified that the plaintiffs' lead levels were high enough to cause brain damage and had reduced their IQs. *Id.* at 337–38. The Court of Special Appeals determined that the trial court had not abused its discretion by permitting Dr. Chisholm's testimony about average IQ loss. *Id.* at 338. It concluded that “[a]ny reference to studies of average IQ loss—while not dispositive of the children’s injuries—offered ‘appreciable help’ to the jury to determine the extent of the injuries [the plaintiffs] sustained, and was not mere conjecture or speculation.” *Id.* In offering his opinion, Dr. Chisholm considered other factors that could have affected the plaintiffs' cognitive development, such as parental alcoholism and their home environment. *Id.* By including these factors, Dr. Chisholm's opinion had a sufficient basis. *See id.* (citing *Simmons v. State*, 313 Md. 33, 43 (1988); 6 Lynn McLain, *Maryland Practice* § 703.1 (1987)).

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<sup>26</sup> The Court of Appeals did not address the issue of IQ loss. *See Richwind Joint Venture 4 v. Brunson*, 335 Md. 661 (1994). *Richwind* was subsequently overruled in *Brooks v. Lewin Realty III, Inc.*, 378 Md. 70, 86–89 (2003), wherein we concluded that a tenant is not required to show that a landlord had notice of housing code violations to establish a *prima facie* case of negligence.

We addressed this issue in *Roy v. Dackman*, 445 Md. 23, 49 (2015), when we considered whether an expert witness, Dr. Sundel, could offer opinions regarding medical causation. Dr. Sundel relied on the Lanphear Study to offer an opinion about IQ loss due to lead exposure. *Id.* at 51. The respondents challenged whether the Lanphear Study proved support for Dr. Sundel’s opinion because other peer-reviewed studies had contradicted the Lanphear Study and disagreed that it could be applied to an individual without “real-life evidence of actual IQ point loss.” *Id.* at 51 n.16. We concluded that:

Dr. Sundel’s reliance on the Lanphear study does not invalidate the entire basis of his opinion, even if the Lanphear study is contrary to the results of other studies as alleged by Respondents. Such is the **grist for cross-examination and dueling experts and for resolution by the relative weight assigned by the fact-finder.**

*Id.* (emphasis added).

Following *Roy*, in *Rochkind v. Stevenson*, 229 Md. App. 422, 465–66 (2016), *rev’d on other grounds by Rochkind v. Stevenson*, 454 Md. 277 (2017), the Court of Special Appeals considered, *inter alia*, whether Dr. Hall-Carrington had an adequate factual basis for her testimony about IQ loss under Md. Rule 5-702. *Rochkind* contended that Dr. Hall-Carrington “improperly” relied on “general population studies” to offer an opinion about the plaintiff’s IQ loss. *Id.* Dr. Hall-Carrington used the Lanphear Study to calculate that, based on BLLs, the plaintiff lost approximately 5 to 6 IQ points. *Id.* at 466–67.

The Court of Special Appeals determined that the trial court had not abused its discretion when it accepted Dr. Hall-Carrington’s methodology, “extrapolating from epidemiological studies quantifying IQ loss resulting from lead exposure in the general

population to estimate the range of IQ loss in an individual[,]” as “sound.” *Id.* at 469. She “fully explained the basis for her calculation” during cross-examination.<sup>27</sup> *Id.* at 470. As such, her opinion was “supported by an adequate factual basis.” *Id.*

We most recently addressed this issue in *Levitas v. Christian*, 454 Md. 233 (2017). An expert witness, Dr. Klein, was prepared to offer an opinion that lead exposure caused the plaintiff’s intellectual disability and a loss of 7.4 to 9.4 IQ points. Dr. Klein reached this assessment by averaging the plaintiff’s BLLs and applying the Lanphear Study’s findings on average lifetime BLLs and IQ loss. *Id.* at 239–40. The Circuit Court excluded this opinion because, among other reasons, Dr. Klein lacked a sufficient factual basis under Md. Rule 5-702. *Id.* at 241.

We examined whether Dr. Klein had a sufficient factual basis to testify about the plaintiff’s IQ loss.<sup>28</sup> *Levitas* argued that because the Lanphear Study was population-based, it could not be used to calculate an individual’s IQ loss. *Id.* at 254. We concluded that *Roy* controlled this question: despite other reputable studies that disapproved of its use in calculating individual IQ loss, relying on the Lanphear Study did not invalidate the basis

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<sup>27</sup> The Court of Special Appeals observed that Rochkind’s experts also used the plaintiff’s BLLs to offer an opinion regarding lost IQ points, although they claimed that the plaintiff lost fewer IQ points than Dr. Hall-Carrington’s estimate. *Rochkind v. Stevenson*, 229 Md. App. 422, 470 (2016), *rev’d on other grounds by Rochkind v. Stevenson*, 454 Md. 277 (2017).

