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Current Opinion in Behavioral Sciences

Cerebellar roots of aggression in violent psychopathic offenders: evidence from structural neuroimaging studies

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Investigations of structural brain abnormalities in antisocial personality disorder and psychopathy associated with aggression have focused on prefrontal, limbic, and paralimbic regions. In this narrative review, a series of structural neuroanatomical studies are discussed, which points toward an important role of the cerebellum in antisocial and aggressive behavior. Across the reviewed studies, volumetric reduction of the vermis and right posterior cerebellum was a consistent finding in violent psychopathic offenders. The observations agree with results in healthy volunteers, which show that volumes of the vermis and right cerebellar hemisphere are correlated with impulsivity and aggressive behavior. Deviations in cerebellar volumes in violent psychopathic offenders are proposed to be part of a deficient neural circuit implicated in emotion regulation and executive functions.

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Current Opinion in Behavioral Sciences 2024, **55**:101333

This review comes from a themed issue on **Role of the Cerebellum in Cognition and Behavior**

Edited by **Sheeba.Arnold-Anteraper** and **Wietske van der Zwaag**

Available online xxxx

Received: 5 June 2023; Revised: 28 November 2023; Accepted: 4 December 2023

https://doi.org/10.1016/j.cobeha.2023.101333

Introduction

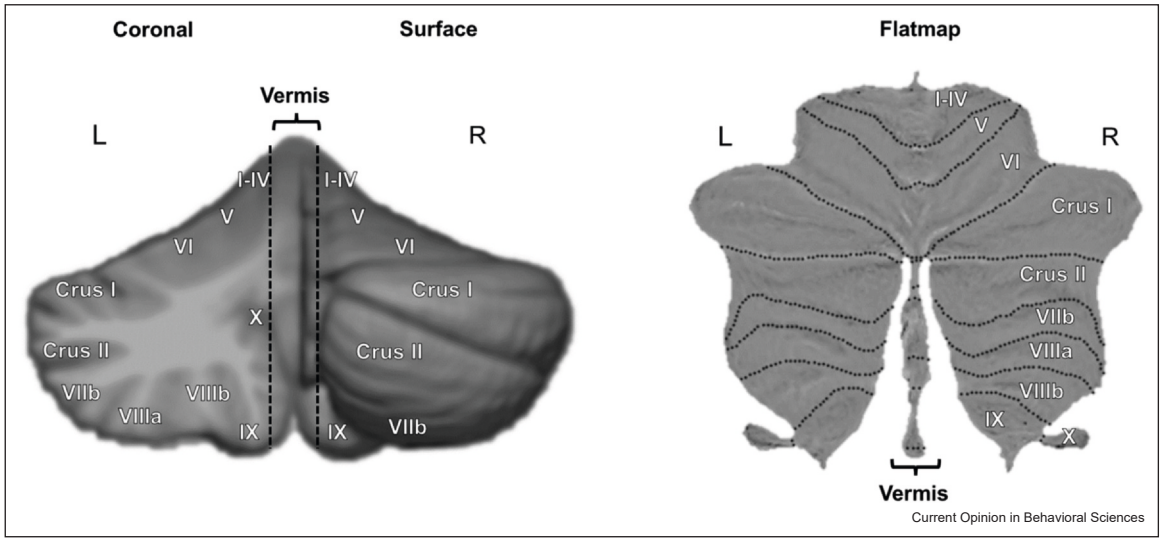
Aggression is an omnipresent behavior in the mammalian species with the evolutionary purposes of resource accessibility and dominance assertion [1]. Primates and humans display a variety of frustration- and threat-related behaviors directed at others with the intent to inflict physical or psychological pain. At the extreme of the aggression spectrum, individuals with psychopathic tendencies tend toward impulsive violence and are more likely to engage in violent crimes, such as homicide, assault, and rape, while at the same time exhibiting little-to-no remorse for their actions [2,3]. Consequently, research on psychopathy in adults has focused on the assessment of incarcerated violent offenders who are often diagnosed with antisocial personality disorder (ASPD) and, to a lesser extent, psychopathy [4,5]. The latter is most commonly assessed using the Psychopathy Checklist-Revised (PCL-R) [3] and discussed in terms of either the total score or its two underlying factors. The interpersonal-affective facet assesses individuals' selfishness, callousness, manipulativeness, and lack of empathy, while the antisocial-deviant lifestyle facet assesses impulsiveness, delinquency, and lack of behavioral control. Importantly, individuals with ASPD *and* psychopathy do not show a strong phenomenological distinction from individuals with ASPD without psychopathy [6], suggesting that psychopathy may not be a distinct disorder but rather constitutes a more extreme form of ASPD [7].

