Intimate Partner Violence Perpetration Corresponds to a Dorsal-Ventral Gradient in Medial PFC Reactivity to Interpersonal Provocation

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Abstract

Intimate partner violence (IPV) perpetration is often preceded by perceived interpersonal provocations such as slights, insults, and rejections. Yet the neural mechanisms that link provocation to IPV remain unclear. In the context of interactions with strangers, the medial prefrontal cortex (MPFC) has been repeatedly shown to respond to provocation, with more dorsal activation associated with more aggressive reactions and more ventral activation associated with less aggressive reactions. We used functional brain imaging to test whether this dorsal-ventral MPFC reactivity gradient would also correlate with greater aggression towards an unexamined target: intimate partners. To do so, 61 undergraduates (27.87% male, age range: 18-22) reported whether they had ever committed various acts of IPV perpetration (e.g., punching, hitting, shoving) and then were repeatedly provoked by a stranger while undergoing functional MRI (fMRI) scanning. Individuals with a disproportionately dorsal, rather than ventral, MPFC response were more likely to have perpetrated IPV and had perpetrated more kinds of IPV, even when controlling for gender. These findings provide further evidence that the dorsal-ventral MPFC gradient is a critical, biological indicator of whether an individual is more or less likely to react aggressively and suggest new avenues for understanding and potentially preventing IPV perpetration.

***Keywords*:** intimate partner violence, provocation, medial prefrontal cortex, fMRI, aggression

Introduction

Intimate partner violence (IPV) is perpetrated when individuals attempt to harm their intimate partner against that partner's will (Anderson & Bushman, 2002). IPV can span sexual, psychological, and physical forms of harm, as well as stalking (Black et al., 2011). Across the world, approximately 22 to 35 percent of women and 7 to 29 percent of men experience IPV in their lives (World Health Organization, 2013). Despite the prevalence and cost of IPV, the psychological and neural mechanisms that explain IPV perpetration remain poorly understood. The current study fills this gap by identifying a potential neural mechanism that links interpersonal provocation with IPV perpetration.

Provocation and IPV

Interpersonal provocation is a broad category that can include verbal insults or threats, physical forms (e.g., shoving), and social exclusion or rejection, amongst others. Interpersonal provocation is a remarkably reliable cause of aggression against strangers (e.g., Denson, Pedersen, Friese, Hahm, & Roberts, 2011). This holds true for IPV as well. On days when male and female members of an intimate relationship felt most provoked by their partner, they stuck the most sharp pins in a voodoo doll that represented their partner (DeWall et al., 2013). This effect replicated when male and female participants were verbally insulted by their intimate partner, or were angered in an argument with their partner (DeWall et al., 2013). Simply imagining a hypothetical situation in which one's intimate partner was flirting with an attractive individual was enough to increase the tendency for female and male members of an intimate relationship to express aggressive intentions toward their partner (Finkel, DeWall, Slotter, Oaten, & Foshee, 2009). An actual provocation, such as offering a meager sum

of money for an intimate partner's painting, increased the duration with which individuals

assigned their intimate partner to experience physical pain (Finkel et al., 2009). Other

laboratory studies have also replicated the causal effect of provocation on greater IPV

perpetration by female and male participants (Finkel et al., 2012; Slotter et al., 2012).

Among women incarcerated for IPV perpetration, one of the most common reasons they

provided for their IPV perpetration was provocation from their intimate partners (Stuart,

Moore, Hellmuth, Ramsey, & Kahler, 2006).

Yet is provocation from one's intimate partner a unique experience that promotes

IPV, or do individuals' responses to provocation from other types of individuals

generalize to the intimate partner context? Indeed, provocations can arise from our

friends, family members, romantic partners, and even strangers. *To our knowledge, no*

studies have systematically varied the source of provocation and examined its impact

on IPV perpetration. The source of provocation might play a critical role in the neural

and behavioral responses and more work is needed to articulate these potential source

effects. Although there are many causes of IPV perpetration, these various sources of

evidence suggest that to understand the mechanisms of IPV perpetration, provocation

is an excellent starting point.