<sup>28</sup> *Levitas* argued that Dr. Klein’s basis was inadequate because he had not personally examined the plaintiff to form his opinion and instead relied on the neuropsychologist’s report. *Levitas v. Christian*, 454 Md. 233, 253–54 (2017). We rejected this argument, pointing out that *Roy v. Dackman*, 445 Md. 23 (2015) and other cases contemplated that an expert’s factual basis can arise from a number of sources, including reports prepared by another doctor. *Levitas*, 454 Md. at 254.

of an expert’s opinion. *Id.* at 254–55 (citing *Roy*, 445 Md. at 51–52 & n.16). Opposing counsel, we explained, was free to attempt to undermine that opinion through cross-examination and testimony from contrary experts, leaving the matter in the fact finder’s hands. *Id.*

*Levitas* and *Roy* demonstrate that a properly qualified expert witness can rely on the Lanphear Study methodology, as well as other accepted scientific research, as a factual basis for an opinion that a plaintiff’s elevated BLLs caused the loss of a specific number of IQ points. *See id.* at 254–55; *Roy*, 445 Md. at 51 n.16; *see also Rochkind*, 229 Md. App. at 469–70. Legitimate scientific disputes go not to the admissibility of testimony, but to the weight of the evidence. *See Levitas*, 454 Md. at 254–55; *Roy*, 445 Md. at 51 n.16; *Exxon Mobil Corp.*, 433 Md. at 483.

### ***Liles’s IQ Loss***

Sugarman contends that affirming the Court of Special Appeals’ decision “invites any litigant to prove brain injury with only evidence of BLLs and no evidence of actual cognitive injury through neuropsychological evaluation or otherwise.” Sugarman also forecasts dire consequences, specifically that we would “eviscerate” fundamental rules of injury and causation.

Sugarman argues that Liles has not shown sufficient evidence of injury to satisfy specific causation because Dr. Blackwell-White assumed Liles lost IQ points solely by linking his exposure to the epidemiological data. Liles’s full-scale IQ score was 94, which Dr. Kraft characterized as in the “average range of intelligence . . . .” Sugarman attempts to distinguish the cases above by pointing out that some plaintiffs had far lower IQs than



Liles. *See Roy*, 445 Md. at 34 (full-scale IQ of 78); *Rochkind*, 229 Md. App. at 434 (full-scale IQ of 76). The IQs in *Roy* and *Rochkind* were sufficient evidence of IQ loss because they were lower than would otherwise be expected. Sugarman asserts that Liles’s IQ has not been shown to be lower than expected.

Sugarman suggests that plaintiffs with average or higher IQs could also prove injury by demonstrating an IQ deficit through a “neuropsychological evaluation that finds a statistically significant discrepancy between the IQ measure and other cognitive measures, such that the IQ measure, even though it is average, is lower than would otherwise be expected . . . .” Sugarman has not offered any legal authority to support limiting proof of injury to these two methods. We decline to adopt such a restrictive approach.

Dr. Kraft and Dr. Blackwell-White testified about Liles’s cognitive function, as well as the specific deficits that Dr. Kraft identified. Dr. Kraft explained that he found a “significant” discrepancy in Liles’s scores on the WAIS-IV test. Liles obtained a “perceptual reasoning index score of 104.” By contrast, on the “two index scores that are most sensitive to attention and concentration, which are working memory and processing speed,” Liles obtained a “standard score of 86 on both of those measures.” Dr. Kraft stated that those scores were “statistically, significantly lower than his performance in perceptual reasoning.” From this, he concluded that Liles had a mild brain impairment—deficits in auditory encoding and information processing speed.

Dr. Kraft testified that despite Liles’s average full-scale IQ, he could not rule out brain damage because of the discrepancy in Liles’s scores. As Sugarman points out, Dr. Kraft acknowledged on cross-examination that he did not find “deficits in IQ.” But

immediately following that, he stated that “it’s the **pattern of the scores that suggest deficit** to me.” (Emphasis added). This acknowledgment might detract from, but does not obliterate, Dr. Kraft’s opinion on brain impairment. When we place this testimony in context, we see that Dr. Kraft interpreted Liles’s scores as indicative of cognitive injury.

