No. 25-732

IN THE
# Supreme Court of the United States

AMOS WELLS,
*Petitioner*,
v.

ERIC GUERRERO, DIRECTOR,
TEXAS DEPARTMENT OF CRIMINAL JUSTICE,
CORRECTIONAL INSTITUTIONS DIVISION,
*Respondent*.

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

## BRIEF OF PROFESSORS NITA A. FARAHANY, STEVEN E. HYMAN, JOSHUA A. GORDON, GENE E. ROBINSON, AND 25 OTHER SCIENTISTS AND SCHOLARS AS *AMICI CURIAE* IN SUPPORT OF PETITIONER

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## TABLE OF CONTENTS

## TABLE OF AUTHORITIES

## INTEREST OF *AMICI CURIAE*[1]

*Amici* are scholars representing a variety of disciplines, including genetics, psychiatry, and law. *Amici* have an interest in ensuring that reliable scientific evidence is introduced in the courtroom—particularly in cases implicating the death penalty where widely discredited science is used as aggravating evidence that undermines the defendant's mitigating evidence. A list of *amici* is included in an Appendix to this brief.

## INTRODUCTION AND SUMMARY OF ARGUMENT

Petitioner Amos J. Wells III, a Black man, was sentenced to death after his defense counsel introduced expert testimony by a psychiatrist, Dr. William Bernet, who told the jury that Mr. Wells was genetically predisposed to violence. Specifically, Dr. Bernet testified that Mr. Wells was "four and a half times more likely" than the "typical person" to be violent in the future, based on his variant of the *monoamine oxidase A* ("*MAOA*") gene—the so-called "warrior gene"[2] —and childhood maltreatment. Pet. 6, 24 (citing ROA.13033, ROA.13073, ROA.13076-13078).[3]

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[1] Pursuant to Supreme Court Rule 37.6, counsel for *amici* represent that they authored this brief in its entirety and that none of the parties or their counsel, nor any other person or entity other than *amici* or their counsel, made a monetary contribution intended to fund the preparation or submission of this brief. Pursuant to Rule 37.2(a), counsel for *amici* also represent that all parties were provided notice of *amici*'s intention to file this brief at least 10 days before it was due.

[2] Nicholas Scurich & Paul S. Appelbaum, *State v. Yepez: Admissibility and Relevance of Behavioral Genetic Evidence in a Criminal Trial*, 72 Psychiatric Servs. 853, 853 (July 2021).

[3] "ROA" refers to the Record on Appeal filed in the court of appeals.

Dr. Bernet based this opinion on a 2002 study by psychologist Avshalom Caspi and his colleagues (the "Caspi Study")[4] and follow-on studies concerning the purported connection between *MAOA* gene variants, childhood maltreatment, and violent behavior. *See* ROA.13056-13061.

As explained by petitioner, by the time Mr. Wells's counsel presented this prejudicial evidence, the Caspi Study and similar *MAOA* studies had been "strongly and publicly" criticized by the scientific community. Pet. 24 (quoting ROA.15144). Because Mr. Wells's *MAOA* gene is an immutable characteristic, the scientifically dubious evidence regarding Mr. Wells's genetic makeup constituted "constitutionally impermissible" evidence of future dangerousness. *Zant v. Stephens*, 462 U.S. 862, 885 (1983). In addition, the wide and public criticism of that evidence helps demonstrate that defense counsel acted unreasonably by introducing it, where the evidence only helped the prosecution "establish[] a prerequisite for the death penalty while providing no meaningful benefit in mitigation." Pet. 14. Put simply, "[n]o competent defense attorney would introduce such evidence about his own client." *Buck v. Davis*, 580 U.S. 100, 119 (2017).

*Amici* offer additional context regarding the scientific consensus discrediting the Caspi Study and related *MAOA* studies on which Dr. Bernet relied. *First*, these studies are methodologically flawed, and their results have not been consistently reproduced. *Second,* in the context of human behavioral and psychiatric genetic studies, the scientific community has largely discredited the "candidate-gene approach" used in the Caspi Study and related *MAOA* studies.

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[4] Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 Sci. 851 (2002).