Mechanisms of the Provocation-IPV Link

Psychological mechanisms. Provocation reliably increases aggression, but it

does so through many different mechanisms. Experimental evidence suggests that

provocation impairs self-control, which renders individuals more likely to succumb to

their aggressive impulses towards their intimate partners (Denson et al., 2011; Finkel et

al., 2012; cf. Lievaart, Huijding, van der Veen, Hovens, & Franken, 2017). Forms of

provocation that approximate rejection or exclusion tend to be truly painful, which can correlate with greater subsequent aggressive retaliation (Chester et al., 2014). Further, individuals tend to angrily ruminate about the provocative experience, which also fosters increases in aggression towards both the provocateur and innocent bystanders (Denson et al., 2011; Pedersen, Gonzales, & Miller, 2000).

Neural mechanisms: Dorsal MPFC. Angry rumination is subserved by a specific network of brain regions, which largely converge on a specific hub: the dorsal medial prefrontal cortex (DMPFC; Denson, Pedersen, Ronquillo, & Nandy, 2009). Indeed, the DMPFC shows heightened activity during both the experience of provocation itself and angry rumination about the provocation after it has occurred (Denson et al., 2009). Activity in the DMPFC, during the provocation incident, was associated with the tendency to act aggressively towards innocent individuals who played no part in the provocation (Denson et al., 2009). Other studies have revealed that DMPFC activity during provocation is positively correlated with retaliatory aggression against provocateurs (Chester & DeWall, 2016; Krämer, Jansma, Tempelmann, & Münte, 2007; Lotze, Veit, Anders, & Birbaumer, 2007). As such, the DMPFC is a likely candidate for a neural mechanism that links instances of provocation with the perpetration of IPV.

Neural mechanisms: Ventral MPFC. If the dorsal MPFC appears to promote aggressive responses to provocation, then the ventral MPFC (VMPFC) serves to inhibit such angry responses to provocation and facilitate prosocial responses. VMPFC responses to images of angry faces are negatively associated with noise blasts administered to an opponent (Beyer, Münte, Göttlich, & Krämer, 2014). Further, VMPFC

activity during aggressive acts are associated with greater reactivity to the suffering of the victim and suggests a compassionate role for this brain region in the context of aggressive interactions (Lotze et al., 2007). Indeed, structural VMPFC damage impairs empathic processes (Leopold et al., 2011) and is a key neural substrate of the effective regulation of negative emotions (e.g., anger) that often motivate aggression (Bachevalier & Loveland, 2006). Even naturally-occurring deficits in VMPFC gray matter density is associated with greater aggressive behavior (Chester, Lynam, Milich, & DeWall, 2017).

Potential explanations for the dorsal-ventral distinction. These findings paint a clear dorsal-ventral gradient to the MPFC, with dorsal regions facilitating antagonistic responses to provocation and ventral regions constraining such reactions and orienting individuals towards compassion and communion. Yet why would this dorsal-ventral distinction influence acts of aggression such as IPV? Seminal work on this neuroanatomical gradient of the MPFC indicated that judgments about the self were subserved by the VMPFC and judgments about others subserved by the DMPFC (Denny, Kober, Wager, & Ochsner, 2012). Further, thinking about others that are similar to the self, recruit ventral MPFC and mentalizing with dissimilar others recruits dorsal MPFC regions (Mitchell, Macrae, & Banaji, 2006). Thus, VMPFC responses to provocations might inhibit subsequent retaliation by imbuing the provocateur with greater self-other overlap. Conversely, more dorsal MPFC responses would magnify self-other distinctions, facilitating aggressive acts, as such self-other distinction is a reliable correlate of antisocial behavior (Galinsky, Ku, & Wang, 2005). Further, more dorsal MPFC responses to social threats tend to up-regulate autonomic threat

responses and ventral MPFC responses to such threats tend to down-regulate such

threat responses (Wager et al., 2009). These differential roles in threat responding may

also be at play in influencing whether a provocation is met with an aggressive response.

Present Research

The present research tested the hypothesis that more-ventral-than-dorsal MPFC

reactivity to experiences of interpersonal provocation would be negatively associated

with greater IPV perpetration. To do so, a sample of undergraduate participants

reported their lifetime perpetration of different acts of physical IPV. Afterwards, these

participants completed a competitive computer task in which an opponent repeatedly

provoked them, while undergoing functional MRI. To assess if our observed effects

reflected a tendency to displace aggression from provocateurs onto innocent targets,

participants also completed a trait measure of displaced aggression. This study was

part of a larger project on the role of impulsivity on externalizing behaviors (described

more fully here: Chester et al., 2016).

