No. 19-8105

IN THE
Supreme Court of the United States

JUSTIN ANDERSON,
Petitioner,

v.

DEXTER PAYNE, Director,
Arkansas Division of Correction.
Respondent.

On Petition for a Writ of Certiorari to the United States Court of Appeals for the Eighth Circuit

BRIEF OF NATIONAL ORGANIZATION ON FETAL ALCOHOL SYNDROME AS *AMICUS CURIAE* IN SUPPORT OF PETITIONER

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INTEREST OF *AMICUS CURIAE*

The National Organization on Fetal Alcohol Syndrome ("NOFAS") is a nonprofit public health advocacy organization committed to preventing prenatal exposure to alcohol, drugs, and other harmful substances. NOFAS educates and supports women (before and during pregnancy) and works to secure access to therapeutic services for individuals, families, and communities living with the effects of Fetal Alcohol Spectrum Disorders ("FASD") and other preventable intellectual and developmental disabilities.[1] FASD is the leading preventable cause of developmental disabilities and birth defects, and a leading cause of learning disabilities. Nearly 100,000 newborns in the United States enter the world every year having been exposed to a mother's heavy or binge drinking.

Petitioner Justin Anderson is one of them. The defects in his mental functions reflect classic FASD characteristics. So does the fact of his incarceration. Approximately 60% of people suffering from FASD have a history of legal trouble; and 50% have been confined to a jail, prison, treatment facility, or psychiatric hospital. Natalie Novick Brown et al.,

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[1] NOFAS files this brief pursuant to Rules 37.2(a) and 37.3(a). Counsel of record received timely notice of intent to file this brief, and all parties to the appeal have consented to the filing of this brief. Pursuant to Rule 37.6, NOFAS certifies that no counsel for any party authored this brief in whole or in part; no party, or counsel for any party, made a monetary contribution intended to fund the preparation or submission of this brief; and no individuals or organizations other than NOFAS, its members, or its counsel made a monetary contribution intended to fund the preparation or submission of this brief.

Prenatal Alcohol Exposure: An Assessment Strategy for the Legal Context, 42–43 Int'l J.L. & Psychiatry 144, 144 (2015). Because prenatal alcohol exposure so often leads to long-term legal consequences, NOFAS—like the American Bar Association—believes that law enforcement officials, courts, jurors, and corrections officers have an obligation to consider FASD during all stages of an individual's encounter with the criminal justice system, including sentencing, confinement, mitigation, diversion, and exclusion from the death penalty. Am. Bar Ass'n, FASD Resolution and Report (Aug. 7, 2012), https://www.americanbar.org/groups/public_interest/child_law/resources/attorneys/fasd-resolution/.

Mr. Anderson's case is a stark reminder of the costs, both personal and societal, of a judicial process that fails to recognize and account for FASD's devastating effects. FASD left Mr. Anderson with permanent brain damage, impairing his ability to function as an adult, yet Mr. Anderson's capital defense team did nothing to explore FASD's impact on his culpability. NOFAS hopes that a heightened understanding of FASD can inform both the Court's review of Mr. Anderson's sentence and lead to more appropriate outcomes for vulnerable individuals across the criminal justice system.

SUMMARY OF THE ARGUMENT

FASD causes permanent organic brain damage that severely impairs an individual's ability to function in society. It is undisputed that Mr. Anderson suffers from a disabling form of FASD known as Partial Fetal Alcohol Syndrome ("PFAS"),

which results in severe brain damage and accompanying deficiencies in adaptive behavior and executive functioning. Although Mr. Anderson's IQ score is above the range for an intellectual disability diagnosis, the record establishes that his adaptive-functioning defects mirror those of a person with intellectual disabilities. The record also indicates that Mr. Anderson's affliction left him with the mental functioning of a child at the time of the offenses for which he was convicted—closer to a nine- or eleven-year-old than the adult of nineteen who stood accused.

This Court has prohibited the execution of children and individuals with intellectual disabilities on the grounds that they act with diminished moral culpability. Mr. Anderson and others with FASD operate at a level of cognitive function equal to that of children and people with intellectual disabilities, and they thus face a similar risk of wrongful execution. Because Mr. Anderson has the diminished moral culpability of an intellectually disabled person or a child, evidence of his FASD-induced brain damage should carry significant mitigating weight.