Dr. Blackwell-White built on Dr. Kraft’s testimony when offering her own specific causation opinion. She agreed that population studies do not necessarily mean that each individual child in the population could show a measurable effect from lead exposure, and that neuropsychological testing may not be able to measure some damage. She explained that while she believes that “every child who’s exposed to lead does lose IQ points. . . . [the] very specific psychological tests that are given to these children . . . may or may not pick up the deficits that these children have.” On cross-examination, Dr. Blackwell-White stated that, in her opinion, the results of Liles’s neuropsychological testing demonstrated brain injury.

**[Defense Counsel]:** Okay. Would you agree with me that you do not have the quantifiable evidence to say to a reasonable degree of medical probability that Mr. Liles’[s] lead exposure caused him to have academic difficulties?

**[Dr. Blackwell-White]:** Dr. Kraft, in his evaluation of Chauncey Liles, found a discrepancy between—among some of his IQ—some of his test results. He found a perceptual reasoning index of 104, which is solidly average. But he found a processing speed—but he found a processing speed index and a working memory index that were in the low average range, 86. I mean that’s a significant difference. In my opinion, that is a quantifiable indicator of brain injury. Again, when there is a discrepancy between subtests of neuropsychological testing, that often indicates some brain deficit or some brain injury in very—in a very specific area.

Although Dr. Blackwell-White offered broad opinions about lead-exposed plaintiffs and IQ loss, here, she also offered specific testimony that the results of Liles's IQ testing revealed brain injury and explained why Liles's specific deficits were more consistent with an injury than another cause.

Like the experts in *Levitas* and *Roy*, Dr. Blackwell-White relied on Liles's documented BLLs, the results of his neuropsychological evaluation, and the Lanphear Study, to estimate a specific IQ point loss. She did not look only to his elevated BLLs, assume he had a cognitive defect, and then extrapolate a point loss based on the Lanphear methodology. Rather, Dr. Blackwell-White relied on evidence that Liles—individually—had already manifested cognitive deficits. This gave her enough reason to use her knowledge and expertise to extrapolate a loss of IQ points according to the methodology outlined in the Lanphear Study. Sugarman presented contradictory experts and extensively cross-examined Liles's experts about their conclusions. That is what we contemplated in *Levitas* and *Roy*. See *Levitas*, 454 Md. at 254–55; *Roy*, 445 Md. at 51 n.16.

Dr. Blackwell-White's testimony provided sufficient evidence for the jury to draw the inference that, more likely than not, Liles's elevated BLLs caused a measurable loss of IQ points. For that reason, the trial court appropriately denied Sugarman's motions and allowed the question to go to the jury.

## **DAMAGES**

Sugarman also contends that Liles did not put forth sufficient evidence to prove damages. Even if lead exposure caused Liles's injuries, Sugarman argues, Liles did not prove any damages beyond mere speculation. Sugarman focuses on the testimony of

Lieberman, Liles’s vocational rehabilitation expert, and argues that Lieberman “expressly made no assessment of Mr. Liles’s likely pre-injury (pre-lead exposure) educational and vocational capacity . . . .” Rather, he avers, Lieberman should have employed a different methodology—the same methodology used by Sugarman’s vocational rehabilitation expert—that takes into account parental benchmarks to measure a child’s likely outcomes absent injury. Sugarman insists that Lieberman’s opinion rested on the “baseless” assumption that without deficits, Liles would have obtained an Associate’s degree.

Here, Liles offered evidence, in the form of Lieberman’s testimony, that he will not earn an Associate’s degree and that, without the deficits caused by his exposure to lead, he would have earned a degree. He then offered Dr. Conte who explained the difference in lifetime earnings between a person with an Associate’s degree and a person without an Associate’s degree. Accordingly, Liles has attempted to show a loss in earning capacity.<sup>29</sup>

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<sup>29</sup> This Court has not yet discussed how a plaintiff can prove a loss in earning capacity. Comment d to the Restatement (Second) of Torts § 924, however, provides helpful guidance:

The extent of future harm to the earning capacity of the injured person is measured by the difference, viewed as of the time of trial, between the value of the plaintiff’s services as they will be in view of the harm and as they would have been had there been no harm. This difference is the resultant derived from reducing to present value the anticipated losses of earnings during the expected working period that the plaintiff would have had during the remainder of his prospective life, but for the defendant’s act. . . . Accordingly, the trier of fact must ascertain, **as nearly as can be done in advance**, the difference between the earnings that the plaintiff probably would or could have received during his life expectancy but for the harm and the earnings that he will probably be able to receive during the period of his life expectancy as now determined. In this

That is, he tried to prove that his lead exposure reduced the amount of money he will earn over his lifetime. A claim for lost earnings differs from that of lost wages:

Evidence in [a] wage loss claim reflects loss of specific opportunities, such as those represented by an existing job. Proof typically shows past wage and future prospects in the job, coupled with proof that the plaintiff can no longer work or can work only part time. . . . For example, evidence might show that the plaintiff was studying to become an engineer and because of his injury he could no longer master the materials, or general capacity for advancement which has been limited by the injury. In either kind of claim, the evidence must be sufficient to permit a **reasonable estimate** of the loss claimed.