<p align="center">**Methods**</p>

Participants

Participants were 80 right-hand dominant, undergraduate psychology students,

61 of whom actually completed our IPV measure (44 females, 17 males; $M = 18.61$, SD

$= 0.84$, range: 18-22) who were compensated with images of their brain, course credit,

and money. Participants racial identity was as follows: 46 White, 7 African-American, 4

Asian-American, 2 'Other', and 2 were missing racial identity data. Two of the

participants identified as Hispanic, whereas 57 identified as Non-Hispanic, and such

data were missing from two others. Data was not collected regarding participants'

socioeconomic status, education, intimate relationship status, or sexual orientation.

Potential participants were screened using an online questionnaire that assessed their

ability to be comfortable and safe in the MRI environment, and whether they possessed

neurological or psychological pathologies that might undermine the validity of our data.

Procedure

      Initial laboratory session. Participants arrived at our laboratory where they

provided informed consent according to guidelines set by the University of Kentucky's

Office of Research Integrity and were again screened for safety in the MRI environment.

Then, participants completed a battery of questionnaires that included the Crime and

Analogous Behavior scale (CAB), which has been validated in multiple studies on

interpersonal aggression (Miller & Lynam, 2003, 2006) and intimate partner violence

(Derefinko, DeWall, Metze, Walsh, & Lynam, 2011; Jones & Miller, 2012; Miller, Dir,

Gentile, Wilson, Pryor, & Campbell, 2010). The CAB quantifies an individual's past

antisocial behaviors, which includes six items that measure past acts of IPV.

Specifically, the CAB's IPV subscale contains the items that ask whether the individuals

has *ever* "thrown something at a romantic partner", "twisted a romantic partner's arm or

hair", "pushed or shoved a romantic partner", "grabbed a romantic partner", "slapped a

romantic partner", and "punched or hit a romantic partner with something that could

hurt". Participants responded to each item using a Yes or No scale, in which Yes was

coded as 1 and No was coded as 0. These six items were summed to create an 'IPV

severity' composite score. This severity score was also re-coded into a binary (1 = ever

perpetrated any form of IPV, 0 = never perpetrated any form of IPV) score that was

used to determine if individuals had ever engaged in any type of IPV perpetration.

Participants also completed the Displaced Aggression Questionnaire, which includes a 10-item subscale that quantifies the dispositional tendency to displace aggressive thoughts, feelings, and acts from the provocateur onto innocent third parties (Denson et al., 2006). Participants rated their agreement with various statements along a 1 (extremely uncharacteristic of me) to 7 (extremely characteristic of me) response scale, which were averaged to create a displaced aggression index.

MRI laboratory session. Several days after the initial laboratory visit, participants arrived at the University of Kentucky's Magnetic Resonance Imaging and Spectroscopy Center. Participants were told that they would complete a task in the MRI scanner against a same-sex University of Kentucky student who was also in an MRI scanner connected over the internet. Participants then completed a well-validated provocation paradigm, the Taylor Aggression Paradigm, which took the form of a block-based fMRI task previously validated by other studies (Chester & DeWall, 2016; Krämer et al., 2007).

In this task, participants repeatedly competed against a fictitious opponent to see who could press a button faster. As a putative motivational tool, participants would receive an aversive noise blast if they were slower than their opponent on the button-pressing competition. Participants, and their fictitious opponents, could set the volume of the noise blast punishment, from 1 (not audible) to 4 (aversively loud). Provocation took the form of whether participants viewed a loud (i.e., high provocation: 3 or 4) or soft (low provocation: 1 or 2) volume setting from their opponent.

Each of the task's 12 blocks began with a fixation trial that modeled baseline neural activity (20s), which was then followed by a volume selection trial (7.5s). Then,

participants viewed a blank screen with a jittered duration (0.5/1.0/1.5s) then competed

to quickly press a button when a red square appeared on the screen (3.5/4.0/4.5s).