Cerebellar neuroanatomy of violent behavior

Neuroscientific research has explored whether antisocial and aggressive behaviors are associated with a particular structural anatomical composition of the brain. An extensive body of work has found evidence for reduced gray matter (GM) volume, cortical thinning, and impaired white matter (WM) connections in fronto-temporal, limbic, and paralimbic brain regions of violent psychopathic individuals [8–11]. These abnormalities have been associated with dysfunctions in emotion processing and executive functioning, and may in part underlie behaviors resulting from, for example, suboptimal decision-making, impulsivity, increased reward sensitivity, and deficits in affective empathy [2,12].

In addition to the proposed deficits in cortico-subcortical circuits, converging evidence from animal and human studies points toward a role of the cerebellum in driving antisocial and aggressive behavior [13–16]. The cerebellum, located in the posterior fossa, consists of the midline (vermis) and two hemispheres, and anatomically can be divided into ten lobules (Figure 1). A dedicated role of the cerebellum in violent individuals was already

Figure 1



Cerebellar lobules and vermis illustrated in coronal and surface view (left) and projected on a flatmap [90] (right).

proposed in the late 19th century, with anatomists reporting an enlarged vermis [17] and reduced occipital lobe-to-cerebellum ratio in criminals [18]. Importantly, these findings were restricted to a series of case studies based on postmortem examinations [19]. By comparing structural magnetic resonance images of violent offenders with psychopathic traits and/or ASPD to those of a control group, voxel-based morphometry (VBM) and related techniques offer a more sensitive way to examine subtle yet relevant structural differences across groups [20]. The aim of this narrative review is to discuss neuroanatomical evidence of structural cerebellar abnormalities in violent psychopathic offenders. Specifically, we will examine empirical reports on individuals convicted for violent offenses and/or diagnosed with ASPD relative to healthy controls (HC) as well as cerebellar associations with aggressive behavior in nonclinical samples.

To identify relevant articles, the PubMed database was searched using different combinations of the following search terms: 'structural MRI', 'voxel-based morphometry', 'sMRI', 'VBM', 'grey matter volume', 'antisocial', 'violent offenders', 'aggression', 'anger', 'impulsivity', 'cerebell*'. Moreover, reference lists of already-identified studies were consulted to gain more results. Studies were included if they reported an association between cerebellar volumes and the examined sample. Owing to the narrative nature of the present review, no data were collected concerning studies that did not report cerebellar abnormalities. Table 1 provides details on the studies discussed hereafter.

Among the studies that investigated GM of the cerebellum in aggression and psychopathy, volumetric reductions of the cerebellar hemispheres are among the most consistently reported findings. Compared with HC, violent offenders with psychopathic traits were shown to have lower GM volumes in the left anterior cerebellum and right posterior lobule VIIIb [21,22]. Moreover, the presence and severity of psychopathic traits in violent offenders have been associated with reduced absolute and relative GM volumes in bilateral posterior cerebellum [22–24]. Relatedly, another study found that individuals scoring higher on psychopathic traits such as callousness and superficial charm (i.e. the interpersonal-affective factor of the PCL-R) had significantly lower GM volume in the right anterior cerebellum (lobule V) [25]. Subsequent analyses dividing this factor into an interpersonal and affective component, respectively, further indicated that individuals scoring higher on this scale had lower GM volume in right Crus I. Interestingly, structural volume reductions in the cerebellum associated with anger and aggression are not restricted to violent offenders, but have also been reported for patients with intermittent explosive disorder (IED) [26] and bipolar disorder (BP) [27]. Moreover, lower GM volume in right lobules VIIb and VIIIa has been linked to higher self-reported physical aggression in a healthy adolescent sample [28]. A study in neurotypical male volunteers confirmed an association between psychopathic traits and volumetric reductions in cerebellar GM [29]. Male subjects scoring low and high on self-reported reactive aggression were selected from a larger sample of nonclinical volunteers. Volumetric comparisons found that next to reduced GM volumes in bilateral amygdala and right putamen, men with high levels of reactive aggression and callous–unemotional traits had lower GM volumes in bilateral lobule VI and left lobule V [29].

Table 1

Voxel-based morphology and volumetry studies associating cerebellar volumes with violence, psychopathy, aggression, and impulsivity.