Dan B. Dobbs, *Law of Remedies* § 8.1(2), at 364 (2d ed. 1993) (emphasis added) (footnotes omitted).

In a personal injury action, a plaintiff must prove that injury and damages were proximately caused by the negligent acts of the defendant. *See, e.g., Washington Metro. Area Transit Auth. v. Seymour*, 387 Md. 217, 223 (2005). Damages must be actual, not speculative, remote, or uncertain. *See Mount Royal Cab Co. v. Dolan*, 166 Md. 581, 584 (1934) (“[T]he recovery is limited to those consequences which have actually and naturally ensued the tort, or which may certainly or reasonably and probably result as a proximate

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computation, there are considered the type of work that the plaintiff has done and the type of work that, in view of his physical condition, education, experience and age, he would have been doing and will be likely to do in the future during the working period of his life, together with all other matters reasonably relevant.

(Emphasis added).

consequence of the act, but not consequences which are merely possible, and so speculative or conjectural.”); *see also Jones v. Malinowski*, 299 Md. 257, 268–69 (1984) (same).

Harper, James, and Gray, in their fundamental Torts treatise, have explored the level of certainty required to prove future damages. *See Fowler V. Harper, Fleming James & Oscar Gray, Harper, James and Gray on Torts*, §25.3, 590–603 (3d ed. 2007). Their discussion focused on lost profits. “Originally,” they explained, “the speculative or contingent nature of [lost] profits was regarded as a **complete bar** to their recovery in any case.” *Id.* at 591 (emphasis added). Rules have developed however, that tend to “ameliorate the harshness of restrictive [damages] rules,” while “others are more lenient toward the plaintiff than are general canons of proof.” *Id.* The rigid prohibition on claiming lost profits, for example, has “given way to a more flexible requirement of ‘reasonable certainty.’” *Id.* at 591–92. But such evidence requires more supporting evidence beyond “conjecture or speculation.” *Id.* at 593–97.

Professor McCormick, in his treatise on damages, also explained that the “harsh” rule of certainty gave way to various “subdoctrines” applicable in proving future harm:

There are various modifications to the rule of certainty. They enable the courts, while holding up a high standard of certainty as an ideal, to avoid harsh applications of it. Among them are: (a) if the fact of damage is proved with certainty, the extent or amount may be left to reasonable inference[;] . . . (c) Mere difficulty in ascertaining the amount is not fatal[;] (d) Mathematical precision in fixing the exact amount is not required[;] and (e) If the best evidence of the damage of which the situation admits is furnished, this is sufficient.

McCormick, *Handbook on the Law of Damages* § 27, at 101–02 (1931). In claims for lost earning capacity, “since the extent and hence the value, of future earning power, depends

on probabilities, and cannot be reduced to even a reasonable certainty, the courts should—and do—scan with much more charitable eyes, the sufficiency of proof of this item of damage . . . .” *Id.* at 309.

We have explored the certainty of proof required in a claim for lost earning capacity. In *Adams v. Benson*, 208 Md. 261, 265–66 (1955), the plaintiff injured her hand when she fell into an unprotected electric fan at a tavern. The plaintiff’s expert, a dermatologist, explained that the plaintiff’s hand was likely to remain permanently tender and sensitive due to scarring. *Id.* Her injury led the packing company that employed the plaintiff to reduce her salary after the accident. *Id.* at 272. The defendants excepted to the trial court’s instruction that the jury could consider: “(1) how far plaintiff’s injuries might disable her in her employment; and (2) any loss of earnings which she has sustained and might sustain as a result of her injury.” *Id.* at 270. They argued there was no evidence that she would be unable to work as a domestic servant and that there was no “certainty of future loss of earnings.” *Id.*

