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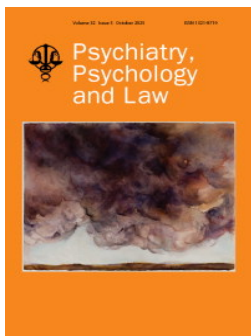
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Forensic neuroimaging in childhood abduction, rape and murder case and trial outcome

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Impulse control can be affected by factors including neurological disorders or developmental experiences. As such, a combination of neurodegenerative disorder, neurodevelopmental disorder, and childhood abuse can potentially lead a person to commit violent crimes. In this case report, we discuss the case details, biopsychosocial history, and neuroimaging of 61-year-old Donald Smith who was convicted for kidnapping, sexual assault, and murder of an eight-year-old girl. Using quantitative volumetric analysis, fluorodeoxyglucose positron emission tomography, and diffusion tensor imaging, we found neurologic evidence consistent with traumatic encephalopathy syndrome, autism spectrum disorder and neurological sequelae of adverse childhood experiences, which aligned with Smith's neuropsychiatric testing and reported personal and medical history. Although evidence of neurological structural abnormalities has influenced mitigations in previous homicide trials, in this case, Smith was sentenced to death. We explore neurological underpinnings that could have led to his deviant behaviour, along with a discussion regarding reasons behind the final verdict.

Keywords: Neurolaw; paediatric sex offender; diffusion tensor imaging; positron emission tomography; traumatic encephalopathy syndrome; autism spectrum disorder; adverse childhood experiences.

Article History: Received 17 September 2024; Accepted 14 August 2025

Introduction

On 21 June 2013, eight-year-old Cherish Perrywinkle was kidnapped, sexually assaulted, and murdered. The perpetrator was 61-year-old Donald Smith. The pictures shown of Perrywinkle's body during Smith's trial made many jurors turn away and beg the question of just how a person could have it in them to commit such an act. One might consider, even, that there must be brain abnormalities present in someone willing to partake in the aforementioned crimes. With the advent of modern technology, we are able to explore such thoughts. Thus, this case report will focus on the various pathologies that are

consistent with the brain findings present in the indicted Donald Smith.

Neuroscience has had a place in the courtroom since the early 1900s, with electroencephalogram (EEG) first being used in 1940, in a case where the defendant had epilepsy. In 1981, John Hinckley, who assassinated President Ronald Reagan, was found not guilty by reason of insanity, with evidence from computed tomography (CT) scans used to argue that he had schizophrenia. There exist many more examples where evidence of mental illness or brain abnormalities led to mitigation of legal decisions (1). As neuroscience has progressed, different neuroimaging techniques used in the legal context have also

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progressed and now include fluorodeoxyglucose positron emission tomography (FDG-PET, PET for short), diffusion tensor imaging (DTI), and magnetic resonance imaging quantitative volumetrics (MRI QV). PET is a technique that utilises measurement of glucose uptake and metabolism to extrapolate functional processes in the brain. It has been shown to detect differences after traumatic brain injuries (TBI) (2). MRI DTI is a non-invasive imaging technique that measures water diffusion to analyse the properties of biological tissues and track white matter pathways; it has also been used to elucidate neuropsychiatric disorders. MRI DTIs measure fractional anisotropy (FA), designated a value between 0 and 1, which represents the integrity of the white matter tracts (with 1 being the highest integrity) (3,4).

QV allows measurement of different brain structure volumes with high accuracy and has been used for identifying brain atrophy in TBI (5,6). It can also be used to assess white matter hypointensities (WMH) which are believed to signify malformations in neurodevelopment and are increased in autism spectrum disorder (ASD) (7).

Brain imaging using the above studies has influenced court decisions in many homicide cases by providing evidence of structural brain abnormalities such as TBI, emotional neglect, and foetal alcohol syndrome (5,8). Particularly, it is important to assess for structural brain abnormalities consistent with published findings for ASD in the courtroom because individuals with ASD sometimes do not realise their sexual behaviour may be unethical (9,10). In many cases, an extreme preoccupation with a specific interest, rather than intent to commit crime, results in criminal behaviour in those with ASD (11). The obsession can also have a negative influence on understanding social cues, impulse control, and empathy, all of which can help explain sexual misconduct in the ASD population (9). Furthermore, if the obsession is of a sexual nature, this may also contribute to sexual offences (11). These explanations suggest that

increasing courtroom knowledge about the unique challenges and behaviours people with ASD face could help jurors make more educated decisions during trials. Historically, ASD is a behavioural syndrome that is diagnosed with behavioural assessments, but research has been finding a place for neuroimaging as a tool to support structural identification of the autistic brain (12,13). Thus, in terms of ASD, TBI, and other brain abnormalities, neuroimaging evidence has utility in exploring aberrant behaviour of defendants and influencing outcomes of a court decision. This case report explores one such instance where neuroimaging, in corroboration with clinical and behavioural history, can help explain the lack of impulse control and other psychological and neurological abnormalities that ultimately led to the kidnap, murder, and sexual assault of a minor by defendant Donald Smith. All information discussed is public record due to Florida's sunshine law and was publicly disclosed at trial and available to any interested party and therefore not subject to the Health Insurance Portability and Accountability Act (HIPAA) regulations.

Case details

Cherish Perrywinkle, 8 years-old, was kidnapped from her family at a local Walmart on 21 June 2013, by Donald Smith, age 61. Earlier that day, the older man had seen Rayne Perrywinkle with three of her daughters at the dollar store. After watching her put back clothes that she could not afford, he offered to purchase the girls clothes at Walmart, saying his wife would meet them there with a \$150 gift card (14).