Study	Experimental group	Control group	Method	Findings	Assessment of violence, psychopathy, substance abuse, aggression, or impulsivity
Adults: violent criminal convicts					
Bertsch et al., 2013 [21][a]	Male violent criminal offenders: ASPD with BPD (ASPD+BPD, $n = 13$), ASPD with psychopathy (ASPD+P, $n = 12$)	Male HC ($n = 14$)	VBM	Offenders vs. controls: lower GM in left vermis I–IV, lobule VIIb (ASPD+BPD), left lobules I–IV, V, and right lobule VIIIb (ASPD+P) ASPD+P vs. ASPD+BPD: higher GM in right Crus II in ASPD associated with high psychopathy traits compared with ASPD+BPD	Violent crimes: severe bodily injury such as murder, manslaughter, robbery, or rape. Offenders met ASPD criteria and scored higher on aggression (FAF)
Ermer et al., 2012 [24]	Incarcerated males ($n = 296$)	-	VBM	Lower GM in the left cerebellum associated with higher psychopathy (PCL-R) total scores, results corrected for substance dependence	PCL-R was used to assess psychopathy
Gregory et al., 2012 [22]	Male violent offenders with (ASPD+P, $n = 17$) and without psychopathy (ASPD-P, $n = 27$)	Male nonoffenders ($n = 22$)	VBM	Violent offenders ASPD+P vs. ASPD-P: lower GM in bilateral cerebellum	Violent crimes: murder, rape, attempted murder, and grievous bodily harm. Offenders had more alcohol and drug abuse and/or dependence compared with controls
Hofhansel et al., 2020 [39][a]	Male violent offenders ($n = 27$)	Male HC ($n = 27$)	VBM	Within offenders: higher GM in left Crus I associated with higher BPA angerscores, lower GM in right Crus I associated with higher BPA hostility scores	Violent crimes: armed robbery, assault, burglary, sexual offence, or manslaughter. Offenders scored higher on the BPA and RPQ
Klöckner et al., 2021 [41]	Male CSO pedophilic (CSO+P, $n = 22$) and nonpedophilic (CSO-P, $n = 21$)	Male NSO: nonsexual violent offenders ($n = 20$)	SBM, volumetry	CSO+P vs. CSO-P: lower GM bilateral posterior cerebellum and lobule V Within the NSO: lower GM in right lobule VIIb ⇔ higher number of crimes (marginal)	NSOs had committed at least one homicide and no sexual offenses. CSO-P scored higher on PCL-R compared with CSO+P and NSO. No difference in alcohol abuse (14–30%), CSO-P had highest substance abuse (29% vs. 5–10%)
Kolla et al., 2014 [23][a]	Male violent offenders with (ASPD+P, $n = 9$) and without psychopathy (ASPD-P, $n = 15$)	Male nonoffenders ($n = 13$)	VBM	ASPD+P offenders vs. ASPD-P offenders: lower GM in right lobule VI	Violent crimes: murder, rape, attempted murder, and grievous bodily harm. ASPD+P scored higher on antisocial-deficient affect and interpersonal facets (PCL-R) compared with ASPD-P, no difference in substance use disorder
Leutgeb et al., 2015 [35][a]	Male high-risk violent offenders ($n = 40$)	Male nondelinquent controls ($n = 37$)	VBM	Offenders vs. controls: higher GM in right lobule IX and vermis I–IV Within offenders: higher GM in right Crus I associated with higher PCL-R factor 1, higher GM in vermis I–VI associated with higher VRAG risk/VRS static/STAXI anger-out scores, higher GM in left lobule VI associated with higher VRAG risk/STAXI temperament	High-risk violent offenders with one or more index convictions for violence, sexual offenders excluded. Higher psychopathy scores (PCL-R), STAXI on trait anger not significantly higher. History of alcohol/drug abuse led to exclusion
		Male controls ($n = 19$)	VBM		

Table 1 (*continued*)

Study	Experimental group	Control group	Method	Findings	Assessment of violence, psychopathy, substance abuse, aggression, or impulsivity
Nummenmaa et al., 2021 [51]	Male convicted offenders ($n = 19$) with high psychopathic traits, ASPD in $n = 4$			Offenders vs. controls: lower WM density in the cerebellum	Violent crimes: murder, manslaughter, and grievous bodily harm.
Pera-Guardiola et al., 2016 [44][a]	Male psychopaths, violent offenders ($n = 19$)	Male HC ($n = 20$)	VBM	Within offenders: lower GM in right lobules I–IV and V associated with lower emotion recognition performance	Violent crimes: murder, violent robberies. Psychopathy measured with PCL-R. Emotion recognition: sadness, fear, happiness, surprise, anger, and disgust
Puri et al., 2008 [60]	Violent offenders with schizophrenia ($n = 13$)	Nonviolent schizophrenia patients ($n = 13$)	VBM	Offenders vs. nonoffenders: lower GM in bilateral cerebellum	Violent crimes: homicide, attempted murder, or wounding with intent to cause grievous bodily harm; no substance abuse.
Sajous-Turner et al., 2020 [40][a]	Male incarcerated offenders convicted for homicide ($n = 203$)	Male incarcerated offenders convicted for violent nonhomicide offenses ($n = 475$) or nonviolent nonhomicide offenses ($n = 130$)	VBM	Violent homicide vs. violent nonhomicide: lower GM in left lobule VI, left Crus I, and right lobule VI/ Crus I	Violent nonhomicide crimes: aggravated battery, robbery, assault, any crimes with serious physical contact, armed robbery, domestic violence (with weapon or great bodily harm), kidnapping, unlawful confinement, and arson
Tiihonen et al., 2008 [34]	Male persistent violent offenders ($n = 26$) diagnosed with psychopathy or ASPD	Male HC ($n = 25$)	Whole-brain volumetry	Offenders vs. controls: higher GM in the right cerebellum, higher WM in the left cerebellum Within offenders: lower WM in the left cerebellum associated with higher Impulsive Irresponsible Lifestyle score (PCL-R)	Recurrent violent crimes: murder, manslaughter, assault, and armed robbery.
Adults: psychiatric/neurologic diagnoses with symptomatic aggressive and impulsive tendencies					
Beckwith et al., 2018 [52]	Adults with childhood lead exposure ($n = 155$)	-	VBM	Lower WM in the cerebellum associated with higher PPI scores	PPI was used to assess psychopathy
Coccaro et al., 2016 [26][a]	IED patients ($n = 57$)	Psychiatric controls ($n = 58$), HC ($n = 53$)	VBM	IED vs. all controls: lower GM in right Crus I/II	IED patients showed higher LHA aggression, BPA aggression, LHIB, and BIS-11 impulsivity
Cope et al., 2012 [25][a]	Participants from probation, parole, and drug-treatment centers ($n = 66$)	-	VBM	Lower GM in right Crus I associated with higher PCL-R Facet 1 (interpersonal– affective) Lower GM in right lobule V associated with higher PCL-R Factor 1 (interpersonal + affective)	Comorbidities: ASPD ($n = 38$), drug abuse ($n = 66$), and alcohol abuse ($n = 49$)
Kuhlmann et al., 2013 [36][a]	Female patients with BPD ($n = 30$)	HC ($n = 33$)	VBM	Patients vs. controls: lower GM in vermis X; higher GM in left lobules VI, VII, and vermis IV–VI	Patients scored higher on FAF spontaneous/reactive aggression and irritability, but not inhibition. STAXI: higher anger scores
Lapomarda et al., 2021 [33][a]	BPD patients ($n = 46$)	HC ($n = 60$)	SBM	BPD vs. controls: lower GM concentration in a parietal–occipital and cerebellar network (cerebellar tonsil, declive, pyramis, tuber, and uvula); lower GM concentration in parietal–occipital–cerebellar network associated with higher BIS-11 scores (all scales)	Measures of impulsivity: BIS-11, BART. Patients scored higher on the BIS-11 compared with controls. No substance dependence within the past six months

