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Research report

Physical aggressiveness and gray matter deficits in ventromedial prefrontal cortex

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ABSTRACT

What causes individuals to hurt others? Since the famous case of Phineas Gage, lesions of the ventromedial prefrontal cortex (VMPFC) have been reliably linked to physically aggressive behavior. However, it is unclear whether naturally-occurring deficits in VMPFC, among normal individuals, might have widespread consequences for aggression. Using voxel based morphometry, we regressed gray matter density from the brains of 138 normal female and male adults onto their dispositional levels of physical aggression, verbal aggression, and sex, simultaneously. Physical, but not verbal, aggression was associated with reduced gray matter volume in the VMPFC and to a lesser extent, frontopolar cortex. Participants with less gray matter density in this VMPFC cluster were much more likely to engage in real-world violence. These findings suggest that even granular deficits in normal individuals' VMPFC gray matter can promote physical aggression.

Aggression has remained in the human behavioral repertoire despite substantial cultural advances. *Physical* aggression, in which aggressors seek to harm others' bodies against their will, is a remarkably pernicious and costly form of aggression (Anderson & Bushman, 2002). The stubborn and tenacious nature of such behavior suggests that physical aggression arises, in part, due to alterations in individuals' underlying brain structure. To date, investigations of physical aggression's neuro-structural underpinnings have focused on the small percentage of the population that comprises clinically- and criminally-aggressive populations. However, limiting such investigation to these populations ignores physical aggression's continuous nature, which exhibits substantial individual differences across humankind. In the present research, we sought to assess the gray matter correlates of physical aggressiveness in a relatively large sample of normal young adults.

1. The VMPFC and aggression

1.1. *Evidence from lesion patients, violent criminals, and aggressive psychopathology*

Extending back to the famous case of Phineas Gage, injuries of the brain's frontal lobe have shown how readily physical aggressiveness can emerge from structural damage to the brain (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). Indeed, one of the most common biological causes of

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aggressive behavior is lesions of the brain's frontal lobe (Tateno, Jorge, & Robinson, 2003). Combat veterans with lesions of the ventromedial prefrontal cortex (VMPFC) exhibited substantially greater aggressive behavior than individuals with lesions of other brain regions or no brain lesions (Grafman et al., 1996). VMPFC dysfunction reliably differentiates violent from non-violent criminal offenders (Bufkin & Luttrell, 2005). Individuals diagnosed with intermittent explosive disorder exhibited reduced VMPFC activity and VMPFC-amygdala connectivity while viewing angry faces (Coccaro, McCloskey, Fitzgerald, & Phan, 2007). Across these samples of violent patients and offenders, the VMPFC appears to play a clear and causal role in promoting physical violence.

1.2. *Functional evidence from normal adults*

Empirical support for the link between VMPFC deficits and physical aggression does not purely come from studies of lesion patients, violent criminals, and individuals diagnosed with violent psychopathology. Normal young adults who imagined an aggressive act exhibited reduced cerebral blood flow in the VMPFC, as compared to when they imagined a non-violent act (Pietrini, Guazzelli, Basso, Jaffe, & Grafman, 2000). Among a similar population, VMPFC activity was negatively associated with financial forms of aggression during behavioral economics (Mehta & Beer, 2009). VMPFC reactivity to aggressive interactions was selective for prosocially reacting to the opponent's suffering due to the aggression (Lotze, Veit, Anders, & Birbaumer, 2007). However, these studies used *functional* neuroimaging, leaving unexplored the structural basis of physical aggression among normal individuals. The current research fills this gap in the literature by providing a comprehensive test of the hypothesis that normal individuals' aggressiveness and real-world aggressive behavior relates to structural deficits in the VMPFC.

1.3. *Overview*

Based on previous research implicating VMPFC deficits in the promotion of physical aggression, we predicted that normal, young adults' dispositional physical aggression would be associated with reduced gray matter in this region. To test this hypothesis, participants had a high-resolution structural scan taken of their brain using magnetic resonance imaging (MRI) and then reported their trait levels of physical and verbal aggressiveness, as well as their sex. These two latter variables served as crucial covariates to assure our results were specific for trait physical aggression. As an additional outcome measure, a subset of participants reported whether they had ever previously been in a physical fight. We predicted that VMPFC gray matter density would be inversely associated with prior engagement in physical fights.

2. Materials and methods

2.1. *Participants*

Participants were 138 normal, right-handed, young adults (91 females, 47 males; age: $M = 19.42$, $SD = 2.10$, range: 18–30). This sample size is quite large for an MRI analysis that tested a brain-behavior correlation and was determined by combining participants from two existing MRI datasets that contained our study variables. As such, sample size was not determined based on a power analysis, but on the available data. Participant enrollment occurred using the stop rule of reaching 60 participants in the first dataset and 80 participants in the second dataset.

Participants were either undergraduates recruited through the introductory psychology subject pool in exchange for credit towards their course's research requirement and an image of their brain or general community members recruited in exchange for money and an image of their brain. Potential candidates were excluded from the study if they met any of the following criteria as assessed by an online questionnaire: any major medical conditions, developmental disorders, body mass index above 30, claustrophobia, current or past neurological or psychiatric disorder, metallic objects in the body, prior head trauma, and psychoactive medication use.

3. Materials

3.1. *Brief Aggression Questionnaire*

To measure trait physical aggression, we employed the Brief Aggression Questionnaire (Webster et al., 2014). The Brief Aggression Questionnaire contains twelve items that comprise four factors: anger (sample item: I have trouble controlling my temper), hostility (sample item: I sometimes feel that people are laughing at me behind my back), physical aggression (sample item: Given enough provocation, I may hit another person), and verbal aggression (sample item: My friends say that I'm somewhat argumentative). Participants responded to each item along a 1 (disagree) to 7 (agree) Likert-type scale.

3.2. *Procedure*

Participants arrived at the University of Kentucky's Magnetic Resonance Imaging and Spectroscopy Center where they had the study explained to them and were screened to ensure they would be safe and comfortable in the MRI environment. Participants were then placed in an MRI scanner and had a high-resolution structural scan taken of their brain. Participants then exited the scanner and completed a battery of questionnaires including the Brief Aggression Questionnaire and a demographics survey. A sub-sample ($N = 64$) also completed a single item measure of actual physically aggressive behavior, in which they answered 'yes' or 'no' to the item "Have you ever been in a physical fight?" Yes responses were coded as 1 and No responses were coded as 0. Finally, all participants were debriefed and dismissed.

3.3. *MRI data acquisition*

All MRI data were obtained using a 3.0 T Siemens Magnetom Trio scanner. Structural images consisted of a T1-weighted

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