Participants then viewed their opponents' pre-programmed volume settings (7.5s) and

then saw whether they won or lost the competition (7.5s), receiving a noise blast if they

lost at the volume selected by the computer. The 12 blocks were characterized by 6

high and 6 low provocation trials that were randomly ordered yet held constant across

participants.

Functional MRI Data Acquisition, Preprocessing, and Analysis

  Acquisition. All MRI data were obtained using a 3.0-tesla Siemens Magnetom

Trio scanner using a 32-channel head coil. Echo planar BOLD images were acquired

with a T2*-weighted gradient across the entire brain with a 3D shim (matrix size = 64 x

64, field of view = 224mm, echo time = 28ms, repetition time = 2.5s, slice thickness =

3.5mm, 40 interleaved axial slices, flip angle = 90°). To allow for registration to native

space, a coplanar T1-weighted MP-RAGE was also acquired from each participant

($1mm^3$ isotropic voxel size, echo time = 2.56ms, repetition time = 1.69s, flip angle =

12°).

  Preprocessing. The Oxford Center for Functional MRI of the Brain (FMRIB)'s

Software Library (FSL version 5.0) was used to conduct all preprocessing and fMRI

analyses (Smith et al., 2004; Woolrich et al., 2009). The first functional volume was

removed to facilitate BOLD signal equilibration. Reconstructed functional data

underwent head motion correction to the median volume. Non-brain tissue was then

stripped from all functional and structural data. Functional volumes underwent slice-

timing correction, pre-whitening, spatial smoothing with a 5mm full width half maximum Gaussian kernel, and high-pass temporal filtering (100s cutoff).

Participant-level analysis. Preprocessed fMRI data from the aggression task were then analyzed using a two-level general linear model approach. First, each participant's BOLD signal was modeled with a fixed-effects analysis which modeled high provocation and low provocation trials as events using a canonical double-gamma hemodynamic response function with a temporal derivative. Volume-setting, pre-competition, competition, and outcome trials, along with all six motion parameters were included as nuisance regressors into the model. Fixation trials were not modeled in this analysis. Linear contrasts then compared high provocation to low provocation trials (i.e., High Provocation > Low Provocation). Resulting contrast images from this analysis were first linearly registered to native space structural volumes and then spatially normalized to a Montreal Neurological Institute (MNI) stereotaxic space template image.

Group-level analysis. Then, each participant's High Provocation > Low Provocation contrast volumes were fed into a group-level, mixed-effects analysis which created group average contrast maps across the entire brain. Cluster-based thresholding was then applied to this group contrast map (cluster threshold: $Z > 2.3$, $p <$ .05; Heller, Stanley, Yekutieli, Rubin, & Benjamini, 2006; Worsley, 2001), followed by family-wise error multiple comparison correction based on Gaussian random field theory.

Computing a ventral-dorsal gradient score. Parameter estimates from the High Provocation > Low Provocation contrast, in percent signal change units, were extracted from the DMPFC and VMPFC using 10mm spherical region-of-interest masks,

centered on peak voxels reported in each of these two regions by Denson and

colleagues (2009; Figure 1). Parameter estimates from the DMPC were subtracted from

the VMPFC to create a ventral-dorsal difference score, in which larger values indicated

more VMPFC-than-DMPFC responses to provocation, and smaller values indicated

more DMPFC-than-VMPFC responses to provocation.

Figure 1. ROI masks for the dorsal (red) and ventral (blue) MPFC spheres. X coordinate