The four entered Walmart first and as they were shopping, Rayne noticed some disturbing behaviour from Smith. For example, he suggested that Cherish buy heels that were even taller than ones Rayne herself would wear. At around 10.30pm, Smith offered to buy the girls McDonald's from inside the Walmart as they were hungry. Cherish followed him there, and moments later the two are seen on surveillance camera leaving the store. That was the last time Cherish was seen alive (15). About

20 minutes later, Rayne realised the McDonald's in the store was already closed and began to panic. At 11.18pm she called 911 and reported Cherish missing (16). Smith's wife never showed up with the gift card.

On 22 June 2013, at 9.05am, Smith was spotted driving his white van and taken into custody. He was sopping wet from the waist down and Cherish was not in the van. Fifteen minutes later, Cherish's body was found by the K9 team (16). She was submerged in water in a marshy area behind a church, her body inflicted with multiple injuries, including evidence of strangulation, rape, sodomisation, and blunt force trauma. Smith was a face that the police knew well – he was a registered sex offender with a history of lewd behaviour towards a minor and attempted kidnapping. In fact, he had been released from jail only 21 days earlier (14). He was indicted on 22 June 2013, for kidnap, murder, and sexual assault of a minor.

Donald Smith's trial began on 5 February 2018. Evidence was presented, including photos of Cherish's ravaged body which caused some members of the jury to turn away or even cry (14). On 14 February 2018, after less than 15 minutes of deliberation, the jury returned a guilty verdict, and on 20 February, the penalty phase began (17). On 2 May 2018, the judge sentenced Smith to death. He filed an appeal on 23 May 2018, but this was denied by the Florida Supreme Court on 22 April 2021 (18). As of the time this paper was written, Smith is an inmate at Union Correctional Institution.

Early life

Donald Smith had many adverse childhood events. His biological father was estranged from the family before he was born and his mother remarried three times before he was 25 years old. His mother and first stepfather would physically punish him, and he felt verbally abused by two of his stepfathers. As a child, he moved around a lot, which made it difficult for him to establish meaningful relationships with anyone but his mother. He was

often bullied as the new kid and felt 'alienated, despondent, and detached'. He even resorted to paying other kids to play with their toys. When he was five years old, he had his first violent urge, and attempted to strangle a neighbour's sleeping baby, stopping when the parent entered the room. At age eight, he was molested by two older neighbourhood boys. They forced him to masturbate and perform oral sex on them, and he also witnessed them sexually molest several girls.

Smith began engaging in criminal acts at age 10, including peeping and stealing. At this point in time, he also already had homicidal urges, mostly involving strangling girls. His mother's second remarriage, when he was around 13 years-old, was a psychiatrist. Smith would steal psychotropic medications from his step-father and began to experiment with cannabis, alcohol, and tobacco. By 10th grade, he had dropped out of school and began to get arrested regularly. His drug use eventually spiralled into crack cocaine addiction, and he was especially violent when high on this substance. At approximately 20 years old, Smith hit an oak tree while driving and his car flipped over – he did not receive treatment. A year later, he was arrested for lewd and lascivious indecent assault on a child under 16 and sentenced to six years. He attempted suicide as soon as he arrived at the prison and ended up serving about one year in the Florida Department of Corrections, after which he was designated as a mentally disordered sex offender and committed to North Florida Evaluation and Treatment Centre. By the time Smith was 40 years-old, he had been charged with kidnapping, showing obscene material to a minor, and was registered as a sex offender in Florida (19).

Materials and methods

PET and DTI MRI imaging

MRI DTIs were obtained on 30 August 2016, a little more than three years after the kidnapping and homicide, at Precision Imaging

Centre on a 3 Tesla GE Signa HDxt MRI scanner with slice thickness of 5 mm in 25 gradient directions. Diffusion b-value was 1000s/mm² and matrix 128 × 128. TR/TE = 6000/88.2 and field of view 25.6 × 25.6 cm. Five mCi of fluorine-18 deoxyglucose (FDG) was administered intravenously during resting state for the FDG-PET. Scans were normalised before analysis using the Functional Magnetic Resonance Imaging of the Brain Software Library (FSL). Both MRIs were compared to those of age- and sex-matched neurologically normal controls (15 controls for PET, 42 controls for DTI obtained from the Functional Biomedical Informatics Research Network (FBIRN) data repository). Positive and negative Z-maps were constructed using Statistical Parametric Mapping (SPM) software (see Figure 1). Areas of metabolic activity and FA, respectively, in regions of interest (ROIs) and the brain as a whole were also compared for abnormalities using VINCI software (against all PET controls and select 15 controls matched more closely by sex and age for DTI). The software TrackVis was also utilised to construct a corpus callosum tractography.

Quantitative volumetrics

MRI QV was performed using CorTechs Labs NeuroQuant software (20). The volumes of various brain structures were calculated, including cerebral white matter, cerebral gray matter, cerebellar white matter, and cerebellar gray matter, and compared to healthy controls obtained from the FBIRN data repository.

Other statistical analysis

A two-step correction for statistical parametric mapping for familywise error correction was used which includes a height threshold of $p < .01$ and a second extent threshold of 30 contiguous adjointed voxels. In previous publications, an area under the curve receiver operator characteristic analysis was performed which showed that this combination of height and extent threshold has an acceptable area under

the curve, suggesting that the chance of false positives are low (21). No other commercial PET or DTI analysis software has published area under the curve receiver operator characteristic analysis for brain injury which show such high specificity and sensitivity. The familywise error correction described here was used for both DTI and PET scans.