Indeed, this Court and multiple federal courts have expressly recognized the uniquely compelling nature of brain damage as mitigating evidence in death penalty cases. FASD is fundamentally *different in kind* from other mitigating evidence because FASD causes permanent brain damage that inherently renders a defendant less culpable. Even though FASD was a well-known medical condition and form of mitigation evidence at the time of Mr.

Anderson's trial—and despite numerous indications that his mother consumed alcohol during pregnancy—Mr. Anderson's capital defense team failed to investigate for FASD altogether. Such evidence would have carried persuasive and unique mitigating weight because it would have offered the jury a biological explanation for Mr. Anderson's conduct and likely would have reduced the jury's appraisal of his moral culpability.

ARGUMENT

I. FASD ADDS SIGNIFICANT MITIGATING WEIGHT TO A CAPITAL-SENTENCING PROFILE

a. FASD is an organic brain condition caused by fetal alcohol exposure

FASD is a spectrum of "conditions that can occur in individuals whose mother drank alcohol during pregnancy." ABA Resolution and Report Approved August 7, 2012, at 2 (citing National Institute on Alcohol Abuse and Alcoholism, Alcohol Alert, Fetal Alcohol Spectrum Disorders: Understanding the Effects of Prenatal Alcohol Exposure, No. 82). People with FASD are born with significant brain damage. *Id.* at 2–3; *see also* Burd and Edwards, *Fetal Alcohol Spectrum Disorders: Implications for Attorneys and the Courts*, Crim. Just. (Fall 2019) at 27.

While FASD is typically associated with certain facial characteristics that can be measured and ranked, those outward features often fade as the

individual ages.[2] Svetlana Popova, Danijela Dozet, and Larry Burd, *Fetal Alcohol Spectrum Disorder: Can We Change the Future?*, Alcohol Clin. Exp. Res. (Vol. 44, No. 4 2020), at 817–18. Thus, the most reliable indicator of FASD is the permanent brain damage it causes and the accompanying deficiencies in adaptive behavior and executive functioning. *See* Petitioner's Eight Circuit Exhibits, Volume II ("8th Cir. Exs."), Ex. 55 at 26 ("it is executive functioning . . . that determines learning capacity").

Because it is "genetically programmed," FASD is a permanent, severe developmental disability that cannot be outgrown. Burd and Kerbeshian, *Fetal Alcohol Spectrum Disorders*, Int'l Jour. of Alc. and Drug Res. (2013), at 3. FASD causes a "dysfunction of or damage to" the central nervous system, resulting in permanent developmental impairments. This affects an individual's cognitive, emotional, and social brain functions. Burd and Kerbeshian, "Fetal Alcohol Spectrum Disorders" (2013), at 3. In short, FASD permanently alters an individual's brain structure. *See* Hoyme et al., "Updated Clinical Guidelines for Diagnosing Fetal Alcohol Spectrum Disorders" (2016) at 6.

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[2] Facial features associated with FASD include a smooth philtrum (the vertical groove between the nose and upper lip), thin vermillion border of the upper lip, and small palpebral fissures (the space between the two eyelids). *See* Hoyme et al., *Updated Clinical Guidelines for Diagnosing Fetal Alcohol Spectrum Disorders*, Pediatrics (Vol. 138, No. 2 2016) at 3. While Mr. Anderson displays some of these external features, including a smooth philtrum and thin upper lip, his do not reflect the most extreme forms. 8th Cir. Exs., Ex. 42 at 9.

People with FASD "are typically impulsive and have difficulty predicting the consequences of their actions." *See* ABA Resolution and Report Approved August 7, 2012, at 6 (citing Substance Abuse and Mental Health Services Administration: A Fetal Alcohol Spectrum Disorders Center for Excellence. *What You Need To Know: Fetal Alcohol Spectrum Disorders and Juvenile Justice: How Professionals Can Make a Difference*. DHHS Pub. No. (SMA)-06-4240 (Rockville, MD: 2007)). There is no cure for the brain damage associated with FASD, which occurs before birth. *See* ABA Resolution and Report Approved August 7, 2012, at 5 (citing Blair Paley and Mary J. O'Connor, *Neurocognitive and Neurobehavioral Impairments in Individuals with Fetal Alcohol Spectrum Disorders: Recognition and Assessment*, 6 Int'l J. Disabil. Hum. Dev. 127, 130 (2007)). Not only do the deficits associated with FASD "follow children into adulthood," *see id.*, they also become more severe throughout that individual's lifetime. Kambeitz et al., "Association of adverse childhood experiences and neurodevelopmental disorders in people with fetal alcohol spectrum disorders (FASD) and non-FASD controls" (2019), at 6 ("FASD symptomatology increases in severity over time"). FASD is a "lifelong disability." *See* ABA Resolution and Report Approved August 7, 2012, at 2.