Table 1 (*continued*)

Study	Experimental group	Control group	Method	Findings	Assessment of violence, psychopathy, substance abuse, aggression, or impulsivity
Lee et al., 2011 [37][a]	Male psychiatric patients ($n = 35$)	Male HC ($n = 18$)	VBM	Within patients: higher GM in vermal areas IV–VI associated with higher motor impulsivity	Patients with self-control problems characterized by impulsivity (e.g. substance abuse, aggression, or other criminal behavior). Patient group scored higher on impulsivity (BIS-11)
Okada et al., 2015 [30][a]	OCD patients ($n = 37$)	HC ($n = 37$)	VBM	Within OCD: lower GM in right Crus II ⇔ aggression and checking	Patients with presence of aggression/checking (DY-BOCS), one of the six OCD symptom dimensions
Scharmüller et al., 2013 [45][a]	HD patients ($n = 18$)	HC ($n = 18$)	VBM	Patients vs. controls: lower GM of total cerebellum and vermis, higher GM in bilateral lobule X and vermis VII Within patients: higher GM in the vermis and bilateral lobules IV, V, III, and right lobule IX associated with better anger recognition	Patients showed impairment in anger recognition when tested on emotion recognition (happiness, fear, sadness, anger, disgust, surprise, and neutral)
Soloff et al., 2008 [27][a]	BPD patients ($n = 34$; 12 males, 22 females)	HC ($n = 30$; 11 males, 19 females)	VBM	All patients vs. controls: lower GM in right lobule VI, covarying for aggression Male patients vs. controls: lower GM in right lobule V Female patients vs. controls: higher GM in right lobules I–IV	Patient group scored higher on LHA and BIS-11. Comorbid ASPD more frequent among male patients (58.3%) compared with female patients (9.1%)
Healthy populations					
Bobes et al., 2013 [29][a]	Males with high RPQ scores ($n = 25$)	Males with low RPQ scores ($n = 29$)	VBM	High- vs. low- aggression groups: lower GM in bilateral VI and left lobule V	RPQ-reactive aggression scores measured in a healthy sample of $n = 230$. Two groups formed based on high and low scores
Grecucci et al., 2023 [32]		HC ($n = 212$)	Machine learning: Kernel ridge regression	Whole-brain network predicted anger-out scale, including vermis lobules I–II and IV–VIII and left lobule III (no direction for higher/lower GM concentrations)	Anger in and anger out measured by STAXI
Sorella et al., 2022 [38]	-	HC ($n = 71$)	SBM	Higher GM in a network including fusiform gyrus, cerebellum (tuber, uvula, tonsil, inferior semilunar, and pyramis), and posterior cingulate cortex associated with higher trait anger scores	Trait anger and anger control measured by STAXI
Wolfs et al., 2023 [28]	-	Healthy adolescents and adults ($n = 201$)	SUIT volumetry	Lower GM in right lobules VIIb and VIIIa associated with higher aggression and impulsivity scores, higher GM in vermis associated with higher impulsivity scores	Impulsivity and aggressive traits measured by BIS-11 and BPA