Results

The NeuroQuant analysis of the MRIs shows a number of abnormalities in Smith's brain when compared to neurologically normal controls. One finding is abnormally increased white matter in both his cerebral cortex and cerebellum, placing him in at least the 93rd percentile for white matter volume in both the right and left hemispheres (see Table 1). A further analysis revealed that the white matter volume of his left and right cerebellum is significantly increased – a 30% increase in his left cerebellum and a 50% increase in his right cerebellum compared to controls (see Table 2, mean = 15,189.4, Smith = 20,122; mean = 15,327.6, Smith = 23,000, $p = .0113$ and $p = .00666$ respectively). When comparing the atrophy of both of Smith's brain hemispheres, it was found that there is greater right-sided than left-sided atrophy (see Table 3). The volume of the right forebrain parenchyma is smaller than when compared to the left forebrain, and this difference is significantly greater than it is in controls (mean left-right parenchyma = -0.44, Smith = 0.509998, $p = .0012$). He also has decreased total gray cerebral matter in the right hemisphere, standing at the 4th percentile (see Table 1). When examining more discrete brain structures, Smith was found to have an enlarged thalamus, amygdala, and hippocampus (Table 1). His left thalamus places at the 98th percentile while his amygdala is at the 99th percentile bilaterally. His hippocampus places in the 97th percentile in the left hemisphere and the 98th percentile in the right hemisphere.

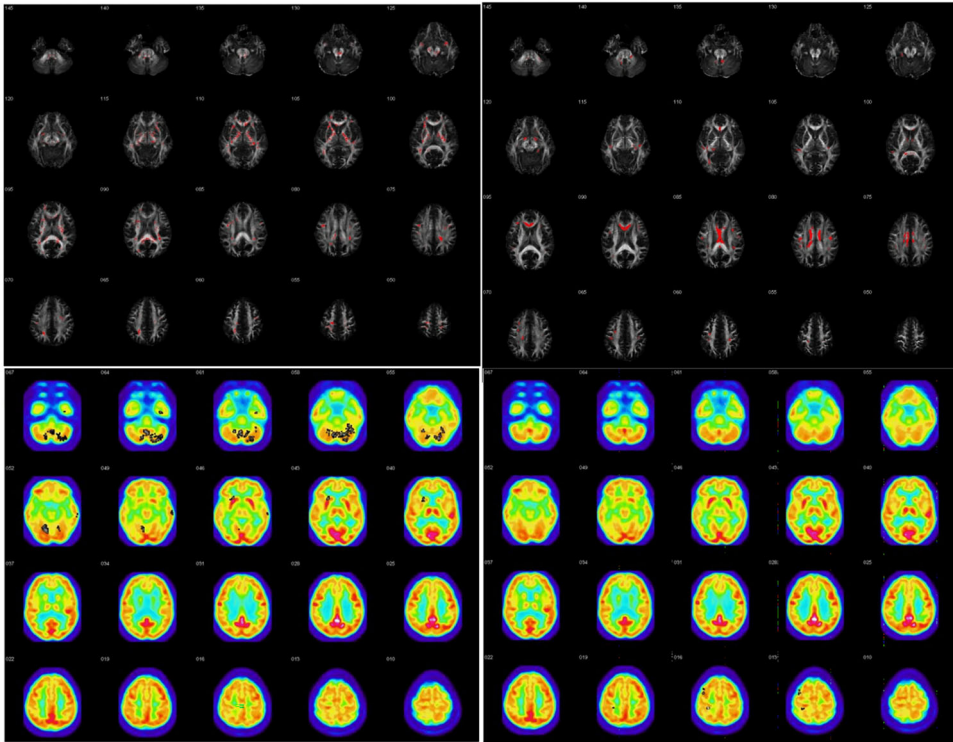


Figure 1. MRI DTI and PET Z-maps of areas of increased FA measurements (top left), decreased FA measurements (top right), increased FDG uptake (bottom left) and decreased FDG uptake (bottom right) as compared to age- and gender-matched healthy controls. Areas of possible significance are noted in red (DTI) or black (PET).

Table 1. Cerebral and cerebellar white matter volumetric comparison between Donald Smith and healthy controls as measured by NeuroQuant.

Brain structure	LH z-score	LH percentile	RH z-score	RH percentile
Total cerebral white matter	>1.65	98	1.48	93
Total cerebral gray matter	-1.55	6	<-1.65	4
Total ventricle	-0.55	29	-0.64	26
Cerebellar white matter	>1.65	97	>1.65	96
Cerebellar gray matter	0.05	52	0.33	63
Amygdala	>1.65	99	>1.65	99
Brainstem	1.64	95	1.41	92
Thalamus	>1.65	98	-0.03	49
Ventral diencephalon	1.08	86	1.41	92
Hippocampus	>1.65	97	>1.65	98

LH = left hemisphere, RH = right hemisphere. Bolded values have a z-score greater than 1.65. White matter in cerebrum and cerebellum abnormally increased. Total gray matter decreased. Enlarged amygdala and left thalamus also noted.

Table 2. Absolute volume of various brain structures as measured by NeuroQuant.