b. FASD causes people to operate at the level of the intellectually disabled

People with FASD operate cognitively and functionally at the level of the intellectually disabled. Brown et al., *Prenatal alcohol exposure: An*

assessment strategy for the legal context, 42-43 Int'l J. of L. and Psych. 144, 144 (2015). Even though some people with FASD have IQ scores within the normal range, they nonetheless typically exhibit deficits in adaptive behavior and suffer from other neurocognitive impairments that make it difficult for them to function at an age-appropriate level. *See* ABA Resolution and Report Approved August 7, 2012, at 5 (citing Natalie Novick Brown, Anthony P. Wartnik, Paul D. Connor, and Richard S. Adler, *A Proposed Model Standard for Forensic Assessment of Fetal Alcohol Spectrum Disorders*, 38 J. of Psych. & L. 383, 387 (2010)); *see also* Stephen Greenspan et al., FASD and the Concept of "Intellectual Disability Equivalence" (2016) at 241 (recognizing that FASD is the equivalent of an intellectual disability because "people with FASD have adaptive deficits and support needs identical to those with [intellectual disabilities]"). In recognition of these deficits, some states have codified FASD as a developmental disability. Stephen Greenspan et al., FASD and the Concept of "Intellectual Disability Equivalence" (2016) at 250, 256; Minn. Stat. § 252.27; Alaska Stat. § 47.20.290.

c. FASD stunts people at the social and emotional level of children

Because people with FASD have a permanent, severe disability that cannot be "cured," they often remain stunted, unable to increase their level of functioning as they enter adulthood. *See* Larry Burd, *Fetal alcohol spectrum disorder: complexity from comorbidity*, The Lancet (Vol. 387, No. 10022 2016), at 1. This distinguishes FASD from more

familiar and better understood conditions, such as ADD/ADHD and PTSD, which can be readily treated and frequently overcome. By contrast, "[m]ost people with FASD require care over much of their lifetime." *Id.*; *see also* Brown et al., "Prenatal alcohol exposure: An assessment strategy for the legal context," at 145 ("as such deficits stem from permanent brain damage, competency restoration may be impossible"). People with FASD lag behind their peers in brain development and generally do not achieve self-sufficiency as an adult because of their organic brain damage. Streissguth et al., "Understanding the Occurrence of Secondary Disabilities in Clients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE)" (1996), at 4.

II. BECAUSE HE HAS PFAS, MR. ANDERSON HAS THE DIMINISHED CULPABILITY OF A CHILD OR THE INTELLECTUALLY DISABLED

It is undisputed that Mr. Anderson suffers generally from FASD and specifically PFAS, which is among the more severe manifestations of FASD. *See* 8th Cir. Exs., Ex. 42 at 1 ("Mr. Anderson'[s] condition can be characterized as being Partial Fetal Alcohol Syndrome (PFAS)"); 8th Cir. Exs., Ex. 55 at 23 (noting that Mr. Anderson's neurological profile is consistent with FASD); ABA Resolution and Report Approved August 7, 2012, at 3 (citing National Institute on Alcohol Abuse and Alcoholism, Alcohol Alert, Fetal Alcohol Spectrum Disorders: Understanding the Effects of Prenatal Alcohol Exposure, No. 82). Individuals with PFAS, like those with other forms of FASD, suffer from organic

brain injury and display all the physical and emotional deficits associated with FASD. ABA Resolution and Report Approved August 7, 2012, at 3.

Despite Mr. Anderson's IQ exceeding the range for an intellectual disability diagnosis,[3] the record demonstrates that Mr. Anderson's adaptive-functioning defects are equivalent to those of a person with intellectual disabilities. *See* 8th Cir. Exs., Ex. 55 at 23 ("Mr. Anderson's level of adaptive functioning . . . is equivalent to individuals diagnosed with Intellectual Disability"). Brain damage associated with PFAS, which Mr. Anderson's testing specifically confirmed, imposes a "biological ceiling" on his ability to regulate his own behavior. *See* Hoyme et al., "Updated Clinical Guidelines for Diagnosing Fetal Alcohol Spectrum Disorders" (2016) at 9; Brown et al., "A proposed model standard for forensic assessment of Fetal Alcohol Spectrum Disorders," at 390.