The plaintiff’s evidence—injury and reduced wages—was sufficient to show a reduction in her earning capacity. We said, “[t]he determination of the extent of impairment of earning power as a result of injury, although involving contingencies and matters of opinion, is an ordinary function of the triers of fact.” *Id.* at 272. In many cases, evidence of income before and after injury will be available for comparison. “[S]uch a comparison,” however, “is not essential to proof of diminished earning power, but **all relevant facts must be considered.**” *Id.* at 272–73 (emphasis added). *See also Ihrie v. Anthony*, 205 Md. 296, 305–07 (1954) (jury had sufficient evidence to conclude that the plaintiff suffered a loss in earning capacity, even when the plaintiff was not working at the

time of injury); *Maryland, D. and V. Ry. Co. v. Brown*, 109 Md. 304, 315 (1909) (it was “certainly proper” for the jury to know a plaintiff’s “earnings at the time of his injury” and those “he was capable of earning and did receive afterwards”).

In *Bender v. Popp*, 246 Md. 65, 71 (1967), the plaintiff testified that she sustained a neck injury following a car accident. She also said that she stopped working more than two years after the accident, due to her neck injury, and did not intend to go back to work. *Id.* at 69. Both the defendant’s doctor and the plaintiff’s doctor testified that the injury was permanent. *Id.* at 71. From this, we concluded that the jury should have been instructed regarding lost earning capacity. We said that “the undisputed evidence of a permanent injury, **even though close to minimal limits**, renders the question of its effect on future earning capacity and job opportunities a matter within the purview of the jury.” *Id.* (emphasis added). In such a situation, the effect of the injury “on the question of damages is fair game for both adversaries to argue to the trier of fact.” *Id.* at 72.

More recently, the Court of Special Appeals has considered the sufficiency of proof of damages in a claim for lost earning capacity. In *Anderson v. Litzenberg*, 115 Md. App. 549, 573–77 (1997), the plaintiff was injured while driving behind a piece of construction equipment owned and operated by the defendants. *Id.* at 557–58. At the time of the injury, the plaintiff was employed as a construction worker with his father’s company and ran his own rental property business. At trial, the plaintiff testified that his injuries prevented him from working on the rental property business, which had yet to turn a profit. *Id.* at 574. Nonetheless, he offered an expert who testified to the cost of hiring a replacement property



manager for the business. Ultimately, the jury awarded the plaintiff \$349,400 in damages, \$213,000.00 of which was compensation for future lost earnings. *Id.* at 559.

The defendants argued that the plaintiff could not recover future profits from an unprofitable business. The intermediate appellate court rejected this argument and explained:

It is generally recognized that impairment of earning capacity seeks to compensate the plaintiff for a reduction in his **ability to earn** through his personal services. Once the fact of impaired earning capacity is established, the plaintiff must submit evidence so that the extent of the impairment can reasonably be determined. The prevailing proper measure of lost earning capacity is the difference between the amount that the plaintiff was capable of earning before his injury and that which he is capable of earning thereafter.

*Id.* at 573 (emphasis added) (citations omitted). In cases seeking recovery for lost earning capacity, “[a]s a general rule, any evidence is admissible that would assist the fact finder in determining the plaintiff’s earning capacity before the injury and the potential decrease in that capacity after the injury . . . .” *Id.* Relying on these principles, the Court of Special Appeals concluded that the plaintiff could recover in his claim of lost earning capacity even though the business he owned had yet to turn a profit. *Id.* at 576–77.

A few years later in *Lewin Realty III, Inc. v. Brooks*, the Court of Special Appeals again upheld an award for lost earning capacity after concluding that the plaintiff’s vocational rehabilitation expert had provided sufficient evidence of a reduction in earning capacity. 138 Md. App. 244 (2001), *aff’d* 378 Md. 70 (2003), *abrogated on other grounds by Ruffin Hotel Corp. of Md., Inc. v. Gasper*, 418 Md. 594 (2011). The plaintiff called Lieberman as his vocational rehabilitation expert, and he opined, within a degree of

reasonable certainty, that the plaintiff had suffered a reduction in earning capacity as a result of his exposure to lead. *Id.* at 279.

Lewin Realty argued that Lieberman’s opinion was “too speculative” because the plaintiff had “no history of working,” and “numerous forces . . . could intervene before the infant plaintiff enters the work force . . .” *Id.* at 279. Lieberman based his opinion on facts “personal to [the plaintiff] as an individual.” *Id.* at 284. He reviewed a doctor’s neuropsychological evaluation of the plaintiff, his educational records, medical records, and achievement of developmental milestones. *Id.* Lieberman also considered the plaintiff’s mother’s work history and educational background. Based on **all** this information, Lieberman reasoned that, without a lead-caused disability, the plaintiff would have attained an educational level between the 9th and 12th grade. With the disability however, the plaintiff was likely to drop out of high school at age 16 and would likely find work in “very basic manual labor.” *Id.* at 285.