Absolute Volume (mm ³)	Smith	Mean	SD	Z-score	P-value
Left cerebellum white matter	20122	15189.4	1948.3	2.531746	1.13E-02
Left cerebellum cortex	50250	50365.5	3230.486	-0.03575	
Right cerebellum white matter	23000	15327.6	2827.706	2.713295	6.66E-03
Right cerebellum cortex	51308	52053.25	4013.68	-0.18568	
WM hypointensities	5081	1757.2	694.8876	4.78322	1.73E-06
non-WM hypointensities	10	23.5	14.11792	-0.95623	
Corpus callosum posterior	1131	975.85	188.3525	0.823722	
Corpus callosum mid posterior	524	429.45	84.52061	1.118662	
Corpus callosum central	595	433.65	67.95453	2.374382	1.76E-02
Corpus callosum mid anterior	510	452.75	90.85088	0.630154	
Corpus callosum anterior	286	896.7	152.8753	-3.99476	6.48E-05

Of note are the increases in cerebellar white matter ($p = 1.13\text{E-}02$ and $6.66\text{E-}03$ for LH and RH respectively) and the increase in white matter (WM) hypointensities ($p = 1.73\text{E-}06$).

Table 3. Relative volume of brain structures as compared to the whole intracranial volume in Donald Smith and controls as measured by NeuroQuant.

Relative (% ICV)	Smith	Mean	SD	Z-score	P-value
Left forebrain parenchyma	32.87	33.18	0.79	-0.39	
Right forebrain parenchyma	32.36	33.63	0.9	-1.41	
Left-right forebrain parenchyma	0.509998	-0.44	0.39	3.23	0.0012

Freesurfer can also be used to measure WMH as mentioned above. Quantification of Smith’s WMH revealed that he has almost three times the normal amount of WMH as compared to controls (Table 2, mean = 1,757.2, Smith = 5,081, $p = 1.73\text{E-}06$).

Measurements of FA from the MRI DTI showed that Smith had a significantly decreased FA of 0.32 in his mid corpus callosum (see Figure 2, mean = 0.63, $p = 3.8\text{E-}14$). His FA was 50% lower than when compared to neurologically normal controls. He also had a significantly increased FA of 0.67 in the right internal capsule (see Figure 2, mean = 0.59, $p = 5.8\text{E-}05$).

When looking at measurements from the MRI PET, it was found that there was a decreased neocortex to cerebellum metabolism ratio of 0.85 (Figure 3, mean = 1.04, $p = 9.2\text{E-}04$) as well as increased metabolism of 1.02 in the right hemisphere corresponding with the

right cerebrum, sub-lobar area, or claustrum (Figure 3, mean = 0.88, $p = 4.8\text{E-}03$). There is a noticeable gap in his corpus callosum tractography (see Figure 4).

Discussion

Analysis of Donald Smith’s brain revealed several abnormalities that could help explain his unusual sexual preferences and lack of impulse control. Many of his imaging results including DTI, PET, and QV analysis corroborated with a history of multiple TBIs which is associated with a high risk for traumatic encephalopathy syndrome (TES), a progressive neurodegenerative disorder caused by a history of repeated brain injury described as the clinical presentation of suspected chronic traumatic encephalopathy (CTE) (22,23). The clinical presentation of TES can include memory/attention

