

**CAPITAL CASE  
IN THE TENNESSEE SUPREME COURT  
AT NASHVILLE**

STATE OF TENNESSEE, )  
)  
Movant, )  
)  
v. )  
)  
BYRON LEWIS BLACK, )  
)  
Respondent. )

**EXECUTION DATE:**  
October 8, 2020  
  
Case No. M2000-00641-SC-DPE-CD

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**Motion to Reset Execution Date  
Due To COVID-19 Pandemic**

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Byron Black, by counsel, moves this Court to reset his execution date scheduled for October 8, 2020. Because of the COVID-19 pandemic, Mr. Black’s counsel cannot properly prepare for his competency to be executed hearing required to take place during the middle of August. Nor can counsel prepare for clemency proceedings which must take place simultaneously. This motion is made under the Sixth, Eighth, and Fourteenth Amendments to the United States Constitution; Article 1, §§ 8, 16, and 32 of the Tennessee Constitution; this Court’s inherent authority under Tenn. Code Ann. § 16-3-503; and Tennessee Supreme Court Rules 12.4(A), 12.4 (E). In support of his motion, Mr. Black states the following.

## Introduction

1. The COVID-19 pandemic poses unprecedented challenges to the legal community. This Court appropriately continued the judicial state of emergency through May 31, 2020, and ordered the suspension of jury trials until July 3, 2020, subject to further order of the Court. *In Re: COVID-19 Pandemic*, No. ADM2020-00428, Order Modifying Suspension of In-Person Court Proceedings and Further Extension of Deadlines (Tenn. April 24, 2020). The continuing suspension of in-person judicial proceedings for all but the most emergent cases makes sense.

2. As of Tuesday, August 28, 2020, the Tennessee Department of Health reported 10,005 positive cases state-wide with 188 deaths. <https://www.tn.gov/health/cedep/ncov.html> (last visited April 28, 2020).

3. Sunday, April 26, was the largest jump in positive cases in our state to date. *Id.* Nashville reported 2,588 COVID-19 cases on Tuesday. <https://www.asafenashville.org/> (last visited April 29, 2020). Twenty-four Nashvillians have died. *Id.* As new cases of the virus continue to rise, Nashville cannot determine the date that phased reopening can safely begin. Samantha Max, *Nashville Coronavirus Cases Continue to Mount, Slowing Plans to Reopen*, WPLN (April 28, 2020), <https://www.tn.gov/correction/frequently-asked-questions-regarding-covid-19.html> (last visited April 29, 2020).

4. COVID-19 is inside Tennessee prisons. Bledsoe County has one of the worst outbreaks in the nation. Steven Hale, *The Bledsoe County Prison COVID-19 Outbreak is One of the Worst in the Country*, Nashville Scene (April 27, 2020),

<https://www.nashvillescene.com/news/pith-in-the-wind/article/21130409/the-bledsoe-county-prison-covid19-outbreak-is-one-of-the-worst-in-the-country> (last visited April 29, 2020). The Tennessee Department of Correction reports that as of April 28, 2020, only 3,808 inmates have been tested. TDOC Inmates COVID-19 Testing, Attachment A. 756 prisoners have tested positive. *Id.* Only two inmates have been tested at Riverbend Maximum Security Institution which houses death row inmates. On April 27, 2020, the department revealed that one of those inmates is positive for COVID-19.

5. New cases of the virus are rising in prisons at a startling rate. The Marshall Project reports “[t]he number of new cases among prisoners is more than doubling each week, showing that the curve used to measure when the virus is under control is still soaring in prisons[.]” Kate Park, Tom Meagher, Weihua Li, *Tracking the Spread of Coronavirus in Prisons*, (April 24, 2020, 3:05 PM), <https://www.themarshallproject.org/2020/04/24/tracking-the-spread-of-coronavirus-in-prisons> (last visited April 29, 2020).

6. As the State begins to gradually and cautiously reopen, it is clear that social distancing, the wearing of face coverings and gloves, and admonishments for those who can to stay at home will continue throughout the summer. White House Coronavirus Task Force Coordinator Deborah Birx stated on Sunday “social distancing will be with us through the summer to really ensure that we protect one another as we move through those phases.” Felicia Sonmez, Paige Winfield Cunningham and Meryl Kornfield, *Social Distancing Could Last for Months, White House coronavirus coordinator says*, *The Washington Post*

(April 26, 2020, 6:08 PM),  
[https://www.washingtonpost.com/politics/social-distancing-could-last-months-white-house-coronavirus-coordinator-says/2020/04/26/ad8d2f84-87de-11ea-8ac1-bfb250876b7a\\_story.html](https://www.washingtonpost.com/politics/social-distancing-could-last-months-white-house-coronavirus-coordinator-says/2020/04/26/ad8d2f84-87de-11ea-8ac1-bfb250876b7a_story.html) (last visited April 27, 2020).

7. Experts agree that the country can expect a second wave of the pandemic to coincide with the fall flu season. Dr. Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases and a member of the White House’s Coronavirus Task Force stated, “We will have coronavirus in the fall ... I am convinced of that because of the degree of transmissibility that it has, the global nature. What happens with that will depend on how we’re able to contain it when it occurs.” Savannah Behrmann, *“Convinced”: Fauci Says There Will Be Coronavirus in the Fall After Trump Says “It May not Come Back,”* USA Today (April 23, 2020, 9:56 AM) (last visited April 27, 2020).

8. While Tennessee is beginning to reopen the economy, Tourist Department Commissioner Mark Ezell stated, “we are not returning to business as usual.” Joel Ebert, *Tennessee Pledge: Gov. Lee Rolls Out Reopening Guidelines for Restaurants, Retail Stores*, The Tennessean (April 24, 2020, 2:31 PM),  
<https://www.tennessean.com/story/news/politics/2020/04/24/tennessee-governor-reveals-reopening-guidelines-restaurants-stores/3019032001/> (last visited April 27, 2020).

9. None of us knows what the future holds, but we all have an obligation to minimize the risk of spreading the virus. As Chief Justice Bivens said in a statement posted to the Court’s website:

[T]he Court strongly encourages all courts to continue to operate through remote proceedings such as video or audio conferencing whenever possible, even if the matter can be handled in-person in compliance with the judicial district’s approved plan. We have a long way to go in defeating this virus, and the more social distancing that can be done, the better it will be for everyone.

<https://tncourts.gov/press/2020/04/24/supreme-court-extends-judicial-emergency-may-31-allows-judicial-districts-submit> (last visited April 27, 2020).

10. Governor Bill Lee asks Tennesseans to continue to practice social-distancing, wear face coverings when in public, and for businesses to allow those who can to work from home. “Social distancing works, and as we open up our economy it will be more important than ever that we keep social distancing as lives and livelihoods depend on it.”

<https://www.fox13memphis.com/news/local/safer-home-order-tennessee-will-expire-april-30-governor-says/5DUO6HYX5BC3ZKL6TKX476OWRI/> (last visited April 27, 2020).

Noting that the “threat from COVID-19 is far from over,” in Executive Order #30 Governor Lee emphasizes that “[s]taying at home when possible and limiting unnecessary activity [is] still critically important to maintaining a healthy and economically vibrant Tennessee.” Exec. Order 30, *An Order Allowing Tennesseans to Return to Work Safely While Encouraging Continued Adherence to Health Guidelines to Limit the Spread of COVID-19*, at 2-3 (April 28, 2020). Governor Lee’s Order explains “avoiding unnecessary public interactions is critical to

protecting the health and safety of Tennesseans[.]” *Id.*, at 3. To that end, Governor Lee “strongly encourage[s] [Tennesseans] to continue to stay at home and to minimize in-person contact with people not in the same household, except when engaging in critical activities[.]” *Id.* The Governor’s order “strongly urge[s]” employers to the greatest extent practicable to equip, encourage, allow, or require employees to work remotely or via telework from home.” *Id.*, at 2.

11. Consistent with the guidance of the CDC and orders from State and local governments, the Federal Public Defender’s Office in Nashville has directed its staff to work remotely and to limit in-person contact to emergency situations. Only a skeleton crew remains on site. Working remotely presents several challenges for representation of clients under a sentence of death whose case files are voluminous. The office is not designed to operate with remote staff, and technological obstacles are inevitable and on-going.

12. Daily life is severely disrupted. These disruptions negatively affect Mr. Black’s counsels’ ability to prepare for his competency to be executed hearing or his case for clemency.

### **A Competency Hearing Is Impossible At This Time**

13. The United States and Tennessee Constitutions prohibit the execution of incompetent prisoners. *Madison v. Alabama*, 139 S.Ct. 718 (2019); *Van Tran v. State*, 6 S.W.3d 257 (Tenn. 1999). This Court recognizes that Mr. Black has a constitutional right to an adjudication of his colorable claim.

Mr. Black has ... raised the issue of his present competency to be executed and requests a competency hearing under *Van*

*Tran v. State*, 6 S.W.3d 257 (Tenn. 1999). In accordance with the procedures adopted by this Court in *Van Tran v. State* and the standard set forth in *State v. Irick*, 320 S.W.3d 284 (Tenn. 2010); see also *Madison v. Alabama*, 139 S.Ct. 718 (2019), the issue is remanded to the Criminal Court for Davidson County, where Mr. Black was originally tried and sentenced, for a determination of his present competency, including the initial determination of whether he has met the required threshold showing. To ensure the determination of Mr. Black's competency to be executed occurs in close proximity to his scheduled execution date, the filing requirements established in *Van Tran* are hereby modified as follows. Mr. Black shall file his petition alleging incompetency to be executed in the trial court no sooner than July 24, 2020 and no later than July 28, 2020. In all other respects, the proceedings shall be conducted in accord with the procedures and timelines set forth in *Van Tran*.

Order, *State v. Black*, No. M2000-00641-SC-DPE-CD (Tenn. Feb. 24, 2020).

14. The timeframe for the adjudication of Mr. Black's competency to be executed claim is driven by the *Van Tran* requirements which tether the competency adjudication to the execution date. Based upon this Court's setting of the October 8 execution date, this Court ordered that Mr. Black file his petition between July 24 and July 28, 2020. Further, the order requires that the petition be supported by contemporary data.

The likely hearing on the petition is mandated to occur in August. The hearing will require the testimony of expert witnesses.

15. Mr. Black's counsel cannot responsibly or safely comply with this timeline given the nationwide outbreak of COVID-19 and its presence within Riverbend Maximum Security Institution.

16. *First*, Mr. Black's identified expert witnesses reside out of state, including the states of California, Pennsylvania, and Georgia. Interstate travel is highly discouraged by the CDC. Three of Mr. Black's experts are in the high-risk group of individuals advised to stay at home even as the states begin to open. Mr. Black's experts cannot travel to Nashville and conduct a contemporary evaluation in July.

17. *Second*, a key component of Mr. Black's claim is the extent of his brain damage. "The State does not contest that Black currently has brain damage. But the source of his condition is highly disputed." *Black v. Bell*, 664 F.3d 81, 88 (6<sup>th</sup> Cir. 2011).<sup>1</sup> Mr. Black requires neuroimaging studies to demonstrate the nature and progression of his brain damage. Mr. Black's counsel has successfully arranged for similar studies, including in Mr. Black's case.

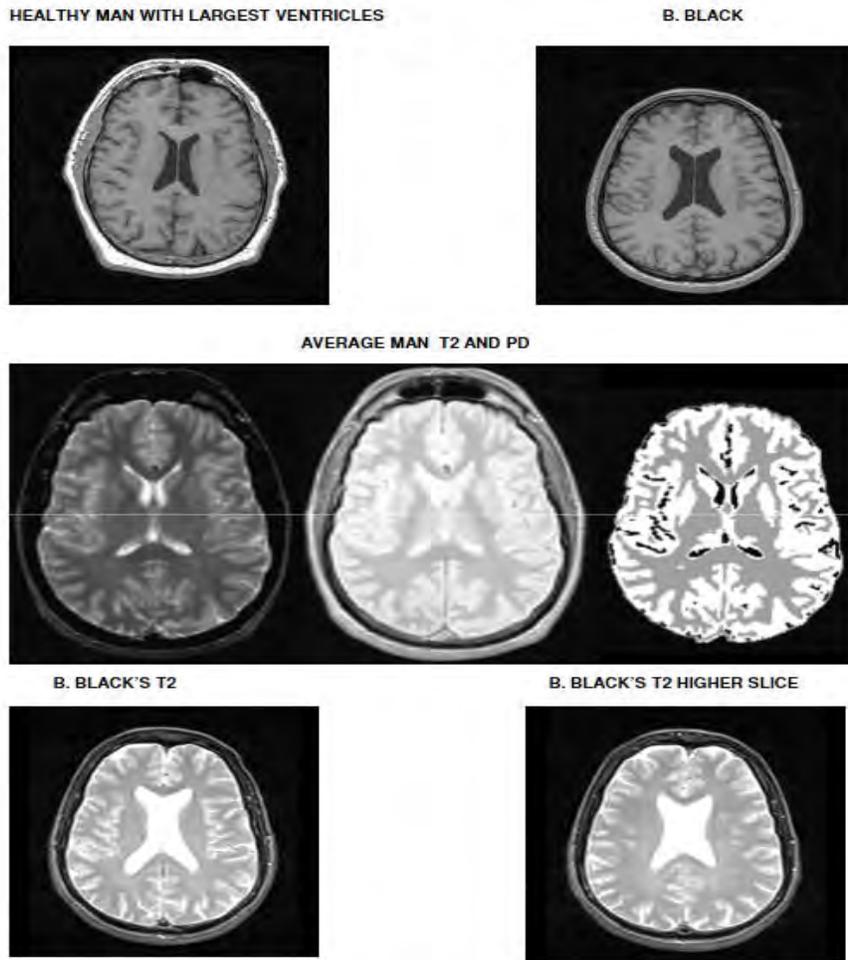
18. Neuroimaging studies in 2001 revealed that Mr. Black has significant brain damage. The studies showed several areas of damage including "ventricular atrophy." Deposition of Dr. Ruben Gur, at 51,

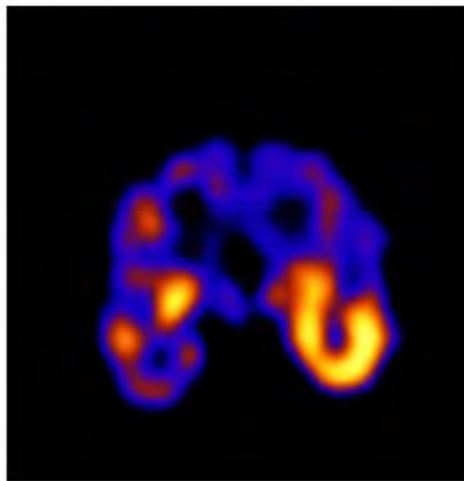
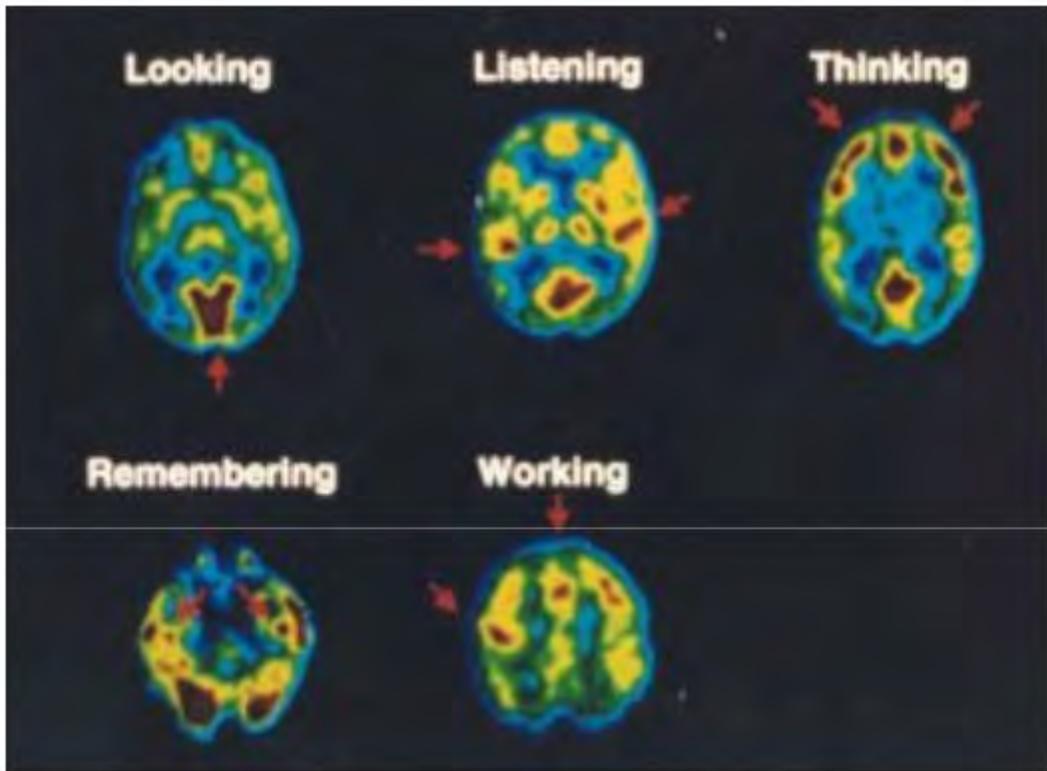
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<sup>1</sup> State expert Dr. Susan Vaught "admitted that [Mr. Black] has a relatively impaired brain." *Black v. State*, No. M2004-01345-CCA-R3PD, 2005 WL 2662577, at \*10 (Tenn. Crim. App. 2005). Dr. Vaught postulated that Mr. Black suffered from some sort of degenerative brain disease.

Attachment B. Dr. Gur explained “Large Ventricles are a cardinal sign of schizophrenia, but appear in mental retardation and various forms of cerebral dystrophy or atrophy related disorders.” *Id.* Dr. Gur “interpreted [Mr. Black’s] MRI as being severely abnormal[.]” *Id.*, at 60.

19. Dr. Gur prepared several visual aids which summarize his findings and document Mr. Black’s organic brain damage:





**Epilepsy**

Figure 1 PET regional ratio (R/WB) results for MEAN  $\pm$ SD of cerebral metabolic rates for glucose (CMRgl) in healthy males and Mr. Byron Black (BB).

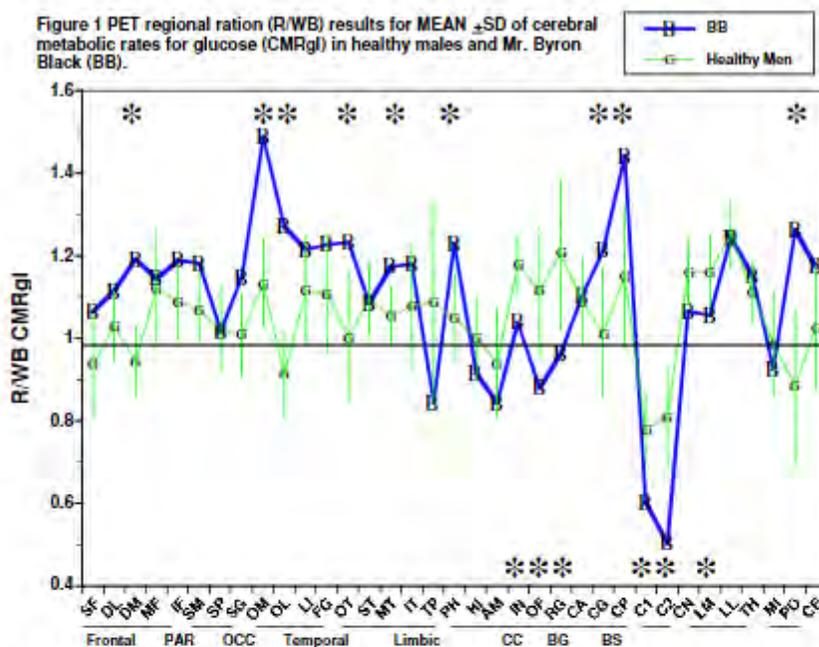
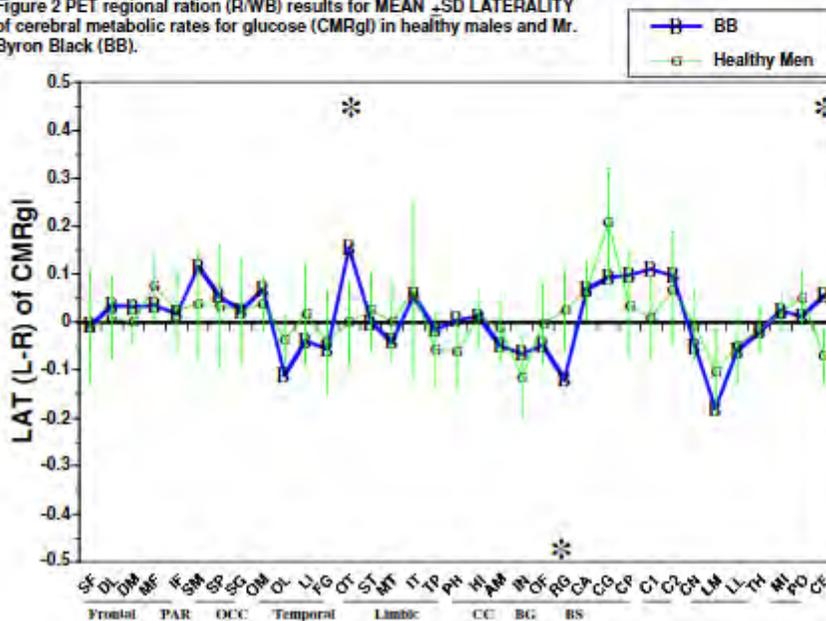
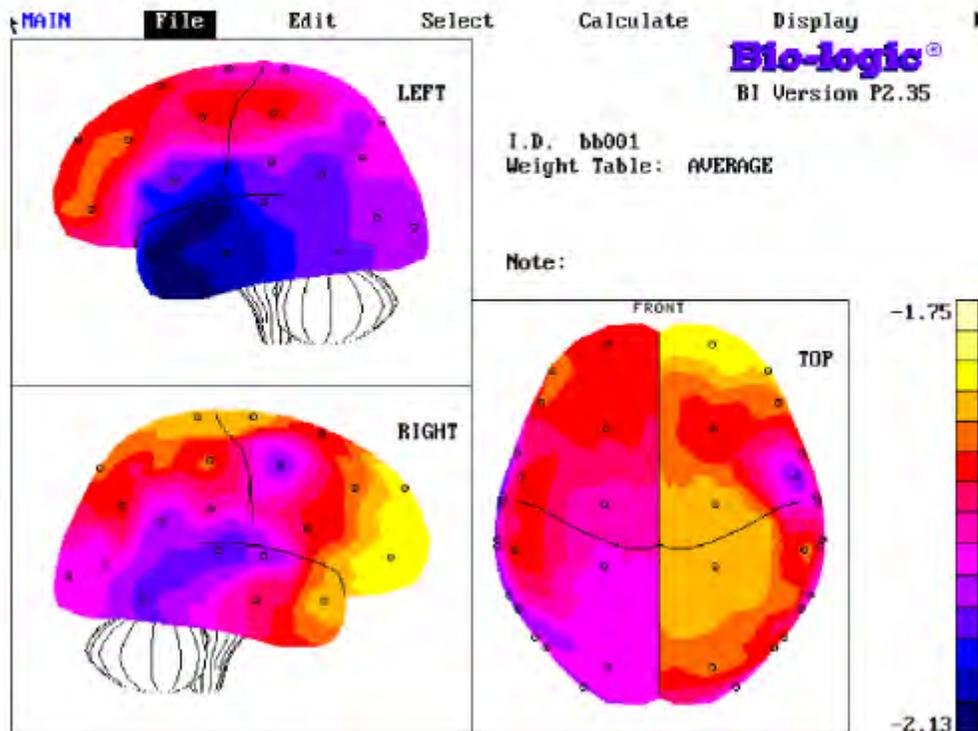


Figure 2 PET regional ratio (R/WB) results for MEAN  $\pm$ SD LATERALITY of cerebral metabolic rates for glucose (CMRgl) in healthy males and Mr. Byron Black (BB).



## Figure NP-1. Behavioral image of B. Black

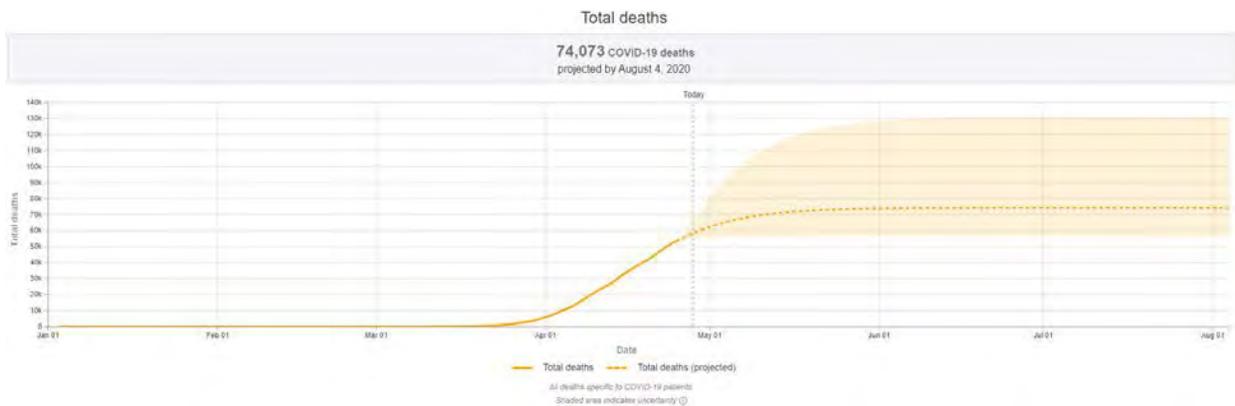


20. To obtain current neuroimaging studies counsel will need to arrange for the testing with a qualified imaging facility. This testing cannot be performed at this time because hospitals are reserving the use of their imaging equipment for COVID-19 and other emergency patients. Further, it is unlikely that any health care facility would permit Mr. Black to undergo imaging given the outbreak of COVID-19 in the prison system.

21. The transportation of Mr. Black for neuroimaging studies poses a significant risk of transmission of the disease. Mr. Black would

be transported to the facility by a team of corrections officers. Mr. Black and the officers would be at risk to contract the disease at the hospital. This further risks the spread of the disease within the prison. The dangers are not just to the prisoners but also to the officers and their friends and family.

22. *Third*, Mr. Black’s witnesses cannot travel to Nashville and testify in person for the hearing in August given their own health conditions and the predicted spread of the disease through the summer months. On April 27, the IMHE at the University of Washington adjusted their predictions to reflect the impact of increased mobility within the country. The number of predicted deaths rose from 60,000 to 74,073 by August 4, 2020.



<https://covid19.healthdata.org/united-states-of-america> (last visited April 28, 2020). At the time of this writing, the CDC reported the number of COVID-19 cases in the country was reported at 981,246 with 55,258 deaths. <https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/cases-in-us.html> (last visited April 29, 2020).

23. Expert testimony in this case cannot be effectively presented via Zoom. Video-conferencing software has been utilized within the

courts to address emergency, and often routine, matters, but it is ill-suited for a competency to be executed hearing in a capital case. This hearing will involve the presentation of numerous physical exhibits and complex testimony regarding Mr. Black's mental and physical ailments.

24. Mr. Black has a constitutional right to a full and fair hearing on his claim which he cannot obtain during the COVID-19 pandemic with an October 8, 2020 execution date. *Panetti v. Quarterman*, 551 U.S. 930 (2007).

**The COVID-19 Pandemic is Interfering With Mr. Black's Ability to Prepare His Case for Clemency**

25. Mr. Black also has a right to request executive clemency. "It is an unalterable fact that our judicial system, like the human beings who administer it, is fallible." *Herrera v. Collins*, 506 U.S. 390, 415 (1993). "Executive clemency [provides] the 'fail safe' in our criminal justice system." *Id.*, quoting K. Moore, *Pardons: Justice, Mercy, and the Public Interest* 131 (1989).

26. A central theme in his clemency request will be Mr. Black's intellectual disability and how the courts failed to provide Mr. Black a full and fair adjudication of his claim because of the evolving nature of the case law and ineffectiveness of his lawyers at the 2005 *Atkins* hearing. Proof of Mr. Black's intellectual disability was presented piecemeal. Because of procedural technicalities, Mr. Black has been denied the opportunity to have proof from the nation's foremost experts heard in a hearing in open court. *See* Declaration of Dr. Stephen Greenspan, Attachment C and Declaration of Dr. Marc Tasse,

Attachment D. These are matters properly addressed to the Governor in clemency.

27. Mr. Black's most recent IQ score was 67 on WAIS-IV administered by an expert witness who frequently testifies for the State. This same expert found evidence of neurocognitive deficits.

28. Mr. Black will also submit evidence of his brain damage to the Governor in his clemency petition. He requires neuroimaging, as discussed above, to fully establish his case.