Indeed, by 2016, leading scientific journals and organizations had sharply criticized and recommended abandoning the human candidate-gene framework. *Third*, recent genetic studies adopting the "genome-wide association" approach have also failed to replicate the earlier findings of *MAOA* candidate-gene studies.

In sum, recent literature has only confirmed what was true when defense counsel presented Dr. Bernet's testimony in Mr. Wells's case: the Caspi Study and related *MAOA* studies purporting to link genetic makeup and future violence have been consistently discredited. These studies therefore do not provide reliable information in support of the conclusion that Mr. Wells is genetically predisposed to future dangerousness, and they offered no benefit to Mr. Wells.

## ARGUMENT

### I. Defense Counsel Relied On Genetic Studies That Have Been Widely Discredited For Decades And That Only Helped The Prosecution

By the time Dr. Bernet testified in Mr. Wells's case, the scientific record was clear that the findings of the Caspi Study could not be reproduced consistently and that the methodology it employed was questionable. In other words, defense counsel introduced expert testimony that rested on widely discredited genetic studies. That scientifically dubious testimony deprived Mr. Wells of effective assistance of counsel, because it only served to prove Mr. Wells's supposed genetic predisposition to future violence while severely undermining his mitigation evidence.

### A. The Caspi Study Has Not Been Consistently Replicated

The Caspi Study employed the "candidate gene" or "candidate gene environment" approach—which tests for associations between a pre-specified genetic variant, environmental factors, and an observable trait or behavior.[5] Based on that approach, Caspi and his colleagues concluded that there is a connection between the *MAOA* gene, childhood maltreatment, and violent behavior: those with a genetic variant called *MAOA-L* (the low-activity form of the *MAOA* gene) were more likely to exhibit violent behavior if they had been maltreated as children, compared to those with a genetic variant called *MAOA-H* (the high-activity form of the *MAOA* gene) who had also experienced child maltreatment.[6]

The scientific community initially lauded this discovery as a breakthrough in the field of psychiatric genetics. Indeed, a few early follow-on studies successfully replicated the Caspi Study results. But many others did not, leading to growing concern about the reliability and validity of those results. For example, a 2005 study attempting to replicate the Caspi Study's findings was "unable to confirm the hypothesis that differences in the MAOA promoter region plays a moderating role in the relationship between maltreatment as a child and conduct problems in adolescence and young adulthood."[7] Similarly, in 2006,

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[5] *See* Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 Sci. at 852.

[6] *See id.* at 853.

[7] Brett C. Haberstick et al., *Monoamine Oxidase A (MAOA) and Antisocial Behaviors in the Presence of Childhood and Adolescent Maltreatment*, 135 Am. J. Med. Genetics Part B 59, 62-63 (2005).

scholars found "no genetic-environmental interaction with genotype for maltreatment."[8] A 2008 study with a sample size of 1,002 men also failed to replicate the Caspi Study findings, leading the authors to raise "doubts about the robustness of this finding."[9] In 2014, another study reported that, in its sample of 4,316 men, "there were no main effects of *MAOA* genotype" on antisocial behavior, and "*MAOA* genotype was not a significant moderator of the relationship between maltreatment and antisocial behaviors."[10] As the authors of that study described, "[a]lthough there have been numerous attempts at replicating [the Caspi Study's] observation, results remain inconclusive."[11]

In a nutshell, despite early enthusiasm about its novel findings, the Caspi Study has not been consistently replicated. By the time Mr. Wells's counsel introduced the Caspi Study—nearly a decade and a half after its publication—it was clear that the replication rate of the Caspi Study and similar follow-on studies was unreliable.

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[8] Susan E. Young et al., *Interaction Between MAO-A Genotype and Maltreatment in the Risk for Conduct Disorder: Failure to Confirm in Adolescent Patients*, 163 Am. J. Psychiatry 1019, 1019 (2006).

[9] Zoë Prichard et al., *No Evidence for Interaction Between MAOA and Childhood Adversity for Antisocial Behavior*, 147 Am. J. Med. Genetics Part B 228, 230, 232 (2008).