The Court of Special Appeals reasoned that Lieberman’s opinion was based on more than mere generalized statistical data regarding the likely employment outcomes of the population at large. The intermediate appellate court concluded:

[n]otwithstanding that [the plaintiff] had no work history or track record of employment, the combination of evidence specific to [the plaintiff] and general to the population that was adduced at trial was such as to permit a reasonable finding that, more likely than not, [the plaintiff’s] future earning[s] would be less than it would have been if he were not injured. The evidence was reasonably certain and was not based on speculation or conjecture.

*Id.*

Other courts have assessed and approved various offers of proof as sufficient for a claim of lost earning capacity. See *Andler v. Clear Channel Broadcasting, Inc.*, 670 F.3d 717, 726–28 (6th Cir. 2012) (expert’s testimony based on specific analysis of the plaintiff and labor statistics was not unreasonable); *Hammons v. Paul*, 101 So. 3d 1006, 1011–12 (La. Ct. App. 2012) (testimony from plaintiff’s vocational rehabilitation expert sufficient where he reviewed medical records and performed an independent vocational assessment); *Klingman v. Kruschke*, 339 N.W.2d 603, 604–05 (Wis. 1983) (vocational rehabilitation expert’s testimony sufficient to support a finding of lost earning capacity where expert relied upon an interview, testing, and review of medical records). In other cases, courts have rejected—due to insufficiency—claims for loss of earning capacity. *Hughes v. Pender*, 391 A.2d 259, 262–63 (D.C. 1978) (expert’s opinion relying on statistical data alone insufficient to demonstrate a loss of earning capacity); *Bulala v. Boyd*, 389 S.E.2d 670, 677–78 (Va. 1990) (plaintiff who relied on “statistical averages alone” had not proven damages within a reasonable degree of certainty because the “evidence must be grounded upon facts specific to the individual whose loss is being calculated”).

Sugarman alleges that Liles’s damages claim should not have been submitted to the jury because Lieberman’s opinion was “baseless.” We disagree, and apply the standards from *Anderson* and *Lewin Realty*, as those cases are entirely consistent with our earlier rulings in *Adams* and *Bender*. As in those cases, Lieberman’s opinion was based on substantial material. He interviewed Liles, conducted additional vocational testing, and reviewed his educational and medical records. He also reviewed and relied upon the neuropsychological evaluation and conclusions of Dr. Kraft. Additionally, Lieberman

relied on his years of experience as a vocational rehabilitation counselor during which he has helped thousands of students attend college. After reviewing this data, he concluded that Liles was not likely to receive a college degree due to the attention problems Dr. Kraft identified. He further proffered that, in his expert opinion, Liles would have been able to earn a college degree without his disabilities. Dr. Conte, the economics expert, then testified regarding the financial earnings of an individual with a college degree versus those of an individual without a college degree.<sup>30</sup>

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<sup>30</sup> Sugarman asserts that Dr. Conte based his opinion regarding Liles's economic earnings on an assumption that cannot be drawn from Lieberman's opinion. Sugarman takes issue with the following exchange:

**[Defense Counsel]:** So your testimony is that—you're saying that what Mr. Lieberman is saying is that even if he gets a four-year degree, he will only be able to compete as a high school graduate with some college? Is that the—is that the assumption you're proceeding under?

**[Conte]:** No, I'm not making any assumptions about whether Mr. Lieberman admits . . . if Mr. Liles is going to be able to obtain a college degree. I heard him say he's probably not. But nonetheless, the important part of Mr. Lieberman's opinions—and this is the only part of Mr. Lieberman's opinions upon which I'm specifically relying, is that—**and in parens, I will put, irrespective of his actual educational attainment, but that's just in parens—he will most likely be able to command a wage commensurate with that of an individual with high school and plus some college.**

(Emphasis added). Despite Sugarman's assertion otherwise, Dr. Conte relied on Lieberman's conclusion that Liles would never receive a college degree, and his answer above says just that. Dr. Conte's testimony that Liles will not command a wage of a college graduate is entirely consistent with Lieberman's opinion that Liles will not graduate from college.