29. A case for clemency also includes information from family, friends, co-workers, and other third parties with information that courts cannot consider, but which is relevant to the case for mercy. This investigation is entirely separate from the investigation performed in post-conviction court proceedings. A typical clemency effort consumes hundreds of hours of staff time, extensive travel to locate and develop witnesses who have relocated away from middle Tennessee, and face to face interviews. Many of those individuals are of an age as to put them at high risk for COVID-19.

30. It would be irresponsible and against the public's interest to conduct the necessary investigation during this pandemic. Mr. Black's team cannot conduct the work necessary to fulfill their obligation to him without putting themselves and others at risk. There is a tension between counsels' obligation to Mr. Black and to their own personal safety and that of their families and coworkers.

31. Clemency plays a vital role in our system of capital punishment. An inadequate investigation and presentation deprives the

condemned of his rights and deprives the Governor of critical information necessary to carry out his most solemn duty.

32. No efforts can, or should, be accomplished without frequent in person contact with Mr. Black. This is particularly important given the need to provide current data regarding Mr. Black's mental status. Legal visits at Riverbend Maximum Security Institution have been suspended until further notice. <https://www.tn.gov/correction/frequently-asked-questions-regarding-covid-19.html> (last visited April 29, 2020). And for good reason. In the words of the Department of Correction, prisoners are a "vulnerable population." *Id.* Governor Lee's April 28, 2020 Executive Order #30 states "[s]pecial care should be taken to protect vulnerable populations." Exec. Order # 30, at 4. The legal team could inadvertently introduce the virus into the institution. Conversely, the legal team could contract the virus and spread it in the community. Telephone access is difficult while the legal team is working remotely. Telephone conversations lack the privacy necessary to protect the attorney/client privilege and are wholly inadequate given the stakes at hand.

33. The limitations resulting from the pandemic have vexed death penalty attorneys nationwide. Emily Olson-Gault, Director and Chief Counsel of the American Bar Association Death Penalty Representation Project, explains the unique responsibilities of counsel representing men with imminent execution dates in a declaration dated April 3, 2020:

Underlying much of the ABA Guidelines is the recognition that defending capital cases requires extraordinary time and effort at every stage of a capital proceeding, including

postconviction, habeas corpus, and once an execution warrant has issued. See Guideline 1.1, Commentary (“Every task ordinarily performed in the representation of a criminal defendant is more difficult and time-consuming when the defendant is facing execution.’ ... Due to the extraordinary and irrevocable nature of the penalty, at every stage of the proceedings counsel must make ‘extraordinary efforts on behalf of the accused’”) (quoting, first, Douglas W. Vick, *Poorhouse Justice: Underfunded Indigent Defense Services and Arbitrary Death Sentences*, 43 *BUFF. L. REV.* 329, 357-58 (1995) and, second, *ABA Standards for Criminal Justice: Defense Function*, Standard 4-1.2(C), (3d ed. 1993)). The need for time and resources to prepare the defense is due in part to the tremendous amount of investigation that the capital team must complete to adequately represent a person under a death sentence.

Attachment E, at 2-3. Ms. Olson-Gault continues:

During the month of March 2020, I have spoken with capital defenders and pro bono attorneys all over the United States as they attempt to cope with the unprecedented situation created by the COVID-19 global pandemic. My understanding from these conversations is that most capital defense teams are unable to conduct the large majority of the investigation and expert work required in capital representation (*see* ¶¶18-31, *infra*). This is due to restrictions set in place by state and local governments, as well as departments of corrections and

institutional defender offices and law firms, out of a concern for public health and the welfare of employees. As a result, the already extremely limited time available to capital teams has been truncated significantly because of health concerns related to COVID-19.

Time is a scarce resource in all capital representation, and never more so than at the post- conviction or habeas corpus stages, or when an execution warrant has issued. *See* Guideline 1.1, Commentary (“Post-judgment proceedings demand a high degree of technical proficiency, and the skills essential to effective representation differ in significant ways from those necessary to succeed at trial. In addition, death penalty cases at the post- conviction stage may be subject to rules that provide less time for preparation than is available in noncapital cases. Substantive pleadings may have to be prepared simultaneously with, or even be delayed for, pleadings to stay the client’s execution.”); Guideline 10.15.1, Commentary, n.335 (“When a capital case enters a phase of being ‘under warrant’—i.e., when a death warrant has been signed—time commitments for counsel increase, “due in large part to the necessary duplication of effort in the preparation of several petitions which might have to be filed simultaneously in different courts.”).

When the already limited time is further truncated, whether by operation of the legal system or by something wholly external like a natural disaster, counsel will not have adequate time to prepare their case and this, in turn, jeopardizes due process and fairness in capital cases. See Guideline 6.1, Commentary (“Regardless of the context, no system that involves burdening attorneys with more cases than they can reasonably handle can provide high quality legal representation. In the capital context, no such system is acceptable.”).

The Guidelines’ description of the nature of investigation required at every stage of a capital proceeding provides insight into the extraordinary need for time in all capital proceedings.

*Id.*, at 3.

34. The State of Texas has now stayed six executions because of COVID-19. Associated Press, *6<sup>th</sup> Execution Delayed As Attorneys Cite Pandemic* (April 28, 2019), <https://apnews.com/984c818a009a7a9064719584abf01402> (last visited April 29, 2020). This Court postponed the execution of Oscar Smith to February 4, 2021. Order, *State v. Smith*, No. M2016-01869-SC-R11-PD (Tenn. April 17, 2020).

### **A Stay of Execution Benefits the Prison**

35. A stay of Mr. Black’s execution will benefit the prison at this time. The risk of spreading the coronavirus inside a penal institution is

enormous. Recognizing this danger, this Court ordered each county to present a plan for the reduction of pre-trial detainees in recognition of the inherent risk posed by institutional confinement.

The presiding judge or the designee of the presiding judge of each judicial district shall develop a written plan to affirmatively address issues regarding the incarceration of nonviolent offenders in furtherance of efforts to reduce the jail population, including but not limited to bond reductions or eliminations, deferred sentences, and suspended sentences.

*In Re COVID-19 Pandemic*, Order Continuing Suspension of In-Person Court Proceedings and Extension of Deadlines, No. ADM2020-00428 (Tenn. Mar. 25, 2020). As described above, the virus is spreading rapidly in Tennessee prisons and prisons nation-wide.

36. An execution takes prison staff away from their day-to-day duties. Every staff member pulled away from his or her primary responsibility of keeping the institution safe represents an opportunity for the virus to infiltrate the institution. Once inside a prison environment, the virus will spread like wildfire. *See* Associated Press, US Prisons Called a Coronavirus “Tinderbox,” Courthouse News Service (Mar. 19, 2020), <https://www.courthousenews.com/us-prisons-called-a-coronavirus-tinderbox/> (last visited April 29, 2020). Inmates and staff alike are at risk.

37. Conducting an execution during a pandemic presents needless additional risk to the staff and the almost 800 inmates of Riverbend Maximum Security Institution. Tennessee Code Annotated § 40-30-116 requires the admission of media witness and witnesses

representing the victims' families to any execution. The execution protocol does not address the carrying out of executions in the time of the COVID-19 pandemic. Prison staff will require additional training in providing safeguards to protect witnesses to the execution, the media, lawyers for the state and defense, and others who will attend the execution.

38. Should the Court fail to stay Mr. Black's execution, additional members of the public—unconnected to any penological purpose or security measure—will enter the facility presenting additional risk to the staff and incarcerated people—a risk that could be obviated by a delay in Mr. Black's execution.

39. "Death, in its finality, differs more from life imprisonment than a 100-year prison term differs from one of only a year or two. Because of that qualitative difference, there is a corresponding difference in the need for reliability in the determination that death is the appropriate punishment in a specific case." *Woodson v. North Carolina*, 428 U.S. 280, 305 (1976).

40. An execution is irreversible. It is only the order of this Court that demands that the execution be carried out on October 8, 2020. This Court has the authority to stay the execution to permit the virus outbreak to run its course and allow for Mr. Black's legal team to conduct its crucial work in representing him.

41. District Attorney General Glenn Funk, who represents the interests of the State at the competency to be executed and clemency proceedings, has authorized the undersigned to state that he takes no position on this motion.

42. Mr. Black faces irreparable harm absent a stay.

WHEREFORE, the motion should be granted.

Respectfully submitted this 29th day of April, 2020.

KELLEY J. HENRY, BPR #21113  
Supervisory Asst. Federal Public Defender  
FEDERAL PUBLIC DEFENDER  
FOR THE MIDDLE DISTRICT OF  
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Email: Kelley\_Henry@fd.org

BY: /s/ Kelley J. Henry  
Counsel for Mr. Black

### **CERTIFICATE OF SERVICE**

I, Kelley J. Henry, certify that a true and correct copy of the foregoing was served via email, on this the 29th day of April, 2020, to opposing counsel, Amy Tarkington, Associate Solicitor General, P.O. Box 20207, Nashville, Tennessee, 37202 and District Attorney General Glenn Funk, Washington Square, Suite 500, 222 2<sup>nd</sup> Avenue North, Nashville, Tennessee 37201.

BY: /s/ Kelley J. Henry  
Counsel for Mr. Black

# Attachment A

TDOC INMATES COVID-19 TESTING							
April 2020							
By Location	#Tested	#Positive	#Negative	Pending			
<b>EAST REGION</b>							
Bledsoe County Correctional Complex	2,322	583	1,703	36			
Morgan County Correctional Complex	0	0	0	0			
Northeast Correctional Complex	1	0	1	0			
<b>MIDDLE REGION</b>							
Lois M. Derry Special Needs Facility	5	0	5	0			
Riverbend Maximum Security Institution	2	1	1	0			
Tennessee Prison for Women	4	0	4	0			
Turney Center Industrial Complex	275	38	235	2			
Turney Center Industrial Complex-Annex	38	2	35	1			
<b>WEST REGION</b>							
Mark Luttrell Transition Center	2	1	1	0			
Northwest Correctional Complex	902	38	826	38			
West Tennessee State Penitentiary	1	0	1	0			
Women's Therapeutic Residential Center	2	0	2	0			
<b>CONTRACT &amp; PRIVATE MANAGED</b>							
Hardeman County Correctional Facility	2	0	2	0			
South Central Correctional Facility	2	0	1	1			
Trousdale Turner Correctional Center	248	93	148	7			
Whiteville Correctional Facility	2	0	2	0			
<b>TOTAL</b>	<b>3,808</b>	<b>756</b>	<b>2,967</b>	<b>85</b>			

Updated April 28, 2020

<b>Race/Ethnicity Breakdown</b>	
White	2,376
Black	1,222
Hispanic	53
Asian	8
Pacific Islander	6
Unavailable	143

**TDOC INMATES COVID-19 TESTING**

March 2020

By Location:	#Tested	#Positive	#Negative	Pending	Gender	Race/Ethnicity
<b>EAST REGION</b>						
Bledsoe County Correctional Complex						
Morgan County Correctional Complex						
Northeast Correctional Complex	1		1		Male	1-White
<b>MIDDLE REGION</b>						
Lois M. Derry Special Needs Facility						
Riverbend Maximum Security Institution						
Tennessee Prison for Women	2		2		Female	2-White
Turney Center Industrial Complex						
Turney Center Industrial Complex-Annex	2		2		Male	2-White
<b>WEST REGION</b>						
Mark Luttrell Transition Center						
Northwest Correctional Complex	4		4		Male	3-White, 1-Hispanic
West Tennessee State Penitentiary						
Women's Therapeutic Residential Ctr.						
<b>CONTRACT &amp; PRIVATE MANAGED</b>						
Hardeman County Correctional Facility	2		2		Male	1-Black, 1-White
South Central Correctional Facility	1		1		Male	1-Black
Trousdale Turner Correctional Center	2		2		Male	1-Black, 1-White
Whiteville Correctional Facility	8		8		Male	8-Black
<b>TOTAL</b>	<b>22</b>	<b>0</b>	<b>22</b>	<b>0</b>	20-M, 2-F	11-Black, 10-White, 1-Hispanic

## Attachment B

IN THE FIFTH CIRCUIT COURT FOR DAVIDSON COUNTY, TENNESSEE

BYRON LEWIS BLACK,

Plaintiff

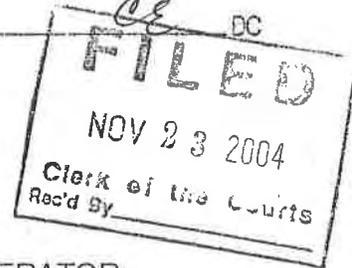
vs.

STATE OF TENNESSEE,

Defendant

2004 APR -8 PM 3:57

No. 88-S-1479 NCE CLERK



CERTIFICATE OF VIDEOTAPE CAMERA OPERATOR

The deposition of Ruben Gur, Ph.D. was taken before me, Steven W. Prince, Notary Public, at 810 Broadway, Nashville, Tennessee, on March 19, 2004, in the above captioned matter.

There appeared on behalf of plaintiff, attorneys John C. Zimmermann and Harold R. Donnelly; and on behalf of defendant, attorneys Donald E. Dawson and Catherine Y. Brockenborough.

Ruben Gur, Ph.D., after being first duly sworn by the Notary Public and Court Reporter, Florence A. Kulbaba, testified as recorded on the videotapes marked "Ruben Gur, Ph.D." These videotapes are the original audio-visual recording of the deposition.

\*\*\*\*\*

I, Steven W. Prince, a Notary Public at Large in and for the State of Tennessee, hereby certify that the videotapes marked "Ruben Gur, Ph.D." are a true and accurate record of the testimony of Ruben Gur, Ph.D. before me and that said Ruben Gur, Ph.D. was first duly sworn by a Notary Public, Florence A. Kulbaba.

I further certify that I am not a relative, employee, attorney or counsel to any of the parties to the action in which the foregoing deposition was taken, and that I am not a relative or employee of any such attorney or counsel, and that I am not financially interested in such action.

Steven W. Prince, Notary Public  
My Commission Expires: 07/29/06

1 IN THE FIFTH CIRCUIT COURT FOR DAVIDSON COUNTY,  
2 TENNESSEE, AT NASHVILLE

**FILED**  
NOV 23 2004  
Clerk of the Courts  
Rec'd By

3 BYRON LEWIS BLACK, )  
4 )  
5 Plaintiff, )  
6 vs. )  
7 STATE OF TENNESSEE, )  
8 Defendant. )

No. 88-S-1479

9 -----  
10 Deposition of:

11 RUBEN C. GUR, Ph.D.

12 Taken on behalf of the Defendant

13 March 19, 2004

2004 APR - 8 PM 3:57  
DAVID L. JOURNALISE CLERK  
DC

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25

1           The deposition of RUBEN C. GUR, Ph.D., was  
2 taken by counsel for the Defendant, pursuant to notice,  
3 at 610 Broadway, Nashville, Tennessee, on March 19,  
4 2004, for all purposes under the Tennessee Rules of  
5 Civil Procedure.

6           The formalities as to notice, caption,  
7 certificate, et cetera, are waived. All objections,  
8 except as to the form of the questions, are reserved to  
9 the hearing.

10           It is agreed that Florence A. Kulbaba, being  
11 a court reporter and notary public for the State of  
12 Tennessee, may swear the witness, and that the reading  
13 and signing of the completed deposition by the witness  
14 are waived.

15

16

17

18   \* \* \*

08:33:08 19

08:37:59 20

08:45:30 21

08:45:39 23

08:45:39 24

08:45:44 25

  VIDEOGRAPHER: We're now on the record.  
This deposition is being videotaped by Steven Prince of  
Nashville's Media Services. My business address is  
526-B Third Avenue South in Nashville, Tennessee.  
Today's date is March 19, 2004 and the time is 8:44  
a.m. Our location is 810 Broadway, Nashville,

08:45:47 1 Tennessee. The caption of this case is, In The Fifth  
08:45:51 2 Circuit Court for Davidson County, Tennessee, Byron  
08:45:57 3 Lewis Black versus State of Tennessee, Case No.  
08:45:58 4 88-S-1479. The deponent is Ruben Gur, Ph.D. This  
08:46:03 5 deposition is being taken on behalf of the defendant.

08:46:09 6 Would the attorneys please make voice  
08:46:11 7 introductions?

08:46:13 8 MR. DAWSON: This is Donald Dawson for  
08:46:16 9 Byron Black, and I'm assisted by Catherine  
08:46:16 10 Brockenborough.

08:46:19 11 MR. ZIMMERMANN: And I'm John Zimmermann  
08:46:21 12 with the District Attorney's Office, representing the  
08:46:26 13 State of Tennessee, and with me is Harold Donnelly.

08:46:26 14 VIDEOGRAPHER: The court reporter is  
08:46:28 15 Florence A. Kulbaba of Cleeton-Davis Court Reporters.

08:46:28 16 Would you please swear the witness?  
17

18 RUBEN C. GUR, Ph.D.  
19

20 was called as a witness on behalf of the Defendant and,  
21 having been first duly sworn, testified as follows:  
22

23 DIRECT EXAMINATION

24 QUESTIONS BY MR. DAWSON:

08:46:43 25 Q. Dr. Gur, would you please state your full name

08:46:45 1 for the record?

08:46:45 2 A. Ruben Gur, G-u-r.

08:46:48 3 Q. And Dr. Gur, where are you employed?

08:46:52 4 A. University of Pennsylvania.

08:46:54 5 Q. And what is your -- in what capacity are you  
08:46:58 6 employed at the University of Pennsylvania?

08:47:00 7 A. I'm a professor in the -- with tenure at the  
08:47:06 8 School of Medicine with primary appointment in  
08:47:09 9 Psychiatry and secondary appointments in Neurology and  
08:47:13 10 Radiology, and I'm directing the Brain Behavior  
08:47:15 11 Laboratory at the Medical Center.

08:47:18 12 Q. Dr. Gur, what is your educational background?

08:47:22 13 A. I received my Bachelor's Degree in Psychology  
08:47:27 14 and Philosophy at The Hebrew University of Jerusalem in  
08:47:32 15 Israel, and Master's Degree in Psychology at Michigan  
08:47:37 16 State University in 1972, and a Ph.D. at Michigan State  
08:47:43 17 University, 1973. I did Post-Doctoral Fellowships at  
08:47:49 18 Stanford University and the University of Pennsylvania.

08:47:54 19 Q. Dr. Gur, you indicated that you also worked  
08:47:59 20 with the Brain Behavior Laboratory at the University of  
08:48:03 21 Pennsylvania. Could you explain what that organization  
08:48:06 22 is engaged in?

08:48:07 23 A. The laboratory was founded in 1980 with the  
08:48:13 24 mission of exploiting the new developments in imaging  
08:48:19 25 to better understand the way in which the brain

08:48:23 1 regulates behavior, focusing on human behavior.

08:48:29 2 Q. Dr. Gur, just very briefly, since we will --  
08:48:39 3 ultimately, we'll ask to put your curriculum vitae into  
08:48:44 4 evidence in this matter, could you just briefly  
08:48:46 5 describe any specific awards or honors which are  
08:48:48 6 pertinent to your testimony in this case?

08:48:50 7 A. I suppose the kinds of awards that reflect  
08:48:56 8 recognition of peers would include the Erickson Prize  
08:49:10 9 for Scientific Excellence. The Logan Award for  
08:49:10 10 Contribution to Research in Schizophrenia. Membership  
11 in Study Section of NIMH. I've served -- I'm now  
12 serving a second term.

08:49:26 13 Q. The NIMH is the National Institute of Health;  
08:49:29 14 is that correct?

08:49:29 15 A. That's the National Institute of Health and,  
08:49:32 16 specifically, the branch of Mental Health. And the  
08:49:35 17 Study Section is a group of experts that meet three  
08:49:40 18 times a year to review all grant submissions for  
08:49:48 19 support from NIMH and decide the priority of these  
08:49:54 20 grants. Which grants are scientifically sound, and if  
08:49:58 21 they are, what is the priority for funding those  
08:50:05 22 grants.

08:50:06 23 Other awards would include election to Fellow  
08:50:11 24 status of the American Psychological Association, the  
08:50:15 25 American Psychological Society and the American Academy

08:50:26 1 of Neuropsychopharmacology.

08:50:26 2 Q. And, Dr. Gur, what is the relevance of being  
08:50:31 3 elected to Fellow status?

08:50:35 4 A. It means that -- Fellows are a small group of  
08:50:42 5 the organization that are considered leadership, and  
08:50:47 6 you have to be nominated, and discussed, and your  
08:50:53 7 credentials reviewed, and you have to -- you have to  
08:50:58 8 show that you are a leader in the field.

08:51:03 9 Q. And what is the National Academy of  
08:51:06 10 Neuropsychologists?

08:51:11 11 A. That's an organization of neuropsychologists  
08:51:16 12 that has the mission of integrating research with high  
08:51:23 13 levels of clinical practice. It's -- I don't know how  
08:51:35 14 to say, an association like many others.

08:51:39 15 Q. I think you said you were elected Fellow  
08:51:44 16 status in the American Academy of Neuropharmacologists?

08:51:49 17 A. Neuropsychopharmacology.

08:51:51 18 Q. What is that?

08:51:53 19 A. That actually is quite a deal. It's a small  
08:51:56 20 organization that has a limit on the number of its  
08:52:01 21 members. So, literally, someone has to pass away for  
08:52:06 22 new members to come in. And it's a very, very detailed  
08:52:13 23 process. And even to become a member, you have to show  
08:52:17 24 evidence of scientific excellence, and then to be  
08:52:22 25 elected Fellow of that is quite a deal.

08:52:26 1 Q. And these limitations are in their bylaws as  
08:52:30 2 to the total number that can be members of Fellows in  
08:52:33 3 that organization?

08:52:34 4 A. Yes.

08:52:36 5 Q. Are you board certified in any field of  
08:52:41 6 psychology or neurology?

08:52:43 7 A. I'm certified by the American Board of  
08:52:49 8 Professional Psychology, with specialty in Clinical  
08:52:59 9 Neuropsychology.

08:52:59 10 Q. Dr. Gur, could you again briefly discuss  
08:53:06 11 whether you have any editorial or advisory positions  
08:53:10 12 that are relevant to this deposition?

08:53:13 13 A. I'm on the Editorial Board of Journals, which  
08:53:20 14 means that I get to referee papers.

08:53:29 15 The way it works is when someone submits a  
08:53:32 16 paper to a journal, it goes to the editor, and the  
08:53:35 17 editor decides who on the board is most related to that  
08:53:41 18 area. And then that member would get the manuscript  
08:53:48 19 and will come up with three or four experts in the  
08:53:52 20 field and ask them to review the paper and comment on  
08:53:57 21 it. And then we'll decide, based on these reviews,  
08:54:01 22 whether to accept the paper, which almost never  
08:54:06 23 happens, or to reject it, which is usual, or to send it  
08:54:13 24 back to the authors with the comments of the reviewers  
08:54:16 25 and ask them to fix it if the editor thinks those

08:54:20 1 difficulties are fixable. And then the manuscript goes  
08:54:24 2 back and the author revises it, sends it back. And  
08:54:28 3 finally, when that -- that member of the board decides  
08:54:31 4 that the paper is ready for publication, they forward  
08:54:35 5 it to the editor, who then makes the final  
08:54:39 6 determination.

08:54:40 7 Q. What particular publications have you served  
08:54:45 8 -- or do you serve on the editorial board for?

08:54:48 9 A. Well, I'm what's called an Action Editor for  
08:54:54 10 The Journal of Brain Recognition, which is a leading  
08:54:58 11 journal in the field of linking brain and behavior, and  
08:55:03 12 I review imaging studies that are submitted to that  
08:55:07 13 journal. I'm also on the Editorial Board of  
08:55:16 14 Schizophrenia Research, and it's about the same deal.

08:55:21 15 Q. And do you also do ad hoc reviewing for other  
08:55:26 16 organizations or publications?

08:55:28 17 A. Yeah, I do -- so I do serve as one of those  
08:55:35 18 people that the editors send the journal and article to  
08:55:39 19 to review in order to make the decision, and I have  
08:55:43 20 reviewed for -- I have done reviews for many journals,  
08:55:50 21 including all the major journals in my field, as well  
08:55:54 22 as general scientific journals such as Science And  
08:55:59 23 Nature.

08:55:59 24 Q. Tell us about Science And Nature; where do  
08:56:03 25 they fit in terms of the overall journals in the, I

08:56:06 1 guess, scientific community, particularly those that  
08:56:09 2 would report on matters involving brain function?  
08:56:12 3 A. Well, the way it works is if you did a study  
08:56:15 4 that, depending on the results, if you think the study  
08:56:19 5 was done well and the results are meaningful for your  
08:56:22 6 field, you will submit it to a specialty journal in  
08:56:25 7 your field. But if you think that your results are far  
08:56:29 8 reaching and may be of relevance to other fields, other  
08:56:33 9 scientific fields, you would consider a broader journal  
08:56:37 10 that is read by a more diverse group of scientists.  
08:56:44 11 And the value, the importance of a journal is judged by  
08:56:55 12 the so-called impact factor.

08:56:58 13 There is an Institute for Scientific  
08:57:02 14 Information that collects data every time a paper that  
08:57:06 15 was published is cited by -- in another paper, then  
08:57:11 16 they assign a point to that paper. They also assign a  
08:57:17 17 point to that journal. So counting how many times on  
08:57:20 18 the average papers published in that journal are being  
08:57:24 19 cited is an index of the impact of that journal on the  
08:57:29 20 scientific community.

08:57:30 21 So Science and Nature are the journals among  
08:57:38 22 the highest impact on the scientific community. So if  
08:57:43 23 you published a paper in Science it, first of all,  
08:57:43 24 means that it must be a very good paper because they  
08:57:46 25 reject 99 -- they probably reject 99 percent of the

08:57:53 1 submissions, and then -- but if it gets in, then it's  
08:57:58 2 very likely to be read by a lot of scientists. And so  
08:58:03 3 they will use it in different fields. And so that's  
08:58:07 4 how the impact -- your own impact and the impact of the  
08:58:12 5 journal is enhanced.

08:58:13 6 Q. Now, have you been published in the Journal of  
08:58:16 7 Science?

08:58:16 8 A. Yes.

08:58:16 9 Q. Approximately how many articles have you had  
08:58:20 10 published by that publication?

08:58:21 11 A. I have five.

08:58:25 12 Q. Now these are the articles, then, that were in  
08:58:29 13 your field, based on your research?

08:58:31 14 A. Yes.

08:58:32 15 Q. But would be applicable to people beyond just  
08:58:35 16 the brain imaging field; is that right?

08:58:38 17 A. That is correct.

08:58:39 18 Q. And then are there other articles that you  
08:58:41 19 have submitted to be published in more specific  
08:58:43 20 journals that deal more specifically with the area of  
08:58:47 21 brain imaging or neuropsychology?

08:58:49 22 A. Yes.

08:58:50 23 Q. And what are some of the -- in terms of -- you  
08:58:53 24 indicated there is a scoring system of how often that  
08:58:58 25 they're cited by other articles. In terms of the

08:59:02 1 journals that you have published in, which journals are  
08:59:06 2 the most cited? The ones that are just specifically  
08:59:09 3 related to the field of neuropsychology or brain  
08:59:12 4 imaging?

08:59:13 5 A. Probably the journal with the highest impact  
08:59:17 6 in neuropsychology would be a journal called -- well,  
08:59:25 7 it's sort of a toss between Brain Incognition and a  
08:59:30 8 journal called Neuropsychologia.

08:59:33 9 The top journals in the field of  
08:59:36 10 psychiatry in terms of impact are the Archives of  
08:59:40 11 General Psychiatry and followed by the American Journal  
08:59:44 12 of Psychiatry. And in the field of neuroimaging, a  
08:59:49 13 journal that is called Neuro-Image has a very high  
08:59:53 14 impact factor, in general. In the neuroscience, the  
08:59:57 15 journal with the highest impact factor is the Journal  
09:00:00 16 of Neuroscience.

09:00:03 17 Q. Have you been published in all of those  
09:00:06 18 journals that you're talking about?

09:00:07 19 A. Yes. I have published in all of those  
09:00:10 20 journals.

09:00:10 21 Q. One of things I have noticed in looking  
09:00:12 22 through your curriculum vitae, as far as the journals  
09:00:16 23 that -- the articles that you have published, some of  
09:00:18 24 them were published with more than one author; is that  
09:00:21 25 correct?

09:00:21 1 A. Correct. Yes.

09:00:22 2 Q. And just somebody reviewing this, your  
09:00:30 3 curriculum vitae, what -- is there a significance to  
09:00:32 4 the order in which the names appear on the articles?

09:00:35 5 A. The order -- the significance of the order  
09:00:40 6 depends on the field. If you think there are a lot of  
09:00:44 7 authors in some of those papers, you should see the  
09:00:47 8 last issue of Physics Review. You would find there,  
09:00:51 9 literally, a paper where the number of authors exceeds  
09:00:55 10 the number of words in the article. And in there they  
09:01:01 11 list the authors alphabetically. So people with the  
09:01:06 12 name of Zura are really in trouble, or Zimmermann, I  
09:01:11 13 suppose.