[a] included in Figure 2. All changes reported are changes in volume unless indicated otherwise. *Abbreviations*: BART, balloon analog risk task; BIS, Barratt impulsiveness scale; BPA, Buss–Perry aggression questionnaire; BPD, borderline personality disorder; CSO, child sexual offenders; DY-BOCS, dimensional Yale–Brown obsessive–compulsive scale; EIS, Eysenck impulsivity scale; FAF, factors of aggressiveness; LHA, life history of aggression; LHIB, life history of impulsive behavior; NSO, nonsexual offenders; PPI, psychopathic personality inventory; RPQ, reactive–proactive aggression questionnaire; SBM, source-based morphometry; SDQ, strengths and difficulties questionnaire; STAXI, state trait anger expression inventory; SUIT, spatially unbiased atlas template of the cerebellum and brainstem; VRAG, violence risk appraisal guide; VRS, violence risk scale; vs., versus.

Moreover, patients with obsessive–compulsive disorder (OCD) have been found to have a higher tendency to check on not acting out on their aggressive–impulsive thoughts as a function of decreased GM volume in right Crus II [30]. In addition, recent work on structural brain networks of uncontrollable behavior has highlighted a role of the cerebellum. One study identified a cortico-cerebellar structural network that may be linked to trait aggression in healthy volunteers [31]. In this study, the left centromedial and superficial amygdala, amygdaloid subregions thought to promote aggressive behavior, exhibited structural covariance with medial anterior portions of the cerebellum and lateral right Crus I, respectively. Similarly, a structural network, including the vermis and fronto–parieto–temporal regions, has been linked to one's self-reported tendency to express anger [32]. Extending this work to psychiatric patients, reduced GM concentrations were reported in a structural network, including vermal and posterior right regions of the cerebellum in bipolar patients compared with HC [33]. Notably, lower GM concentration was associated with increased self-reported impulsivity scores in the patient group. This again suggests that a reduction in GM may be linked to a continuous loss of behavioral control.

To the best of our knowledge, only two studies in violent offenders have reported volumetric *increases* of GM in the cerebellar hemispheres. One study found higher right-hemispheric GM volumes in a group of violent offenders with ASPD or psychopathy compared with HC [34]. Similarly, higher GM volume was reported in a right posterior cluster around lobule IX and anterior vermis in violent offenders with ASPD and substance dependence relative to HC [35]. These findings of GM volume increases are complemented by results from studies in other psychiatric populations. Compared with HC, higher GM volumes in left lobules VI, VIII, and vermis IV–VI have been found in female patients with bipolar disorder presenting with high reactive aggression, irritability, and state anger [36]. In psychiatric patients with different diagnoses (e.g. bipolar disorder, major depressive disorder, and attention-deficit hyperactivity disorder), higher motor impulsivity was associated with increased volumes of the anterior vermis [37]. Finally, a recent study in healthy volunteers reported a positive association between GM volume in a whole-brain network, including inferior parietal, occipital, posterior cingulate cortex, and bilateral Crus I, and self-reported trait anger [38].

Cerebellar GM volumes have also been linked to specific character traits associated with psychopathy and aggression. In violent offenders convicted for at least one violent crime, lower GM volume in right Crus I was associated with higher levels of hostility and distrust toward others [39]. By contrast, the same study found a positive association between GM volumes in left Crus I and self-reported anger and irritability. Similarly, another study observed that higher manipulativeness, lack of empathy, and shallow affect were correlated with larger GM volume of right Crus I in violent offenders [35]. In the same group, higher risk for violence recidivism and the tendency to behave aggressively were associated with higher GM volume in left lobule VI and anterior vermis. Two other studies also highlighted a role of the bilateral posterior cerebellum as a function of the nature and severity of the committed crimes. Lower GM volume in bilateral posterior cerebellum with peaks in Crus I and lobule VI was found in incarcerated offenders who committed or attempted homicides, relative to prisoners charged for violent, but nonlethal crimes [40]. Similarly, GM volume reductions spanning large portions of the bilateral posterior cerebellum were found in child sex offenders compared with nonsexual violent offenders [41].

A notable feature of psychopathic traits is the individuals' aberrant response to other people's facial expressions and to distress cues more specifically [42,43]. Comparing violent offenders with psychopathic traits to HC, worse recognition of emotional faces has been linked to reduced GM volume in bilateral anterior cerebellum [44]. By relating impaired behavioral performance to structural changes, these results may suggest a role of the anterior lobe in emotion recognition deficits in violent psychopathic offenders. In line with this, patients with Huntington's disease (HD) often exhibit impaired recognition of angry expressions, yet higher GM volume of the bilateral anterior cerebellum and vermis was related to better anger recognition [45]. Moreover, compared with HC, HD patients had lower total cerebellar volume. Importantly, agitation, irritability, aggression, and impulsive behavior are well-documented nonmotor symptoms of HD [46]. While these symptoms are typically considered part of a frontal dysexecutive syndrome, the lower-than-normal cerebellar volume in HD patients as compared with non-affected controls suggests that cerebellar atrophy may play a role in these symptoms as well.