Further, the record demonstrates that Mr. Anderson had the mental functioning of a child at the time of the offenses for which he was convicted. Because Mr. Anderson's FASD caused his brain development to lag eight to ten years behind that of

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[3] At age fifteen, Mr. Anderson's full-scale IQ was found to be 65. 8th Cir. Exs., Ex. 55 at 24–25. When the State of Arkansas tested Mr. Anderson at age twenty, it found his IQ to be 91. *Id.* At age thirty-one, his IQ was tested by his defense team and found to be 85, and the same full-scale IQ score was reported by Dr. Novick Brown in Mr. Anderson's 2015 neurodevelopmental assessment. *Id.*

his peers, Anderson's brain was "more like a child's brain than a typical 19-year-old's brain" when he committed the offense. 8th Cir. Exs., Ex. 55 at 23. Mr. Anderson's mental functioning more closely resembled that of a nine- to eleven-year-old child.

This Court prohibits the execution of individuals with intellectual disabilities and children. *Atkins v. Virginia*, 536 U.S. 304, 320 (2002); *Roper v. Simmons*, 543 U.S. 551, 572–73 (2005). The Court has recognized that intellectually disabled individuals "do not act with the level of moral culpability that characterizes the most serious adult conduct" due to their deficiencies in reasoning, judgment, and impulse control. *Atkins*, 536 U.S. at 306. This Court has also recognized that the diminished culpability of children requires a different approach to sentencing. *See Roper*, 543 U.S. at 570 (prohibiting the execution of juvenile offenders because they act with less culpability due to their "susceptibility . . . to immature and irresponsible behavior").

To the extent courts do not adequately understand or properly consider FASD, it likewise poses a risk of wrongful execution. Because Mr. Anderson had the mental functioning of a child or an individual with an intellectual disability due to his FASD-induced brain damage, his diminished moral culpability should have been considered during sentencing.

III. FASD IS A UNIQUE MITIGATING FACTOR THAT IS DIFFERENT IN KIND FROM OTHER TYPES OF MITIGATING EVIDENCE

a. The Supreme Court and multiple federal courts recognize the unique mitigating value of FASD evidence

The Eighth Circuit's decision below directly conflicts with federal case law by failing to recognize the crucial power of FASD evidence. Indeed, this Court has expressly recognized evidence of brain damage as a uniquely compelling mitigating factor. *See Abdul-Kabir v. Quarterman*, 550 U.S. 233, 259–60 (2007) (finding neurological damage was relevant mitigating evidence because it showed how defendant's violent propensities were caused by factors beyond his control); *see also Rompilla v. Beard*, 545 U.S. 374, 392 (2005) (explaining how FASD-induced brain damage reduced defendant's "capacity to appreciate the criminality of his conduct or to conform his conduct to the law").

Decisions in the Fourth, Tenth, and Eleventh Circuits have followed the Court's lead by also recognizing the uniquely compelling nature of brain damage as mitigating evidence. *Williams v. Stirling* is illustrative. 914 F.3d 302, 318 (4th Cir. 2019), *cert denied*, 140 S. Ct. 105 (2019). In *Williams*, the petitioner suffered from the same condition as Mr. Anderson: PFAS. *Id.* at 308. Despite multiple "red flags," including evidence of maternal alcohol abuse and evidence of Williams' brain damage, Williams' trial counsel "failed to connect the indicators suggesting further investigation" of FASD and thus,

failed to raise FASD-related brain damage as mitigating evidence. *See id.* at 315. The Fourth Circuit concluded that counsel's failure to investigate FASD was unreasonable and the absence of evidence of brain damage was prejudicial. *Id.* at 318. The Court explained that FASD evidence has independent mitigating weight because the diagnosis "could have provided to the jury evidence of a neurological defect that *caused* Williams' criminal behavior" and that "[w]ithout this information, the jury . . . would have assigned greater moral culpability to [Williams] for his criminal behavior." *Id.* *See also Littlejohn v. Trammell*, 704 F.3d 817, 864 (10th Cir. 2013) ("Evidence of organic mental deficits ranks among the most powerful types of mitigation evidence available."); *Hooks v. Workman*, 689 F.3d 1148, 1205 (10th Cir. 2012) (finding that defendant's intellectual disability should have been presented as mitigating evidence); *Jefferson v. GDCP Warden*, 941 F.3d 452, 484 (11th Cir. 2019) (quoting *Middleton v. Dugger*, 849 F.2d 491, 495 (11th Cir. 1988) (evidence of FASD "has the potential to totally change the evidentiary picture by altering the causal relationship that can exist between mental illness and homicidal behavior").