09:01:12 14 But in my field, the -- and the neurosciences  
09:01:17 15 in general, the order is that the person, the senior  
09:01:22 16 author is the first author, the second and the third  
09:01:25 17 author, and the last author. So the last author is  
09:01:29 18 usually the person who's lab work was done. And  
09:01:34 19 depending on that individual, sometimes this person  
09:01:41 20 could have even written the article and stated as last  
09:01:46 21 author, or it could have been someone who's barely read  
09:01:48 22 the article and be the last author. So --

09:01:50 23 Q. And Dr. Gur, I have noticed that many of the  
09:01:53 24 articles that you have published have as two of the  
09:01:56 25 authors, Gur, R.C., and Gur, R.E; could you explain who

09:01:59 1 those two individuals are?

09:02:01 2 A. Yes. Gur, R.C. is myself and the other Gur is  
09:02:09 3 my better half, Racquel E. Gur. We have studied  
09:02:18 4 together, and despite the strong advice of our  
09:02:24 5 post-doctoral advisor, we continued to work together.  
09:02:30 6 He warned us not to publish together because people  
09:02:33 7 will forever try to figure out who is the real brain.  
09:02:38 8 And he suggested we best go into different fields.  
09:02:42 9 While she established preeminence for herself, she went  
09:02:49 10 and got the medical degree and did residencies in  
09:02:53 11 neurology, and then in psychiatry.

09:02:56 12 And so nobody confuses us. They know we  
09:03:00 13 worked together but they -- I think most people have  
09:03:09 14 come to accept that it's possible that both of us are  
09:03:13 15 intelligent and still work together.

09:03:16 16 Q. Okay. You had indicated earlier, you  
09:03:19 17 mentioned the term that -- of, I believe you said  
09:03:24 18 referee journals, and are they also called peer review  
09:03:31 19 journals?

09:03:32 20 A. Yes.

09:03:32 21 Q. And are there other journals that are not peer  
09:03:36 22 review?

09:03:36 23 A. Yes. There are quite a few of those.

09:03:38 24 Q. And what is the difference, basically, between  
09:03:41 25 those two types of journals?

09:03:42 1 A. It's -- the other types of journals would have  
09:03:48 2 very minimal quality control of the papers, and they  
09:03:54 3 usually would charge for publishing there.

09:03:58 4 So it's a business. But they have similar  
09:04:06 5 names. They look the same. And so if someone wants to  
09:04:12 6 be proud of having a paper that appeared in a journal  
09:04:15 7 that has a name and a number and all that stuff, they  
09:04:18 8 can publish there. It's unlikely that anybody will  
09:04:22 9 cite work that was published in one of those journals.  
09:04:26 10 But sometimes there are some interesting studies that  
09:04:32 11 the author just decided, "I can't -- I can't fight with  
09:04:36 12 those reviewers. I think it's interesting. I think  
09:04:40 13 it's important. The heck with it. I'll send it to one  
09:04:43 14 of those journals."

09:04:44 15 So you may find, occasionally, something  
09:04:46 16 interesting in them, except that they would not be  
09:04:50 17 included in the Institute of Scientific Information  
09:04:55 18 database. So you really have to go out of your way to  
09:04:58 19 find those papers.

09:05:00 20 Q. Let me go back a minute just to ask about your  
09:05:12 21 -- you had indicated, I think in the beginning, that  
09:05:15 22 you were, I believe, a tenured faculty member at the  
09:05:18 23 University of Pennsylvania?

09:05:19 24 A. Yes.

09:05:20 25 Q. What is the significance of being tenured?

09:05:23 1 A. Well, it's been the stated criteria for  
09:05:31 2 receiving tenure at the level of associate professor.  
09:05:35 3 So when you are hired, you're hired as an assistant  
09:05:38 4 professor and then you get -- if you don't have  
09:05:41 5 clinical duties, you get seven years. If you do have  
09:05:47 6 clinical duties, you can get up to 10 years. And  
09:05:51 7 during that time, in order to be promoted you have to  
09:05:55 8 become -- as is stated, you have to become a national  
09:05:59 9 leader in the field with international reputation. In  
09:06:06 10 order to be promoted from associate professor to full  
09:06:10 11 professor, you have to be a recognized international  
09:06:14 12 leader in your field. So there are a lot of people who  
09:06:19 13 have been promoted to associate professor and stayed at  
09:06:22 14 that level for the remainder of their career.

09:06:26 15 Q. You are now a full professor?

09:06:29 16 A. Yes.

09:06:29 17 Q. When were you promoted to full professor?

09:06:32 18 A. I believe 1988 was it? Such an important  
09:06:39 19 thing and I don't remember anymore.

09:06:40 20 Q. It's been some time that you have been a full  
09:06:44 21 professor?

09:06:44 22 A. 1988. I do. 1988.

09:06:58 23 Q. In terms of the work that you do at the  
09:07:11 24 university, are you involved in requests for grants to  
09:07:16 25 pursue your research?

09:07:18 1 A. Well, the kind of research that I do is very  
09:07:24 2 expensive. I mean, a single pet study would be in the  
09:07:30 3 neighborhood of \$2,300, if you do everything that you  
09:07:35 4 would want to do.

09:07:36 5 So you need to get support for the work, and  
09:07:39 6 the way you get support is by applying for grants from  
09:07:44 7 one -- either the National Institute of Mental Health  
09:07:48 8 or the National Science Foundation. And in the grant  
09:07:54 9 you have to describe what your hypotheses are, what  
09:07:59 10 your plan is, describe in detail what studies you plan  
09:08:02 11 to do, and then it gets reviewed. Those applications  
09:08:08 12 get reviewed three times a year by a Study Section that  
09:08:12 13 consists of other experts in your field and they  
09:08:18 14 scrutinize your application very thoroughly because  
09:08:23 15 usually they, literally, see that money as coming out  
09:08:27 16 of their pocket, not just as taxpayers, but any penny  
09:08:33 17 that goes into your lab is a penny less available for  
09:08:36 18 their work. And they would assign a score to the grant  
09:08:41 19 that ranges from one, outstanding, to five, acceptable,  
09:08:46 20 if they approve the grant. If they consider that the  
09:08:50 21 ideas are sound and the design is sound. If they  
09:08:54 22 don't, then they just disapprove it.

09:08:58 23 So just to give you a feel, in order to get  
09:09:02 24 the grant funded today, you have to score better than  
09:09:09 25 about 1.5. So there will have to be about 20 to 25

09:09:14 1 people your field who will think that this idea -- 1 to  
09:09:19 2 1.5 is the outstanding range. So if anybody thinks  
09:09:25 3 that your proposal is not outstanding, but only  
09:09:28 4 excellent, you only have about 15, 20 percent chance of  
09:09:33 5 getting the money. If they think it's only very good,  
09:09:36 6 you have to read their comments and hope that you can  
09:09:40 7 change and revise your application in order to convince  
09:09:45 8 them the next round that they should fund you. You're  
09:09:49 9 allowed to revise the application twice.

09:09:53 10 Q. And what particular grants have you or your  
09:09:59 11 center been -- well, you, individually, been awarded?  
09:10:01 12 Let's talk about that first.

09:10:03 13 A. Well, the sort of most challenging and  
09:10:13 14 prestigious grant to get is called the RO1 which is an  
09:10:18 15 individual grant where one person gets -- their team  
09:10:24 16 gets the money to carry out a specific focus study.

09:10:31 17 Second, is a Center Grant where you put  
09:10:38 18 together a group of projects around a specific theme,  
09:10:44 19 and there the challenge is how to get different  
09:10:47 20 laboratories to work together and use different means  
09:10:51 21 in order to attack the same problem. So there has to  
09:10:56 22 be a central theme and you have to show that by funding  
09:10:59 23 all these laboratories to work together, you get  
09:11:02 24 something that is more than the sum of its parts,  
09:11:05 25 rather than, "Why don't we fund this lab to do that?"

09:11:08 1 Something specific. And this, "Why should we make it a  
09:11:12 2 center?"

09:11:12 3 So that is very difficult to get. And there  
09:11:18 4 is a national competition for those. So the center  
09:11:22 5 that we got, the County Center for the Neuroscience,  
09:11:33 6 they were not -- in the round that we got it, there  
09:11:36 7 were 20 applications in from all over the United  
09:11:42 8 States, and actually in that round we were the only one  
09:11:47 9 -- the only center that was funded out of them.

09:11:49 10 Q. And what was that grant to do?

09:11:54 11 A. This specific grant proposes to really crack  
09:12:01 12 the fundamental abnormalities in the brain of people  
09:12:06 13 who suffer from schizophrenia by using a combination of  
09:12:12 14 molecular biology and integrating it all the way to the  
09:12:19 15 clinical phenomenon.

09:12:22 16 So there are several studies that work with  
09:12:27 17 mice, monkeys, and one project that works on single  
09:12:33 18 neurons, all the way to studies that use functional  
09:12:38 19 imaging and structural imaging. And we are testing a  
09:12:42 20 very specific hypothesis that, if it turns out that we  
09:12:47 21 are on the right track, it will really, at least the  
09:12:52 22 judgment of our peers, it will really open new avenues  
09:12:56 23 for understanding the brain dysfunction of  
09:13:00 24 schizophrenia and how to treat it.

09:13:02 25 Q. This is an ongoing grant that began in 2001

09:13:06 1 and continues to 2006?

09:13:10 2 A. That's correct. And then you can get it  
09:13:11 3 renewed for five more years. But again, you have to  
09:13:14 4 submit your plans and show that you did what you  
09:13:18 5 promised and that now is the time to keep going in that  
09:13:24 6 direction.

09:13:24 7 Q. In addition to this ongoing Center Grant, you  
09:13:27 8 also have ongoing RO1 Grants, correct?

09:13:31 9 A. Yes.

09:13:31 10 Q. Are they related to the Center Grant or are  
09:13:35 11 they separate from the research of the center?

09:13:37 12 A. They are separate. It's an independent study.  
09:13:41 13 Although one advantage of having several grants around  
09:13:45 14 the same area is that you get an economy of resources  
09:13:49 15 so that -- for example, for my RO1, I do a certain  
09:13:55 16 procedure during functional MRI. For the County  
09:14:01 17 Center, they need another procedure and we can combine  
09:14:05 18 both procedures in the same session, and here we have  
09:14:08 19 one research participant who can contribute valuable  
09:14:12 20 data to different projects and that cuts the cost of  
09:14:19 21 the study by half.

09:14:20 22 Q. And how many research projects, I guess, are  
09:14:24 23 you presently engaged in through either the Center  
09:14:27 24 Grant or through individual grants?

09:14:29 25 A. Well, as the head of the laboratory, I also

09:14:32 1 help junior faculty and collaborate with them on their  
09:14:40 2 project. And several of them have now already obtained  
09:14:45 3 their own grant support, including RO1's and what is  
09:14:50 4 called K-Award, or Research Scientist Development  
09:14:55 5 Award.

09:14:55 6 If a beginning scientist thinks that they have  
09:15:00 7 a chance to become independent leaders in the field,  
09:15:04 8 they can submit an application to the NIMH describing  
09:15:11 9 who their mentor is going to be, and what are their  
09:15:14 10 plans to advance their career, and what studies they're  
09:15:18 11 going to do in the next five years. And then it goes  
09:15:21 12 through the same kind of a Study Section. If a Study  
09:15:25 13 Section decides this is an excellent candidate who is  
09:15:28 14 likely to make major contributions with a good mentor,  
09:15:31 15 then they basically paid that person's salary for five  
09:15:35 16 years. And so the University can release that person  
09:15:39 17 from teaching or clinical duties and allow that person  
09:15:44 18 to focus on their research.

09:15:47 19 So, I'm mentor on several of those and I  
09:15:52 20 collaborate with more junior faculty, and sometimes  
09:15:57 21 more senior faculty who work in other projects and need  
09:16:00 22 input in my area.

09:16:04 23 Q. Now, what is an RSDA?

09:16:08 24 A. That stands for a Research Scientist  
09:16:11 25 Development Award, as I've just described.

09:16:14 1 Q. And how many people are you currently  
09:16:17 2 supervising with those?

09:16:18 3 A. Four.

09:16:19 4 Q. What about your faculty work at the University  
09:16:26 5 of Pennsylvania; are there any particular committees  
09:16:29 6 that you are on that are of particular significance, in  
09:16:32 7 your own mind?

09:16:33 8 A. Well, over the years, probably the most  
09:16:37 9 important committee I was on was the Senate Committee  
09:16:41 10 On Academic Freedom And Responsibility, which I served  
09:16:46 11 on twice.

09:16:48 12 The reason I considered that important is that  
09:16:55 13 it's a sign my colleagues consider me a person of  
09:16:59 14 integrity because the role of that committee is to  
09:17:02 15 adjudicate disputes among faculty about issues such as  
09:17:09 16 plagiarism, scientific misconduct, as well as  
09:17:15 17 adjudicate issues on tenure. So, if someone has been  
09:17:23 18 denied tenure and they think there was something wrong  
09:17:26 19 about that decision, it was an unfair procedure, then  
09:17:32 20 they will file a grievance, and if they win the  
09:17:36 21 grievance, then it comes to the Senate Committee On  
09:17:40 22 Academic Freedom to decide and recommend what should be  
09:17:44 23 done about it.

09:17:47 24 Another committee that I'm proud of  
09:17:52 25 participating in is the University Scholars Council.

09:17:57 1 The University Scholars Council is a very special  
09:18:02 2 program at Penn designed to identify early individuals  
09:18:09 3 within the undergraduate student body who are likely to  
09:18:14 4 become major people in their fields. And to get  
09:18:23 5 accepted into it, the student has to, of course, do  
09:18:28 6 very well in the courses, but also show evidence that  
09:18:34 7 they have a focus and a direction and a plan of  
09:18:38 8 implementing either a scientific or a community or an  
09:18:44 9 artistic project that will make a big difference. And  
09:18:49 10 if their plan is accepted, then we fund it. So we have  
09:18:52 11 funded people to travel all over the world to do  
09:18:57 12 studies. We have funded them to spend summers in a lab  
09:19:03 13 somewhere to learn a specific method. We have funded  
09:19:07 14 someone to make a movie, and it was the first Penn  
09:19:12 15 undergraduate to produce a full length commercial  
09:19:19 16 movie.

09:19:19 17 So those are the kinds of issues that the  
09:19:24 18 University Scholars Council deals with. And it's  
09:19:29 19 really a pleasure to serve on it.

09:19:33 20 Q. And Dr. Gur, at this point in your career how  
09:19:40 21 many articles have you had published in journals?

09:19:42 22 A. I think I just passed the 200 mark.

09:19:47 23 Q. And are these all in referee journals?

09:19:53 24 A. Yes.

09:19:53 25 Q. In addition, did you have some other

09:20:01 1 publications in various things that were not at the  
09:20:09 2 same level, in terms of professional standing, as these  
09:20:12 3 journal articles?

09:20:14 4 A. Yes. I mean, these are book chapters for  
09:20:20 5 consideration of tenure, and those kinds of things.  
09:20:24 6 They don't count, except if there are chapters in  
09:20:29 7 textbooks, and these are actually quite as prestigious  
09:20:36 8 as individual -- as refereed articles because they  
09:20:42 9 indicate that somebody who writes the textbook of your  
09:20:47 10 field thinks that there is an area that you are the  
09:20:50 11 best person to contribute to.

09:20:53 12 So they are listed separately on the chapters,  
09:20:59 13 but they do include several textbook chapters. And  
09:21:04 14 then commentaries, they are refereed, but they don't  
09:21:09 15 really contribute new data. So again, it's not the  
09:21:14 16 same class of publication.

09:21:18 17 Q. So they're not based on your ongoing research,  
09:21:21 18 these are based more on commenting on somebody else's  
09:21:25 19 research, or something of that nature?

09:21:27 20 A. Yes. They may be reflections on the field,  
09:21:33 21 thoughts on how the field can do better. Could be  
09:21:40 22 arguments with other investigators who came up with a  
09:21:47 23 certain idea, and you don't think this is such a great  
09:21:51 24 idea, you write a paper, and if the journal decides,  
09:21:55 25 they will publish it.

09:21:59 1 Sometimes they are invited. So Contemporary  
09:22:03 2 Psychology is a journal that invites people to review.  
09:22:06 3 And that actually is considered quite prestigious but,  
09:22:10 4 you know, when you review it is refereed and all that,  
09:22:14 5 but it's not, again, the same status as contributing a  
09:22:20 6 scientific paper.

09:22:25 7 MR. DAWSON: And at this point, I think  
09:22:27 8 we have concluded the credentials for Dr. Gur.

09:22:35 9 Mr. Zimmermann, do you have any objections?

09:22:37 10 MR. ZIMMERMANN: I have no objections.

09:22:41 11 MR. DAWSON: And then I would assume that  
12 you don't have any objections to him testifying as an  
09:22:41 13 expert in the field of neuropsychology?

09:22:44 14 MR. ZIMMERMANN: No. I have no objection.

09:22:46 15 MR. DAWSON: Thank you.

16 BY MR. DAWSON:

09:22:50 17 Q. And, Dr. Gur, if you would look, I think the  
09:22:54 18 top document that's sitting there in that pile of  
09:22:59 19 documents that we sat there earlier is your curriculum  
20 vitae? That one that's in the --

09:23:04 21 A. Yeah, I believe she gave me a copy of it.

09:23:06 22 MR. DAWSON: I would just ask that that  
09:23:08 23 be admitted as Exhibit No. 1 to this deposition.

24 (Document marked Exhibit No. 1.)

25 BY MR. DAWSON:

09:23:30 1 Q. Dr. Gur; just very briefly, before we leave  
09:23:32 2 the issue of publications, are you familiar with a  
09:23:39 3 publication called Perceptual and Motor Skills?

09:23:43 4 A. Yes.

09:23:46 5 Q. And did you have an opportunity to try to look  
09:23:51 6 at what the impact factor was on that list that you  
09:23:56 7 indicated that were -- where publications are rated to  
09:23:59 8 see what kind of an impact number that particular  
09:24:03 9 journal has?

09:24:04 10 A. This is one of those journals where you pay to  
09:24:08 11 publish, and the ISI doesn't even list it.

09:24:14 12 Q. And what about another journal called  
09:24:21 13 Cognitive Rehabilitation?

09:24:23 14 A. I could not find that listed.

09:24:25 15 Q. That was not listed, either, on the list?

09:24:27 16 Now, the list that you have of journals that  
09:24:31 17 have been rated under that impact factor, that number  
09:24:34 18 is in the thousands or hundreds or --

09:24:37 19 A. It's close to a thousand. I don't remember  
09:24:43 20 exact number. I can check if I have it.

09:24:46 21 Q. What about another journal called -- or  
09:24:48 22 publication called the Archives of Clinical  
09:24:51 23 Neuropsychology?

09:24:52 24 A. That is listed and has an impact factor of  
09:24:58 25 0.9.

09:24:58 1 Q. And what does the 0.9 impact factor mean?

09:25:01 2 A. Means that, on the average, about one paper  
09:25:07 3 that gets published in their journals would be cited  
09:25:12 4 once.

09:25:12 5 Q. And how would that be, comparatively to, for  
09:25:16 6 instance, the Journal of Science in terms of the number  
09:25:18 7 that it would have?

09:25:19 8 A. The impact factor of Science is approximately  
09:25:24 9 30.

09:25:25 10 Q. So that would mean it would be, an article  
09:25:27 11 published in Science would be -- would be cited by  
09:25:32 12 other scientists writing in that field approximately 30  
09:25:35 13 times?

09:25:35 14 A. Yeah.

09:25:36 15 Q. Is that per year or per decade or what do we  
09:25:45 16 have?

09:25:45 17 A. It's total.

09:25:46 18 Q. Total.

09:25:57 19 A. You have to understand, this average is what  
09:25:59 20 it is. It's an average. So it could be one article in  
09:26:04 21 the Archives that is cited 100 times and none of the  
09:26:09 22 other articles are cited. So, it's an average.

09:26:13 23 Q. Kind of hit or miss, whereas with the Journal  
09:26:18 24 of Science, it would be more likely that an article  
09:26:20 25 there would be cited by somebody sometime?

09:26:22 1 A. Exactly.

09:26:22 2 Q. In terms of the testimony or the research that  
09:26:29 3 you did or the tests that you conducted on Mr. Black,  
09:26:34 4 can you briefly tell us the information that's  
09:26:37 5 necessary to understand the imaging results?

09:26:40 6 A. Yes. I'll be happy to. If I may --

09:26:43 7 Q. Would you like to use PowerPoint at this  
09:26:48 8 point?

09:26:48 9 A. Yes, if I may, because it really will save  
09:26:53 10 time.

09:26:54 11 MR. ZIMMERMANN: Anything that will save  
09:26:55 12 time, Doctor.

09:26:56 13 THE WITNESS: So, what you see here is a  
09:27:00 14 schematic of a neuron, and this is really the  
09:27:04 15 fundamental element of the brain. The brain consists  
09:27:07 16 of the collection of neurons that are packed together.  
09:27:12 17 A neuron includes a cell body, which is here, and out  
09:27:23 18 of the cell body you see protrusions coming out, and  
09:27:29 19 these are called dendrites. And the protrusions are  
09:27:36 20 here in order to get information from adjacent neurons.  
09:27:40 21 So, you can imagine another neuron here. They don't  
09:27:45 22 touch each other. For many years we thought they were.  
09:27:49 23 But it was proven conclusively that they are not.

09:27:53 24 So, somehow information has to get from this  
09:27:57 25 neuron to this neuron. But the way the neuron works is

09:28:01 1 that when it gets input, chemical input through the  
09:28:07 2 dendrites, once those chemical inputs come up to about  
09:28:15 3 a certain threshold, an electrical pulse is generated  
09:28:18 4 and it travels down the membrane of the neuron.

09:28:21 5         And here what you see is an axon, and that is a  
09:28:27 6 long fiber, unlike the dendrites that are short. It's  
09:28:32 7 a long fiber. It can be actually as long as your  
09:28:37 8 entire height because there is one bundle of those  
09:28:40 9 going from your brain all the way to the tip of your  
09:28:44 10 toes. So this can be very long fibers. And so the  
09:28:47 11 electricity needs to travel down this neuron in order  
09:28:52 12 to generate a pulse for the next neuron to fire.

09:28:59 13         So, this is how the brain communicates. The  
09:29:02 14 dendrites are used to communicate with neurons adjacent  
09:29:06 15 to them, and the axons are used to send information for  
09:29:09 16 longer distances.

09:29:11 17         Now, in order to send information for longer  
09:29:14 18 distances you need to insulate the conductor of  
09:29:18 19 electricity, the same way we send electricity long,  
09:29:20 20 long distances. We have to use thick rubber for  
09:29:25 21 shielding the cables. The brain doesn't use rubber for  
09:29:29 22 shielding, it uses fat. So, those red nodes, they  
09:29:34 23 really are white, and they surround the axon and make  
09:29:41 24 sure that the electricity goes through effectively and  
09:29:47 25 doesn't dissipate around, okay? Now --

09:29:56 1 MR. DAWSON: Doctor, at this point I'd  
09:29:57 2 just like to stop you one minute. We have -- I think  
09:30:00 3 the next thing in the stack of documents in front of  
09:30:02 4 you is that picture of the neuron that you just showed,  
09:30:05 5 and I would ask that that be introduced as Exhibit No.  
09:30:10 6 2.

09:30:10 7 MR. ZIMMERMANN: Well, is this what he's  
09:30:12 8 showing is going to be an exhibit?

09:30:13 9 MR. DAWSON: Yes. But, in case somebody  
09:30:16 10 doesn't have the capability of seeing, it's a good idea  
09:30:21 11 to have it in the deposition.

09:30:35 12 (Document marked Exhibit No. 2.)

09:30:38 13 THE WITNESS: So, now how do we study the  
09:30:43 14 brain? How do we try to understand its development and  
09:30:48 15 how that relates to behavior?

09:30:52 16 Up until the era of neuroimaging, the only way  
09:30:59 17 to study the brain was to hope that you get a brain and  
09:31:02 18 you can take a look at it and cut it up and look inside  
09:31:07 19 and try to figure out how it is wired. So this work  
09:31:12 20 was done with post mortem brains. And among the most  
09:31:18 21 pre-eminent investigators of brain anatomy, Yakovlev at  
09:31:26 22 Harvard established big collection of brains of aborted  
09:31:32 23 fetuses and children who died for various reasons, as  
09:31:43 24 well as adults who also died for various reasons. So  
09:31:47 25 he has a lot of premie -- a lot of fetuses in his

09:31:52 1 collection and a lot of old people. Very few  
09:31:56 2 inbetween.

09:31:57 3 But on that basis, he tried to understand how  
09:32:00 4 does the brain develop? And the method he used was to  
09:32:04 5 look at a process called myelination. So if you  
09:32:10 6 recall, if you recall the neuron, this myelin, what it  
09:32:18 7 turns out is that there is really none of it in a  
09:32:21 8 newborn infant. Those fibers are totally exposed. And  
09:32:27 9 this is why if you tickle a baby, they really will  
09:32:32 10 throw up their hands and their feet. They can't really  
09:32:35 11 send the signal to the right part of the body. It goes  
09:32:39 12 all over the place. And as those neurons begin to  
09:32:42 13 myelinate, they can control themselves better so when  
09:32:46 14 they want to grab something, they will get closer and  
09:32:49 15 closer to it. And that exactly goes hand in hand with  
09:32:53 16 the process of myelination.

09:32:55 17 So Yakovlev had a stain that he could use that  
09:33:01 18 he would take a slice of brain, and this stain that he  
09:33:05 19 used is sensitive to fat. It will attach itself to  
09:33:08 20 fat. So if there is a -- if there is a lot of myelin  
09:33:12 21 in the area, it will be dark. If there is very little,  
09:33:15 22 it will be light.

09:33:17 23 And this shows, for example, the corpus  
09:33:20 24 callosum, which is a body of nerve fibers that  
09:33:24 25 connects the two -- you know, we have not one brain but

09:33:27 1 two brains, two hemispheres, and the only thing that  
09:33:30 2 connects them is a body of axons, or neurofibers. And  
09:33:34 3 you can see that these fibers in a baby that was born  
09:33:39 4 at term is gray. There is no fat there. In a two-  
09:33:45 5 year-old you can already see it's quite dark, the  
09:33:49 6 corpus callosum. This banana-shape, here, is the  
09:33:53 7 corpus callosum. In an adult, pitch black.

09:33:57 8 So, it shows how much more myelin was created  
09:34:01 9 there. And that's also -- this is a coronal slice and  
09:34:08 10 you can see here again in a -- this is a baby, 24  
09:34:13 11 gestational weeks. There is no fat here. Now, at term  
09:34:17 12 you see a little dark appearing in the brain, so the  
09:34:23 13 brain begins to myelinate.

09:34:26 14 Now, based on all those data, Yakovlev has  
09:34:34 15 created those developmental trajectories and it turns  
09:34:40 16 out, remarkably, that different regions of the brain  
09:34:45 17 myelinate at a different rate. So it's not that the  
09:34:48 18 entire brain myelinates at the same time, but the  
09:34:51 19 myelination goes in stages. And based on looking at  
09:34:55 20 different brain regions and finding how much myelin  
09:35:01 21 there is at various stages of life, he discovered the  
09:35:06 22 principal that there's essentially three zones of  
09:35:12 23 myelination.

09:35:14 24 So the first zone that gets myelinated is the  
09:35:18 25 part of the brain that deals with sensory and motor

09:35:23 1 functions. So you -- that explains why a baby can  
09:35:28 2 already reach and grab something specific before they  
09:35:31 3 can walk. Walking is already a part of it. So that  
09:35:35 4 development is going hand in hand with the process of  
09:35:39 5 myelination. And there what he found was that the last  
09:35:43 6 regions to myelinate are the cortical regions, the part  
09:35:49 7 of the thinking brain. And the last of those to become  
09:35:54 8 myelinated is the frontal lobe, which is right here, in  
09:35:59 9 the front of the brain. And we'll get to talk about  
09:36:04 10 what the significance of that is in a moment.

09:36:08 11 So because of what happens, you can see that --  
09:36:14 12 this is another post mortem study where they took  
09:36:18 13 hundreds of people and weighed their brains. They just  
09:36:25 14 looked at how many kilograms the brain is. And you can  
09:36:28 15 see that as a function of age, the weight of the brain  
09:36:33 16 climbs. Now, this climb of weight reaches its peak at  
09:36:40 17 around age 21 and, then it levels off for a bit, and  
09:36:45 18 then, as we all know, things go downhill from there.

09:36:53 19 Now with MRI, or magnetic resonance imagining,  
09:36:59 20 we don't have to wait for someone to die in order to  
09:37:02 21 study the brain. We can do a lot of work in  
09:37:06 22 understanding brain structure and function with people  
09:37:09 23 who are alive.