Given the role of the anterior cerebellum (lobules I–V) in sensorimotor processing and the presence of somatotopic representations, it is tempting to speculate about a relation with the brain's mirror neuron matching system [47]. The mirror neuron system maps the observation of others' actions onto the observer's own motor representation that allows to infer the intentions of others. Functional MRI data have provided support that such a mirror matching mechanism is also present in the cerebellum [47]. These findings are in good agreement with an earlier study showing activation of anterior lobule V as well as posterior regions (VI, Crus I, VIIIa, and VIIIb) during the integration of perception- and action-related

signals [48]. Activation of the anterior cerebellum may be more directly linked to action and motor control, whereas the posterior regions may be more associated with (nonmotor) cognitive operations such as mentalizing and theory of mind [48,49]. In agreement with the presently suggested role of the cerebellum in antisocial and aggressive behavior, abnormalities of the motor mirror neuron system have recently been demonstrated in individuals with high psychopathic traits [50].

In addition to deviations in GM volumes, cerebellar WM has also been linked to psychopathy and violent behavior. For example, lower WM volumes in the cerebellum have been found in violent offenders with psychopathic traits compared with HC [51]. Moreover, individuals with childhood lead exposure scoring higher on psychopathic traits were found to have lower WM volume in the cerebellum compared with a HC group [52]. The cerebellum has extensive structural and functional connections to frontotemporal, limbic, and paralimbic cortical regions [53–55]. The supposed relation between WM volume and psychopathic traits could therefore be associated with impairments in connectivity and communication within the responsible neurobiological circuits. Importantly, also *higher* cerebellar WM volume has been reported in violent offenders compared with neurotypical male volunteers [34]. Interestingly, in this study, higher scores on the PCL-R scale Impulsive Irresponsible Lifestyle were linked to lower WM volume in the left cerebellum in violent offenders. This initially paradoxical negative association in light of the overall positive findings of this study was explained by heavier substance (e.g. alcohol) abuse in individuals exhibiting more impulsive behavior. Substance abuse and dependence have been linked to atrophy of the cerebellum [56–58] and introduce a potentially important confound in neuroscientific research on ASPD and psychopathy. Antisocial and psychopathic traits are vulnerability factors for substance abuse and developing substance dependence and show a high degree of comorbidity in ASPD and psychopathy [4,7,59]. Whether the volumetric effects on the cerebellum reflect a behavioral endophenotype, result from the harming effects of substances on the brain, or both, remain a question that needs to be systematically addressed in future studies. In addition, the presence of other comorbidities may also contribute to explaining structural cerebellar abnormalities. Comorbid bipolar disorder, for example, has been associated with lower GM volume in right Crus II compared with violent offenders with ASPD, and lower GM volume in anterior vermis and left Crus II/lobule VIIb compared with controls [21]. In another study, schizophrenic patients with a history of violent offenses were compared with nonviolent schizophrenic patients [60]. Here, lower GM volume in bilateral posterior cerebellum was found. More recently, a comparison of brain-wide differences in GM volumes between violent offenders with and without a diagnosis of schizophrenia or bipolar disorder highlighted fundamental structural dissociations [61]. Both groups reported comparable levels of psychopathic traits, but the group with a comorbid diagnosis of schizophrenia or bipolar disorder was found to have increased GM volumes in bilateral Crus I and posterior vermis. Together, these findings suggest that next to individuals' violent and antisocial tendencies, comorbid mental disorders may be manifested structurally in the brain, including the cerebellum.