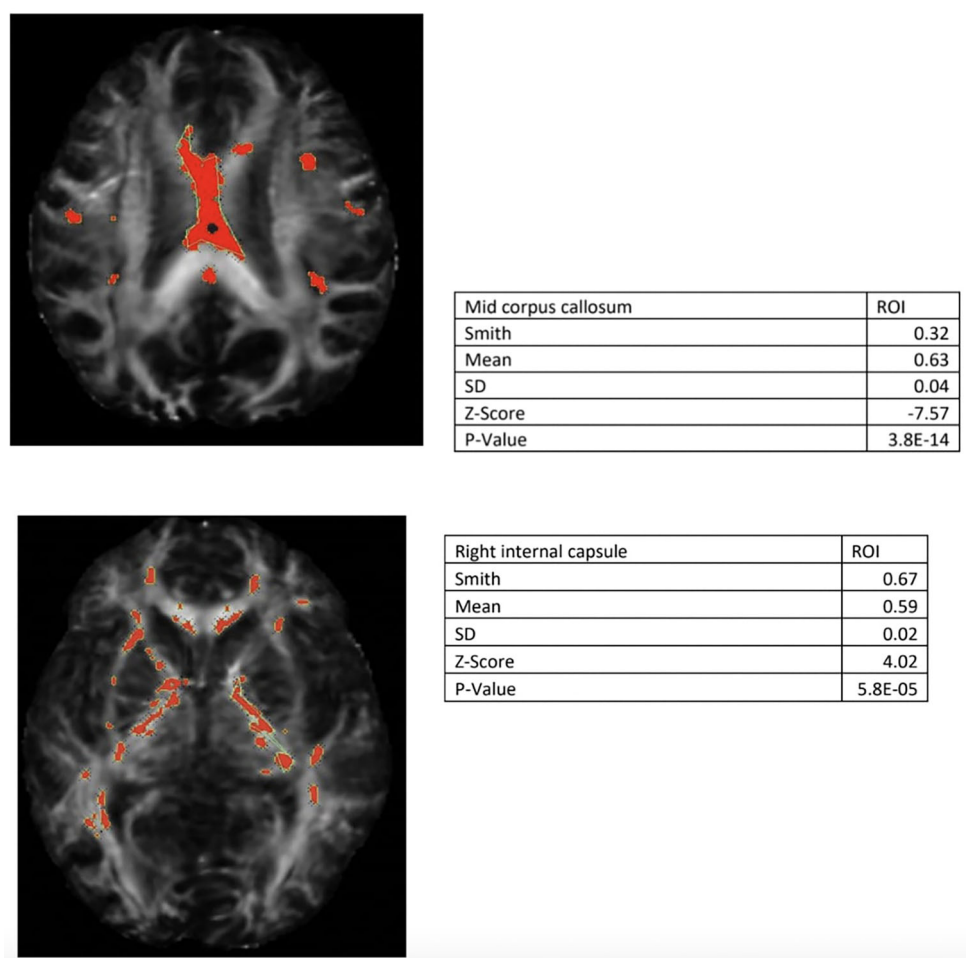


Figure 2. Analysis of FA in mid corpus callosum (top) and right internal capsule (bottom) of defendant Donald Smith. Significantly decreased FA in the red region outlined in yellow is seen in the corpus callosum ($p=3.8E-14$) and significantly increased FA in the red region outlined in yellow is seen in the internal capsule ($p=5.8E-05$).

problems, increased irritability and aggression, suicide risk, and mood disorder, mostly major depressive disorder (22). Poor impulse control and psychosis are also symptoms that have been associated with TES (24). In terms of Smith’s MRI DTI, there was significantly decreased FA in the mid corpus callosum when compared to controls (Figure 2). This finding is consistent with a 2015 study which found decreased FA in the genu of the corpus callosum when comparing patients with mild TBI to healthy controls (25,26).

Tractography of Smith’s corpus callosum (Figure 4) also shows missing white matter tracts in comparison to a neurologically normal 56-year-old male. Identification of alteration of white matter connectivity, number of fibre tracts, and average length of tracts, have been used in studies of tractography and TBI, and show corpus callosum volume and tract loss in subacute and chronic stages of TBI (27). Increased FA in the right internal capsule was also found, but in the literature, there are some mixed findings regarding FA in the

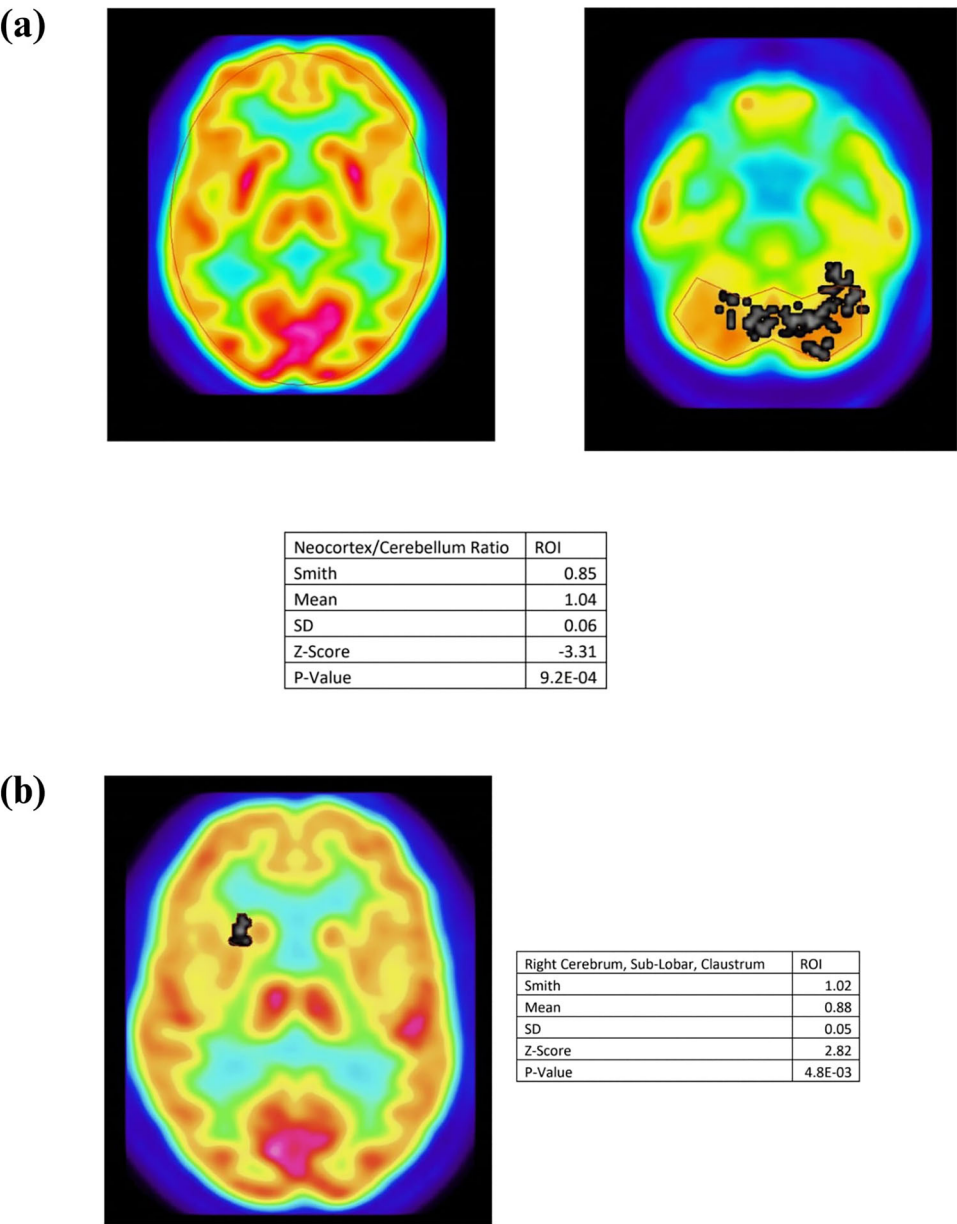


Figure 3. PET comparisons of regions of interests of the defendant to healthy controls. (a) Neocortex to cerebellum ratio. The defendant's cerebellum is shown on the right. The areas in black note areas of increased metabolic activity. The defendant's neocortex (left) does not show any change in metabolic activity from normal. The ratio between the metabolic activity of the neocortex to that of the cerebellum is significantly decreased, indicating a significant amount of metabolic activity increase in the cerebellum ($p = 9.2E-04$). (b) Single ROI comparison. The area in black notes an area of increased metabolic activity ($p = 4.8E-03$). This area corresponds to the claustrum but could also be considered as right cerebrum or sub-lobar.