09:37:11 24 The way it works is that you put the head in a  
09:37:13 25 very strong magnetic field. And so all the molecules

09:37:19 1 in your head align themselves with a strong field, and  
09:37:22 2 then you send a brief pulse that is at an angle to the  
09:37:27 3 main field. When you send that radiofrequency pulse,  
09:37:32 4 the molecules say, "Hey, there is a new boss here."  
09:37:35 5 They try to align themselves with the pulse, but then  
09:37:39 6 it disappears because it's very brief. And so they  
09:37:41 7 will rattle back to the original orientation of the  
09:37:46 8 large field and, like anything that is pliable, like  
09:37:51 9 this pencil, if it goes back it rattles. So those  
09:37:56 10 molecules rattle when they resonate back to the first  
09:38:01 11 field. And there is this principal, of course, that  
09:38:05 12 big bodies move slowly, and that works for everything,  
09:38:09 13 including molecules. So the first to come back to  
09:38:14 14 position are the smallest molecules, which is water.  
09:38:19 15 And then come the bigger molecules, the proteins and  
09:38:24 16 the fats and so on.

09:38:26 17 So you can actually -- you can put an antenna,  
09:38:31 18 like the radio antenna, and listen to the resonance of  
09:38:37 19 those molecules. And from that way, figure out how,  
09:38:40 20 what's the distribution of each molecule in the brain.

09:38:46 21 So, depending on when you listen to the  
09:38:49 22 resonance, to the echo, you can get -- highlight  
09:38:55 23 different aspects of the brain. So if you listen  
09:38:59 24 early, you'll see a lot of water. If you wait a little  
09:39:02 25 bit, you'll get a different kind of contrast. So what

09:39:05 1 is nice about MRI is with the same excitation, the same  
09:39:10 2 pulse, you can get more than one picture of the brain,  
09:39:13 3 each highlighting a different aspect of it.

09:39:17 4 So, we took advantage of that feature and you  
09:39:22 5 can see here is an image that is called a T2 weighted  
09:39:27 6 image where the fluid appears as white, and this is a  
09:39:36 7 proton density image, and when you combine those, you  
09:39:42 8 can get a very clear separation of values. So if you  
09:39:52 9 see a photograph and you try to blow it up and blow it  
09:39:58 10 up and blow it up, you may have encountered this  
09:40:01 11 phenomenon of pixellation. You will start seeing the  
09:40:06 12 dots that make the picture. In MRI, those are -- this  
09:40:09 13 is the picture. So each dot is really dictated by the  
09:40:13 14 computer telling, "I'm seeing something in this value."  
09:40:18 15 And the higher the value, the brighter the spot.

09:40:22 16 So we know the dimensions of each of those  
09:40:28 17 picture elements because we determine them. And we  
09:40:31 18 know -- so, these pixells have a known length and  
09:40:35 19 width. We also know that slice thickness. So these  
09:40:40 20 are not really completely two-dimensional because each  
09:40:46 21 slice has a thickness and we can determine that.

09:40:49 22 So this -- for example, these slices are five  
09:40:53 23 millimeters thick. We can now make one millimeter  
09:40:58 24 slices. Again, it depends on the scanner and what you  
09:41:03 25 can do with it. So, we are dealing with

09:41:09 1 three-dimensional pixells that are called voxels. So  
09:41:13 2 now when you know a voxel's dimension, once you let the  
09:41:18 3 computer determine which voxel is in the range of gray  
09:41:24 4 matter, white matter and cerebral spinal fluid, you can  
09:41:30 5 -- and the computer essentially goes, scans through the  
09:41:34 6 MRI and takes each picture element and throws it into  
09:41:38 7 one pile, either gray matter or white matter or  
09:41:44 8 cerebral spinal fluid, which is called CSF. And, of  
09:41:52 9 course, the rest of it, which is skin and bone, you  
09:41:55 10 throw out -- you can throw away, except in this case it  
09:42:02 11 was the skin and bone of our esteemed chairman of  
09:42:06 12 radiology, Nick Bryan.

09:42:09 13 So if you get computer geeks together, they'll  
09:42:13 14 say, "Hey, we can do something with that, too." And it  
09:42:16 15 turns out that you can actually -- very expensive  
09:42:21 16 photograph, but it's based on the MRI, on Nick's MRI.

09:42:26 17 So, now when you can throw the voxels into  
09:42:31 18 piles of gray matter and white matter you can calculate  
09:42:35 19 the volume of gray matter and white matter and cerebral  
09:42:40 20 spinal fluid in the brain. And this is where Adolph  
09:42:43 21 Pfefferbaum and his group at Stanford have seen two  
09:42:46 22 phenomenon.

09:42:49 23 One is that white matter increases as you grow  
09:42:56 24 in age from zero to 30. The volume of white matter  
09:43:02 25 goes up. The volume of gray matter -- gray matter are

09:43:07 1 the nerve cells, themselves, that we think, other than  
09:43:11 2 the axons. Gray matter goes down. And that's how the  
09:43:18 3 new principal of pruning was discovered. That brain  
09:43:27 4 development involves two complimentary processes. One  
09:43:33 5 is myelination, which is the laying down of the  
09:43:38 6 shielding for the axons. And the other is pruning,  
09:43:41 7 which is actually death of neurons. And pruning, it  
09:43:46 8 turns out, is as important as myelination, similar to  
09:43:52 9 the way it's important in raising a tree. This is not  
09:43:56 10 a trivial analogy because we are dealing with living  
09:44:01 11 organisms, and there are some principals that apply  
09:44:05 12 across.

09:44:05 13 So, if you have a brain region that you haven't  
09:44:09 14 used by age of 10 or 12, then there is probably no  
09:44:14 15 reason to keep it there, and it shrivels off. At least  
09:44:19 16 that's what your biology thinks. And that's as  
09:44:23 17 important as pruning because it makes sure that your  
09:44:26 18 brain works better as a whole and the information  
09:44:30 19 doesn't go to areas that are really not used. But  
09:44:34 20 that's also part of the reason for why it's important  
09:44:36 21 to do certain things while you grow up because if you  
09:44:40 22 didn't do them while you grow up, you will never do  
09:44:43 23 them. That part of the brain that can do them will be  
09:44:47 24 gone. Things such as music, things such as foreign  
09:44:50 25 languages and certain other skills. If you hadn't

09:44:57 1 really done it growing up, then you probably will never  
09:45:03 2 be able to do it exactly right. And, I mean, anybody  
09:45:08 3 who skis knows that you can be a terrific skier even if  
09:45:13 4 you learn to ski when you are old, like myself. I  
09:45:17 5 mean, the part about learning skiing when I was old.  
09:45:21 6 But I know people who have been excellent skiers when  
09:45:25 7 they learn it when they're old, you can still -- you  
09:45:30 8 can immediately see that they didn't learn it when they  
09:45:33 9 were children. Someone who learns skiing when they  
09:45:36 10 were children has an entirely different look on the  
09:45:40 11 slopes and, in fact, on the catwalks is really where  
09:45:43 12 you can tell them apart. It's really hard on those who  
09:45:46 13 learn it late.

09:45:47 14 But the point is that pruning is every bit as  
09:45:51 15 important as myelination in the process of brain  
09:45:54 16 development.

17 BY MR. DAWSON:

09:45:56 18 Q. Dr. Gur, what in terms of the period of time  
09:45:58 19 when this takes place, the myelination and the pruning,  
09:46:01 20 what ages are we looking at that this is occurring?

09:46:05 21 A. Yeah. Well, it turns out that both processes  
09:46:08 22 are about complete at age 21. Between 21 and 22, which  
09:46:13 23 is, I think, quite remarkable because that's what we  
09:46:17 24 picked as an age of maturity for a lot of things. So  
09:46:25 25 that is probably closest to the biological age of brain

09:46:27 1 maturity.

09:46:30 2 Q. Dr. Gur, also, is the brain more susceptible  
09:46:34 3 to damage during this period than it is later in terms  
09:46:37 4 of various insults that it might receive?

09:46:40 5 A. Well, there is a principal in medicine which  
09:46:43 6 is that always the most dangerous time is time of  
09:46:48 7 change because things are in flux and you can easily  
09:46:54 8 derail them. They stand on the balance and every  
09:46:58 9 little thing can derail.

09:47:00 10 Now, I always run into that with  
09:47:06 11 pediatricians. Pediatricians are always optimistic and  
09:47:12 12 they keep telling parents, you know, it's the child,  
09:47:16 13 he'll grow out of it. Everything will be fine. And to  
09:47:20 14 some extent there is a point there because the  
09:47:24 15 developing brain is more flexible, more pliable, can go  
09:47:29 16 into different directions. And so it can overcome  
09:47:33 17 difficulties and has more resources to overcome  
09:47:37 18 difficulties.

09:47:38 19 So, a child may pull out of something that an  
09:47:41 20 adult may not pull out of. But, still, if there is  
09:47:46 21 damage, a disruption of brain development, that the end  
09:47:52 22 result is going to be misshapen, is going to be  
09:47:57 23 something wrong. The same way that you can have a tree  
09:48:01 24 that was hit by lightning at a young age has overcome  
09:48:08 25 it and grows around it, but it's never really a normal

09:48:12 1 tree. You can see it can be big, it can be all that,  
09:48:15 2 but you can see those kinds of rough spots that it will  
09:48:19 3 never, never overcome. An older tree may never have  
09:48:23 4 survived it.

09:48:24 5 So, the question is of -- what is the impact  
09:48:29 6 of damage? In some ways, an impact of damage is worse  
09:48:33 7 when you're growing up because it's hitting a  
09:48:35 8 developing brain, in some respects, is better because  
09:48:42 9 you have enough resources to deal -- to deal with the  
09:48:47 10 illness.

09:48:50 11 Q. Dr. Gur, is the scientific basis of the brain  
09:48:55 12 imaging as you have discussed it, is it accepted in the  
09:49:00 13 scientific community?

09:49:01 14 A. Everything that I have said so far is  
09:49:03 15 standard, textbook stuff, really.

09:49:07 16 Q. And, Dr. Gur, if you would, what then -- how  
09:49:12 17 did you apply this information to an analysis of Byron  
09:49:18 18 Black?

09:49:18 19 A. Okay. Okay. I mean, just this one shoes in  
09:49:26 20 adults this process continues, and you can see gray  
09:49:31 21 matter. This is adults, age 18 to 45. So, even within  
09:49:36 22 that age range you can see gray matter is going down.  
09:49:41 23 This is for men. This is women. And white matter  
09:49:47 24 stays about the same, but cerebral spinal fluid goes  
09:49:51 25 up, and the reason cerebral spinal fluid goes up is

09:49:55 1 that every time a brain cell dies, its place is taken  
09:49:59 2 up by fluid.

09:50:01 3 The cerebral spinal fluid is very important  
09:50:03 4 because it surrounds the brain, around the brain and  
09:50:06 5 inside the brain, and it serves both as a source of  
09:50:10 6 nutrients, although the blood is what supplies most of  
09:50:14 7 the nutrients. But, most importantly, it cushions the  
09:50:19 8 brain against blows. I think the back of the brain is  
09:50:23 9 quite smooth, but in the front you have a lot of nasty  
09:50:27 10 bones, or bits in the nose. So, if the brain is thrown  
09:50:30 11 against them it can be damaged, and if you didn't have  
09:50:35 12 cerebral spinal fluid you could damage your brain  
09:50:37 13 everytime you sneeze.

09:50:38 14 Q. Dr. Gur, what is the ability of the brain to  
09:50:40 15 develop new cells or new neurons, I mean, once you're  
09:50:45 16 born?

09:50:46 17 A. So when I went to school, graduate school, I  
09:50:49 18 was taught that it's zero. There is no regeneration of  
09:50:55 19 nerves. Once a nerve cell dies, it won't -- no new one  
09:51:02 20 will come. And the reason for that is that in order to  
09:51:06 21 build cells you need proteins, which are big molecules,  
09:51:11 22 and there is a barrier, it's called the blood brain  
09:51:15 23 barrier, that doesn't allow large molecules to get into  
09:51:19 24 the brain. So sugar can get in, you know, all those  
09:51:25 25 small molecules, very tiny proteins may get in, but the

09:51:29 1 big proteins you need to build cells don't get in.

09:51:34 2 So, in a muscle, if you kill muscle, for  
09:51:40 3 example when you lift weights, the key is to bring  
09:51:45 4 yourself to muscle failure. Muscle failure means that  
09:51:49 5 your muscles are suffocating and dying and that's when  
09:51:53 6 you feel like you couldn't lift it one more time. If  
09:51:56 7 you can lift it that one more time and you kill enough  
09:52:00 8 muscle cells, that will trigger the growth of new  
09:52:03 9 cells. And this is how you build muscle. So if you  
09:52:07 10 don't reach muscle failure, you really don't build your  
09:52:11 11 muscle, you only improve the tone of your muscle. To  
09:52:14 12 build muscle you have to kill it.

09:52:16 13 In the brain, if you did that, the neurons  
09:52:21 14 will die but no neurons will be built because there  
09:52:25 15 isn't the building blocks.

09:52:27 16 Now, in recent years there have been some  
09:52:30 17 studies suggesting that this is not entirely true.  
09:52:34 18 That you can get regeneration, much more so than we  
09:52:40 19 ever thought was possible. But, still, it's nothing  
09:52:44 20 like what can be done with muscle. The best we can  
09:52:48 21 hope is that some, a little bit regeneration can take  
09:52:52 22 place.

09:52:54 23 Q. And, Dr. Gur, then what did you do in Byron  
09:53:00 24 Black's case? What was the methodology that you used  
09:53:04 25 there?

09:53:05 1 A. I used exactly the same methodology. I can  
09:53:12 2 actually cover the -- I did put two more -- I can  
09:53:17 3 cover them very quickly.

09:53:18 4 Q. Okay.

09:53:19 5 A. This one is from our study in collaboration  
09:53:23 6 with the Japanese group. They were able to get brains  
09:53:27 7 of babies as soon as they were born and those -- they  
09:53:31 8 sent those brains to us, I mean, electronically. And  
09:53:35 9 we analyze the data, and you can see how the brain  
09:53:40 10 myelinates. And the myelination, in this case, will be  
09:53:46 11 reflected in the increase of this gray area. You can  
09:53:50 12 see that as a baby, there is very little gray. There  
09:53:54 13 is some growth of the brain. Most of the growth is  
09:53:57 14 because this white matter is being added here. And in  
09:54:02 15 a 20 -- and you can see, also, that first these regions  
09:54:06 16 get myelinated in the middle, and the last regions,  
09:54:10 17 really, to become myelinated are right here in the  
09:54:13 18 front of the brain, the frontal lobe, and that is  
09:54:16 19 important to bear in mind.

09:54:19 20 And this is a summary. This shows the effect  
09:54:21 21 of pruning. This is the volume for the whole brain.  
09:54:26 22 But look at the frontal lobe and the temporal lobe, in  
09:54:31 23 particular. The blue are babies less than two years,  
09:54:34 24 the green are babies more than two years, and the red  
09:54:37 25 are adults. You can see that for the frontal lobe,

09:54:41 1 that for gray matter the babies, the children between  
09:54:45 2 24 months and 10 years have more gray matter than  
09:54:49 3 either newborn or adults. So, you lose gray matter in  
09:54:55 4 that age.

09:54:56 5 White matter, on the other hand, even those  
09:55:00 6 have not yet reached the adult level of white matter.  
09:55:04 7 In the temporal lobe, you can see that, that there is,  
09:55:11 8 in fact, not that much of pruning as in the frontal and  
09:55:20 9 the myelination is nearly complete by age 10. In the  
09:55:25 10 right hemisphere is the same value as adult, and in the  
09:55:29 11 left it's a little bit lower. So, those arrow bars  
09:55:33 12 indicate how confident you can be in the meat, how much  
09:55:37 13 slack there is in the meat. So, these are big, big  
09:55:42 14 effects.

09:55:42 15 And, finally, my colleague, Sowell, et al,  
09:55:48 16 had, based on their data, made this pictorial  
09:55:52 17 representation that shows what are the regions -- what  
09:55:56 18 is the rate of myelination of regions, and again, they  
09:55:59 19 show the same phenomenon. That the frontal cortex is  
09:56:03 20 the last to get myelinated.

09:56:05 21 And now we come to Mr. Black.

09:56:07 22 Q. Before we get to Mr. Black, let me ask you, in  
09:56:09 23 terms of the frontal lobe being the last to myelinate,  
09:56:14 24 what does that do in terms of -- or what does that  
09:56:17 25 show, I guess, in the way that humans develop certain

09:56:21 1 abilities? I mean, what is controlled by that frontal  
09:56:24 2 lobe and what does the late myelination mean?  
09:56:27 3 A. If I had to use one word to describe the role  
09:56:36 4 of the frontal lobe based on everything we know from  
09:56:39 5 human and animal studies, the word would be executive  
09:56:48 6 functions. And what we mean by executive functions,  
09:56:54 7 being able to abstract a principal out of instances and  
09:57:03 8 apply that principal to your behavior in a way that is  
09:57:08 9 adaptive with respect to the context of the behavior.  
09:57:15 10 And different parts of the frontal lobe approach that  
09:57:20 11 very same task from a slightly different angle. So  
09:57:24 12 there is a part that is called the dorsal lateral  
09:57:29 13 prefrontal region, and that is very narrowly looking at  
09:57:34 14 your so-called scratch pad.

09:57:37 15 So right now as you're listening to me, you  
09:57:41 16 know where you are. What is the context of finding  
09:57:45 17 yourself in such a nice day getting a lecture on brain.  
09:57:52 18 You remember what I just told you and you are ready to  
09:57:57 19 hear what I'm about to say. That's what the dorsal  
09:58:00 20 lateral prefrontal cortex does.

09:58:03 21 So, if I wanted to get it activated in a  
09:58:06 22 functional imaging study, I will ask a subject, give a  
09:58:10 23 subject a task. For example, I'm now going to give you  
09:58:15 24 a series of numbers and I want you to push a button  
09:58:21 25 every time a number that you hear is the same as the

09:58:23 1 number before the number before that one. Okay? Now,  
09:58:29 2 so if you want to get your dorsal lateral prefrontal  
09:58:34 3 grinding, try that with someone and have someone read  
09:58:39 4 to you a list of numbers and try to do that because you  
09:58:42 5 constantly have to remember not just the one that you  
09:58:45 6 just heard, but the one before. And then when the next  
09:58:50 7 one comes, you have to remember this is -- I have to  
09:58:52 8 hit and now scratch the old one and remember the one  
09:58:55 9 inbetween that happened. So if it now happens again, I  
09:58:58 10 have to hit the button.

09:58:59 11 But that's the most fundamental part of the  
09:59:02 12 frontal lobe. And you can see how that introduces more  
09:59:05 13 complex functions to the frontal lobe which is, again,  
09:59:11 14 to understand the context of your behavior and make it  
09:59:15 15 adaptable to that context and in line with your future  
09:59:22 16 goals.

09:59:22 17 So abstraction, mental flexibility, attention,  
09:59:27 18 being able to select what aspect of the environment you  
09:59:30 19 are going to focus on because there are lots of things  
09:59:34 20 happening at any moment. My voice, you may be hungry,  
09:59:37 21 you maybe uncomfortable, hot, worrying about, you know,  
09:59:43 22 the meter running out on your car, and yet you're able  
09:59:47 23 to put all that aside and just listen to me. That's  
09:59:50 24 frontal lobe. And that, also, is the part of the brain  
09:59:55 25 that shows the biggest increase relative to apes and

10:00:01 1 certainly relative to lower species. So the frontal  
10:00:05 2 lobe is what really, many have argued, make us uniquely  
10:00:10 3 human in the sense that we can behave in a larger  
10:00:16 4 context. So we can behave in even considering the  
10:00:20 5 context of our society, of the past, of things that  
10:00:25 6 happened to our nation of things we hope will happen to  
10:00:29 7 our nation. No other animal can think in those terms,  
10:00:33 8 and this is what the frontal lobe contributes to the  
10:00:38 9 human mental abilities.

10:00:41 10 Q. Dr. Gur, now I think we can go to your test  
10:00:45 11 with Mr. Black.

10:00:45 12 A. Okay. So, the first graph here shows the  
10:00:55 13 volume of the entire cranium. So we took the gray  
10:01:00 14 matter, white matter and CSF and everything together.  
10:01:04 15 And this is a sample of -- here you see a sample of 79  
10:01:12 16 healthy men. These are all volunteers for research.  
10:01:16 17 And we have a large program of recruiting volunteers,  
10:01:20 18 and we screen them carefully. So this is the average,  
10:01:26 19 and the blue arrow -- blue bars are standard  
10:01:32 20 deviations. So, really, anything that is away from  
10:01:36 21 this is suspicious and abnormal.

10:01:40 22 As you can see, Mr. Black has a normal size of  
10:01:44 23 head. The whole cranium is a normal size and normal  
10:01:49 24 size of brain. But when you look at cerebral spinal  
10:01:57 25 fluid, circle CSF is normal, but ventricular CSF is

10:02:05 1 hugely abnormal. In fact, it's twice the normal  
10:02:12 2 volume.

10:02:12 3 So to give you an idea, I first thought it was  
10:02:16 4 a mistake. Something went bad with the software  
10:02:21 5 because I have never seen such values. Frankly, I got  
10:02:25 6 the value for each hemisphere and I thought that, by  
10:02:34 7 mistake, it put both hemispheres in each and here --  
10:02:40 8 so, I went and dug up the healthy subject that had the  
10:02:45 9 largest ventricles, ventricular volume in our entire  
10:02:51 10 sample, and it turns out that I know exactly who that  
10:02:55 11 person is, and this is his ventricles. Can you see?  
10:03:06 12 This is his ventricles, and this is Mr. Black's  
10:03:10 13 ventricles in exactly the same slice.

10:03:13 14 Q. Dr. Gur, if you would, I think again in the  
10:03:16 15 documents that were placed on the edge of the desk,  
10:03:18 16 there should be the four pictures, the two on the top  
10:03:21 17 and two on the bottom, that are in that group. If you  
10:03:25 18 would find those so we can --

10:03:27 19 A. Yes.

10:03:35 20 MR. DAWSON: And then, if we could have  
10:03:37 21 that placed in as the next -- the picture -- the one  
10:03:43 22 being the normal person's brain image in that group, if  
10:03:49 23 you could hand that to the court reporter and we would  
10:03:51 24 ask that that be the next numbered exhibit to this  
10:03:55 25 deposition.

10:03:55 1 Is that the other one?

10:03:57 2 THE WITNESS: That's the normal.

10:04:26 3 MR. DAWSON: And then if the one that was  
10:04:29 4 identified as Mr. Black's, if that could be the next  
10:04:31 5 numbered exhibit, please.

6 (Document marked Exhibit No. 3 and 4.)

7 BY MR. DAWSON:

10:05:04 8 Q. Okay. Dr. Gur, now if you would, continue,  
9 then.

10:05:08 10 A. So, this is what I showed you before. This is  
10:05:12 11 the T2 image that highlights -- you can see that here  
10:05:18 12 the T1 image is -- the CSF is black. In the T2 image,  
10:05:26 13 the CSF is white. All right?

10:05:28 14 So, this is a better contrast to see cerebral  
10:05:33 15 spinal fluid. And here, this is an average man. We  
10:05:38 16 took this brain exactly from the -- from the man who  
10:05:43 17 gave us the average values across the board. These are  
10:05:51 18 his ventricles in the T2. These are Mr. Black's  
10:05:55 19 ventricles. And I couldn't get exactly the same slice,  
10:06:01 20 but these are -- these are the two closest slices I  
10:06:04 21 could find.

10:06:06 22 MR. DAWSON: Dr. Gur, if the one on the  
10:06:08 23 left, the first one that you pointed to on the bottom,  
10:06:12 24 the white, if that could be introduced then as the next  
10:06:15 25 numbered exhibit.

10:06:16 1 THE WITNESS: Yeah.

10:06:17 2 MR. DAWSON: And the final one, then, as  
10:06:20 3 the next exhibit. So the next two exhibits, please.

4 (Documents marked Exhibit Nos. 5 and 6.)

10:07:36 5 THE WITNESS: We were so trained by  
10:07:38 6 HIPPA, that we can't even say a patient's name to the  
10:07:42 7 patient. It's really become a --

10:07:46 8 BY MR. DAWSON:

10:07:46 9 Q. Dr. Gur, then, continue on then as to what  
10:07:50 10 this shows us about Mr. Black.

10:07:52 11 A. Now, let me just first conclude the anatomic  
10:08:02 12 findings.

10:08:05 13 Q. All right.

10:08:05 14 A. I'm sorry, were you asking me to --

10:08:07 15 Q. Again --

16 A. What does it mean?

10:08:07 17 Q. If you can explain, briefly, what this  
10:08:09 18 finding, in terms of the size of the --

10:08:12 19 A. The ventricles?

10:08:14 20 Q. -- the ventricles, what does that mean in  
10:08:17 21 terms of where you are on this particular slide?

10:08:20 22 A. Well, what it means, literally, is that there  
10:08:29 23 -- a lot of cells died there, right in the middle of  
10:08:33 24 the brain. If the cells die on the side, you get what  
10:08:38 25 is called sulcal atrophy, and you get that in certain

10:08:43 1 conditions. Ventricular atrophy is something that you  
10:08:48 2 get in several disorders. It's something that happens  
10:08:54 3 early during gestation. Large ventricles are a  
10:09:08 4 cardinal sign of schizophrenia, but appear in mental  
10:09:09 5 retardation and various forms of cerebral dystrophy or  
10:09:19 6 atrophy related disorders. Particularly when the  
10:09:24 7 damage is in regions that are so-called limbic, in the  
10:09:30 8 center of the brain.

10:09:35 9 I have really -- I mean, I have seen literally  
10:09:40 10 thousands of MRI's. I have not seen that size  
10:09:47 11 ventricle. So it's probably not one single factor that  
10:09:53 12 produced that.

10:09:56 13 BY MR. DAWSON:

10:09:56 14 Q. So if I understand it, you talked about  
10:09:59 15 atrophy and I think you said that means the cells have  
10:10:03 16 died?

10:10:03 17 A. Yes. It could also be dystrophy, which means  
10:10:06 18 that cells were never there.

10:10:08 19 Q. So does this tell us where the cells died, or  
10:10:16 20 just that a lot of cells have died within the brain?

10:10:19 21 A. It only tells us that a lot of cells died and,  
10:10:22 22 roughly, it tells us that because it's ventricular  
10:10:26 23 atrophy rather than sulcal atrophy, it tells us that  
10:10:31 24 those medial structures of the brain suffered the  
10:10:37 25 damage rather than the more, what you call the lateral

10:10:42 1 aspects.

10:10:43 2 Q. And just so that -- so, when you look at these  
10:10:46 3 pictures and we see what you have indicated as a normal  
10:10:51 4 brain but on the large end of the ventricle for the  
10:10:53 5 normal brain?

10:10:53 6 A. Yes.

10:10:56 7 Q. This is the first picture of this group. And  
10:10:56 8 that would indicate that that person, at whatever age,  
10:11:01 9 that that's the -- they had a normal loss of brain  
10:11:04 10 tissue throughout life, or whatever got them to that  
10:11:09 11 point?

10:11:10 12 A. Well, we also see large ventricles in people  
10:11:14 13 who are at risk for schizophrenia. For example, people  
10:11:20 14 who have schizophrenia in their family and that normal  
10:11:27 15 ventricle is like that, is -- has that sort of an  
10:11:37 16 appearance. So these are people who are functioning  
10:11:40 17 fine. They don't suffer from schizophrenia, or the  
10:11:45 18 disorder. If you get to know them, you realize that  
10:11:50 19 they're a little off. They may have a hard time  
10:11:56 20 forming interpersonal relationships, they may have some  
10:12:02 21 bizarre thoughts and habits. We have a term called  
10:12:09 22 schizotypal. So, someone who can be fine but just  
10:12:13 23 somehow doesn't -- doesn't manage to have it all  
10:12:18 24 together.