[10] Brett C. Haberstick et al., *MAOA Genotype, Childhood Maltreatment, and Their Interaction in the Etiology of Adult Antisocial Behaviors*, 75 Biol. Psychiatry 25, 25 (2014).

[11] *Id.*

### B. The Candidate-Gene Approach Has Been Discredited for Human Behavioral and Psychiatric Genetic Studies

More broadly, the candidate-gene approach used by the Caspi Study and related *MAOA* studies has been discredited in the context of human behavioral and psychiatric genetic studies. In the early 2010s, retrospective analysis of human candidate-gene studies revealed that, while 96% of the initial novel findings were significant, they were replicated only 27% of the time.[12]

In the wake of this replication crisis, modern research techniques have revealed significant methodological problems with candidate-gene studies. For example, human candidate-gene studies typically have used sample sizes that are too small to accurately measure the relationship between a given genetic variant and a complex trait.[13] In general, studies based on small samples may lead to overestimates of effect size and low reproducibility of results.[14] Candidate-gene studies assessing the connection between *MAOA* and violent behavior have typically used small sample sizes—for example, the Caspi Study

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[12] *See* Laramie E. Duncan & Matthew C. Keller, *A Critical Review of the First 10 Years of Candidate Gene-by-Environment Interaction Research in Psychiatry*, 168 Am. J. Psychiatry 1041, 1043 (2011); *see also* Peter T. Tanksley et al., *The Genome-Wide Study of Human Social Behavior and Its Application in Sociology*, 4 Frontiers in Soc. 1, 2 (2019).

[13] *See* Danielle M. Dick et al., *Candidate Gene-Environment Interaction Research: Reflections and Recommendations*, 10 Persp. on Psychol. Sci. 37, 41 (2015); Joel Gelernter, *Genetics of Complex Traits in Psychiatry*, 77 Biol. Psychiatry 36, 37 (2015).

[14] *See* Katherine S. Button et al., *Power Failure: Why Small Sample Size Undermines the Reliability of Neuroscience*, 14 Nature Rev. Neuroscience 365, 365 (2013).

had a sample size of only 1,037 children.[15] (By comparison, a recent study on schizophrenia had a sample size of more than 30,000.[16]) As a result, scholars have expressed concern that "the proportion of 'discoveries' in candidate gene . . . studies that are actually false[] may be unacceptably high."[17]

As a result of their methodological flaws and replication failures, leading scientific journals and organizations have criticized the human candidate-gene approach or recommended abandoning it altogether. In 2012, *Behavioral Genetics*, the leading journal concerned with the genetic analysis of complex traits, published an editorial critical of the human candidate-gene method.[18] That editorial noted:

> The literature on candidate gene associations is full of reports that have not stood up to rigorous replication. This is the case both for straightforward main effects and for candidate gene-by-environment interactions. As a result, the psychiatric and behavioral genetics literature has become confusing and it now seems likely that many of the published findings of the last decade are wrong or misleading and have not contributed to real advances in knowledge.[19]

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[15] *See* Caspi et al., *Role of Genotype in the Cycle of Violence*, 297 Sci. at 852.

[16] *See* Dick et al., *Candidate Gene-Environment Interaction Research*, 10 Persp. on Psychol. Sci. at 41.

[17] *Id.*

[18] *See* John K. Hewitt, *Editorial Policy on Candidate Gene Association and Candidate Gene-by-Environment Interaction Studies of Complex Traits*, 42 Behav. Genetics 1 (2012).

[19] *Id.* at 1 (citation omitted).

The editorial went on to recommend that the journal publish novel candidate-gene studies only if they have been directly replicated or meet certain methodological criteria.[20] *Psychological Science*, the journal of the Association for Psychological Science, similarly adopted these requirements.[21] The Caspi Study did not meet these standards.[22]

Going further, a 2016 working group of the National Institute of Mental Health concluded that "[c]andidate gene studies of psychopathologic, cognitive, or behavioral phenotypes should be abandoned."[23] The group's report explained that "[c]andidate gene studies attempting to find associations" between genetic

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[20] *See id.* (recommending publication of candidate-gene studies testing novel hypotheses only if they include a "direct replication study reported in the same paper" or if "the finding meets the statistical criteria for genome wide significance taking into account all sources of multiple testing" and are based on a sample population large enough to accurately measure results).