The combined information offered by Lieberman and Dr. Conte presented a detailed and individualized analysis of Liles’s employment prospects and future earnings. Unlike the plaintiffs in cases where the evidence has been deemed insufficient to prove damages, Liles set forth an individualized analysis of his likely outcome coupled with statistical data to assist the jury in quantifying his damages. Although an award for lost earning capacity is necessarily less certain than pecuniary damages in other contexts, we view Liles’s evidence as similar to—if not stronger than—the evidence offered by the plaintiffs in *Adams*, *Anderson*, and *Lewin Realty*. Liles put forth evidence that proved the damages that will “certainly or reasonably and probably” result from his injuries. *Adams*, 208 Md. at 272–73.

Sugarman argues that Lieberman’s opinion was insufficient because he did not consider the educational and work history of Liles’s parents. If he had, the argument goes, Lieberman would have realized that neither of Liles’s parents had earned a college degree. Sugarman contends that consideration of parental achievement would invalidate Lieberman’s conclusion that, absent injury, Liles would have earned a degree. Lieberman should have changed his conclusion that Liles would have otherwise earned a college degree merely because his parents did not finish college. Sugarman points to the fact that in both *Lewin Realty* and *Rochkind*, Lieberman considered parental achievement when assessing the plaintiffs’ likely outcomes with and without injury. Although the Court of Special Appeals approved of such parental consideration in *Lewin Realty* and *Rochkind*, we do not view those cases as mandating a **requirement** that a vocational expert must consider parental achievement when offering an opinion as to a plaintiff’s pre-injury outcome. Sugarman offers no authority for such a requirement. Accordingly, we conclude

that Liles set forth sufficient evidence of damages in the form of loss of earning capacity and the trial court correctly submitted the issue to the jury.

### **CONCLUSION**

Unlike the expert's opinion in *Rochkind*, Dr. Blackwell-White's testimony regarding the causation of Liles's attention problems did not suffer from an "analytical gap." Her review of the epidemiological literature, coupled with an analysis of Liles specifically, supported her conclusion that Liles's elevated BLLs caused his IQ loss. In addition, Liles sufficiently demonstrated, beyond mere speculation, the existence of damages as a result of the injury caused by Sugarman's negligence. Accordingly, the trial court did not err in submitting the case to the jury on the issues of causation and damages.

**JUDGMENT OF THE COURT OF  
SPECIAL APPEALS AFFIRMED. COSTS  
TO BE PAID BY PETITIONERS.**

Circuit Court for Baltimore City  
Case No. 24-C-14-005808  
Argued: June 1, 2018

IN THE COURT OF APPEALS  
OF MARYLAND

No. 80

September Term, 2017

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STANLEY SUGARMAN, et al.

v.

CHAUNCEY LILES, JR.

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Barbera, C.J.  
Greene,  
Adkins,  
McDonald,  
Watts,  
Hotten,  
Getty,

JJ.

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Concurring and Dissenting Opinion by Getty, J.

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Filed: July 31, 2018

I concur with two of the majority's conclusions: (1) Dr. Blackwell-White's reliance on documented BLLs, a neuropsychological evaluation, and the Lanphear Study provided sufficient basis for her expert testimony as to specific causation (*i.e.*, that Liles's elevated BLLs caused a loss of IQ points); and (2) Liles provided sufficient evidence of lost earning capacity damages to submit the issue to the jury. However, I respectfully dissent from the Court's holding that Dr. Blackwell-White had a sufficient factual basis to offer an expert opinion as to general causation (*i.e.*, that lead exposure can cause impairments to auditory encoding and information processing speed). Dr. Blackwell-White did not provide a sufficient factual basis to opine specifically that auditory encoding and processing speed fell under the broad term "attention" contemplated by the EPA-ISA study, which found a causal relationship between lead exposure and "attention decrements."

In *Rochkind v. Stevenson*, we analyzed whether epidemiological studies can supply a sufficient factual basis for expert causation testimony as required by Maryland Rule 5-702. 454 Md. 277, 288 (2017). In *Rochkind*, this Court recognized that a "sufficient factual basis" for an expert opinion as to causation involves "two subfactors: an adequate supply of data and a reliable methodology." *Id.* at 286. Because we had not previously decided the issue, this Court looked to *General Electric Co. v. Joiner*, 522 U.S. 136 (1997), in which the Supreme Court considered whether epidemiological studies constituted a sufficient basis for an expert opinion that exposure to polychlorinated biphenyls can cause lung cancer. *See id.* The Supreme Court ultimately held that the epidemiological studies were not sufficient to support the expert's conclusions, noting that:



[C]onclusions and methodology are not entirely distinct from one another. Trained experts commonly extrapolate from existing data. But nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is **connected to existing data only by the ipse dixit of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered.**

*Joiner*, 522 U.S. at 146 (emphasis added).