10:12:22 25 Q. So that the first brain, the one that we

10:12:25 1 showed that was somewhat normal, may have some  
10:12:28 2 tendencies towards schizophrenia, but that would be the  
10:12:33 3 result of normal pruning within the brain, perhaps  
10:12:36 4 normal number of injuries that someone may suffer  
5 during life to the brain without doing any major harm.  
6 Is that basically where we are heading? Where we are?  
10:12:42 7 A. Yes.  
10:12:42 8 Q. And in terms of Mr. Black's brain, this would  
9 indicate a far more, either failure to develop of  
10:12:47 10 tissue or else the death of cells in his brain than you  
10:12:54 11 would expect?  
10:12:55 12 A. Correct. Correct. It's far beyond what you  
10:12:58 13 would see in schizophrenia.  
10:13:00 14 Q. And then were there other tests done? This  
10:13:02 15 was found through an MRI; correct?  
10:13:05 16 A. Correct.  
10:13:05 17 Q. And, Doctor, if you would, I think, again in  
10:13:07 18 that stack of documents there was a report that you  
10:13:11 19 sent this past week which reads, "Summary of qMRI,  
10:13:18 20 Quantitative Analysis For Mr. Byron Black."  
10:13:21 21 A. That incorporates, also, the next couple  
10:13:24 22 slides I was going to show you.  
10:13:25 23 Q. Okay. Why don't we go to the slides and then  
10:13:27 24 we'll come back to the report.  
10:13:28 25 A. Okay. So, the next stage in analyzing the

10:13:31 1 data is called parcellation. After we segment the  
10:13:35 2 brain into gray matter, white matter and cerebral  
10:13:39 3 spinal fluid, we try to see what is the volume of  
10:13:43 4 different brain structures. And that is not easy.  
10:13:49 5 It's far from trivial because while every person has a  
10:13:56 6 brain, and the brains, if you look at them, basically  
10:13:59 7 have the same structure. The degree of variability is  
10:14:04 8 still about the same, if not more than variability in  
10:14:09 9 faces. You can say everybody has the same face,  
10:14:13 10 unmistakably two eyes, a nose, a mouth and two ears,  
10:14:18 11 unless somebody had an accident and one of those got  
10:14:20 12 injured. But still we can see there is a big  
10:14:25 13 difference. And if I try to make one face out of all  
10:14:28 14 of your faces, it will look very strange and this is,  
10:14:31 15 unfortunately, what we have to do. We have to take  
10:14:34 16 each brain and find out where is a particular  
10:14:40 17 structure, trying to extract from our knowledge of many  
10:14:45 18 brains.

10:14:45 19 And, more recently, methods have been  
10:14:48 20 developed to do that and these are called deformation  
10:14:53 21 -- this methodology is called deformation based  
10:14:58 22 morphometry, or DBM. And the idea is that it uses  
10:15:08 23 complex mathematics. But it would take a template, it  
10:15:12 24 can be any -- any arbitrarily chosen brain you can take  
10:15:18 25 as a template. But we take a template of a healthy

10:15:22 1 brain. And then you take any brain, all the other  
10:15:27 2 brains, and try to warp them. Literally, try to squish  
10:15:32 3 and stretch and make that brain fit. Except that when  
10:15:36 4 you are done, you still remember what you had to do to  
10:15:39 5 get there.

10:15:40 6 And so you can put a value on each region  
10:15:44 7 that tells you how much did I have to stretch and how  
10:15:47 8 much did I have to squoosh, or squeeze that region in  
10:15:54 9 order to fit the template. And based on that, you can  
10:15:58 10 calculate the volume of each part of the brain and how  
10:16:03 11 much it is smaller or bigger than what it's supposed to  
10:16:10 12 be than a template.

10:16:11 13 So what my colleague, Christos Davatzikos, has  
10:16:16 14 done, he's among the pioneers of this methodology and  
10:16:21 15 he applied these tools to Mr. Black's brain and has  
10:16:29 16 found that -- I mean, first of all, he ran an automated  
10:16:35 17 classifier. Once you are done with that process, you  
10:16:38 18 can ask the system, if I give you -- here are 79 brains  
10:16:42 19 of healthy people, here are 69 brains of people with  
10:16:47 20 schizophrenia, and here's the brain of Mr. X. Where do  
10:16:51 21 they belong? And the classifier will try to fit into  
10:16:57 22 normal and give you a probability that this is a normal  
10:17:02 23 brain and try to fit into schizophrenia and give you a  
10:17:08 24 probability it's a schizophrenia brain. And usually  
10:17:10 25 you get sort of equivocal results. It can say --

10:17:16 1 chances, yeah, it could be schizophrenia. Chances are  
10:17:20 2 maybe 15 percent. It could be normal, chances are a  
10:17:26 3 little bit better, 40 percent. So you have to decide,  
10:17:27 4 well, all right.

10:17:28 5 In the case of Byron Black, that program  
10:17:31 6 basically bombed. Not normal, not schizophrenic. I  
10:17:37 7 don't know. It's just abnormal. And that is reflected  
10:17:43 8 in the volumetric measures. And you can see, this is  
10:17:46 9 the ventricles, for example. But when you look at the  
10:17:51 10 actual volume, it turns out that the regions that show  
10:17:53 11 the most significant decline are parts of the frontal  
10:18:00 12 lobe that includes the inferior frontal, the medial  
10:18:05 13 frontal, orbital frontal and the anterior cingulate  
10:18:13 14 gyrus. And these are highly significant regions  
10:18:18 15 because this is essentially the system that controls  
10:18:26 16 basically aggression, but also modulates other  
10:18:32 17 impulses.

10:18:34 18 The way it works, this is a three-dimensional  
10:18:39 19 rendering, looks better on my screen than here, but  
10:18:45 20 it's a three-dimensional rendering of the regions that  
10:18:49 21 had to be squished or expanded in order to fit into a  
10:18:54 22 normal brain, and these are the regions that really had  
10:18:58 23 to be expanded. This is a view of the bottom of the  
10:19:01 24 brain, as if you took it out and looked from the  
10:19:03 25 bottom. And this is a view from the top. So you can

10:19:06 1 see that it's the frontal -- the whole bottom of the  
10:19:09 2 frontal lobe and the -- and the so-called orbital part  
10:19:15 3 of it is missing, is really seriously small. Too small  
10:19:25 4 in Mr. Black.

10:19:27 5 Q. So, let's go back to that, if we may. So, what  
10:19:35 6 we're looking at here, then, is what would be the light  
10:19:38 7 gray matter, is the part that is no longer healthy  
10:19:43 8 brain cells that are no longer functioning, is that --

10:19:46 9 A. Not entirely. But this is the part that is  
10:19:51 10 shrunk in Mr. Black.

10:19:55 11 Q. Okay. So if we can go back --

10:19:56 12 A. There could be cells there -- I mean, there  
10:19:57 13 are cells there, there are millions of cells there, but  
10:20:00 14 this is not as many as you supposedly should have.

10:20:02 15 Q. And if we can go back, then, and again mark  
10:20:06 16 exhibits that are in the group of exhibits. I believe  
10:20:08 17 the first slide you had that had the two -- the two  
10:20:14 18 graphs on it, is actually made up of --

10:20:17 19 MR. DONNELLY: Right here, Doctor.

10:20:18 20 THE WITNESS: Yes.

10:20:19 21 MR. DAWSON: -- is actually made up of  
10:20:21 22 two separate pages, but they're no longer --

10:20:25 23 THE WITNESS: Yeah. I combine some for  
10:20:28 24 the purpose of this.

10:20:28 25 MR. DAWSON: Right.

10:20:28 1 THE WITNESS: You want this, right?

10:20:29 2 MR. DAWSON: Yes. That would be the  
10:20:36 3 next.

10:20:36 4 THE WITNESS: Then the next would be  
10:20:38 5 these two, these three.

10:20:40 6 MR. DAWSON: The brain and the CSF, I  
10:20:41 7 guess, is our next exhibit.

10:20:43 8 THE WITNESS: Yeah. So this is the next  
10:20:45 9 and then these.

10:20:45 10 MR. DAWSON: That's correct.

11 Why don't we do the one that has Brain and CSF  
12 as the next exhibit, and then the next three as a  
13 collective exhibit?

14 THE WITNESS: Well, I could tell you, why  
15 don't I just then put the whole report from --

16 MR. DAWSON: That would be fine.

10:21:16 17 THE WITNESS: -- Mr. Davatzikos?

10:21:16 18 I think -- I think it's worth because he doesn't --

19 he's a computer scientist. He doesn't know who Black

10:21:27 20 is, what it is. He's just getting a brain. Tell me

10:21:28 21 what you see. And some of the way he tries to explain

10:21:32 22 it is, I think, worth reading.

10:21:36 23 MR. DAWSON: So --

10:21:37 24 THE WITNESS: I mean, one point that he

10:21:39 25 makes very well is, for example, if you look at these,

10:21:45 1 you cannot consider each comparison in isolation. And  
10:21:51 2 say that, that if none of the regions deviates from  
10:22:00 3 normal, then it's normal.

10:22:05 4 In other words, if you see that they deviate,  
10:22:08 5 certainly it's abnormal, but the brain can be abnormal  
10:22:12 6 even if no individual region deviates because the  
10:22:16 7 relative size of different regions can be abnormal.  
10:22:20 8 And the example he gives is, it is normal to have a  
10:22:24 9 blue eye. It is normal to have a brown eye. It's  
10:22:28 10 abnormal to have both in the same person. So you can  
10:22:33 11 have all the individual regions being normal, but one  
10:22:38 12 of them is really -- should be much higher than the  
10:22:41 13 other, and it's not. That makes sense?

14 BY MR. DAWSON:

10:22:48 15 Q. So then, Doctor, let me ask you first, the  
10:22:50 16 work that Dr. Davatzikos conducts is something that you  
10:22:55 17 commonly rely on for the work that you do; is that  
10:22:58 18 accurate?

10:22:58 19 A. Yes.

10:22:59 20 Q. And Dr. Davatzikos' report goes with the three  
10:23:06 21 bar graphs; correct?

22 A. Correct.

10:23:09 23 MR. DAWSON: If we could, then, perhaps  
10:23:10 24 put it together with the three bar graphs, the colored  
10:23:13 25 bar graphs, the ones that are in yellow, green and blue

10:23:16 1 and make that the collective exhibit.

10:24:46 2 And then I think that the next exhibit, then,  
10:24:48 3 was the photos that you now have on the screen. That  
10:24:55 4 would then be Exhibit No. 9.

10:25:19 5 THE WITNESS: Where do you want to  
10:25:21 6 include my report because it refers to figures?

10:25:24 7 MR. DAWSON: Right. This would be the  
10:25:26 8 qMRI.

10:25:29 9 MR. ZIMMERMANN: We're off the record  
10:26:00 10 here.

11 (Recess taken.)

12 (Document marked Exhibit Nos. 7, 8 and  
10:26:02 13 9.)

10:26:02 14 BY MR. DAWSON:

10:26:03 15 Q. Dr. Gur, and then you have a report that I  
10:26:05 16 think is labeled, "Summary of qMRI Quantitative  
10:26:07 17 Analysis for Mr. Byron Black."

10:26:11 18 A. Yeah.

10:26:13 19 Q. And could you explain what that document is?

10:26:18 20 A. It describes the procedure for scanning and  
10:26:28 21 analyzing and offers an interpretation of the results.

10:26:34 22 Q. And what was your interpretation based upon?  
10:26:39 23 The MRI scanning that was done in Mr. Black's case?

10:26:44 24 A. I interpreted the MRI as being severely  
10:26:52 25 abnormal, both because of the unusual size of the

10:27:00 1 ventricles and the loss of tissue in the -- in the  
10:27:11 2 location of the loss of tissue which would have severe  
10:27:16 3 consequences for behavior. You can have same amount of  
10:27:21 4 damage in other parts of the brain and not have those  
10:27:27 5 severe consequences.

10:27:30 6 So, I mean, every part of the brain is  
10:27:35 7 important, as is every part of the body. It wouldn't  
10:27:43 8 survive all those years of having to struggle with a  
10:27:58 9 changing world if it was -- I mean, the appendix is  
10:28:02 10 probably the only part that you don't need anymore, and  
10:28:05 11 still is there. So, damage anywhere in the brain will  
10:28:10 12 have consequences, but this particular system is, is  
10:28:15 13 really what makes us, in some ways, uniquely -- unique  
10:28:25 14 animals. Unique in the animal world of being able to  
10:28:29 15 plan into the future, be able to consider the future in  
10:28:32 16 our actions, being able to inhibit an impulse to do  
10:28:40 17 something now because of the bigger picture, because of  
10:28:45 18 the context, because of our future plans. This is  
10:28:50 19 exactly the part of the brain that is most severely  
10:28:54 20 damaged in Mr. Black.

10:28:55 21 Q. Dr. Gur, let's go back a little bit.

10:28:58 22 In terms of the MRI that was done in this  
10:29:01 23 case, did you personally conduct the MRI, itself?

10:29:05 24 A. The MRI was performed at Vanderbilt, but I  
10:29:18 25 supplied the imaging parameters and was here to

10:29:26 1 supervise. And the study was done exactly as if it had  
10:29:31 2 been done at Penn. They have an excellent MR facility  
10:29:37 3 and they are very good about shimming and making sure  
10:29:40 4 that the studies are technically of high quality and we  
10:29:44 5 got excellent images.

10:29:47 6 Q. Now, when you say you provided the parameters,  
10:29:50 7 what does that mean?

10:29:51 8 A. Well, I mean if you do a clinical MRI,  
10:29:54 9 clinicians are not terribly interested in history.  
10:29:58 10 They have a problem they want to fix, they have HMOs on  
10:30:02 11 their back. They basically want to see is there  
10:30:05 12 anything that can threaten your life.

10:30:07 13 And so to do an MRI, a standard clinical MRI,  
10:30:12 14 they will do some slices through your brain and usually  
10:30:17 15 they will skip -- there's a -- so each slice is a  
10:30:21 16 certain thickness. And you can have the next slice  
10:30:25 17 right adjacent to it, or you can skip a little bit and  
10:30:29 18 have the next slice. You can slice five millimeters,  
10:30:32 19 skip five, slice another slice, skip five. And so it  
10:30:37 20 gives you fewer pictures, but that usually is enough  
10:30:41 21 because if there is a tumor, it's not going to show up  
10:30:44 22 on one slice. It will show up on another slice and  
10:30:48 23 will produce some changes that you can detect even if  
10:30:50 24 you happen to miss that particular slice.

10:30:52 25 If you have a stroke, similarly it will show

10:30:56 1 changes in several areas. These are the most important  
10:30:59 2 things you want to rule out, there is no tumor and  
10:31:02 3 there is no bleed, because these are things that will  
10:31:05 4 kill you right away.

10:31:06 5 Other abnormalities have very focal  
10:31:10 6 appearance. If you have multiple sclerosis, the MS  
10:31:14 7 plaques, you will be able to pick them up. You don't have  
10:31:17 8 to do the whole brain.

10:31:19 9 But in order to do the kind of analysis we are  
10:31:23 10 doing, volumetric analysis, we really need the entire  
10:31:27 11 brain. Otherwise, our results would be meaningless.  
10:31:30 12 So you have to specify that, the slice thickness, and  
10:31:34 13 there is no skip and you don't -- you have to do the  
10:31:38 14 whole volume of the brain and not just, you know, part  
10:31:42 15 of it. You have to supervise the orientation of the  
10:31:46 16 head in the scanner. I mean, we can re-orient it in  
10:31:51 17 any way we want, but the less re-orientation we need to  
10:31:55 18 do, the better. So you want to make sure they are  
10:31:58 19 scanned according to the suborbital line that,  
10:32:01 20 basically, defines the base of the brain. And you also  
10:32:08 21 specify what will be the echo times that you want to  
10:32:15 22 use, or that you want to have. What times do you want  
10:32:18 23 to listen to the echo, as I described earlier, to get  
10:32:23 24 the best contrast that you are interested in so that  
10:32:29 25 your algorithm will work smoothly.

10:32:33 1 Q. When you were making these decisions as to how  
10:32:36 2 to -- what parameters to use, did you have other  
10:32:41 3 information about Byron Black that was necessary to  
10:32:44 4 determine the type of MRI you wanted to run or the  
10:32:46 5 things that you were looking at? What other  
10:32:50 6 information did you have at that point?

10:32:52 7 A. For doing the specific analyses that I did, it  
10:33:03 8 would be the same parameters. I did nothing special  
10:33:03 9 for Black. If I had suspected certain -- certain  
10:33:09 10 things, I could order different, additional scans or  
10:33:14 11 additional sequences to highlight. I can give  
10:33:19 12 examples, but none of that was relevant.

10:33:23 13 Q. When you -- in your report, you indicate under  
10:33:27 14 your interpretation some consistencies with various  
10:33:35 15 etiology?

10:33:36 16 A. Yes.

10:33:37 17 Q. How did you come about the possible etiologies  
10:33:43 18 in Mr. Black's case?

10:33:44 19 A. Well, you look in the literature and see what  
10:33:47 20 are conditions associated with large ventricles. And  
10:33:56 21 it turns out that probably the most likely condition is  
10:34:03 22 fetal alcohol syndrome, where there is quite a bit of  
10:34:08 23 evidence that the ventricles are enlarged to the point  
10:34:12 24 that the corpus callosum is grossly misshaped. The  
10:34:17 25 corpus callosum is right above the ventricles. Large

10:34:23 1 ventricles, as I mentioned, are also seen in  
10:34:26 2 schizophrenia.

10:34:29 3           You see, the brain doesn't organize itself by  
10:34:37 4 DSM-IV criteria. You can have a disease and it will  
10:34:41 5 impact behavior and you can have another disease, if it  
10:34:45 6 is in the same region, it will impact behavior in the  
10:34:49 7 same way. So you can have symptoms of schizophrenia  
10:34:55 8 without having schizophrenia because you have a lesion  
10:34:59 9 in an area that, if you had schizophrenia, you would  
10:35:02 10 also have the lesion there. But you could have  
10:35:06 11 acquired that lesion for other reasons. Could have  
10:35:09 12 been a head injury, could have been a tumor growing  
10:35:12 13 there, it could be any toxins have reached that point.

10:35:19 14           So just looking at behavior, you can get  
10:35:28 15 really confused in cases of brain damage because if you  
10:35:33 16 didn't know about the brain, you would see symptoms of  
10:35:37 17 all sorts of different disorders and you try to  
10:35:44 18 pigeonhole an individual into the DSM-IV criteria and  
10:35:46 19 it just doesn't work.

10:35:59 20 BY MR. DAWSON:

10:35:59 21 Q.           What, in terms of this report, what did -- was  
10:36:07 22 there anything else that you used to suggest the  
10:36:11 23 possibilities as to what the etiology might be to this  
10:36:15 24 particular brain damage you were seeing in Mr. Black?

10:36:18 25 A.           Well, I was looking for a big head injury with

10:36:22 1 coma. That could be another condition. Would be -- I  
10:36:28 2 have seen big ventricles in people who have had injury  
10:36:31 3 and were in coma for a long time. Several months. But  
10:36:36 4 I didn't see any evidence for that in Mr. Black's  
10:36:41 5 record at any time.

10:36:44 6 So, I mean, the way you make a diagnosis is  
10:36:47 7 that you look at all the information that you have and  
10:36:55 8 you then say, well, what kinds of disorders would have  
10:37:00 9 that? And in the case of Mr. Black -- and then you end  
10:37:08 10 up naming the disorder, that if that is what happened,  
10:37:13 11 it explains everything. And if there is something you  
10:37:19 12 can do, you will treat it at that point. Or sometimes  
10:37:23 13 you say, "Well, we made a perfect diagnosis. We know  
10:37:27 14 what you have, but sorry, there is nothing we can do  
10:37:30 15 for you, other than monitor."

10:37:34 16 In the case of Mr. Black, I would probably say  
10:37:40 17 that fetal alcohol syndrome, plus a series of more  
10:37:44 18 minor head injuries would be very high on my  
10:37:53 19 differential. Probably be, what? If this were a  
10:37:56 20 clinical case, this is probably -- this would have been  
10:38:00 21 my conclusion. My diagnosis.

10:38:03 22 Q. And, Dr. Gur, if we could then make your  
10:38:06 23 report that's entitled, "Summary of qMRI Quantitative  
24 Analysis for Mr. Byron Black," the next exhibit,  
25 please.

10:41:00 1 (Document marked Exhibit No. 10.)

10:41:00 2 (Recess taken.)

10:41:00 3 VIDEOGRAPHER: Standby please. Tape Two

10:41:06 4 of the deposition. The time is 10:40 a.m.

10:41:08 5 BY MR. DAWSON:

10:41:09 6 Q. Dr. Gur, I believe you had also prepared a

10:41:13 7 Declaration in this matter back in 2001; is that

10:41:16 8 correct?

10:41:16 9 A. Yes.

10:41:17 10 Q. And is that a document that's also among those

10:41:20 11 papers that were -- if we can have that made the next,

10:41:31 12 which will be Exhibit 11?

10:41:34 13 A. If you want to keep it in the sequence of my

10:41:37 14 presentation, then first I'll talk about PET.

10:41:40 15 MR. DAWSON: Let's put that in and then

10:41:42 16 we'll come back to that. We can just put it in at this

17 point.

10:42:09 18 (Document marked Exhibit No. 11.)

10:42:09 19 BY MR. DAWSON:

10:42:10 20 Q. Dr. Gur, you had indicated a minute ago that

10:42:13 21 -- you state some of your conclusions that are in your

10:42:17 22 report that's now Exhibit 10 were a result of

10:42:21 23 information that you had learned about Byron Black and

10:42:24 24 about his background?

10:42:24 25 A. Yes.

10:42:25 1 Q. And that information is set out, is it not, in  
10:42:27 2 this report that's now Exhibit 11, as far as the  
10:42:31 3 materials that you had reviewed prior to basically  
10:42:36 4 getting started with Mr. Black?

10:42:38 5 A. Yes. Before we did the imaging, I did an  
10:42:45 6 evaluation of Mr. Black, all the records, and I  
10:42:49 7 evaluated him, myself, and tested him and came up with  
10:42:55 8 the conclusion that we should do imaging because this  
10:43:01 9 fellow has brain damage, in all likelihood.

10:43:03 10 Q. And that also then included reviewing some  
10:43:07 11 neuropsychological testing that had been done earlier  
10:43:11 12 on Mr. Black?

10:43:11 13 A. Correct.

10:43:12 14 Q. Dr. Gur, now if you would, if we could go to  
10:43:16 15 the next slide, there was also a PET scan done in this  
10:43:19 16 case?

10:43:19 17 A. Yes.

10:43:20 18 Q. And if you could, tell us what the PET scan  
10:43:23 19 was for and how that worked, please.

10:43:24 20 A. A positron emission tomography, or PET, is a  
10:43:29 21 method for measuring and imaging the rate of brain  
10:43:38 22 activity. It's a versatile method in that, depending  
10:43:44 23 on the ligand, you can measure different aspects of  
10:43:52 24 brain function. The most frequent use is in  
10:43:56 25 combination with a ligand called fluorodeoxyglucose,

10:44:02 1 and that liegand let's you measure the rate of glucose  
10:44:11 2 metabolism in the brain.

10:44:13 3 Now every time a neuron sends a pulse down  
10:44:17 4 that membrane, it needs energy in order to generate the  
10:44:23 5 new pulse. They get the energy from sugar. Sugar --  
10:44:28 6 if you burn sugar, you get heat, you get energy and  
10:44:34 7 that process of burning sugar, or glucose, is called  
10:44:39 8 glycolosis. In order to break the sugar, it needs  
10:44:44 9 oxygen, both of which are supplied through the blood.

10:44:50 10 Now, after the sugar is broken, the byproducts  
10:44:54 11 of that process are toxic to the cells and they would  
10:44:58 12 attack and kill the cell after feeding it, unless  
10:45:05 13 they're taken away. And that is also what the blood  
10:45:08 14 flow does. The blood flow brings the food and takes  
10:45:13 15 away the garbage.

10:45:15 16 So, in order to understand, in healthy people  
10:45:18 17 there is a very, what we call, tight coupling  
10:45:22 18 between blood flow and metabolism. When metabolism  
10:45:26 19 goes up, blood flow in that region goes up instantly,  
10:45:30 20 actually within two seconds of -- when you activate a  
10:45:33 21 certain area, within two seconds there is a rush of  
10:45:37 22 blood, fresh blood into that area to prevent cell  
10:45:41 23 death.

10:45:41 24 Now to make a complete assessment of someone's  
10:45:45 25 brain, you need to look there for both, rate of blood

10:45:49 1 flow and rate of metabolism, which we would have done  
10:45:54 2 at Penn. Vanderbilt, at that time, was not certified  
10:45:58 3 for doing blood flow studies, but they had tremendous  
10:46:03 4 experience. Dr. Kessler is among the world's most  
10:46:08 5 famous experts in PET, and they have an excellent  
10:46:13 6 facility. He's not that interested in studying blood  
10:46:18 7 flow, so never pushed it. But we couldn't do blood  
10:46:21 8 flow, but we could do an excellent PET scan with  
10:46:25 9 measuring glucose metabolism in Mr. Black.

10:46:32 10 These -- on these slides it's just an  
10:46:36 11 illustration of what you get with a PET scan. They  
10:46:40 12 look similar to MRIs, but they don't tell you anything  
10:46:47 13 about the structure. Even if you see a structure  
10:46:50 14 there, there is no structural data. These data  
10:46:54 15 indicate how much sugar was eaten by this region per  
16 minute.

10:47:00 17 And the way it works is you inject sugar into  
10:47:03 18 the subject, except it's not regular sugar, it's  
10:47:06 19 synthetic sugar that is made not to break down, and you  
10:47:11 20 attach positron emmitter to that sugar. So that sugar,  
10:47:18 21 that synthetic sugar, fools the blood brain barrier and  
10:47:24 22 goes in and fools the cell to think it is real sugar,  
10:47:28 23 and the cell gobbles it up. But then instead of  
10:47:32 24 phosphorilating, instead of breaking down, it keeps  
10:47:41 25 sending out those positrons, which are positively

10:47:42 1 charged particles.

10:47:43 2 As you know, positively charged particles, as  
10:47:48 3 soon as they collide with an electron, a negatively  
10:47:53 4 charged particle, they annihilate each other. And so  
10:47:58 5 out of that annihilation, there are two gamma particles  
10:48:04 6 that travel apart. And what's lucky for us is they  
10:48:08 7 travel 180 degrees apart from each other. So when we  
10:48:12 8 put detectors on both sides, we surround the brain with  
10:48:17 9 detectors and every time two detectors of opposite  
10:48:20 10 sides get hit at the same time, we know that there was  
10:48:23 11 sugar in that, in that line. That's how we can  
10:48:27 12 reconstruct a picture of the whole brain, telling us  
10:48:32 13 how much sugar went to each part.

10:48:34 14 So a healthy brain, this is when you're just  
10:48:37 15 looking at objects and you can see that there is  
10:48:41 16 increased activity in the visual area. The visual area  
10:48:47 17 is in the back of the brain. When you're listening,  
10:48:51 18 there is increased activity in the temporal lobe, which  
10:48:54 19 is where the ears, the auditory input goes, and so on.  
10:49:00 20 But this gives you an idea.

10:49:02 21 And this is the brain of a patient with  
10:49:05 22 epilepsy, and what you usually see is these are the  
10:49:10 23 temporal lobes and if the slice goes this time like  
10:49:12 24 that, it's a coronal slide. These are called axial  
10:49:20 25 slices. You can slice the brain axially, coronally or

10:49:25 1 sagittally. Chop it up in all sorts of ways. So, in  
10:49:32 2 epilepsy it's usually in the temporal lobe. And there  
10:49:37 3 is a region of hyperactivity and there is region of  
10:49:39 4 hypoactivity. And this is abnormal high and abnormal  
10:49:44 5 low metabolism is what can give rise to seizures.  
10:49:48 6 Q. And that the hyper is abnormally high?  
10:49:50 7 A. Correct.  
10:49:51 8 Q. And hypo is abnormally low?  
10:49:52 9 A. Abnormally low.  
10:49:55 10 Now, this is Mr. Black's brain. So you can  
10:50:09 11 appreciate, I just put -- we took each, the metabolic  
10:50:30 12 rate in each region and divided it by the metabolic  
10:50:34 13 rate for the whole brain. So you get a feel for the --  
10:50:38 14 for how the regions vary. So if the metabolism is  
10:50:42 15 average, then it's one. Above one is above average and  
10:50:47 16 below one is below average. And these are our healthy  
10:50:51 17 volunteers. And this is Mr. Black. And there is a  
10:50:57 18 star every time the value is significantly abnormal.  
10:51:03 19 And as you can see, this is not a normal brain. There  
10:51:09 20 are nearly as many regions with abnormal as with normal  
10:51:14 21 metabolism, and it's quite chaotic.  
10:51:19 22 Within that, you can see that there is  
10:51:21 23 particularly reduced metabolism in the orbital, frontal  
10:51:28 24 and rectal gyras and insula, which are the major  
10:51:32 25 frontal structures responsible for inhibiting

10:51:34 1 aggression, and what we talked about, considering the  
10:51:43 2 outcome of your actions.

10:51:48 3 And also in the basal ganglia and in the  
10:51:53 4 corpus callosum, and I believe that this low value,  
10:51:57 5 really, very low value of the corpus callosum is not  
10:52:03 6 -- it couldn't be real in the sense that it couldn't --  
10:52:06 7 it would be dead if it were metabolizing at that rate.  
10:52:12 8 Well, not dead, but, really.

10:52:16 9 So what we see here is what we call partial  
10:52:18 10 volume effect. Because the corpus callosum is so  
10:52:22 11 close to ventricles, and ventricles have zero metabolic  
10:52:26 12 rates, then the values spill over. I mean, the  
10:52:29 13 resolution of the PET is not perfect. It's -- the PET  
10:52:35 14 scanner here, at Vanderbilt, has a resolution of about  
10:52:40 15 1.7 centimeters. So it's fairly coarse. It's not  
10:52:45 16 quite as fine as MRI, where we have sub-millimeter  
17 resolution.