internal capsule post-TBI (Figure 2). A meta-analysis conducted by Wallace found that non-penetrating/blunt head trauma resulted in decreased FA in the internal capsule greater than three months post injury, while two other studies have found an increase in FA of the internal capsule four months to two years post injury, in patients with blast-related mild TBI and in those with mild TBI with persistent cognitive impairment (28–30). Another study from Lipton et al. (31) saw both areas of low and high FA in mild TBI patients, with areas of low FA being consistent with recognised patterns of traumatic axonal injury and high FA areas in deep and subcortical white matter. However, an increased FA in the right internal capsule is also associated with depression and obsessive compulsive disorder, which might be contributing to the findings in Smith's imaging (30,32–35). Additionally, although not specific to the internal capsule, it has been shown that abnormally high FA post-TBI may be suggestive of post injury compensatory processes (36).

Aside from DTI findings consistent with multiple TBIs, Smith's QV and PET also show evidence of neurobiological underpinnings to Smith's behaviour, possibly related to TBI. As shown in Table 1, Smith's left thalamus had an increase in volume when compared to healthy controls. A 2021 study showed that longitudinal increases in brain volume occur after TBI, including in subcortical regions such as the thalamus (37). Although other studies have found that repeated head trauma is associated with smaller thalamic volumes in professional fighters, another study in 2013 found that veterans with a history of TBI plus suicidal behaviour had significant thalamic enlargement when compared to veterans with TBI and no suicidal behaviour or to healthy controls (38,39). Since Smith falls into the category of having suicidal behaviour, this study also supports the possibility that his thalamic enlargement could be connected to TBI. Smith also has decreased total cerebral gray matter, a finding that has

been found to be associated with CTE in football players (40,41). Atrophy in CTE is typically most severe in the frontal lobes (as well as the anterior temporal lobes) which could help explain the decreased neocortical to cerebellar PET ratio observed in Smith's scans (41,42). Additionally, hypometabolism in primarily the frontotemporal region has been noted in patients with TES who are at risk for CTE (43).

Neuropsychological assessment and personal history of Smith are also consistent with TES. Smith reported being in a motor vehicle accident at 20 years old and a bike accident at nine years old, either of which could have led to TBI; however, he was never treated for these two accidents so medical records are unavailable (19). Testing revealed moderate impairment in some measurements of nonverbal memory and mild to severe impairment of executive function in addition to severe depression and psychoticism (44). Smith himself also endorsed a history of depression with a suicide attempt and auditory/visual hallucinations in 2011 per released medical records. All these symptoms of mood disorder, executive function impairment, and suicide risk are consistent with a possible presentation of TES (22). Furthermore, Smith's stated history of multiple brain injuries at a young age put him more at risk for longtime neurodegeneration (45). Greater severity of TBI is associated with greater behavioural problems, and TBI in younger children results in an even more increased risk (46). Smith's sustained injuries at a young age with no appropriate care likely placed him at great risk for future behavioural problems. His history of physical and sexual abuse could also have contributed to a lack of impulse control and suicide risk later in life (47,48).

While analysis of Smith's brain is strongly consistent with CTE, other findings were also of note and can help explain his behaviour. QV analysis revealed that Smith has right-sided brain atrophy (Table 3), while PET showed increased metabolism in a brain area