Applying that standard to the facts before it in *Rochkind*, this Court determined that there was not a sufficient factual foundation for the expert’s reliance on the EPA-ISA study as support for the expert opinion that lead exposure can cause ADHD. 454 Md. at 290. Specifically, we concluded that:

The jump from attention deficits and hyperactivity to a clinical ADHD diagnosis may seem reasonable, but we have explained that “just because a conclusion is reasonable does not mean that a court must permit an expert to make it.” Because of the added weight a jury might give to testimony from a designated expert, the trial court “ought to insist that a proffered expert bring to the jury more than the lawyers can offer in argument.” **In equating attention deficits and hyperactivity with a clinical ADHD diagnosis, Dr. Hall-Carrington painted an inaccurate picture of the scientific research regarding lead poisoning—she overstated the known effects of lead exposure. Her testimony suffers from the same “analytical gap” described in *Joiner*.**

*Id.* at 291 (emphasis added) (citations omitted).

In this case, Dr. Blackwell-White relied on the identical EPA-ISA study considered in *Rochkind* in her attempt to provide expert testimony as to general causation. The EPA-ISA finds that the numerous epidemiologic studies are “sufficient to conclude that there is a causal relationship between [lead] exposure and effects on attention” and “attention decrements.” U.S. Env’tl. Protection Agency, Integrated Science Assessment for Lead

(“EPA-ISA”), EPA/600/R-10/075F (June 2013), <https://perma.cc/5ZRA-W54H> at 4-294, 4-289. As to what the study means by “attention,” the EPA-ISA only states that “[a]ttention is the ability to maintain a consistent focus on an activity or relevant stimuli and can be assessed by examining sustained attention, concentration, or distractibility.” EPA-ISA at 4-153. However, the study does not specifically list which attention decrements the individual epidemiological studies assessed; in other words, the EPA-ISA never identifies auditory encoding and processing speed as two types of “attention decrements” caused by lead exposure.

Prior to Dr. Blackwell-White’s testimony, Liles offered Dr. Kraft as an expert in psychology and neuropsychology. Dr. Kraft testified that he looked at Liles’s “performance on the two index scores that are most sensitive to attention and concentration, which are working memory and processing speed.” Dr. Kraft explained that the results of Liles’s performance “suggest[] to [him] that [Liles] has a mild impairment with respect to auditory encoding of information in the working memory, and information processing speed. Both of those are factors of attention.”

Liles subsequently offered Dr. Blackwell-White as an expert in pediatrics and childhood lead poisoning. Dr. Blackwell-White testified that she relied, in part, on the EPA-ISA study, which concluded that lead exposure can cause attention problems. In addition, Dr. Blackwell-White noted that Dr. Kraft “associated” auditory encoding and processing speed “to measures of attention.” Dr. Blackwell-White provided only the following testimony to link the factors of attention identified by Dr. Kraft to the EPA-ISA: “Not only the EPA, but a good much of the lead literature involving children speaks to

attention. They don't parse it out as to what kind of attention. They are using the umbrella term 'attention.' **And so it is my opinion that the deficits that Dr. Kraft found are part of that umbrella term, 'attention.'**" (Emphasis added). Therefore, Dr. Blackwell-White testified that it was her opinion that deficits to auditory encoding of information and information processing speed were two attention decrements that the EPA-ISA study considered when it concluded that lead exposure is causally related to attention.

In my view, there was insufficient information bridging the "analytical gap" between the attention decrements that the EPA-ISA study found to be caused by lead exposure and the specific "factors of attention" of auditory encoding and processing speed. *See Rochkind*, 454 Md. at 291. The only evidence that the jury heard connecting the "attention decrements" in the study to the two specific cognitive deficits from which Liles suffered was Dr. Blackwell-White's singular statement that it is her opinion that auditory encoding and processing speed are part of the umbrella term "attention" as used in the EPA-ISA. This type of unfounded statement was exactly what the Supreme Court cautioned against in *Joiner*: "Trained experts commonly extrapolate from existing data. **But nothing . . . requires a [] court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert.**" 522 U.S. at 146 (emphasis added).

Respectfully, I believe that Dr. Blackwell-White's reliance on the EPA-ISA study along with her unsupported statement that Liles' cognitive deficits fall under the attention decrements identified in the study do not provide a sufficient factual basis to offer an expert opinion as to general causation. I would instead hold that this case suffers a somewhat

distinct, but nonetheless significant, analytical gap as was present in *Rochkind*, 454 Md. at 291.