10:52:49 18 So what we see here is a combination of  
10:52:55 19 abnormally low callosal metabolism, and the effect of  
10:53:00 20 the large ventricles, and that is precisely what has  
10:53:03 21 been described for fetal alcohol syndrome. That's why  
10:53:07 22 I'm tending so strongly toward that diagnosis. And  
10:53:14 23 that PET finding will support that.

10:53:17 24 MR. DAWSON: And, Dr. Gur, I'd like to  
10:53:20 25 then make the -- there should be a copy of the slide

10:53:24 1 in the documents, as well as the document called,  
10:53:27 2 "Summary of PET Quantitative Analysis for Mr. Byron  
10:53:31 3 Black." If that report and that graph could be put  
10:53:36 4 together into the next exhibit, which would be 12.

5 (Document marked Exhibit No. 12.)

10:54:09 6 BY MR. DAWSON:

10:54:10 7 Q. Dr. Gur, then I think you have another slide  
10:54:14 8 that --

10:54:14 9 A. Yep. The last slide relates to my  
10:54:20 10 neuropsychological evaluation, and that actually  
10:54:26 11 summarizes what all the other testing indicates. The  
10:54:35 12 testing that was done on Mr. Black before I tested him,  
10:54:42 13 myself. And the way this has been processed is using  
10:54:49 14 an algorithm called behavioral imaging. It's nothing  
10:54:55 15 -- it's just a way of presenting the data.

10:54:59 16 Essentially, when a neuropsychologist looks at  
10:55:04 17 the battery of tests, they make links between scores of  
10:55:12 18 each test and the brain region that is most likely  
10:55:16 19 damaged. So if, for example, it turns out that if you  
10:55:22 20 have a lesion in the third frontal convolution of left  
10:55:28 21 hemisphere, you will become very -- it will become very  
10:55:33 22 difficult for you to talk. You will just lose your  
10:55:37 23 verbal fluency. This is called a form of aphasia,  
10:55:42 24 classical Brocha's Aphasia.

10:55:46 25 So I can test someone and ask them to tell me

10:55:49 1 how many words here. You have a minute. Write down  
10:55:52 2 all the words you can come up with that starts with  
10:55:56 3 "C." And now give me all the words that start with  
10:56:00 4 "F." Now give me all the words that start with "L."  
10:56:02 5 And then you count how many different words they  
10:56:05 6 produce and compare it to normal. And if it's below  
10:56:09 7 normal, you say, well, it looks like there is a problem  
10:56:12 8 in that third convolution of the left hemisphere.

10:56:18 9 Similarly, patients who have lesions in the  
10:56:20 10 right parietal may lose the ability to read maps, or to  
10:56:27 11 find themselves. They get lost all the time. So you  
10:56:29 12 have a test that measures your orientation, how you are  
10:56:32 13 able to judge orientation. And if you score abnormally  
10:56:37 14 you say, oh, that's maybe where the problem is.

10:56:38 15 And so you go over all those tests and begin  
10:56:41 16 to accumulate evidence in favor of one or another  
10:56:45 17 region. And if you are well-trained, you can make  
10:56:49 18 pretty decent predictions that will eventually be  
19 verified by MRI, or PET.

10:56:57 20 What we have done for behavioral imaging is  
10:57:00 21 done exactly the same process, except without having  
10:57:04 22 any particular patient in mind. And also we just took  
10:57:08 23 the four leading neuropsychologists at the time.  
10:57:15 24 Fortunately, all of them are still alive and doing  
10:57:19 25 well, but they were considered at the time, and still

10:57:25 1 are, as the sort of, the big grandparents of the field.  
10:57:30 2 Well, there was -- grandparents and parents. There  
10:57:34 3 were two generations of experts. And we did the study  
10:57:39 4 basically giving them all the tests that were --  
10:57:45 5 neuropsych tests that were available at the time, and  
10:57:46 6 these are exactly the same tests that are still being  
10:57:49 7 used, and asked them, okay, so here's someone showing  
10:57:55 8 up with a bad score on this test. Give me a picture of  
10:57:59 9 which part of the brain is implicated. You know, just  
10:58:02 10 give me 10, if it's very likely implicated, and  
10:58:06 11 everything else give me lower numbers. So probability  
10:58:10 12 that the lesion in that area is responsible for bad  
10:58:15 13 score on that. And those weights were entered into a  
10:58:20 14 database. We waited a year, convened them again and  
10:58:25 15 have them do the exact same exercise again so we could  
10:58:29 16 calculate their reliability. And it turned out those  
10:58:34 17 folks were highly reliable.

10:58:36 18 And so, now, when we get a specific test, we  
10:58:41 19 can basically get a free consultation of those experts  
10:58:45 20 by feeding that particular score of the person in  
10:58:51 21 question and seeing how they would read it without, of  
10:58:58 22 course, knowing that Byron Black ever existed, or will  
10:59:02 23 exist.

10:59:04 24 So, this is the result of the behavioral image  
10:59:09 25 on Byron Black and it indicates substantial damage that

10:59:22 1 seems to be focused in the orbital, frontal, temporal  
10:59:28 2 area. I tested him, myself, and actually now reading  
10:59:37 3 the report I wrote then, I'm -- I don't -- I can't  
10:59:48 4 think of a graceful way of saying that, but I was quite  
10:59:52 5 impressed with myself, because these are the regions  
10:59:58 6 that I thought would be implicated. I mentioned them  
11:00:02 7 by name. The orbital, frontal, these are the areas  
11:00:06 8 that seemed most suspicious based on the cognitive --  
11:00:10 9 on the neuropsychological tests that previous people  
11:00:15 10 have done, and my own testing. And these turned out to  
11:00:19 11 be the exact regions that subsequently showed reduced  
11:00:24 12 volume and reduced -- and abnormal activity on the MRI  
11:00:30 13 and PET, respectively.

11:00:33 14 MR. DAWSON: And if we, then, could ask  
11:00:35 15 that the document in the pile that reflects the slide  
11:00:39 16 that's currently on the screen, if that be made the  
11:00:43 17 next numbered exhibit, which I believe is 13.

11:01:09 18 (Document marked Exhibit No. 13.)

11:01:09 19 Q. Dr. Gur, I gather from your discussion of the  
11:01:13 20 Declaration that you did in 2001, which is now Exhibit  
11:01:18 21 11, and the results of the MRI and the PET scan that  
11:01:23 22 were done in this case, that we see a continuity or  
11:01:28 23 agreement among those, I guess, three different methods  
11:01:32 24 of looking at the brain as to where there is damage in  
11:01:35 25 Mr. Black's brain?

11:01:36 1 A. Yes.

11:01:37 2 Q. And is that also true of the amount of damage  
11:01:39 3 that we see in Mr. Black's brain? Do each of these  
11:01:44 4 show a significant abnormality?

11:01:45 5 A. Yes, it is. So had I done the studies in  
11:01:50 6 reverse, had I encountered the MRI not knowing anything  
11:01:56 7 about Mr. Black, I would say I would have expected  
11:02:02 8 profound deficits, and that's what we find on testing.

11:02:07 9 Q. Let's talk a little bit.

11:02:09 10 In terms of mental retardation, are there  
11:02:13 11 different causes of mental retardation so that it  
11:02:19 12 varies among various people that are classified as  
11:02:23 13 mentally retarded?

11:02:24 14 A. Oh, yes. There are several causes of mental  
11:02:28 15 retardation. In fact, quite a few. The most  
11:02:35 16 conspicuous one is, so-called, Down's Syndrome or  
11:02:42 17 Mongoloid. And these are people that you could  
11:02:45 18 recognize right away. It's a known genetic anomaly.  
11:02:54 19 We know the chromosome, we know quite a bit about it.  
11:02:59 20 And it is associated with very distinct appearance  
11:03:05 21 including a Mongoloid-looking face, slit in the tongue  
11:03:18 22 and other such anomalies. But the majority of people  
11:03:18 23 with mental retardation appear perfectly normal.

11:03:26 24 In fact, Penn has been always accused of  
11:03:34 25 trying to stay in it's ivory towers and has recently

11:03:40 1 made a lot of effort to become more integrated in the  
11:03:45 2 community, and as part of that we have a program for  
11:03:49 3 hiring people with mental retardation for jobs at Penn.  
11:03:54 4 And so I know quite a few of them, and there are a few  
11:04:01 5 who are Mongoloid, but there are a few, most of them  
11:04:05 6 you couldn't tell that they are mentally retarded  
11:04:10 7 without spending a substantial amount of time with  
11:04:13 8 them.

11:04:13 9 In fact, one chap has been working now for a  
11:04:17 10 long time and he's dressed impeccably, and if it hadn't  
11:04:24 11 been that he was pulling a cart, most people would  
11:04:28 12 think he's one of the doctors. And I would challenge  
11:04:30 13 anybody to talk with him for 10 minutes and talk to  
11:04:35 14 nine other doctors that I won't name and I bet you that  
11:04:38 15 they will pick somebody else as being the mentally  
11:04:44 16 retarded.

11:04:44 17 He's full of wit, he always has a pearl of  
11:04:53 18 wisdom to share. He has very good taste in clothes and  
11:05:01 19 keeps giving me solicited and unsolicited advice on my  
11:05:07 20 own attire. But you couldn't tell he's retarded.

11:05:12 21 Q. Dr. Gur, you have had an opportunity to review  
11:05:16 22 the reports that were provided for this case by Dr.  
11:05:22 23 Eric Engum?

11:05:24 24 A. Yes.

11:05:24 25 Q. And Dr. Vaught, correct?

11:05:26 1 A. Yes.

11:05:26 2 Q. Just in terms of looking at Dr. Engum's report  
11:05:30 3 and some of the things that Dr. Engum mentioned, he was  
11:05:46 4 concerned about malingering; is that correct?

11:05:49 5 A. Yes.

11:05:50 6 Q. And what is your observation of that issue  
11:05:55 7 with regards to the way that Dr. Engum approached it?

11:05:59 8 A. Well, I didn't think -- I didn't get the  
11:06:06 9 impression that Mr. Black was malingering when I tested  
11:06:11 10 him. I thought he was putting forth a lot of effort,  
11:06:18 11 and some of the deficit comes out because he's so --  
11:06:24 12 trying so hard to do well that he takes too much time.  
11:06:31 13 So I didn't see reason to test for malingering. But if  
11:06:40 14 I had suspected malingering, there are several tests  
11:06:44 15 out there that are standard, well validated, off the  
11:06:49 16 shelf, you could test and see if he's malingering.

11:06:56 17 So I, for the life of me, can't understand if  
11:07:00 18 Dr. Engum thought that if Mr. Black was malingering,  
11:07:04 19 why didn't he test for malingering?

11:07:06 20 Q. Well, his comment that in several times on  
11:07:12 21 the tests that were given by other people, that  
11:07:16 22 Mr. Black seemed to miss some easy issues but was  
11:07:19 23 apparently able in some more complicated areas. Do you  
11:07:23 24 have a comment on that approach to mental retardation?

11:07:27 25 A. Well that's -- that statement really got me

11:07:31 1 going back to looking at his C.V. because I can't  
11:07:36 2 imagine anybody trained as a neuropsychologist would  
11:07:40 3 make a statement like that. Usually in my first  
11:07:45 4 lecture on neuropsychology I point out that this is the  
11:07:49 5 first type of brain damage. That most people are  
11:07:54 6 arranged in a systematic fashion. If they know -- if  
11:07:59 7 they know an item of difficulty -- of difficulty level,  
11:08:06 8 say D, they will know the items that are easier, and  
11:08:12 9 will -- and that's it.

11:08:13 10 I mean, there's a fundamental concept in  
11:08:19 11 psychometrics called Guttman's Scaling which is that  
11:08:23 12 you should construct all your tests in a way so that if  
11:08:27 13 someone has passed item N, you know that they have  
11:08:32 14 passed item N minus one. So if they pass an item of --  
11:08:37 15 a level of -- given level of difficulty, you know that  
11:08:42 16 they have passed all the easier items.

11:08:45 17 Now that is not always like that. There is  
11:08:46 18 always some deviation. Occasionally, somebody would  
11:08:50 19 know something hard and not know something easy. But  
11:08:53 20 this is very unusual. And so when you get a testing  
11:08:59 21 and you see there are those pockets of excellence  
11:09:03 22 against pockets of deficit, that's exactly what you're  
11:09:08 23 looking for when you -- when you -- that's exactly when  
11:09:13 24 you send your patient to get an MRI because that's not  
11:09:17 25 normal. And that's the first cardinal sign you look

11:09:21 1 for in brain damage.

11:09:23 2 So I have no -- I have no explanation of why  
11:09:31 3 someone would take that effect and consider that as a  
11:09:35 4 evidence for anything else. I mean, if you were  
11:09:40 5 malingering you would know not to know the hard items.  
11:09:47 6 I don't understand why, what is the logic behind it and  
11:09:52 7 I know for a fact that this is not what is being taught  
11:09:54 8 in neuropsychology.

11:09:58 9 Q. Now, would that be the difference of looking  
11:09:58 10 at somebody that's a Down's Syndrome person and,  
11:10:04 11 therefore, mentally retarded because of that, versus  
11:10:05 12 somebody that is brain damaged and mentally retarded  
11:10:09 13 due to brain damage or brain injury, or is it --

11:10:12 14 A. That could be. If you look at -- I mean, yes.  
11:10:21 15 If someone is retarded because of Down's Syndrome, it's  
11:10:28 16 still -- you'd still get pockets, you'd still get that  
11:10:33 17 strange pattern of certain things they are able to do  
11:10:36 18 that are quite amazing. And that is really -- you see,  
11:10:40 19 when your brain is damaged it's something that the  
11:10:45 20 brain -- you -- you are your brain, and your brain is  
11:10:48 21 trying to correct itself. I mean, the purpose of its  
11:10:52 22 being here is to guide us, control our behavior. So  
11:10:57 23 when it's damaged, it tries to make due with what is  
11:11:03 24 not damaged as much as possible, and that's why many  
11:11:10 25 times people with brain damage, particularly those

11:11:13 1 acquired early, develop some areas in which they are  
11:11:18 2 excellent. They recognize intuitively what are those  
11:11:25 3 things that they can do well, and if they find  
11:11:28 4 something they do well, they work it to perfection.

11:11:32 5 So if you have someone who grew up with the  
11:11:35 6 fetal alcohol syndrome, or another form of retardation,  
11:11:42 7 and they discover that they can draw nicely, that part  
11:11:47 8 of the brain that controls drawing is not damaged, wow,  
11:11:50 9 they'll start drawing and they'll start drawing and  
11:11:54 10 they'll become superb drawers. They can become  
11:12:00 11 excellent even. I mean, usually it's relatively  
11:12:02 12 speaking. We don't no really big geniuses who are  
11:12:06 13 otherwise mentally retarded. It doesn't happen. But  
11:12:10 14 relative to them, you say here is someone who can  
11:12:13 15 barely understand what you say to him, doesn't know  
11:12:15 16 what you're talking about when you mentioned President  
11:12:18 17 and elections and all those things, but he can sit and  
11:12:22 18 in two minutes draw a perfect portrait, you know, that  
11:12:28 19 stands out.

11:12:29 20 Q. This would also be true of things such as good  
11:12:34 21 penmanship? That somebody who was mentally retarded --

11:12:34 22 A. Penmanship is an excellent example.  
11:12:37 23 Penmanship is something that if you get somebody who  
11:12:41 24 has incredibly good penmanship, it's almost a suspicion  
11:12:46 25 for brain damage because they must be really working

11:12:49 1 hard to excel in something. It's important for  
11:12:53 2 everybody to feel that they are special and that they  
11:12:57 3 have abilities, you know, that make them unique, make  
11:13:01 4 them -- make them themselves. It's important if you  
11:13:05 5 have a brain damage or if you don't have brain damage,  
11:13:08 6 sometimes it's more important when you do have brain  
11:13:10 7 damage.

11:13:11 8 So penmanship is something that if they -- I  
11:13:14 9 mean, suppose a kid like that goes to school and,  
11:13:17 10 really, the whole thing goes by him but his teacher  
11:13:21 11 says, "Wow, you write nicely." They identify that area  
11:13:25 12 of strength and they'll become -- you know, they'll get  
11:13:31 13 there "A" in penmanship.

11:13:33 14 Q. What about -- I think another thing about both  
11:13:36 15 Dr. Engum and Dr. Vaught mentioned was that in terms of  
11:13:40 16 their conclusion that Mr. Black was not mentally  
11:13:43 17 retarded, was that he had passed his driver's test.  
11:13:46 18 Does that seem unusual to you for somebody that would  
11:13:49 19 be mentally retarded?

11:13:51 20 A. Well, we all know in our lab of a woman with  
11:13:55 21 Alzheimer's Disease that my wife, Raquel, was worried,  
11:14:05 22 really losing sleep over this lady driving, and her  
11:14:09 23 family was losing sleep over it. She was liable to get  
11:14:12 24 up in the middle of the night, get into her car and  
11:14:16 25 drive and then run out of gasoline somewhere in the

11:14:20 1 middle of Pennsylvania.

11:14:20 2           So her life was in danger. And so finally we  
11:14:25 3 went through the legal procedure of taking away her  
11:14:29 4 driver's privileges. And the judge agreed under the  
11:14:37 5 stipulation that she'll have to take the test, and if  
11:14:40 6 she passes, well, he can't take away the privilege.  
11:14:46 7 But guess what? She passed with flying colors. It was  
11:14:50 8 a real problem.

11:14:52 9           So first of all, driving is -- it's not an  
11:15:01 10 easy thing. You can't teach a monkey to drive a car,  
11:15:05 11 but once you learn how to drive it, it's -- it's the  
11:15:11 12 kind of learning that's called procedural learning.  
11:15:14 13 It's like riding a bicycle or skiing, once you learn  
11:15:18 14 it, you know it. You don't have to -- you don't have  
11:15:21 15 to think about it. And I'm sure everybody here, as I  
11:15:28 16 have, driven home on a commute and all of a sudden you  
11:15:31 17 are home and you say, "Wait a second. Was I sleeping  
11:15:34 18 the whole way? I don't ever remember anything that  
11:15:37 19 happened on the way. I don't remember, you know,  
11:15:41 20 moving the wheel, shifting gears and all that stuff."  
11:15:45 21 And the reason is you just do it, you don't think about  
11:15:47 22 it. It doesn't require any executive functions once  
11:15:52 23 it's learned. Until you learn it, it's not easy. But  
11:15:56 24 once you learn it, it sort of becomes part of nature,  
11:16:00 25 like walking, like running. And so you can be

11:16:06 1 profoundly demented and have only half your brain, you  
11:16:08 2 can still -- if you knew how to drive before it  
11:16:11 3 happened, you can still drive.

11:16:13 4 Q. What about another concern that both Dr.  
11:16:18 5 Vaught and Dr. Engum had was that Mr. Black had held  
11:16:21 6 several jobs, one being for an insurance company where  
11:16:25 7 he was a courier. He took the money bag, essentially,  
11:16:29 8 with checks and cash from the company, from the people  
11:16:31 9 that collected it and put it in the -- to the bank,  
11:16:35 10 gave it to the teller. And picked up orders. When  
11:16:38 11 someone needed supplies, he went to the warehouse and  
11:16:41 12 somebody handed him an order, he'd put it in a bag and  
11:16:44 13 gave it to whoever needed it. Are these activities  
11:16:47 14 inconsistent with mental retardation?

11:16:52 15 A. Well, I don't know the statistics, precisely,  
11:16:55 16 but at least my impression from looking at the retarded  
11:16:59 17 work force at Penn, is that these are exactly the jobs  
11:17:02 18 that they get and excel at. In fact, these are jobs  
11:17:06 19 that they do better than people without retardation  
11:17:10 20 because it's routine, they know where they have to --  
11:17:15 21 what they have to do. They are less likely to stop on  
11:17:19 22 the way and chit chat and do other sorts of things,  
11:17:25 23 less likely to -- those messengers, a lot of times, can  
11:17:31 24 be tempted to steal, and they are less likely to do  
11:17:35 25 that. So they are almost ideal for these kinds of

11:17:40 1 positions and this is -- these are the kinds of  
11:17:45 2 positions that they get and do very well.

11:17:48 3 Q. What about the other two, I think, employments  
11:17:51 4 that he had, according to his record were in  
11:17:53 5 construction, and then at the time of his arrest he was  
11:17:56 6 working at an auto wash company doing car detail. Are  
11:18:00 7 either one of those --

11:18:01 8 A. Right. He passed the postal service. And  
11:18:06 9 these are the kinds of jobs we also try to find for  
11:18:10 10 people with schizophrenia. It's not easy to find jobs  
11:18:13 11 for them. People are afraid. Both -- the same problem  
11:18:17 12 with mental retardation. And so the kinds of jobs you  
11:18:22 13 look for are jobs that require minimal executive  
11:18:29 14 functions because this is exactly the problem with  
11:18:32 15 mental retardation, is the executive functions.

11:18:37 16 So if they don't have to make decisions, they  
11:18:42 17 can do very well. If it's routine, they can do very  
11:18:47 18 well. So these jobs are routine. If you told me he  
11:18:50 19 was a master bricklayer, I would say that's not  
11:18:53 20 consistent. I don't -- I don't think someone with  
11:18:57 21 mental retardation could become a master builder or --  
11:19:04 22 because you need to make a lot of decisions during your  
11:19:10 23 work. But a construction worker, not otherwise  
11:19:16 24 classified, is exactly the right job for them.

11:19:20 25 Q. One of the other things that Dr. Engum said, I

11:19:24 1 think we looked at his Paragraph 28 in his report, and  
11:19:28 2 he was trying to make a point that with all the  
11:19:31 3 advances in diagnosis, why are there differences, or  
11:19:36 4 lack of concordance in the diagnoses that were done on  
11:19:37 5 Byron Black throughout the course of, I guess, really,  
11:19:39 6 this litigation starting with the pre-trial to the  
11:19:42 7 first post-conviction and then now? Do you make any  
11:19:49 8 significance of that in terms of, really, the value of  
11:19:53 9 the prior diagnosis?

11:19:55 10 A. Well, as someone who works in so-called  
11:19:59 11 tertiary care hospital, we really -- big chunk of our  
11:20:05 12 clinical practice is fixing mistakes. Most people  
11:20:13 13 won't come to the university hospital for a routine  
11:20:16 14 problem. I'm not at all surprised. I have seen people  
11:20:23 15 who have been -- who have come for years being  
11:20:27 16 misdiagnosed, undiagnosed, untreated for conditions  
11:20:32 17 that were imminently treatable and diagnosable.

11:20:40 18 And it's not just in the field of psychiatry.  
11:20:43 19 It's in medicine, as well. My colleagues in surgery,  
11:20:47 20 internal medicine, tell me that 80 percent of their  
11:20:52 21 work is fixing mistakes done in area hospitals of  
11:20:57 22 misdiagnosis, of wrong diagnosis. It's not easy to  
11:21:01 23 make a diagnosis. And in the case -- if Mr. Black  
11:21:06 24 would have been an ordinary case of mental retardation,  
11:21:10 25 if he had Down's Syndrome, you wouldn't need a

11:21:14 1 professional to make the diagnosis. But he has a more  
11:21:20 2 rare form of mental retardation. And I don't think it  
11:21:27 3 necessarily because the clinicians who evaluated him  
11:21:32 4 were bad, although some of them left something to be  
11:21:38 5 desired. But even the best clinicians could miss a  
11:21:43 6 complicated case like that. And it doesn't mean what  
11:21:48 7 Mr. Engum says it means. It means that this is not a  
11:21:53 8 case that happens so often that anybody with clinical  
11:21:57 9 experience will see, will see them.

11:22:02 10 Q. Were you particularly surprised or -- in this  
11:22:05 11 case to learn that neither Dr. Engum or Dr. Vaught had  
11:22:09 12 actually seen Mr. Black and interviewed him in any way  
11:22:16 13 or tested him in any way?

11:22:18 14 A. I think surprise would probably be a vast  
11:22:21 15 understatement of my reaction when I discovered that.  
11:22:28 16 Incredulous is more like it. Appalled is probably  
11:22:33 17 closer even because I don't know how they have the  
11:22:40 18 audacity, arrogance or whatever to make a diagnosis  
11:22:47 19 without examining the patient. This is -- I have never  
11:22:54 20 heard of anybody doing that.

11:22:58 21 I mean, I'm a very -- I hope you can believe  
11:23:03 22 that I have -- I'm leading a fairly busy life and the  
11:23:08 23 clinical work is -- occupies about 20 percent, at most,  
11:23:14 24 of what I do. I do research all the time. I have to  
11:23:20 25 travel to conferences, I have to present my studies, I

11:23:23 1 have to supervise people doing various experiments and,  
11:23:28 2 frankly, to do a clinical evaluation of a patient I  
11:23:33 3 need like I need a hole in my head. I mean, this is  
11:23:38 4 not a top priority for me, and yet I do it every time.  
11:23:46 5 I have never signed a report that includes a diagnostic  
11:23:50 6 impression of someone that I have not examined myself.  
11:23:55 7 And that's just even to venture a diagnosis, let alone  
11:24:02 8 venture opinions about somebody else's diagnosis and  
11:24:07 9 somebody else who has seen the patient. How can you  
11:24:11 10 say that Mr. Black is malingering? That other people  
11:24:17 11 didn't know what they were seeing, without talking to  
11:24:22 12 him? If he was wondering, if the guy is retarded, why  
11:24:27 13 didn't he spend 10 minutes, half an hour talking with  
11:24:31 14 him? If he thought he was malingering, why didn't he  
11:24:34 15 use the opportunity to give him a test of malingering?  
11:24:38 16 I mean, this is unheard of in any circles I've  
11:24:43 17 been in. And even in cases where we are not dealing  
11:24:51 18 with life and death decisions, this could be a decision  
11:24:54 19 as to whether to put the kid on Ritalin, or not? It's  
11:24:58 20 not life and death. Should you go to further testing?  
11:25:03 21 Not life and death. Important. You always do it. And  
11:25:12 22 here we are dealing with someone in a capital case and  
11:25:17 23 they think they can make a diagnostic impression  
11:25:22 24 without seeing that individual?  
11:25:25 25 I mean, I have been in the field, well, since

11:25:32 1 1970, and I was not trained that way and I'm not  
11:25:39 2 training my students that way. And, frankly, I haven't  
11:25:47 3 heard of anything like that.

11:25:51 4 Q. And what do the results of your work tell us  
11:26:02 5 about the likelihood that Mr. Black is mentally  
11:26:04 6 retarded?

11:26:07 7 A. Well, they offer the diagnosis of mental  
11:26:12 8 retardation. They support strongly both the cognitive  
11:26:19 9 testing and the behavioral testing.

11:26:22 10 I think there is a lot of confusion about --  
11:26:26 11 and I'm sure it will eventually have to clear itself  
11:26:31 12 through the courts, about the definition of mental  
11:26:36 13 retardation. There is consensus among experts, enough  
11:26:43 14 to make diagnosis, but it's not the kind of consensus  
11:26:52 15 where you can say give me one number and if it's above,  
11:26:57 16 it is, and if it's below, it isn't. You are tempted to  
11:27:01 17 use the IQ as that number. If you put the gun to a  
11:27:07 18 psychologist's head and say, "Give me a number," after  
11:27:12 19 struggling and trying to get out of your control and  
11:27:15 20 realizing that that gun is going to fire, a decent  
11:27:19 21 psychologist will say 70.

11:27:25 22 But, it's really a number that can be very  
11:27:33 23 valuable and missed in a lot of cases of mental  
11:27:37 24 retardation because an IQ test -- there are several IQ  
11:27:41 25 tests and they differ in many ways from each other.

11:27:45 1 They all give you a number so you get in the comfort  
11:27:48 2 zone of, "I have a number." And roughly, the number  
11:27:55 3 means the same thing, namely how many standard  
11:27:58 4 deviations are you away from the mean? But some tests  
11:28:03 5 will have a lot of executive -- measures of executive  
11:28:08 6 functions, some tests will have very few measures of  
11:28:12 7 executive functions. So, IQ test may include measure  
11:28:17 8 of memory, may include a measure of motor speed, may  
11:28:23 9 include motor dexterity.

11:28:25 10 Now, the reason we think people with mental  
11:28:28 11 retardation are incapable, incapable of making reason  
11:28:36 12 decisions is not because their memory is poor, it's not  
11:28:40 13 because they can't orient themselves in space, it's not  
11:28:46 14 because they can't run fast, it's not because they  
11:28:49 15 can't coordinate their hands, but it's because they  
11:28:55 16 cannot appreciate the consequences of their actions in  
11:28:59 17 the context of what they do.

11:29:05 18 Now, in an average IQ test you may have two or  
11:29:08 19 three measures of that and a lot of measures of those  
11:29:12 20 other things that are irrelevant to the issue of  
11:29:20 21 culpability. So if we take the example of someone who  
11:29:24 22 is retarded because of brain damage and it turns out  
11:29:28 23 that that person happens to have spared a region that  
11:29:31 24 is measured by an IQ test -- for example, I know some  
11:29:36 25 of these people who -- where they are able to remember

11:29:44 1 words. So they study the dictionary. They are  
11:29:47 2 profoundly retarded but they have incredible  
11:29:51 3 vocabularies. They will know words you never dreamed  
11:29:55 4 existed. So if there is a test of the vocabulary on  
11:30:02 5 the IQ test, they will score very high and the whole IQ  
11:30:07 6 test -- the whole IQ score will squeak above 70 because  
11:30:13 7 of that one subscale.