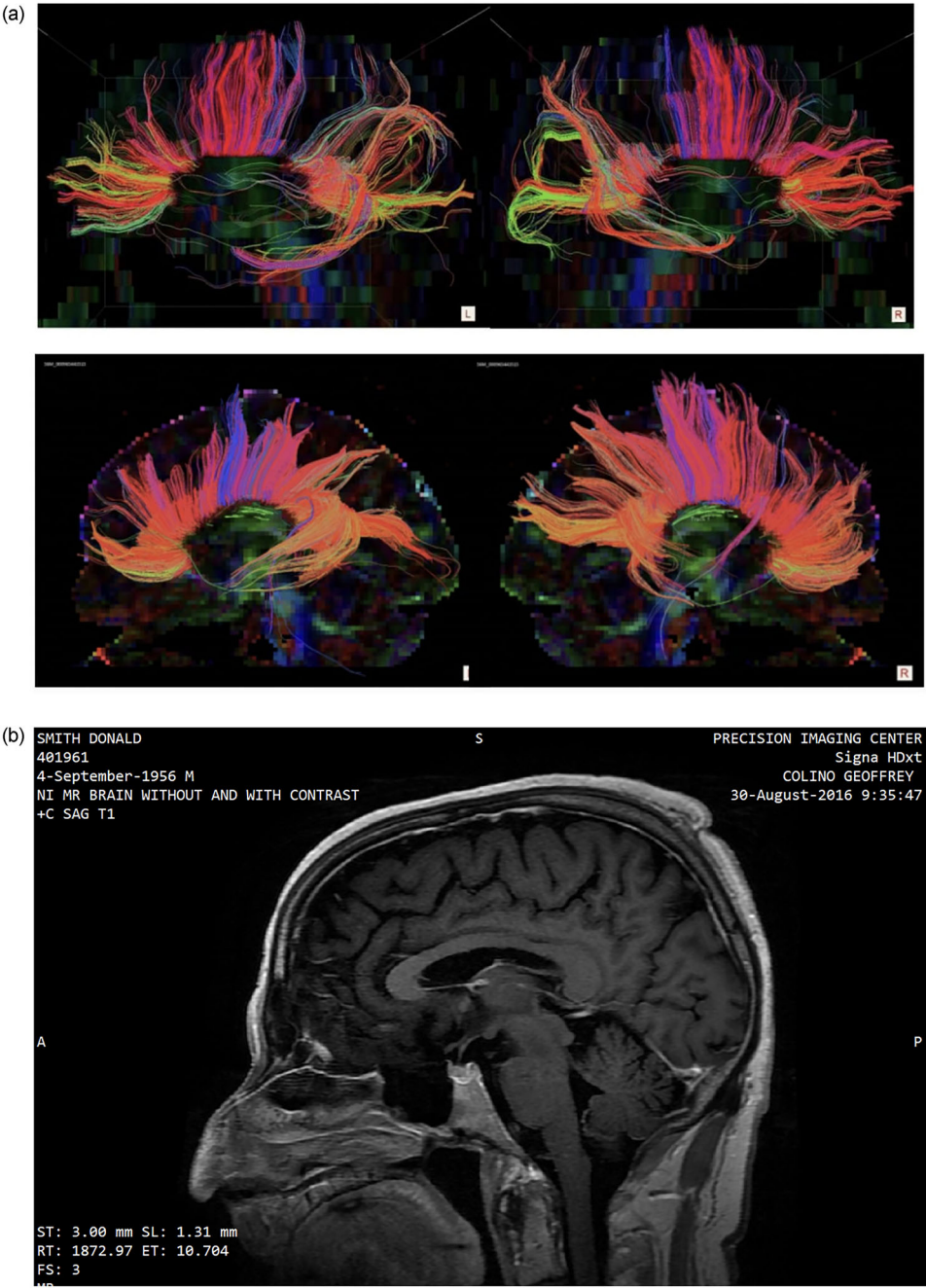


Figure 4. (a) Corpus callosum tractography of Donald Smith (top) and a normal neurological 56-year-old male (bottom). A black gap indicates areas where there are no corpus callosum fibres present, suggestive of damage or injury. (b) Mid-sagittal T1-Weighted image of Donald smith's corpus callosum.

corresponding to the claustrum (Figure 3). Severity of disinhibition in patients with neurodegenerative disease has been associated with right-sided brain atrophy and loss of regulation of social and emotional behaviour (49,50). Right temporal lobe dysfunction more than left, as measured by hypometabolism in PET scans, has also been found to correlate with paedophilia in at least two other case reports, so it is possible that Smith's right-sided atrophy suggests a structural component to his deviant sexual preferences (51). Research on the claustrum has revealed its role in sexual arousal, such as in male tactile genital stimulation, desire for sexual activity, and processing sexual arousal (52–55). Findings regarding hypermetabolism in the claustrum have also been correlated with sexual arousal, particularly the right claustrum (56,57). Furthermore, a study of neural networks and paedophilia found that there is disrupted functional connectivity in networks for sexual processing, which includes the claustrum, in paedophilia (55,58). Thus, Smith's right brain atrophy and hypermetabolism in the claustrum appear to constitute a neurologic basis for his sexual behaviour, but at this point it is difficult to ascertain whether these changes were idiopathic or acquired.

Aside from TES-related psychological disorders, there is evidence that Smith might also have other psychiatric disorders that contributed to his behaviour, namely schizophrenia and ASD. While Smith was never formally diagnosed with schizophrenia, he has a history of auditory and visual hallucinations, which is a criteria for the disorder (59). However, the decrease in FA of the mid corpus callosum (body) seen on DTI analysis of Smith's brain is actually of greater magnitude than that seen in previous DTI studies on patients with schizophrenia, and thus more likely due to TBI from the motor vehicle accident possibly causing psychosis secondary to brain injury rather than primary schizophrenia (24,60,61). Thus, DTI provides a reason to explore the possibility of schizophrenia in Smith, but in

the context of his history of TBI, congruent mood disorders, and substance use, of which cocaine is also known to cause psychosis, it is not heavily supported (62–64).

Neuroimaging corroboration with ASD in Smith's scans is slightly more substantial. On QV, Smith was found to have significantly more white matter hypointensities (WMH) than controls (Table 2). WMH have been correlated with ASD and have also been found to be increased in patients with increased fixation and repetitive behaviours (7). Smith also has increased cerebellar white matter (Table 1 and Table 2) which has been found in children with ASD. Although evidence overall appears to be equivocal, the magnitude of Smith's white matter increase is abnormal and comparable to some studies on a subgroup of ASD patients with a mutation in the PTEN gene who have abnormally (37% greater) increased cortical white matter compared to normal controls (65–69). No other commonly known neurological or psychiatric disorder is associated with abnormal increases in white matter volume besides autism. Increased white matter has also been implicated in violent offenders with antisocial personality disorder (ASPD) (70). While Smith can be said to have sociopathic tendencies, according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (71), a diagnosis of ASPD requires patients to have no underlying medical cause that could explain the behaviour, which may disqualify Smith from this diagnosis given his potential brain injuries and his potential diagnosis of ASD. Lastly, Smith's QV showed both amygdala and hippocampal enlargement (Table 1), which has been associated with ASD in various degrees of development from children to adults, although there have been mixed findings (72–76). Findings in children have been largely consistent regarding amygdala enlargement (72,73). However, many studies disagree on findings in adolescents and older age groups, with some describing continued amygdala enlargement while others find no difference in volume compared to healthy