The ABA Guidelines have similarly acknowledged the persuasive mitigating value of FASD evidence. As the ABA Guidelines emphasize, "it is critically important to construct a persuasive narrative in support of the case for life, rather than to simply present a catalog of seemingly mitigating factors." ABA Guidelines, *reprinted in* 31 Hofstra L. Rev. 913, 1061 (2003). Expert witnesses are

particularly useful for this purpose, as they "may assist the jury in understanding the significance of [a client's social history]." *Id.* For example, expert testimony explaining "the permanent neurological damage" caused by FASD "and the effects of these impairments on the client's judgment and impulse control" could "lessen the defendant's moral culpability for the offense or otherwise support[] a sentence less than death." *Id.* at 1060–61.

b. FASD is powerful mitigation evidence because it involves organic brain damage

The Eighth Circuit majority failed to recognize the unique power of FASD evidence. It found that Anderson's FASD diagnosis would not have altered the sentencing outcome here because the jury heard other mitigating factors related to abuse and neglect Mr. Anderson experienced as a child. *Anderson v. Kelley*, 938 F.3d 949, 958 (8th Cir. 2019). But Mr. Anderson's FASD "is more than 'one more' mitigation argument"; indeed, it is "more powerful than any of the mitigating evidence presented at [Anderson's] resentencing." *Id.* at 963–64 (Kobes, J., dissenting). FASD is fundamentally different from other mitigation evidence, such as an abusive childhood, because FASD causes organic, permanent brain damage that inherently renders a defendant less culpable. Thus, unlike the other mitigating evidence presented at trial, FASD is powerful, outcome-determinative mitigation evidence that would have offered the jury a biological explanation for Mr. Anderson's conduct. This would have significantly reduced the jury's appraisal of Mr. Anderson's moral culpability, which is central to

sentencing. *See Rompilla*, 545 U.S. at 393 (citing *Strickland v. Washington*, 466 U.S. 668, 694 (1984)) (unpresented mitigating evidence, including organic brain damage caused by fetal alcohol syndrome, is "sufficient to undermine confidence in the outcome" of jury's deliberations); *Castro v. Oklahoma*, 71 F.3d 1502, 1516 (10th Cir. 1995) ("more complete picture of [defendant's] mental health," including organic brain damage and a possible diagnosis of fetal alcohol syndrome, "likely would have changed the jury's intuitive calculus").

Further, "[i]t's not just the quantity, but the quality of mitigating evidence that can make the difference between life and death." *Anderson*, 938 F.3d at 965 (citing *Blanton v. Quarterman*, 543 F.3d 230, 236 (5th Cir. 2008)). Evidence of the abuse and instability Mr. Anderson suffered for his first nineteen years of life is relevant mitigating evidence, but evidence of his FASD diagnosis carries significantly more mitigating weight because it explains that Mr. Anderson's actions were attributable to his severe, permanent brain damage.

IV. TRIAL COUNSEL FAILED TO CONDUCT A REASONABLE INVESTIGATION OF FASD, WHICH WAS A WELL-KNOWN MEDICAL CONDITION AND FORM OF MITIGATION EVIDENCE

At the time of Mr. Anderson's 2002 trial, FASD was a well-defined medical condition, having been documented as early as 1973. *See* Kenneth Jones et al., "Pattern of Malformation in Offspring of Chronic Alcoholic Mothers," The Lancet (1973). Medical experts published the authoritative criteria for

FASD in 1996—six years before Mr. Anderson's trial. *See* Stratton K, Howe C, Battaglia F, eds. Institute of Medicine. Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment. Washington, DC: National Academies Press; 1996. In fact, FASD was being raised as a possible defense in Arkansas as early as 1995. *See Miller v. State*, 942 S.W.2d 825, 827–28 (Ark. 1997). And there can be no doubt that by the time of Mr. Anderson's resentencing in 2005, FASD as a form of compelling mitigation evidence was well known to the capital defense bar, as evidenced by the 2003 American Bar Association Guidelines for the Appointment and Performance of Defense Counsel in Death Penalty Cases ("ABA Guidelines")—a "guide[] to determining what is reasonable" in capital cases. *Strickland*, 466 U.S. at 688.