11:30:14 8 Now, we don't say that mentally retarded  
11:30:17 9 people are less culpable because their memory is poor,  
11:30:22 10 right? We say they are less culpable because of other  
11:30:26 11 things that are not measured by vocabulary.

11:30:30 12 Q. Dr. Gur, in terms of looking at this --

11:30:32 13 A. I'm sorry. I meant vocabulary, rather than  
11:30:32 14 memory.

11:30:34 15 Q. In terms of analyzing the issue of mental  
11:30:37 16 retardation in the context of Mr. Black and in the  
11:30:41 17 context of the death penalty, are you familiar with the  
11:30:45 18 case of Atkins versus Virginia?

11:30:45 19 A. Yes.

11:30:48 20 Q. And that, of course, is the case that was  
11:30:51 21 determined by the Supreme Court that it was  
11:30:54 22 unconstitutional to violate -- I mean, execute the  
11:30:55 23 mentally retarded.

11:30:57 24 A. You showed me the entire case.

11:30:59 25 Q. And was there some language in that case that

11:31:01 1 you found particularly instructive as to interpreting  
11:31:06 2 Byron Black's situation?

11:31:07 3 A. Yes. I was struck by the Supreme Court's  
11:31:13 4 description of the reason for why we are holding mental  
11:31:18 5 retarded individuals as less culpable. And in their  
11:31:25 6 description of the reasons they eloquently describe the  
11:31:30 7 frontal lobe. As a neuroscientist, I'd be asked to  
11:31:38 8 describe the function of the frontal lobe, I would use  
11:31:41 9 exactly those same terms. Adaptive, being able to  
11:31:45 10 adjust to the context, be able to plan, be able to  
11:31:48 11 realize the consequences of your actions, be able to  
11:31:52 12 inhibit impulses. This is what the frontal lobe does.

11:31:58 13 So, I think the judges really meant when they  
11:32:03 14 said that mentally retarded people have less  
11:32:12 15 culpability, they really meant to single out those  
11:32:16 16 factors that relate to the frontal lobe except, you  
11:32:23 17 know, they're not -- they're not neuroscientists, so  
11:32:27 18 they just didn't add the word, frontal lobe. But  
11:32:31 19 that's basically those functions that are -- that are  
11:32:37 20 the functions of the frontal lobe.

11:32:40 21 Now, I gave him an IQ test, as well. And one  
11:32:42 22 of the tests I gave him was the Raven's Progressive  
11:32:48 23 Matrices, which is probably the second most valid --  
11:32:51 24 most valid test after the Reys, and that loads heavily  
11:32:55 25 on executive functions, and he scored 2.7 standard

11:33:03 1 deviations below the mean, which would put his estimate  
11:33:07 2 of IQ at around 60.

11:33:10 3 Q. And based upon the brain damage that you have  
11:33:17 4 been able to find in your work in analyzing Mr. Black,  
11:33:23 5 would a person with this type of brain damage be likely  
11:33:28 6 to be mentally retarded?

11:33:31 7 A. Yes.

11:33:31 8 Q. Is it possible that a person with this type of  
11:33:34 9 brain damage that Mr. Black has could have an IQ as  
11:33:39 10 high as 80 or 85?

11:33:42 11 A. Well, as I explained, it's quite possible  
11:33:45 12 depending on the specific IQ test that there were --  
11:33:51 13 items -- there were scales administered that loaded on  
11:33:58 14 abilities that do not relate to executive functions.  
11:34:02 15 And to that extent, it's possible to get an average  
11:34:06 16 score higher than 70. I'd be very surprised by it if  
11:34:14 17 somebody who's brain looks like that would score as  
11:34:17 18 much as 85. But if you told me this happened, I have  
11:34:24 19 been surprised before, so it's possible.

11:34:26 20 Q. In terms of your review of the information  
11:34:28 21 about Mr. Black and your interview with Mr. Black, your  
11:34:32 22 testing of Mr. Black, is there anything about Mr. Black  
11:34:35 23 that would suggest to you that he has gained any  
11:34:39 24 especially high competence in any of the areas of the  
11:34:43 25 subtests that might push his scores up?

11:34:45 1 A. I haven't seen much, any evidence of -- I  
11:34:50 2 mean, the best he did in my testing was slightly below  
11:34:55 3 normal. And these were -- interestingly, I can look  
11:35:07 4 more -- I can look at the detail. But I remember -- I  
11:35:17 5 remember he did well in object learning, surprisingly  
11:35:25 6 well in object learning, and face, face memory. If you  
11:35:43 7 want, I can go in my notes. But the point, the areas  
11:35:48 8 where he did particularly poorly, which is also  
11:35:51 9 pertinent to Atkins is recognizing of emotions on the  
11:35:58 10 face. He scored -- and this is a simple test. You see  
11:36:02 11 a face and you have to say what is the emotion that is  
11:36:08 12 shown, that it shows? And he worked hard. He sweated  
11:36:14 13 it. So he was able to get most of the answers right,  
11:36:23 14 but he took so much time, just unbelievable to watch  
11:36:29 15 him try to do that, and it was tremendous effort for  
11:36:34 16 him. Even simple emotions like happy, sad, anger,  
11:36:39 17 fear.

11:36:39 18 So, he has diminished ability to understand  
11:36:45 19 how other people are feeling, and that is what -- how  
11:36:52 20 are they feeling? How are they thinking? And that is  
11:36:55 21 another -- that's called, in modern terms, called  
11:37:02 22 theory of mind. That is also a uniquely human  
11:37:05 23 function, being able to empathize with somebody else,  
11:37:12 24 and that is also a function that is impaired with  
11:37:18 25 frontal lobe damage and is specifically mentioned by

11:37:22 1 the Supreme Court in the Atkins case.

11:37:26 2 Q. So in terms of then looking at what are the  
11:37:29 3 issues of deficits in adaptive behavior, would it be  
11:37:35 4 your conclusion that the testing that was done and the  
11:37:38 5 various information about Mr. Black would indicate that  
11:37:42 6 there, in fact, is a deficit in adaptive behavior that  
11:37:46 7 would put him into the mentally retarded range?

8 A. Yes.

11:37:50 9 Q. And one question that I need to go back to a  
11:37:52 10 little bit. One of the questions I had forgotten to  
11 ask was one of the tests that both, I think, of the  
11:37:57 12 experts from the State were concerned about in terms of  
11:38:03 13 the issue of whether it was accurate, I think it's the  
11:38:07 14 PPVT? The Peabody Picture --

11:38:10 15 A. Picture Vocabulary Test.

11:38:12 16 Q. And that's the test where Mr. Black apparently  
11:38:16 17 missed things like a flamingo and a canoe. What, in  
11:38:20 18 terms of your information about Mr. Black, your testing  
11:38:23 19 of Mr. Black, your knowledge of Mr. Black do you find  
11:38:26 20 this surprising?

11:38:27 21 A. No. That's another example of exactly what  
11:38:30 22 happens with brain damage. You get those pockets of  
11:38:36 23 excellence and pockets of ignorance that are  
11:38:40 24 surprising. I mean, you can -- one of the signs that  
11:38:43 25 you have somebody with brain damage is you talk to them

11:38:47 1 -- you can talk to them intelligently about, you know,  
11:38:51 2 presidential candidates, and so on, and then you find  
11:38:55 3 out that they don't know what the year is, or they  
11:39:01 4 don't -- for the life of them, can't tell you where the  
11:39:08 5 nearest supermarket is.

11:39:10 6 So that's exactly the kinds of discrepancies  
11:39:15 7 that are reflective of brain damage.

11:39:23 8 Q. In terms of possible damage to Mr. Black's  
11:39:28 9 brain, would his playing football be a possible  
11:39:32 10 contributor to brain damage?

11:39:34 11 A. There is no doubt at this point it was a  
11:39:40 12 finding that was first startling. I think the first  
11:39:44 13 paper on that came, I think, in 1984 by Wayne Alves  
11:39:53 14 and his colleagues at University of Kentucky, and they  
11:39:57 15 studied -- they followed several football teams,  
11:40:06 16 varsity football in college, and did neuropsych testing  
11:40:13 17 when they just started and then continued year after  
11:40:18 18 year. And in 1984 they published the first two-year  
11:40:24 19 follow-up and that's when, you know, it hit the media  
20 in a big way because their conclusions were that, I  
11:40:32 21 think you lose about five IQ points per year of playing  
11:40:35 22 football in college. And now this effect has been  
11:40:43 23 replicated numerous times in several other studies, as  
11:40:48 24 well, and it's about right.

11:40:50 25 I mean, maybe again this IQ thing is more of

11:40:55 1 an embarrassment than of pride for psychologists. Just  
11:41:01 2 the comfort of a single number. But they documented  
11:41:06 3 several groups had documented progressive deterioration  
11:41:13 4 of cognitive abilities related to frontal lobe and  
11:41:17 5 related specifically to number of concussions that they  
11:41:23 6 suffer, the football players suffered during the game.  
11:41:29 7 So this is no longer a controversy. The controversy  
11:41:32 8 now, the issue now is how to try to build safer  
11:41:36 9 equipment and introduce better safety in the coaching,  
11:41:42 10 and so on, to reduce that problem. But I don't believe  
11:41:45 11 there is serious doubt now that there is an effect.

11:41:50 12 Q. You indicated earlier that you felt that one  
11:41:53 13 of the -- or the most likely cause for the brain damage  
11:41:56 14 that we see in Mr. Black's case would be the result of  
11:42:00 15 fetal alcohol, of his mother drinking during pregnancy.  
11:42:05 16 Have there been studies that have looked at that in  
11:42:11 17 some depth as to the type of damage and how you seek  
11:42:17 18 diagnosis?

11:42:17 19 A. Yes. Alcohol, each substance has its favorite  
11:42:22 20 location in the brain, and the favorite location for  
11:42:27 21 alcohol, as well as cocaine, is the frontal lobe. And  
11:42:34 22 they attack it. So if you have an adult chronic  
11:42:39 23 alcoholic, you will see shrinkage of the frontal lobe.  
11:42:45 24 Children born to mothers with -- and you also see  
11:42:48 25 enlargement of the ventricles, reflecting loss of

11:42:51 1 tissue in the middle of the brain. So this is what you  
11:42:53 2 expect to see in children of mothers who have been  
11:42:58 3 abusing alcohol during pregnancy. Alcohol or cocaine  
11:43:02 4 probably would have very similar -- so the crack babies  
11:43:08 5 would probably have the same type of appearance. And  
11:43:12 6 the severity of it depends on the severity of the abuse  
11:43:17 7 of alcohol.

11:43:21 8           There are several studies that demonstrate  
11:43:27 9 ventricular volume increase to the point of inducing  
11:43:35 10 abnormal shape of the corpus callosum. So my  
11:43:40 11 colleague, Fred Bookstein, University of Michigan, has  
11:43:46 12 looked at the shape of the callosum in children of  
11:43:52 13 parents with fetal alcohol -- children with fetal  
11:43:56 14 alcohol syndrome and, indeed, there is an abnormal --  
11:44:07 15 characteristic abnormality in the shape of the corpus  
11:44:11 16 callosum that can be documented with his method. But,  
11:44:15 17 really, all it reflects is what we could measure more  
11:44:18 18 directly which is the expansion of cerebral spinal  
11:44:22 19 fluid that sort of pushes the callosum to a misshaped  
11:44:29 20 appearance.

11:44:29 21           So that Bookstein's work would really fall in  
11:44:35 22 line with the diagnosis of fetal alcohol syndrome in  
11:44:38 23 the case of Mr. Black.

11:44:40 24           MR. DAWSON: I'm told that some people  
11:44:42 25 need a break. I have got about two questions left, but

11:44:46 1 we can take a break and then come back to those.

11:44:46 2 (Recess taken.)

11:44:57 3 BY MR. DAWSON:

11:45:05 4 Q. And, Dr. Gur, I believe there's three articles  
11:45:11 5 that are in that pile of papers that was on the court  
11:45:16 6 reporter's desk there. Are those articles that you're  
11:45:19 7 referring to by Dr. Bookstein?

11:45:28 8 A. Two.

11:45:29 9 Q. Just two?

11:45:31 10 A. But these are the main -- this should suffice  
11:45:34 11 for describing the --

11:45:37 12 Q. What are those articles?

11:45:38 13 A. One is an article in the anatomical record,  
11:45:46 14 "Midline Corpus Callosum Is a Neuroanatomical Focus of  
11:45:51 15 Fetal Alcohol Damage." And the other is an article in  
11:45:57 16 Teratology, "Geometric Morphometrics of Corpus Callosum  
17 and Subcortical Structures in the  
11:46:01 18 Fetal-Alcohol-Affected Brain."

11:46:08 19 Q. Doctor, we have one other article here, if you  
11:46:12 20 would look at this, the third article. It's in  
11:46:12 21 NeuroImage?

11:46:16 22 A. Yeah. That's an article in NeuroImage,  
23 "Corpus Callosum Shape and Neuropsychological Deficits  
11:46:19 24 in Adult Males with Heavy Fetal Alcohol Exposure."

11:46:26 25 Q. And these are the type of documentation or

11:46:29 1 articles that you rely on in your scientific studies?

11:46:32 2 A. These are excellent journals. First rate.

11:46:35 3 MR. DAWSON: I would ask these be marked  
11:46:36 4 as a collective exhibit 14.

11:46:45 5 (Documents marked Exhibit No. 14.)

11:46:45 6 THE WITNESS: I'm not saying that I  
11:46:46 7 endorse Bookstein.

11:46:48 8 MR. DAWSON: Right.

11:46:48 9 THE WITNESS: That's really not my field.  
11:46:50 10 That's a statistical method.

11:46:53 11 MR. DAWSON: I see.

11:46:56 12 THE WITNESS: This is why we have referee  
11:46:58 13 journals. I know that if NeuroImage published it,  
11:47:01 14 there are people who eat those mathematics for  
11:47:04 15 breakfast, lunch and dinner and should be fine.

11:47:13 16 BY MR. DAWSON:

11:47:13 17 Q. So, Doctor, then based on your involvement in  
11:47:16 18 this case, your examination of Mr. Black, your testing  
11:47:21 19 that was done and the conclusions drawn by the  
11:47:24 20 neuroimaging that you conducted and participated in,  
11:47:29 21 would it be your opinion, to a level of scientific  
11:47:32 22 certainty, that Mr. Black has serious brain damage?

11:47:36 23 A. Yes.

11:47:37 24 Q. Would it also be your opinion, to a reasonable  
11:47:41 25 degree of scientific certainty, that Mr. Black, because

11:47:43 1 of that brain damage, is mentally retarded?

11:47:46 2 A. Yes.

11:47:48 3 Q. I have one final question. And again, all of  
11:47:51 4 the work that you did is accepted, the type of work and  
11:47:57 5 the scientific analysis is accepted in the community of  
11:48:00 6 brain neuroimaging and neuropsychology as  
11:48:05 7 scientifically appropriate?

11:48:06 8 A. Yes. For this analysis for the case of Byron  
11:48:08 9 -- Mr. Black, we have used only methods that are  
11:48:13 10 accepted. We have not used -- we have some more tools  
11:48:18 11 in our bag, but we only applied accepted methods. None  
11:48:24 12 of these methods is controversial.

11:48:27 13 MR. DAWSON: I have no further questions  
11:48:28 14 for Dr. Gur.

15 (Recess taken.)

11:56:46 16 MR. DAWSON: I have one more item before  
11:56:47 17 we turn you over to the State. I would ask that you  
11:56:54 18 produce a compact disc of the PowerPoint presentation  
11:56:59 19 that's part of this deposition and make that Exhibit  
11:57:01 20 Number 15.

11:57:02 21 THE WITNESS: I can do it. How should I  
11:57:50 22 name it? Black v. Tennessee. Or Gur's testimony on  
11:58:02 23 Black?

11:58:03 24 MR. DAWSON: Yeah. That sounds good.

11:58:43 25 THE WITNESS: Anything else I should put

11:58:45 1 on that while I'm --

11:58:46 2 MR. DAWSON: No. I think that's it.

11:58:48 3 THE WITNESS: Put my reports?

11:58:48 4 MR. DAWSON: Well, yeah, might as well  
11:58:56 5 put the reports on it.

6 (Document marked Exhibit No. 15.)

7 (Recess taken.)

8

9 CROSS EXAMINATION

10 QUESTIONS BY MR. DONNELLY:

12:01:28 11 Q. Just to clarify a couple things on your  
12:01:30 12 testimony before, did you administer any IQ test to  
12:01:34 13 Mr. Black?

12:01:34 14 A. Yes.

12:01:35 15 Q. Which test?

12:01:36 16 A. I administered one test that is a measure of  
12:01:43 17 just IQ. But all the tests that I administered in one  
12:01:46 18 way or another would be represented in an IQ test. But  
12:01:53 19 the one test that is just considered an alternative  
12:01:57 20 measure of IQ is the Raven's Progressive Matrices Test.

12:02:04 21 Q. Raven's?

12:02:06 22 A. R-a-v-e-n-'s Progressive Matrices Test.

12:02:16 23 Q. Okay. But I believe I'm quoting here that you  
12:02:22 24 said that, "Mr. Black measured slightly below normal"  
12:02:25 25 on most of the tests that you administered?

12:02:27 1 A. Well, varying from slightly below to some of  
12:02:31 2 them were -- most of them were slightly below, but some  
12:02:35 3 of them were profoundly below.  
12:02:37 4 Q. On another point, I think you said alcoholism  
12:02:41 5 in adults causes which area of the brain to be  
12:02:46 6 affected?  
12:02:47 7 A. Front. Frontal brain.  
12:02:49 8 Q. The same --  
12:02:50 9 A. Same.  
12:02:51 10 Q. -- same one, say, that we see in Mr. Black?  
12:02:52 11 A. Yes. Correct.  
12:02:53 12 Q. Okay. And also, you have referred  
12:02:57 13 occasionally to an "MR" facility. We have had a lot  
12:03:02 14 of testimony about MRIs in this case.  
12:03:04 15 A. I'm sorry.  
12:03:05 16 Q. You mean, something else in this case?  
12:03:06 17 A. Yeah, I mean magnetic resonance instead of  
12:03:12 18 mental retardation.  
12:03:13 19 Q. Thank you for pointing that out.  
12:03:15 20 A lot of your testimony about mental  
12:03:18 21 retardation and the people that you've seen, that's  
12:03:21 22 what I would characterize as antidotal testimony. In  
12:03:25 23 other words, the stories you're telling about people  
12:03:28 24 that you've seen. That mental retardation doesn't seem  
12:03:31 25 to be the focus of your practice; would that be a fair

12:03:33 1 statement?

12:03:34 2 A. That is correct, yes.

12:03:35 3 Q. And in this case you're an expert in  
12:03:39 4 neuropsychology and on your C.V, it's mentioned an  
12:03:44 5 expert in neuroimaging. Have you ever been qualified  
12:03:46 6 as an expert, specifically, in mental retardation?

12:03:49 7 A. No.

12:03:49 8 Q. One other little area I wanted to clarify, the  
9 normative group, these tests, the slides you have shown  
12:04:08 10 us on Mr. Black's data that -- I believe I read 79  
12:04:09 11 healthy young men, and later you went on to clarify  
12:04:12 12 it's volunteers that you have come in and they're  
12:04:16 13 allowed to undergo these tests. When you say young  
12:04:18 14 men, what do you mean? What age group are they?

12:04:20 15 A. 18 to 45.

12:04:23 16 Q. So Mr. Black, I believe, when he was tested,  
12:04:26 17 he was 45, mid 40's. He'd be the upper range of this  
12:04:30 18 group of people you're comparing him to?

12:04:33 19 A. Yes.

12:04:33 20 Q. Now, are any of these men incarcerated?

12:04:36 21 A. No.

12:04:37 22 Q. Or have they been incarcerated for long  
12:04:40 23 periods of time? Twenty years?

12:04:42 24 A. No.

12:04:44 25 Q. Would it be fair to say that one's

12:04:49 1 surroundings and cultural situation, level of poverty  
12:04:55 2 versus wealth affects one's brain?

12:04:59 3 A. I think that's fair to say. There is not  
12:05:02 4 strong evidence for it, but there is some.

12:05:07 5 Q. And by your previous Declaration that was  
12:05:10 6 admitted into evidence in the Federal Appeals -- but it  
12:05:11 7 was also a part of the evidence of this case under  
12:05:16 8 Direct Exhibit No.1, you talk a little bit about how he  
12:05:20 9 may have grown up in poverty and the housing he grew up  
12:05:24 10 in, etcetera, that -- I'm just kind of including that  
12:05:27 11 in cultural.

12:05:28 12 A. Sure.

12:05:28 13 Q. Okay.

12:05:29 14 A. Well, then there is much stronger evidence. I  
12:05:33 15 thought you were talking specifically about cultural  
12:05:39 16 influences after one reaches adulthood.

12:05:43 17 Q. I am. And comparing that to your sample  
12:05:47 18 group, it seems like it would be a much different group  
12:05:50 19 Mr. Black belongs to than what you're comparing him to?

12:05:54 20 A. In the sense that they have not been  
12:05:56 21 incarcerated for 20 years, that is correct.

12:06:00 22 Q. You would think that laying in --

12:06:02 23 A. But they all -- I mean, our volunteers, we  
12:06:07 24 make a special effort to have them comparable to our  
12:06:12 25 patients. And so both demographically and social

12:06:15 1 demographically they are comparable. So we don't -- I  
12:06:22 2 mean, a lot of people use samples of convenience, which  
12:06:27 3 is mostly college students and their colleagues for a  
12:06:34 4 normative sample, and we don't do that. We try to get  
12:06:38 5 the plumber to come in and have his brain scanned.

12:06:42 6 Q. They're willing to subject themselves?

12:06:45 7 A. Correct.

12:06:46 8 Q. Okay.

12:06:47 9 A. And you know how tough it is, but that's what  
12:06:49 10 we are trying to do, and I think we do it well.

12:06:51 11 Q. It's fair to say that your sample does not  
12:06:54 12 include many people that have been in prison,  
12:06:57 13 incarcerated for long periods of time?

12:07:00 14 A. That's fair. I mean, we would exclude someone  
12:07:02 15 with the diagnosable disorder, and if somebody was  
12:07:06 16 incarcerated, that fact is not an exclusion. But  
12:07:09 17 usually it comes with some sort of a diagnosis, and  
12:07:12 18 then they'll be excluded.

12:07:14 19 Q. Okay.

12:07:14 20 A. But these are people who grew up in poverty,  
12:07:19 21 many of them. The West Philadelphia ghetto nearby, a  
12:07:29 22 lot of unhealthy people come from there.

12:07:29 23 Q. You also stated that Mr. Black's form of  
12:07:32 24 mental retardation is, "a rare form."

12:07:34 25 A. Uh-huh.

12:07:35 1 Q. What do you mean by that?

12:07:37 2 A. Well, while I hadn't done research in mental  
12:07:42 3 retardation, I do see a lot of people with mental  
12:07:46 4 retardation in my clinical practice. And I do the same  
12:07:55 5 kind of evaluation that I did with Black, and it is  
12:08:07 6 rare. I mean, you do see brain damage of various  
12:08:13 7 forms. What I meant by rare is that I have not seen  
12:08:18 8 ventricles of this size in anybody, and usually the  
12:08:29 9 history will give us fewer reasons for retardation than  
12:08:36 10 what we see here.

12:08:39 11 Q. Okay. In your report concerning the etiology,  
12:08:47 12 am I pronouncing that correct?

12:08:48 13 A. Yes, sir.

12:08:49 14 Q. Is uncertain, but the primary focus seems to  
12:08:54 15 be before birth or in early childhood. Do you agree  
12:09:02 16 with that?

12:09:02 17 A. Yes.

12:09:04 18 Q. And then you went on to say that may have been  
12:09:12 19 from Mr. Black's mother drinking during pregnancy?

12:09:17 20 A. Yes.

12:09:17 21 Q. And you've kind of focused on that on Direct  
12:09:21 22 Examination, that fetal alcohol syndrome is strong.  
12:09:25 23 Has that always been your opinion in this case?

12:09:27 24 A. No. I thought this was a very high  
12:09:33 25 likelihood, but before I looked and analyzed the

12:09:39 1 images, it was about equally prominent as a possibility  
12:09:46 2 as head injuries or lead exposure. These were the two  
12:09:56 3 other main suspects in those kind of --

12:10:00 4 Q. And you really focused in on those in your  
12:10:03 5 prior report to the Federal Appeals?

12:10:05 6 A. Right. The striking similarity between the  
12:10:08 7 regions affected and what you see with alcoholism is  
12:10:13 8 what tipped the scale in my thinking.

12:10:22 9 Q. Can you give us a clue on the timing of when  
12:10:25 10 you -- the scale was tipped in favor of fetal alcohol  
12:10:29 11 syndrome?

12:10:29 12 A. I can tell you the precise second.

12:10:32 13 Q. Okay. When was that?

12:10:33 14 A. When I saw the three-dimensional rendering  
12:10:41 15 that Davatzikos sent me of the regions that were  
12:10:46 16 shrunk, and that these were what you see in adults with  
12:10:51 17 exposure to alcohol. Interestingly in adults, when  
12:10:54 18 they abstain from alcohol, there is some regeneration  
12:11:00 19 of the volume, normalize --

12:11:04 20 Q. That's consistent with adults drinking --

12:11:06 21 A. Correct.

12:11:07 22 Q. -- the damage to Mr. Black?

12:11:09 23 A. Yeah. Correct.

12:11:10 24 Q. When -- can you give me a date?

12:11:13 25 A. Of when it happened? When I saw that image?

12:11:15 1 Q. Recently, or within the last --

12:11:17 2 A. It was quite recent. Maybe couple, maybe a

12:11:24 3 week ago.

12:11:24 4 Q. Okay. I'm going to hand you this. I just

12:11:39 5 want to ask you if you recognize this document?

12:12:01 6 A. Yes. It's my report on the quantitative MRI.

12:12:17 7 Q. Okay. Is that different from the one that has

12:12:20 8 already been admitted as an exhibit in this case, or is

12:12:23 9 it the same?

12:12:25 10 A. I think it's the same.

12:12:27 11 Q. You might want to look under Interpretation,

12:12:31 12 the second paragraph where it starts, "The etiology is

12:12:35 13 uncertain," but then goes on. I believe in that one it

12:12:40 14 states that it's primarily due to, or possibly --

12:12:44 15 probably due to head injuries and -- maybe in young

12:12:52 16 childhood or at some point?

12:13:05 17 A. Yeah.

12:13:08 18 Q. But the one that was put into as an exhibit by

12:13:13 19 the defense states that it's more focused on the

12:13:19 20 alcohol intake of the mother.

12:13:21 21 A. Yeah.

12:13:22 22 Q. This was received by the State.

12:13:23 23 A. Yeah. This could have been an earlier draft

12:13:26 24 before I --

12:13:26 25 Q. Well, it was sent to us as a -- as your

12:13:29 1 report. But then later the same day we received the  
12:13:33 2 second one focusing on the fetal alcohol possibility.

3 A. Yeah. I mean --

12:13:47 4 Doctor, my question regarding the two reports  
12:13:50 5 and the differences, was there any communication  
12:13:53 6 between yourself and the defense that made you change  
12:13:55 7 your report?

12:13:56 8 A. I sent a draft and received -- and had a  
12:14:09 9 conference call, and there were a couple of questions  
12:14:21 10 that they had, and as I recall, he -- they're saying if  
12:14:27 11 I do have a strong opinion on what the reason is, that  
12:14:32 12 it would be okay to state it. And since -- by that  
12:14:40 13 time I -- you know, it takes a while to put everything  
12:14:44 14 together.

12:14:45 15 Q. Okay.

12:14:46 16 A. So it's quite conceivable that as a result of  
12:14:52 17 the telephone conference, or by myself, when I sent the  
12:14:58 18 final draft, I go over it and if I think that there are  
12:15:06 19 some points that I might have missed or underemphasized  
12:15:10 20 or overemphasized, I change. I don't recall Mr. Dawson  
12:15:19 21 specifically telling me to put something because I  
12:15:25 22 don't react well to those kinds of suggestions --

23 Q. Okay.

12:15:29 24 A. -- and I would have remembered.

12:15:30 25 Q. Okay. Thank you, Doctor. I'd just like to

12:15:33 1 make that the next exhibit number to your testimony,  
12:15:36 2 sir.

3 (Document marked Exhibit No. 16.)

4 BY MR. DAWSON:

12:16:41 5 Q. One last question on that, Doctor, if you  
12:16:43 6 don't mind.

12:16:43 7 Who was on that conference call inbetween the  
12:16:47 8 first report and the second one that was changed?