controls (74,75). Some studies even suggest a smaller or preserved amygdala size in older children with ASD and adults with Asperger's (77,78). It has also been found that children with anxiety and ASD have smaller amygdala volumes than children with autism alone or healthy controls, so these mixed findings could partially be explained by this observation (79). Findings regarding the hippocampus are even more contrasting. For example, Groen found both hippocampal and amygdala enlargement in adolescents with ASD while Murphy found only enlarged amygdala but not hippocampus in people with Asperger's (age 12–47) (76,80). Schumann (72), on the other hand, found that children with ASD have larger amygdalas but not adolescents, while the hippocampus in both children and adolescents is enlarged. Furthermore, the relation of amygdala and hippocampal enlargement to ASD is confounded by the possibility that severe emotional neglect and early adverse experiences can also lead to the enlargement of the two structures (81–83). It appears that hippocampal, along with amygdala, enlargement is associated with physical and sexual abuse and/or neglect in youth and with early life stress in adults (84,85). Although the literature surrounding amygdala and hippocampal enlargement is not definitive for ASD and could alternatively point to a history of adverse childhood events, which Smith experienced, one could explore Smith's behaviour as being consistent with ASD. People with ASD can have obsessions of a sexual nature or paraphilic psychopathology, and Smith could be said to have had an abnormal sexual and homicidal fixation since early childhood (11,86). He also had difficulties forming relationships and had to pay other children to play with him, often feeling alienated as a child, which suggests the social impairment that accompanies ASD. While Smith's childhood history could raise suspicion for ASD according to DSM-5 criteria, at this point we lack substantial evidence to officially diagnose him, especially given that symptoms must appear in childhood

and are often missed (71,87). Were he to have ASD, it might help explain his extreme violence: although there is no evidence suggesting ASD predisposes to violence, it has been shown that among mass shooting perpetrators, a disproportionate amount had ASD (88). The small subgroup of violent offenders with ASD appears to correlate with adverse childhood experiences, which aligns with Smith's history as well (86,89).

The case of Donald Smith illustrates how the combination of childhood abuse, neurodevelopmental disorder, brain injury, and neurodegenerative disorder could possibly interact and lead to a catastrophic failure in impulse control, with all four elements alone known to be associated with impulsivity (24,90–92). However, despite the numerous brain abnormalities consistent with multiple pathologies including TES and ASD, Smith was quickly found guilty and ultimately sentenced to death. Previous studies have shown that knowledge that an aggressive person has ASD leads to more lenient sentencing, but also that other factors like judge's discretion, case details, clinical history, and expert testimony can influence mitigation (93,94). Thus, even if the jury was convinced that Smith may have had ASD, it does not necessarily equate to a mitigated sentencing. Similarly, it would be prudent to maintain caution when interpreting neurological imaging in the context of mitigation so as not to overshadow the presence of other factors such as case details, neuropsychological assessments, and clinical correlation which may provide more utility when assessed alongside neuroimaging (95). Additionally, many of the diagnoses in question are traditionally made clinically and imaging is a means of support rather than diagnostic. While there is not much literature addressing the nature of legal scrutiny of neurological data, there are some precedent cases that may help explain the final decision. In the case of Nikolas Cruz, a mass shooter, evidence of intellectual and mental deficits from foetal alcohol syndrome was enough to

pursue a life sentence rather than the death penalty, and CTE evidence influenced the mitigation phase of Raul Roque who committed two homicides (5,96). In cases of child sexual abuse alone, conviction typically results in terms of on average 14 years, with 2% receiving life (97). The death penalty seems to be reserved only for cases where a victim is killed, but criteria beyond that are broad (98). One aggravating circumstance is the killing of a child victim, typically defined as less than 14 or 13 years-old, which evokes such intense emotions that even wrongful convictions disproportionately involve deaths of children (99). From this data, it can be surmised that mitigation may be more successful in cases of murder and child sexual abuse alone, but not so much in cases of child murder. Thus, perhaps the case of Donald Smith, which contained strong evidence of violent sexual assault and murder of a young child, was just too horrific to consider letting the perpetrator receive anything less than the maximum punishment, no matter how strong the evidence of underlying illness.

Ethical standards

Declaration of conflicts of interest

Janet Nguyen has declared no conflicts of interest.

Dr Joseph Wu was the testifying expert for the public defender for Donald Smith in this case. There are no other conflicts of interest to declare.

Ethical approval

This article does not contain any studies with human participants or animals performed by any of the authors. All imaging was acquired for required clinical reasons and not for research. All clinical and imaging information were also publicly disclosed at trial and are public record according to the Florida Sunshine Law.

Supplemental data

Supplemental data for this article can be accessed online at <https://dx.doi.org/10.1080/13218719.2025.2556102>

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Data availability statement

Data is not available due to undisclosed information embedded in the DICOM metadata that cannot be released in compliance with HIPAA. All information presently discussed in this report was openly disclosed in court trial and thus is within the public domain.

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