The ABA Guidelines explicitly call for an investigation of possible FASD and explain that mitigation cases depend on an "extensive and generally unparalleled investigation into personal and family history" that "begins with the moment of conception." ABA Guidelines, *reprinted in* 31 Hofstra L. Rev. 913, 1022 (2003). Accordingly, the ABA Guidelines instruct counsel to explore a defendant's medical history, including "pre-natal and birth trauma" and "neurological damage," as well as family and social history of substance abuse, among other possible mitigating factors. *Id.* To properly investigate such mitigation evidence, the prevailing standards explicitly require all capital defense teams to include at least one expert "qualified by training and experience" to screen for FASD. *Id.* at 952.

Retention of such an expert is essential because "[c]ounsel's own observations of the client's mental status, while necessary, can hardly be expected to be sufficient to detect the array of conditions . . . that could be of critical importance," including FASD. *Id.* at 956–57.

Here, trial counsel's failure to retain a qualified expert constituted ineffective assistance of counsel because Mr. Anderson's capital defense team did *not include anyone* qualified to test for FASD. *See Anderson*, 938 F.3d at 964 n.5; *see also* Burd and Edwards, "Fetal Alcohol Spectrum Disorders: Implications for Attorneys and the Courts," at 25 ("Counsel may also be rendering ineffective assistance of counsel for failing to hire, appoint, or retain qualified experts who are trained in diagnosing FASD and who can testify forensically about problems with executive functioning and impulse control problems."). Moreover, Mr. Anderson's trial counsel did not request neuropsychological testing, despite ample guidance at the time that "part of the diagnostic process is a comprehensive neuropsychological assessment." National Center on Birth Defects and Developmental Disabilities, "Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis" (2004) at 24; 38 J. Psych. and L. (2010), "A proposed model standard for forensic assessment of Fetal Alcohol Spectrum Disorders," at 389. At the time of Mr. Anderson's resentencing in 2005, testing for FASD was a "pertinent avenue[] for investigation of which [counsel] should have been aware." *Porter v. McCollum*, 558 U.S. 30, 40 (2009).

Counsel's failure to investigate FASD as a mitigating factor is especially unjustifiable given the numerous red flags indicating that Mr. Anderson's mother consumed alcohol during pregnancy. As Judge Kobes bluntly observed, "Anderson's childhood was soaked in alcohol." *Anderson*, 938 F.3d at 963 (Kobes, J., dissenting). Trial counsel knew that Mr. Anderson's mother had a drinking problem that was at least closely contemporaneous with Mr. Anderson's gestation, which should have been sufficient to warrant further investigation of FASD as a cause of Mr. Anderson's behavioral factors. Although "no one specifically told counsel that [Mr. Anderson's mother's] drinking continued during pregnancy," counsel knew that she was an alcoholic, and multiple witness descriptions of her drinking "were so tied to Anderson's infancy that counsel should have investigated further." *Id.* at 963 (Kobes, J., dissenting) (describing statements from Mr. Anderson's brother, who "said his mother drank heavily when she was with Amos Strickland, which was during the earliest years of Anderson's life"). Studies on FASD in the legal context have advised that such evidence of alcohol consumption during pregnancy requires counsel to thoroughly investigate maternal alcohol history, including "by going back three generations." Burd and Edwards, "Fetal Alcohol Spectrum Disorders: Implications for Attorneys and the Courts," at 25 ("If trial counsel has discovered any evidence that the birth mother may have consumed any alcohol during pregnancy, counsel must investigate the maternal alcohol history by going back three generations"); *see also* Brown et al., "Prenatal alcohol exposure: An

assessment strategy for the legal context," at 144–48). Despite glaring evidence of prenatal alcohol exposure, no one on the defense team asked Mr. Anderson's parents if Mr. Anderson's birth mother consumed alcohol while pregnant with him and no one on the team knew how to screen for FASD. As further detailed in Mr. Anderson's petition for certiorari, failure to investigate this powerful mitigating factor was contrary to prevailing standards and constituted ineffective assistance of counsel. *See id.* (explaining that failure to investigate FASD as a mitigating factor despite evidence of maternal drinking "could result in ineffective assistance of counsel for not exploring how FASD may relate to the client's moral culpability").

CONCLUSION

For the foregoing reasons, NOFAS respectfully urges the Court to issue a writ of certiorari to review the Eighth Circuit's judgment.

Respectfully submitted,

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