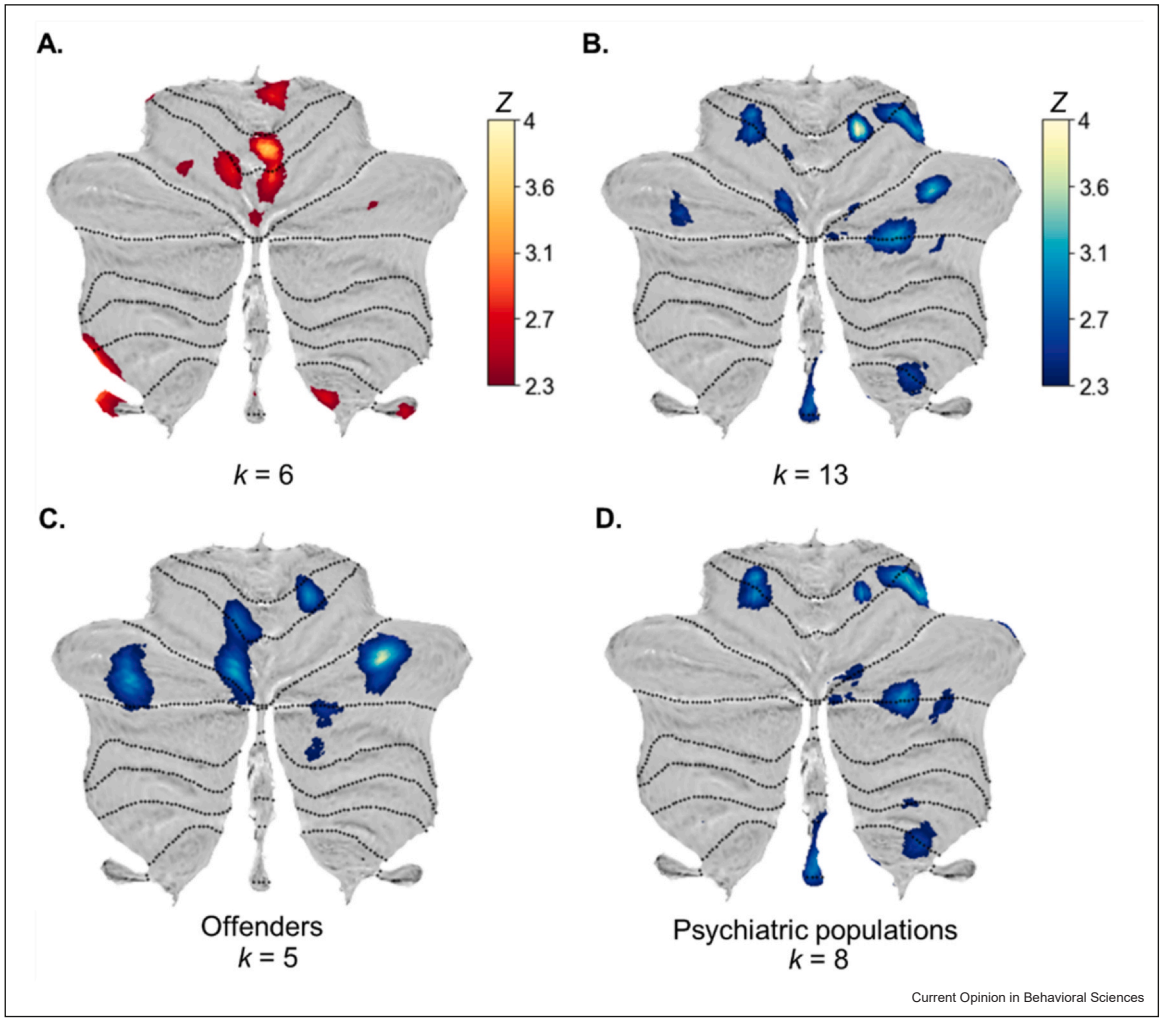
Discussion

The disentanglement of the specific cerebellar abnormalities for the different mental disorders calls for comparison studies. Alternatively, a transdiagnostic approach to find a common mechanism across all mental disorders may be a more viable approach than upholding strict categorical diagnostic criteria. Given the relatively homogeneous cerebellar microarchitecture, the cerebellum is proposed to hold a generic function subserving movement, cognition, and emotion, the so-called universal cerebellar transform [62,63]. From a transdiagnostic viewpoint, the cerebellum thus constitutes a good starting point to study comorbid mental disorders in violent psychopathic offenders.

The observations of lower GM volumes are in agreement with reports on GM volume reductions in frontal and temporal regions observed in ASPD and psychopathy [11,64,65]. The existing anatomical connections between the posterior cerebellar regions and the cerebral cortical association areas may hint at possible dysfunctions in cerebello–thalamo–cortical networks [66–69]. Focal cerebellar volume loss within these networks may be linked to impairments in behavioral control and complex socio-emotional behavior, including decreased empathy and sense of morality. Notably, an association between GM volume reductions in the right posterior cerebellum and aggression in both psychiatric and nonpsychiatric populations appears to be the most consistent finding of the reviewed studies. Figure 2 illustrates convergent volumetric change patterns in the cerebellum across all reviewed studies (increases: Figure 2a; decreases: Figure 2b) and GM volume decreases separately for violent offenders (Figure 2c) and psychiatric populations presenting with aggressive and impulsive symptoms (Figure 2d).

Furthermore, involvement of the right cerebellar posterolateral hemisphere in anger and aggressive behavior was recently established in an activation likelihood estimation (ALE) meta-analysis of functional magnetic resonance imaging (fMRI) studies in healthy volunteers [13]. Reduced volume of right posterolateral regions could signify abnormal emotional appraisal of threatening stimuli [70]

Figure. 2



Topographic maps visualizing cerebellar GM increases and decreases as summated by ALE in NiMARE v0.0.14 [91]. MNI peak coordinates of significant cerebellar clusters were used to estimate convergent volumetric change patterns while accounting for spatial uncertainty and differences in sample size. Each peak was modeled as a three-dimensional probability distribution, combined in an activation map, and tested against a null distribution, with the cerebellum as the region of interest. Maps display *z* values > 2.3 (uncorrected). The different panels show GM increases **(a)** and decreases **(b)** in violent offenders and individuals with aggressive or psychopathic tendencies, GM decreases in violent offenders **(c)**, and GM decreases in psychiatric populations **(d)**. Note that only studies that reported coordinates are included in the figures (indicated with a letter (a) in Table 1).