[21] *See* Ass'n for Psych. Sci., *Submission Guidelines* (July 13, 2016) ("The editors of Behavior Genetics have established perceptive policies regarding candidate gene association and Candidate Gene × Environment interaction studies of complex traits. Submissions to *Psychological Science* that report similar candidate-gene studies are expected to accord with these same policies.") (citation omitted), https://web.archive.org/web/20161119182952/https://www.psychologicalscience.org/publications/psychological_science/ps-submissions.

[22] The Caspi Study paper did not report a direct replication study. *See* Caspi et al., *Role of Genotype in the Cycle of Violence*, 297 Sci. at 853. Nor did it use a sample population large enough for accurate measurement. *See* Dick et al., *Candidate Gene-Environment Interaction Research*, 10 Persp. on Psychol. Sci. at 41.

[23] Nat'l Advisory Mental Health Council, Nat'l Inst. of Mental Health, *Report of the National Advisory Mental Health Council Workgroup on Genomics: Opportunities and Challenges of Psychiatric Genetics*, https://perma.cc/53Y4-52P8.

variants and physical or other traits "have historically been vastly underpowered" because of "serious misunderstandings of the influence of sample size on the robustness and significance of results."[24] The report concluded that human candidate-gene studies have resulted in "the propagation of false, if superficially plausible explanations of psychopathology."[25]

As a recent scholar put it, a "lack of consistency and trust in the results arising from these [candidate-gene] studies led to replication studies and meta-analyses that altogether discredited most candidate gene associations in psychiatry," as well as the conclusion that the candidate-gene approach "hindered the identification of the true biological risk mechanisms underlying psychiatric disorders."[26] As a result, "[p]sychiatric genetics has largely moved away from historical candidate association studies."[27]

## II. Recent Genome-Wide Association Studies Also Have Not Replicated The Caspi Study's Findings

More recent genome-wide association studies have not found a correlation between *MAOA*, childhood maltreatment, and violent or maladaptive behavior. Genome-wide association studies test for correlations between traits and genetic variants across the entire DNA sequence, or genome.[28] Using this approach,

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[24] *Id.*

[25] *Id.*

[26] Rodrigo R.R. Duarte et al., *Ditching candidate gene association studies: lessons from psychiatric genetics*, 42 Braz. J. Psychiatry 342, 342 (2021).

[27] *Id.* at 343.

[28] *See* Dick et al., *Candidate Gene-Environment Interaction Research*, 10 Persp. on Psychol. Sci. at 41.

researchers compare DNA samples from two groups—those with the particular trait of interest, and similar individuals without it—and analyze whether individuals with the relevant trait are more likely to have certain genetic variants.[29] Genome-wide association studies use sample sizes encompassing tens of thousands of participants or more.

Genome-wide association studies to date have not replicated earlier findings connecting *MAOA*, childhood maltreatment, and violence. For example, a 2018 study investigating influence of common genetic variants and childhood maltreatment on childhood antisocial behavior "did not observe any interaction with maltreatment" "in males with a low-activity [*MAOA*]."[30] Therefore, these studies do not support the Caspi Study's discredited findings, either.

## CONCLUSION

Widely and publicly discredited science purporting to link genetic characteristics to propensity for violence has no place in a capital proceeding. There is no scientifically reliable connection between low-activity *MAOA* and future dangerousness. Dr. Bernet's testimony regarding Mr. Wells's genetic makeup was therefore constitutionally impermissible evidence that supported only the prosecution's case. By introducing that evidence, Mr. Wells's counsel provided ineffective assistance. The Court should grant certiorari.

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[29] *Id.*

[30] I. Hyun Ruisch et al., *Interplay Between Genome-Wide Implicated Genetic Variants and Environmental Factors Related to Childhood Antisocial Behavior in the UK ALSPAC Cohort*, 269 Eur. Archives of Psychiatry & Clinical Neuroscience 741, 749 (2018).

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