in MNI space.
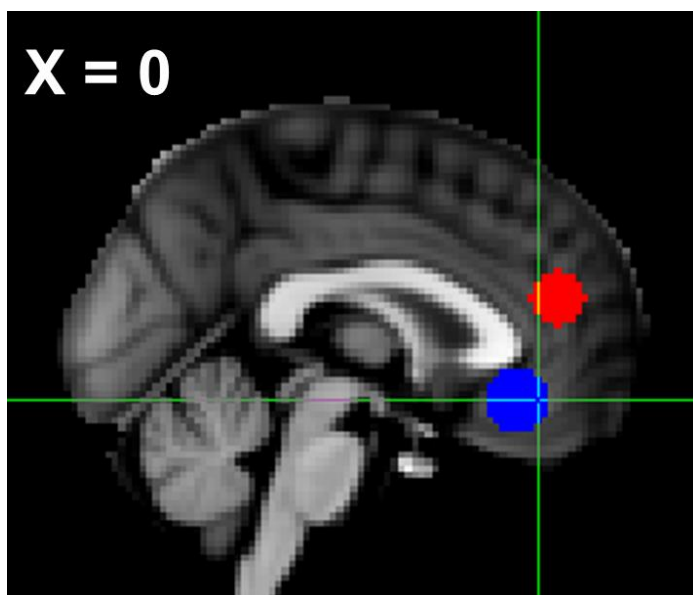
Results

Descriptive statistics. Of all 61 participants, 23 (19 females, 4 males) indicated

that they had perpetrated at least one act of IPV, and 38 (25 females, 13 males)

reported no lifetime IPV perpetration. Of the individual IPV acts, pushing/shoving was

the most frequently endorsed form of IPV perpetration ($n = 15$), followed by throwing

something ($n = 9$), grabbing ($n = 7$), slapping ($n = 6$), twisting arms/hair ($n = 3$), and

punching/hitting ($n = 1$).

Three participants failed to complete the Displaced Aggression Questionnaire.

The IPV severity composite measure exhibited modest internal consistency, $\alpha = .67$,

and the displaced aggression index, $\alpha = .92$. A Kolmogorov-Smirnov test revealed that

the distribution of IPV scores did not meet the assumption of normality as the

distribution was zero-inflated, $M = 0.67$, $SD = 1.17$, range: 0 – 6, and positively skewed,

$k(61) = .34$, $p < .001$ (Figure 2). One female participant with 6 reports of IPV was

removed because her datapoint was 4.57 SDs above the mean, exerting inordinate

influence on our correlational analyses (Figure 2; final $N = 60$; next farthest participant

from mean, Z = 2.86).

Figure 2. Frequency distribution of participants' IPV scores, curved line represents the

normalized distribution.
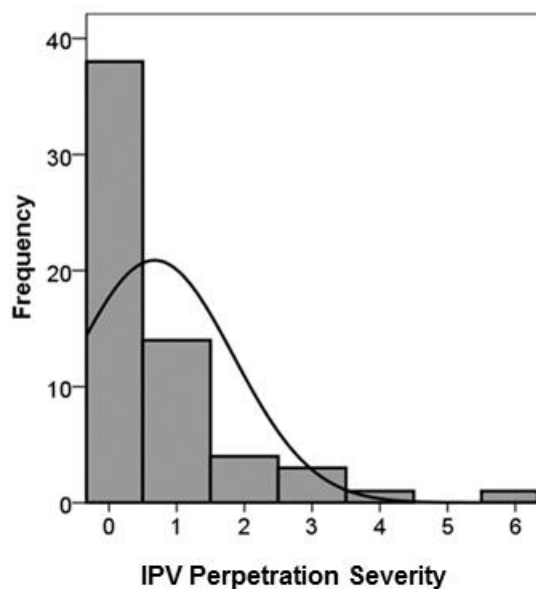
Whole brain provocation analyses.
Provocations from participants' opponents
(i.e., High Provocation > Low Provocation
contrast) were associated with greater activity
in a larger midline cluster that centered
around the supplementary motor area (SMA)
and also included the DMPFC, dorsal anterior
and mid cingulate cortex (ACC), posterior
cingulate cortex and precuneus (PCC). Other

bilateral clusters included the dorsolateral prefrontal cortex (DLPFC), temporoparietal

junction (TPJ), anterior insula (AI), thalami, and dorsal striatum (DS; Figure 3, Table 1).

Figure 3. Brain areas significantly associated with High Provocation > Low Provocation.