12:17:07 9 A. I believe it was mostly Mr. Dawson, and I  
12:17:17 10 think Catherine.

12:17:18 11 Q. And who initiated? Did they call you?

12:17:21 12 A. Yes.

12:17:23 13 Q. Okay. Back to the focus of the probable  
12:17:30 14 causes, I think that's the way you put it in your  
12:17:33 15 report of Mr. Black's -- the findings that you had on  
12:17:37 16 Mr. Black's brain. When you say probable, it kind of  
12:17:42 17 leaves the door open for other causes. Could it have  
12:17:46 18 been lead poisoning, like you said in your other  
12:17:49 19 report?

12:17:49 20 A. Yes.

12:17:50 21 Q. Could it have been, say, the head injuries  
12:17:52 22 from sports?

12:17:53 23 A. Yes.

12:17:54 24 Q. Now, Mr. Black turned 18 when was a junior in  
12:17:59 25 high school. So you're not -- you can't put a date

12:18:02 1 certain on when those sports injuries would have --  
12:18:06 2 could have been any time when he was playing football?  
12:18:09 3 A. Yes.  
12:18:09 4 Q. Could it have possibly been from shaken baby  
12:18:13 5 syndrome?  
12:18:14 6 A. Could be.  
12:18:14 7 Q. Or a car accident?  
12:18:16 8 A. Yes.  
12:18:17 9 Q. Or syphilis?  
12:18:18 10 A. Car accident by itself, unless -- unless -- if  
12:18:24 11 it was -- if it happened later in life --  
12:18:28 12 Q. Okay.  
12:18:29 13 A. -- unless it was followed by coma for a long  
12:18:36 14 time, would not explain -- would not explain that sort  
12:18:42 15 of --  
12:18:42 16 Q. Maybe in conjunction with other minor head  
12:18:45 17 injuries or that kind of thing?  
12:18:46 18 A. Yes.  
12:18:47 19 Q. Or syphilis or some other kind of sexually  
12:18:51 20 transmitted diseases?  
12:18:52 21 A. That a good question. Now syphilis, I have to  
12:19:04 22 -- I have to check some sources on that, but if my  
12:19:11 23 memory serves me right, there have been studies of  
12:19:14 24 imaging brains with syphilis.  
12:19:18 25 Q. That show brain damage?

12:19:19 1 A. That show brain damage.

12:19:21 2 Q. Now, I know you already mentioned that adult  
12:19:25 3 alcohol --

12:19:25 4 A. Yes.

12:19:26 5 Q. -- abuse, that would cause similar findings to  
12:19:29 6 what you found in Mr. Black. Would adult drug abuse  
12:19:33 7 also -- is that in the same category?

12:19:36 8 A. Yes. Actually, the site of action of cocaine  
12:19:41 9 and alcohol are nearly identical. They work both on  
12:19:45 10 the dopamine system, which is the neurotransmitters,  
12:19:50 11 you know when -- I mentioned that there is a gap  
12:19:54 12 between each neuron and the next neuron, and that gap  
12:19:58 13 needs to be traveled. The signal has to jump that gap.  
12:20:03 14 And the way it does, the way the brain does it, is that  
12:20:07 15 the electrical pulse releases molecules from the  
12:20:12 16 sending cells and they travel a distance and find  
12:20:18 17 receptors on the other cells, and once enough receptors  
12:20:22 18 get occupied, that generates the new pulse.

12:20:27 19 Now all that happens very quickly. Those  
12:20:32 20 molecules that travel from one neuron ending to the  
12:20:37 21 other are called neurotransmitters and dopamine is one  
12:20:45 22 of the major ones.

12:20:48 23 Q. And that's what drug abuse and alcohol abuse  
12:20:50 24 affects?

12:20:50 25 A. Exactly. That's what affects it, and that

12:20:53 1 goes in the frontal lobe.

12:20:54 2 Q. So just looking at all these possible causes  
12:20:58 3 along with your probable cause, there's really no way  
12:21:00 4 to say exactly what has caused the brain damage that  
12:21:05 5 you're saying that Mr. Black has with your findings?

12:21:08 6 A. Really, there isn't. I --

12:21:11 7 Q. Now, another kind of similar but -- as far as  
12:21:15 8 timing, again your probable cause is maybe the fetal  
12:21:20 9 alcohol syndrome or lead poisoning, or something, he  
12:21:23 10 fell down, or ate dirt. You know, a lot of different  
12:21:26 11 things were mentioned in these reports that possibly  
12:21:29 12 could have caused some brain damage.

12:21:32 13 A. Uh-huh.

12:21:32 14 Q. But again, with timing, is there any way to  
12:21:35 15 tell exactly what time in his life that this happened?

12:21:41 16 A. No. The only --

12:21:44 17 Q. I'm sorry. Go ahead, Doctor.

12:21:45 18 A. What you can say is that this kind of a brain  
12:21:54 19 doesn't happen overnight. Doesn't even happen to an  
12:21:57 20 adult if they go on a ten-year drinking binge or have  
12:22:04 21 been in a major car accident and coma. Well, if they  
12:22:10 22 have been in prolonged coma and came out and survived  
12:22:14 23 it, you could see a brain like that. So had I had the  
12:22:21 24 history of a severe head injury with loss of  
12:22:27 25 consciousness and coma after age 18, then I would have

12:22:35 1 added it to a possible cause for this.

12:22:39 2 Q. I found a slide you did, and correct me if my  
12:22:44 3 pronunciation is bad, I'm sorry, myelination?

12:22:47 4 A. Yes.

12:22:47 5 Q. And pruning. It's very interesting. And you  
12:22:50 6 correct me if I'm wrong, you said it's mostly complete  
12:22:54 7 at approximately 21 to 23. Then on both of those  
12:22:58 8 graphs you had one of them maybe slightly, or ever so  
12:23:02 9 slightly went up and the other one slightly, or ever so  
12:23:06 10 slightly went down. So it's mostly complete at 21 to  
12:23:10 11 23, but it's never really complete until, I guess until  
12:23:13 12 we pass on?

12:23:14 13 A. Yeah. You can see it that way, but what you  
12:23:18 14 see is a different process taking place. This is no  
12:23:21 15 longer pruning when you have the loss of gray matter  
12:23:25 16 after age 21. This is the real thing. This is the --  
12:23:29 17 you're beginning to lose real branches from the tree  
12:23:33 18 that you don't want to lose. This is part of what  
12:23:37 19 happens with aging. Neurons get old and eventually run  
12:23:42 20 out of steam and expire.

12:23:44 21 Q. That could be the result of, say, a string of  
12:23:47 22 minor head injuries or alcohol abuse, any one of those  
12:23:51 23 things?

12:23:51 24 A. No. That -- that's -- what happens after can  
12:23:57 25 -- you can be a perfectly healthy person and it will

12:24:00 1 happen.

12:24:00 2 Q. And as those --

12:24:01 3 A. And it gets worse after 45.

12:24:04 4 Q. And as that pruning goes on, that's when --

12:24:07 5 A. Well, but that's no longer pruning.

12:24:09 6 Q. That's just natural?

12:24:10 7 A. That's now atrophy due to senesis.

12:24:16 8 Q. So it's the atrophy that can be hurried up by

12:24:19 9 drug abuse?

12:24:19 10 A. Sure. Yes.

12:24:20 11 Q. And as that goes on, when those brain cells

12:24:23 12 are gone, the fluid replaces that?

12:24:27 13 A. Exactly. Exactly right.

12:24:29 14 Q. Okay. Thank you. Just wanted to make sure I

12:24:32 15 had that right.

12:24:33 16 A. You did.

12:24:33 17 Q. A few more questions.

12:24:34 18 A. And it's the same principal. There is a

12:24:37 19 principal in medicine which is that -- in development

12:24:41 20 which is that whatever comes on board last jumps off

12:24:46 21 first.

12:24:47 22 Q. Okay.

12:24:47 23 A. I guess it works in politics, as well.

12:24:50 24 Q. Counting last in, first out?

12:24:52 25 A. Right.

12:24:53 1 So the frontal lobes myelinate first --  
12:24:58 2 myelinate last, and they begin to deteriorate first,  
12:25:03 3 particularly in men. That's one sex difference that's  
12:25:07 4 quite pronounced, that men begin to lose frontal lobe  
12:25:09 5 tissue.  
12:25:12 6 Q. Once that myelination pruning stops between 21  
7 and 30 -- I mean, 21 and 23, that's also the first area  
12:25:14 8 to start --  
12:25:20 9 A. Going down.  
12:25:21 10 Q. In your report you mentioned about when you  
12:25:23 11 have somebody with these findings, under behavioral  
12:25:27 12 consequences, I believe it was, they would have an  
12:25:30 13 inability to control emotion, aggressive impulses,  
12:25:34 14 impaired ability to resolve conflict, aggressive  
12:25:40 15 behavior. Would that type of person have the ability  
12:25:45 16 to hold a steady job?  
12:25:54 17 A. Steady, yes. If the job -- the difficulties  
12:26:02 18 they have is in adaptation. So it's the same -- I  
12:26:07 19 mean, the classic situation, you see that with  
12:26:11 20 Parkinson's Disease. If you know someone with  
12:26:13 21 Parkinson's Disease, it affects exactly the same  
12:26:18 22 regions. It's a disease of the dopamine. But this  
12:26:21 23 time, there is not enough dopamine. And you take a  
12:26:26 24 Parkinsonian patient, okay, they can start walking.  
12:26:28 25 You have to sort of get them to make the first step,

12:26:31 1 but once they make the first step, they will walk fine,  
12:26:35 2 and then their problem will be to stop walking. You  
12:26:37 3 have to sort of stop them because they'll continue  
12:26:41 4 walking.

12:26:41 5 So the adaptability, being able to change your  
12:26:43 6 behavior, is really the hallmark of what the frontal  
12:26:46 7 lobe does, to adapt your behavior to the requirement of  
12:26:50 8 the results.

12:26:50 9 Q. What about the adaptations it would take to  
12:26:54 10 simultaneously date a number of women and keep them all  
12:26:59 11 satisfied; wouldn't that take a high level of adaptation?

12:27:04 12 A. I wish I could answer from personal  
12:27:07 13 experience.

12:27:09 14 Q. Let me go into a similar example.

12:27:12 15 How about to be the go-to person for your  
12:27:15 16 friends to get advice on how to handle their  
12:27:19 17 relationships? That doesn't seem to be consistent with  
12:27:22 18 somebody that would have an impaired ability to resolve  
12:27:26 19 conflict, to be the go-to person for your friends?

12:27:31 20 A. Well, assuming this is the case, I sometimes  
12:27:39 21 -- when I need advice, I go to people who are least  
12:27:43 22 likely to say anything when I talk because, really,  
12:27:52 23 when you go -- most people really know what is good for  
12:27:58 24 them, and going to ask for advice is more to have a  
12:28:01 25 sounding board, really, than to have someone summarize

12:28:08 1 the whole situation for you and tell you, do this.

12:28:10 2 Q. So you want somebody that is going to listen

12:28:13 3 rather than act on impulse, for example?

12:28:16 4 A. I would -- I would -- yeah. I mean, if I'm

12:28:21 5 about to do something stupid, I'll go talk to somebody

12:28:23 6 but I won't expect them to say anything.

12:28:26 7 Q. Okay. Now back to this particular situation

12:28:29 8 of Mr. Black and the PET scans and MRI results, and I

12:28:33 9 believe there is one other test with the rainbow-

12:28:37 10 looking chart that we had. Could a person with those

12:28:41 11 scans and tests score, say, 100 on an IQ test?

12:28:47 12 A. I really don't know. I think it's possible in

12:28:55 13 the way that I explained before, which is that they

12:29:00 14 have developed some areas of competence. And since the

12:29:05 15 IQ score is a fruit salad, you mix a whole bunch of

12:29:10 16 things. You know, if you have a lot of -- if you have

12:29:19 17 a lot of raisins, then when you make a fruit salad, it

12:29:26 18 tastes okay. You know, it looks like it's decent.

12:29:29 19 Q. Really hard to say -- it's hard to say what

12:29:32 20 somebody is going to score on an IQ test just looking

12:29:35 21 at the -- you have to look at the scan?

12:29:38 22 A. Exactly. It's a number that, really,

12:29:41 23 psychologists, well-trained psychologists will only

12:29:46 24 utter, literally, when there is a gun to their head.

12:29:50 25 Q. Now are you familiar with the -- I know you

12:29:52 1 mentioned DSM-IV, I -- I've come to understand that's  
12:29:56 2 the bible when it comes to mental illness or disorders?  
12:29:59 3 A. Well, given that we are now writing the DSM-V  
12:30:06 4 and I have no -- no pretense of writing a bible -- I  
12:30:15 5 think bible is a word that should be reserved  
12:30:20 6 Q. Excuse me?  
12:30:20 7 A. And DSM is not the bible for me.  
12:30:26 8 Q. A book that everybody respects what's in it?  
12:30:29 9 A. Well, it's -- the main contribution is, it  
12:30:33 10 let's a lot of people who were trained in different  
12:30:36 11 places talk the same language and know what they are  
12:30:39 12 talking about. That's pretty much its contribution,  
12:30:42 13 and I don't know want to minimize it. It's tremendous.  
12:30:45 14 But taking that beyond and saying it's -- you know,  
12:30:49 15 science doesn't work that way. I mean, law maybe does,  
12:30:53 16 but not science.  
12:30:54 17 Q. Are you familiar with the AAMR?  
12:30:56 18 A. Sure.  
12:30:57 19 Q. Now both of those organizations, or publishing  
12:31:02 20 companies, however you want to put it, they have  
12:31:06 21 definitions for mental retardation. Three-pronged,  
12:31:10 22 generally, the IQ score, deficits and behavior, and  
12:31:16 23 have to have an onset by the age of 18.  
12:31:20 24 A. Uh-huh.  
12:31:20 25 Q. Here it appears, just by looking at MRI scans

12:31:25 1 and PET scans, just using those alone, it's not -- you  
12:31:29 2 don't have an ability to determine whether or not  
12:31:32 3 somebody is mentally retarded?

12:31:34 4 A. Absolutely agree with you.

12:31:36 5 MR. DONNELLY: That's all.

12:31:37 6 MR. DAWSON: I just have a couple  
12:31:41 7 re-direct questions.

8 RE-DIRECT EXAMINATION

9 QUESTIONS BY MR. DAWSON:

12:31:48 10 Q. You were asked questions about the effects,  
12:31:53 11 really, of incarceration on the brain damage that we  
12:31:57 12 see in Byron Black's case. Is there anything to  
12:32:01 13 suggest that incarceration, itself, would lead to the  
12:32:05 14 type of brain damage that you see in this particular  
12:32:07 15 instance?

12:32:11 16 A. I know of no evidence that incarceration is  
12:32:20 17 associated with changes in brain structure. Now there  
12:32:26 18 is very little data on inmates. For various reasons,  
12:32:34 19 inmates -- I remember when I did my internship in the  
12:32:40 20 State Prison of Southern Michigan in Jackson. It's the  
12:32:44 21 largest indoor prison in the world, I believe. But it  
12:32:50 22 was dwarfed by the campus of drug company facilities  
12:32:56 23 that surrounded it because at that time prison inmates  
12:33:00 24 were the favorite research participants in drug trials.  
12:33:04 25 And that was part of their parole. Being able to prove

12:33:09 1 that they were good citizens was volunteering for those  
12:33:14 2 studies. And then this whole campus vanished. This  
12:33:21 3 changed with the new laws. And the downside of that is  
12:33:23 4 that there is no science, really much done on  
12:33:26 5 incarcerated people.

12:33:30 6 But I think you can, if you're talking about  
12:33:33 7 institutionalization effects, that we have a lot of  
12:33:38 8 data on. So just keeping someone in an institution  
12:33:43 9 where they are treated humanely, like a state hospital,  
12:33:50 10 we have data on that, and we have a lot of MRI studies  
12:33:55 11 done on patients in state hospitals and the damage that  
12:34:01 12 you see in schizophrenia, you see, from the get-go.  
12:34:05 13 You see, in fact, even in some members of the family  
12:34:08 14 who don't have schizophrenia, and there is no -- no  
12:34:12 15 evidence that there is any additional damage that can  
12:34:17 16 be related to the fact that they were  
12:34:22 17 institutionalized.

12:34:22 18 Q. Also, the State went over with you several  
12:34:27 19 things that might cause damage, and one of them they  
12:34:31 20 said was syphilis or other sexually transmitted  
12:34:35 21 disease. When you reviewed the record in Mr. Black's  
12:34:38 22 case, did you find any evidence that there was ever any  
12:34:41 23 syphilis or sexually transmitted disease?

12:34:44 24 A. No, I didn't.

12:34:45 25 Q. And they also asked you about car accidents,

12:34:51 1 or after age 18 -- was there any evidence of any major  
12:34:55 2 car accident with Mr. Black after age 18?  
12:34:58 3 A. There was nothing about a car accident after  
12:35:02 4 age 18.  
12:35:02 5 Q. In terms of other trauma to the brain that may  
12:35:04 6 have occurred in prison, was there any evidence of any  
12:35:07 7 fights or serious injuries that Mr. Black had had while  
12:35:09 8 in prison?  
12:35:10 9 A. None that I could see in the records.  
12:35:14 10 Q. And he also asked you -- and he kept using the  
12:35:30 11 word, fetal alcohol syndrome. Fetal alcohol syndrome  
12:35:34 12 is the particular -- well, syndrome, I guess, it's been  
12:35:38 13 described, it has certain facial appearances and other  
12:35:42 14 situations. And in terms of talking about Mr. Black,  
12:35:48 15 are we talking about fetal alcohol syndrome or fetal  
12:35:52 16 alcohol effect?  
12:35:53 17 A. The latter.  
12:35:55 18 Q. Okay. The difference of those is what, in  
12:35:59 19 terms of -- when we talk about that class of damage?  
12:36:02 20 A. I think it's another example of how medicine  
12:36:10 21 is struggling to -- in the history of medicine you see  
12:36:13 22 a group of symptoms that go together and you believe  
12:36:16 23 that they go together to the point that you call it a  
12:36:19 24 syndrome. And then you really don't have a diagnosis  
12:36:24 25 until you have the biopathology or the neuropathology

12:36:31 1 or whatever the pathology is that is responsible for  
12:36:35 2 it. And when we are lucky, a syndrome has a pathology  
12:36:40 3 and then we have an illness and then we can start  
12:36:44 4 treatment. But this is really the exception rather  
12:36:47 5 than the rule.

12:36:48 6 It turns out that the brain doesn't work that  
12:36:52 7 way. And when you have damage, then when -- but there  
12:36:57 8 is a situation of anoxia, most people will have a  
12:37:02 9 characteristic progression of damage. But the same  
12:37:10 10 amount of damage can cause quite different effects on  
12:37:17 11 different people and different -- you know, different  
12:37:23 12 amount of damage and different types of damage would  
12:37:27 13 cause the same symptom.

12:37:29 14 So you may break a leg because you have a bone  
12:37:36 15 disease or you may break a leg because you are too  
12:37:41 16 adventurous skiing. The pathology underlying is  
12:37:46 17 different, but the end result is the same. You have to  
12:37:50 18 put a cast. You have to walk with -- see that tangle  
12:38:03 19 in my temporal lobe? What are they called that you  
12:38:05 20 have to walk with?

12:38:05 21 COURT REPORTER: Crutches.

12:38:07 22 THE WITNESS: Right. Crutches.

12:38:12 23 So when we talk about things like mental  
12:38:14 24 retardation, we try to come up -- and this is really  
12:38:17 25 what all those position papers, DSM's are trying to do

12:38:19 1 is come up with, let's agree that this is what we call  
12:38:26 2 that. But just saying, let's agree that this is what  
12:38:29 3 we call that, doesn't make that a coherent illness with  
12:38:35 4 one etiology and one form of treatment, or even the  
12:38:41 5 same outcome. And I can say that there is one thing  
12:38:46 6 that is common to all those position papers and  
12:38:52 7 definitions that have been proposed throughout the  
12:38:55 8 history of medicine, and that is that they all proved  
12:38:59 9 out -- proved to be false. So I mean, this specific  
12:39:05 10 one may be the one exception but I rather doubt it.

12:39:09 11 BY MR. DAWSON:

12:39:09 12 Q. But they had -- in terms of the alcohol damage  
12:39:14 13 to the brain, is it fairly well-accepted now that  
12:39:18 14 drinking by a mother, especially large dose drinking  
12:39:24 15 on various occasions, not necessarily all the time, but  
12:39:28 16 periodically during the pregnancy causes specific  
12:39:30 17 damage to the brain?

12:39:31 18 A. If I were giving advice to a pregnant woman, I  
12:39:34 19 would say no alcohol, at all, if you can tolerate it  
12:39:38 20 for the nine and a half months or so.

12:39:41 21 Q. And you were asked questions about this phone  
12:39:43 22 conference that we had that created a difference  
12:39:47 23 between what is now Exhibit 10, which was the report on  
12:39:52 24 the summary of the qMRI Quantitative Analysis for Byron  
12:39:54 25 Black that was dated March 17, 2004, and the prior

12:40:04 1 addition of that report that's now Exhibit 15, I think,  
12:40:10 2 -- or 16 to this deposition.

12:40:16 3 In that conference, is it not true that also  
12:40:20 4 was discussed, the confirmation for your arrival in  
12:40:24 5 Nashville and --

12:40:25 6 A. Yeah. In fact, I remember that conference and  
12:40:28 7 I'm trying to reconstruct it. It happened in the  
12:40:32 8 middle of so many other things happening. I think you  
12:40:37 9 even caught me at the time when I said, well, I'll get  
12:40:44 10 it out of the way because you know if I have him call  
12:40:48 11 me again, then taking more time. So, I'm sorry. I  
12:40:54 12 don't have a very vivid recollection of what we  
12:40:58 13 discussed.

12:40:59 14 But, of course, the main concern was the  
12:41:02 15 travel. And I remember you had -- although I didn't  
12:41:09 16 remember you -- you had some questions about the  
12:41:16 17 report, and I'm trying to remember their vain, but they  
12:41:23 18 were little questions and there was one thing that I  
12:41:30 19 changed. But frankly, this wasn't what I thought. I  
12:41:33 20 think what happened with this alcohol syndrome was when  
12:41:36 21 I said, all right, I'll incorporate what he said and  
12:41:40 22 I'll sit down and write the final draft. And while  
12:41:43 23 doing that, I read in my -- I thought that maybe I  
12:41:52 24 didn't sufficiently clarify what I thought was going  
12:41:56 25 on.

12:41:56 1 Q. And in developing this report and the work  
12:41:59 2 that you did, at times you also talk with Dr. Globus;  
12:42:02 3 correct?  
12:42:02 4 A. Yes.  
12:42:03 5 Q. And you also briefly talked to myself, or  
12:42:08 6 other people --  
12:42:13 7 A. Right.  
12:42:13 8 Q. And you had previously mentioned the serious  
12:42:16 9 concern about fetal alcohol as being the cause of the  
12:42:19 10 damage?  
12:42:19 11 A. Yeah.  
12:42:20 12 Q. Let me ask you a little bit about the role --  
12:42:26 13 A. And that was actually before those Bookstein  
12:42:31 14 studies came out.  
12:42:32 15 Q. The role of the myelination, that also, does  
12:42:40 16 it not, protects the brain from damage once the  
12:42:44 17 myelination takes place? So the frontal lobe, prior to  
12:42:50 18 myelination, is more susceptible to damage if it's  
12:42:51 19 involved in a collision or something of that nature?  
12:42:54 20 A. I don't think so. I don't know the evidence  
12:42:58 21 for that.  
12:42:58 22 Q. Okay.  
12:42:58 23 A. Of course, fat is more robust than the tissue  
12:43:04 24 of neurons. So it's an interesting hypothesis, but I  
12:43:11 25 don't know of any evidence for that.

12:43:14 1 Q. And you also indicated -- you were asked by  
12:43:17 2 the other side about using significant amounts of  
12:43:21 3 alcohol since, you know, in adulthood if that would  
12:43:27 4 cause the type of damage as you're seeing now in  
12:43:30 5 Mr. Black. And then you had indicated that, you know,  
12:43:35 6 that in adults there actually is some regeneration in  
12:43:39 7 the frontal lobe once they stop drinking.

12:43:41 8 A. Uh-huh.

12:43:42 9 Q. So Mr. Black has been basically in prison, or  
12:43:46 10 incarcerated since 1987, and at least on death row, we  
12:43:49 11 assume he has not had significant alcohol intake, no  
12:43:52 12 write-ups for that, certainly. That that would  
12:43:55 13 actually be a possibility, that there could be  
12:43:57 14 regeneration if that were the -- was the cause of  
12:44:00 15 damage?

12:44:00 16 A. Yes. I mean, when the first studies came out  
12:44:04 17 showing this regeneration, they were met with universal  
12:44:08 18 skepticism and almost disbelief. And then they kept  
12:44:17 19 coming out, and I think it's now fairly accepted. I've  
12:44:22 20 been convinced by the studies I've seen that there is a  
12:44:25 21 fair amount of regeneration. Really, has only been  
12:44:29 22 shown with alcohol. Both the loss of frontal lobe  
12:44:34 23 tissue and with abstinence, regeneration of some of it.

12:44:50 24 Q. And in terms of, you were asked about various  
12:44:53 25 IQ scores, etcetera. Is it fair to say that from your

12:45:01 1 knowledge of the neuroimaging that was done, or the  
12:45:06 2 brain imaging that you conducted, and also the other  
12:45:09 3 testing and the review of the data, that Mr. Black's  
12:45:15 4 brain is profoundly impaired?

12:45:17 5 A. Yes.

12:45:18 6 Q. And that it's impaired in areas that deal with  
12:45:23 7 how someone functions and adapts to their environment?

12:45:27 8 A. Yes.

9 MR. DAWSON: No further questions.

10

11

RE-CROSS EXAMINATION

12:45:29 12 QUESTIONS BY MR. DONNELLY:

12:45:30 13 Q. One last question, Dr. Gur. I'm sorry.

12:45:34 14 Back to Wednesday, that's the date both those  
12:45:37 15 reports were written, did you speak to Dr. Globus  
12:45:39 16 between the time of the first report and the second  
12:45:42 17 report --

12:45:42 18 A. No --

12:45:42 19 Q -- or was that some other time?

12:45:43 20 A. I spoke with him -- I actually spoke with him  
12:45:51 21 before he testified --

12:45:52 22 Q. Okay.

12:45:55 23 A. -- in order -- he had some questions and he  
12:45:58 24 thought he would present the PET results and had some  
12:46:03 25 questions about them.

12:46:04 1 And then after he testified, he sent me an  
12:46:08 2 e-mail inviting me to talk with him on the phone about  
12:46:16 3 his experience, and I thought this was a terrific idea  
12:46:21 4 and said back, "Yeah, I would be happy, and I'll call  
12:46:25 5 you." And I just never got around to it.

12:46:28 6 MR. DONNELLY: Thank you, Doctor.

12:46:29 7 THE WITNESS: So, I have not talked to  
12:46:30 8 him since.

12:46:31 9 MR. DONNELLY: Appreciate it. Thank you,  
10 sir.

12:46:33 11 MR. DAWSON: One thing, just, while  
12:46:35 12 counsel is on the record, here, just to make something  
12:46:38 13 clear, and even though this latest report says March  
12:46:44 14 17, it was actually done yesterday because that was the  
12:46:47 15 day I e-mailed it to you, correct? I was out of the  
12:46:50 16 office Wednesday, so yesterday is when I e-mailed it to  
12:46:54 17 you, and that was the day we got the second one -- I  
12:46:56 18 mean, you got it as soon as I got it.

12:46:58 19 MR. DONNELLY: I was just going by the  
12:47:00 20 day that was on the report.

12:47:00 21 MR. DAWSON: I'm just saying, for the  
12:47:01 22 record, saying that it was actually the 18th, not the  
12:47:03 23 17th.

12:47:03 24 MR. DONNELLY: Thank you, Mr. Dawson.

12:47:05 25 VIDEOGRAPHER: This concludes the

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deposition. The time is 12:46 p.m.

FURTHER DEPONENT SAITH NOT.

C E R T I F I C A T E

1  
 2 I, Florence Kulbaba, Court Reporter and Notary  
 3 Public, State of Tennessee at Large, do hereby certify  
 4 that I recorded to the best of my skill and ability by  
 5 machine shorthand the deposition contained herein, that  
 6 same was reduced to computer transcription by myself,  
 7 and that the foregoing is a true, accurate, and  
 8 complete transcript of the deposition testimony heard  
 9 in this cause.

10 I further certify that the witness was first  
 11 duly sworn by me and that I am not an attorney or  
 12 counsel of any of the parties, nor a relative or  
 13 employee of any attorney or counsel connected with the  
 14 action, nor financially interested in the action.

15 This 7<sup>th</sup> day of April, 2004.

16  
 17 Florence Kulbaba  
 18 Florence Kulbaba

19  
 20 My Commission Expires:  
 21 December 9, 2007