and impaired cognitive evaluation of whether to aggress or not. Moreover, while a relative imbalance between left and right frontal cortical activity has been related to increased approach motivation [71–73], we recently proposed that this phenomenon may be reversed in the cerebellum [14,16]. The increased tendency for approach motivation, potentially culminating in aggressive and violent behavior, may thus be linked to structural abnormalities in the left frontal cerebral and right cerebellar cortex. In support of this idea, a VBM study in children in fact reported a negative association between behavioral activation scores as a proxy for approach motivation and a cluster in right Crus I [74]. In addition, the right posterior cerebellum plays a role in social action sequencing, theory of mind, mentalizing, and divided attention [49,75]. Since social cognitive functions are often impaired in ASPD and psychopathy, the right-sided volume reductions of the posterior cerebellum in violent psychopathic offenders are particularly notable.

Next to the posterolateral regions of the cerebellum, structural changes in the posterior vermis have also been documented. Aberrations of the vermis in violent psychopathic offenders concur with previous experimental findings in animals and humans showing involvement of the vermis in emotion regulation, impulsivity, and aggression. Indeed, the extensive cortico-nuclear

projections to the subcortical mid- and forebrain structures, including the periaqueductal gray, ventral tegmental area, amygdala, and hypothalamus, provide a neuroanatomical basis by which the vermis partakes in registering, regulating, and integrating visceral and autonomic responses. These physiological responses are part of the brain's fight-flight mechanism, which in violent psychopathic offenders is by default dominated by approach motivation and fight-related behavior. Indeed, intracranial stimulation and optogenetic studies have shown that the vermis is directly implicated in regulating aggression [76,77].

Vermal structural abnormalities could also point to aberrant reward processing. Recently, an extension of the reward hypersensitivity model for mental disorders [78,79], the cerebellar reward calibration model, was proposed [80]. The model states that overinhibition of the inhibitory Purkinje cells to the deep cerebellar nuclei (i.e. fastigial nuclei) in the vermis leads to an increased excitatory drive of the deep cerebellar nuclei to the brain's reward circuits paralleled by increased reward motivation. Excessive reward drive, in turn, contributes to low frustration tolerance, irritability, sensation-seeking, and aggression. A recent ALE meta-analysis showed that the posterior vermis is involved in the anticipation of reward [81], which could imply abnormal reward expectations that lead to augmented reward drive. The model and the meta-analytic findings provide a theoretical and testable framework for understanding the relation between increased vermal GM volume in psychopathy and aggression in terms of hypersensitivity to reward.[1]

It should be noted that due to the highly foliated structure of the cerebellum, separating gray from white matter can lead to segmentation inaccuracies and imprecise calculation of volumes [83]. In addition, risk of including surrounding vasculature and supratentorial GM could also bias volume estimations [84]. Indeed, a recent study suggests that segmentation routines of structural magnetic resonance images may be susceptible to overestimating GM volumes [85]. Current advances in cerebellar imaging research will undoubtedly lead to more accurate volumetric estimations in the near future. This will significantly increase the signal-to-noise ratio in studies that examine the associations between cerebellar volumes, function, and behavior. Another exciting development is that the study of the cortical layers of the GM is becoming increasingly feasible and will allow studying the cerebellum beyond its lobular divisions [85].

[1] By extension, the vermis–amygdala–ventromedial prefrontal cortex connections provide a functional neuroanatomic account for low fear and punishment sensitivity in (primary) psychopathy [82].

Finally, the studies presented here constitute a non-exhaustive selection of research on structural cerebellar abnormalities in violent offenders and individuals with ASPD. However, it should be noted that a number of studies also failed to find structural changes in the cerebellum related to violent offenses, psychopathy, or ASPD [86–89]. Hence, while there is evidence for structural cerebellar abnormalities, future research is needed to scrutinize the functional and mechanistic role of cerebellar morphology in violent and aggressive behavior.

Conclusions

This narrative review discussed evidence for cerebellar structural alterations in violent psychopathic offenders and aggressive behavior in nonclinical samples. The reported associations between the presence of antisocial and/or psychopathic traits and abnormal cerebellar GM volume suggest that neuroanatomical differences in these populations extend beyond cerebral and subcortical regions. Albeit descriptively, the right posterior hemisphere and vermis were the regions most consistently affected. These findings concur with existing aggression data in healthy and neuropsychological populations and provide new leads into broadening our understanding about the complex neurobiology of antisocial behavior and aggression.

Data Availability

No data were used for the research described in the article.

Declaration of Competing Interest

None.

Acknowledgements

This work was supported by the Dutch Research Foundation (NWO, VI.C.181.005).

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