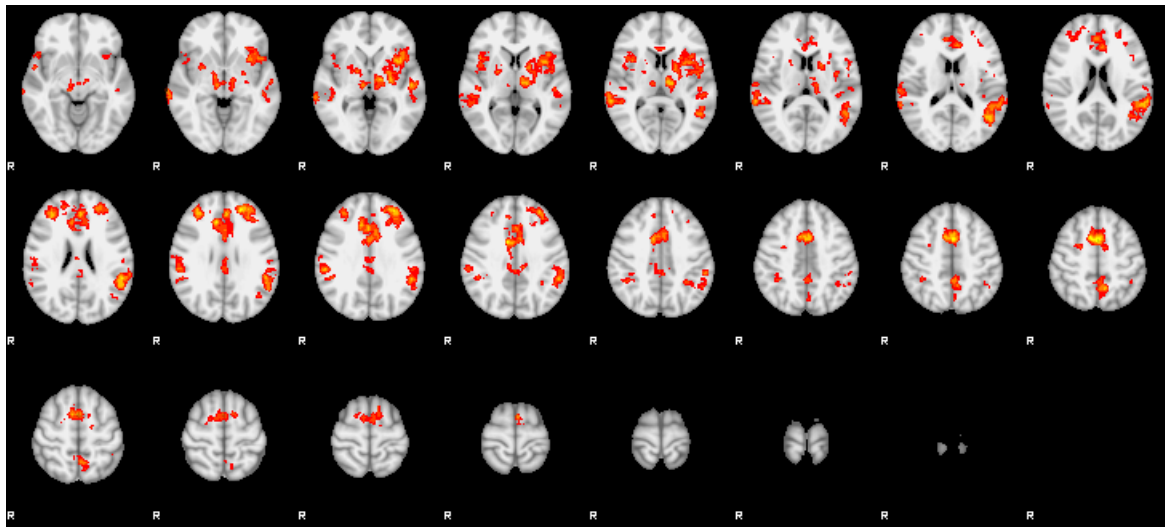*Left and right hemispheres are in scanner space (i.e., flipped).*

Table 1. Whole-brain fMRI main effect results from the High Provocation > Low

Provocation contrast, in descending order of number of voxels within each significant

cluster (11, 082 total voxels).

Brain Region	Voxels	peak Z	peak MNI coordinates (x,y,z)
SMA/ACC/DMPFC/PCC	3,686	4.43	-2, -8, 50
AI/Thalamus/DS	2,023	3.97	-36, 10, 2
TPJ	2,020	4.13	-54, -42, 22
	1,281	4.17	64, -38, 10
DLPFC	813	4.10	-20, 44, 28
	455	4.06	34, 40, 26
Thalamus/DS	421	3.49	8, -18, -2
AI	383	3.26	44, 16, 6

DMPFC = dorsomedial prefrontal cortex, SMA = supplementary motor area, ACC =

dorsal anterior and mid cingulate cortex, PCC = posterior cingulate cortex and

precuneus, DLPFC = dorsolateral prefrontal cortex, TPJ = temporoparietal junction, AI =

anterior insula, DS = dorsal striatum.

   Group comparisons via Welch's *t*-test. Subsequently, analyses were

performed on the ventral-dorsal MPFC index, comparing individuals who had

perpetrated at least one form of IPV to those who had not. A Welch's *t*-test was

employed instead of a Student's t-test, as the number of participants and subsequently

the variance among participants who had and had not perpetrated IPV was not equal

(Delacre, Lakens, & Leys, 2017). The Student's test relies on a variance estimate that is

pooled across both groups. The accuracy of this pooled variance estimation is

contingent on the two groups having mostly equal sample sizes and when this is not the

case, there are marked increases in Type I and II errors (Delacre et al., 2017). The

Welch's test circumvents the issue of unequal sample sizes and consequently, unequal

variance, be using an unpooled estimate of variance that is calculated independently for

each group. Using this statistical approach, IPV perpetrators showed less ventral-than-

dorsal MPFC reactivity to provocation than non-IPV perpetrators, $t(57.27) = -2.49$, $p =$

.016 (Figure 4). Stated otherwise, IPV perpetrators MPFC responses to provocation

from a stranger in the MRI scanner were biased towards the dorsal MPFC and away

from the ventral MPFC.

Figure 4. Histograms depicting greater ventral-than-dorsal MPFC responses to provocation among participants with no history if IPV perpetration than perpetrators.
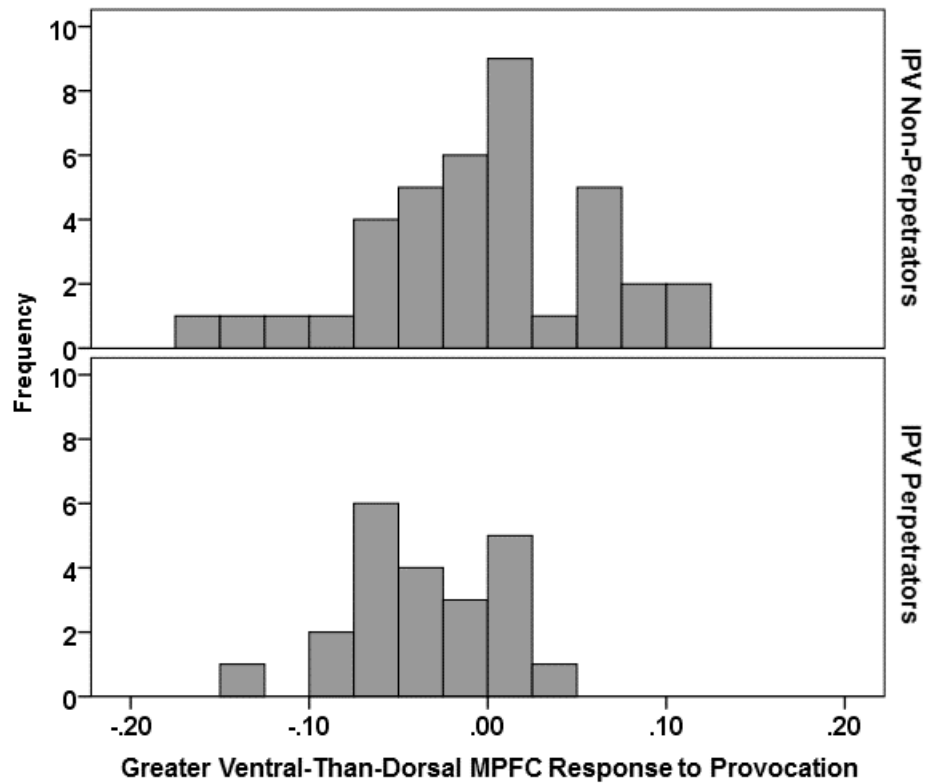
IPV perpetration likelihood via logistic regression models. To test whether more dorsal-than-ventral MPFC reactivity to provocation was able to discriminate between those who had perpetrated IPV at least once in their lifetime and those who had not, we employed binary logistic regression models. Indeed, more ventral-than-dorsal MPFC reactivity was associated with a reduced likelihood of IPV perpetration, $OR < 0.01$, $B = -10.89$, $SE = 5.24$, $X(1, 58) = 4.33$, $p = .038$. Including gender (Female = 0, Male = 1) in this model did not meaningfully change the result, $OR < 0.01$, $B = -10.94$, $SE = 5.33$, $X(2, 57) = 4.22$, $p = .040$. Thus, participants with MPFC responses to provocation that were biased towards the ventral aspect of this region, and away from the dorsal aspect, were much less likely to have perpetrated IPV.

Correlations with IPV perpetration severity via Poisson-based generalized loglinear modeling. Next, we sought to test whether greater ventral-than-dorsal were associated with the number of forms of IPV perpetration (i.e., IPV perpetration severity). Due to the non-normal, zero-inflated, and count nature of the IPV data, generalized linear modeling was employed that specified a log-link function and a Poisson distribution, instead of standard parametric approaches (DeWall et al., 2013). Greater activation in the DMPFC in response to provocation was associated with greater IPV. Due to the non-normal, zero-inflated, and count nature of the IPV data, generalized linear modeling was employed, specifying a log-link function and a Poisson distribution, instead of standard parametric approaches (DeWall et al., 2013). More ventral-than-dorsal MPFC reactivity was associated with a fewer forms of IPV perpetration, $B = -5.96$, $SE = 2.90$, $X^2(1, 58) = 4.21$, $p = .041$ (Figure 5). This effect held when gender (i.e., male = 1, female = 0) was included as a covariate, $B = -5.93$, $SE = 2.92$, $X^2(2, 57) = 4.11$, $p = .043$.

Figure 5. Scatterplot depicting negative association between more ventral-than-dorsal MPFC responses to provocation and the severity of IPV perpetration performed by participants. The regression line is displayed alongside its 95% confidence interval.
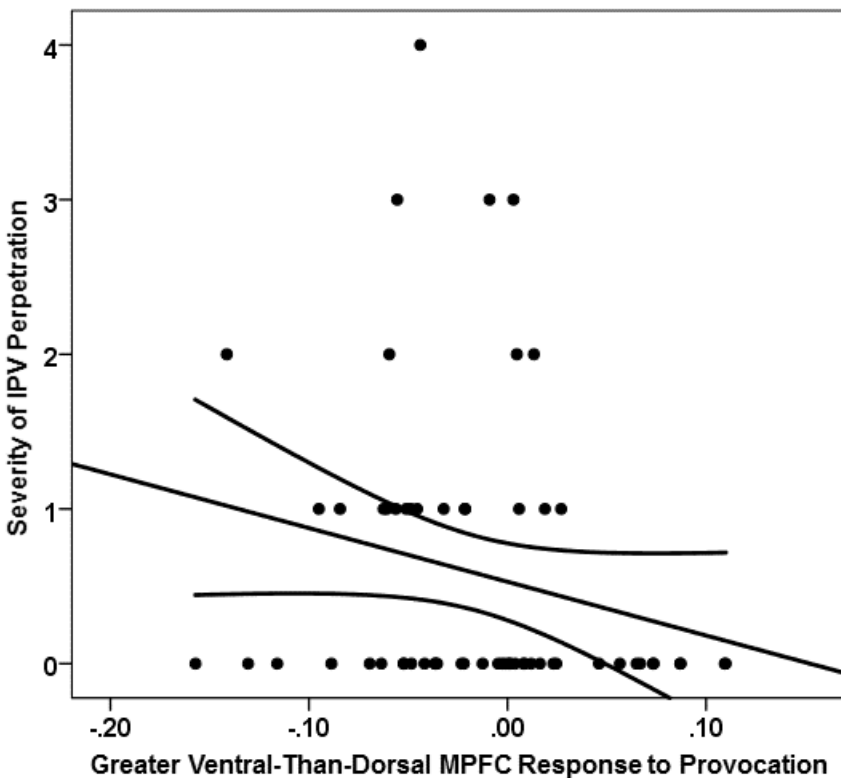
Associations with displaced aggression. Displaced aggression was unassociated with more ventral-than-dorsal MPFC reactivity to provocation, $r(55) = .11$, $p = .403$, likelihood of perpetration at least 1 act of IPV, $OR = 1.39$, $B = 0.33$, $SE = 0.24$, $X(1, 55) = 1.93$, $p = .165$, or IPV perpetration severity, $B = 0.22$, $SE = 0.14$, $X^2(1, 55) = 2.50$, $p = .114$.

Discussion

IPV perpetration is a costly societal ill and research is needed to understand its causes. Situational and personality factors that promote this behavior have been robustly investigated, indicating that *interpersonal provocation* is one of IPV perpetration's more reliable, social causes. However, the mechanisms that link provocation to IPV perpetration remain largely unknown. Using functional brain imaging, we obtained evidence that individuals who had perpetrated at least one act of IPV,

compared to those who had not, exhibited a bias in their neural responses to provocation from a stranger, and that this bias was associated with the likelihood and severity of their IPV perpetration. Building off of prior work, we demonstrated that a bias towards the dorsal aspect of the medial prefrontal cortex (MPFC) and away from the ventral aspect of this region strongly corresponded to various aspects of IPV perpetration. These findings have meaningful theoretical implications, though they must also be considered in the light of several meaningful limitations.

Implications

Neural correlates of provocation. Independent of participants' past IPV perpetration, our neuroimaging results largely replicated those of the seminal fMRI study by Denson and colleagues (2009). Specifically, we observed that provocation was associated with activation in the anterior and posterior cingulate cortex, anterior insula, dorsal MPFC, dorsolateral PFC, and thalamus (Denson et al., 2009). In addition to these regions, we also observed activation in the temporoparietal junction (TPJ), a member of the mentalizing network that largely serves to understand the content of others' mental states (Saxe & Kanwisher, 2003). This TPJ activity may thus represent the attempt to understand the thought process of participants' provocateur, and suggests provocation recruits mentalizing processes more than less antagonistic actions. We also observed activity in the dorsal striatum that included the putamen, globus pallidus, and extended into the tail of the caudate nucleus. It is unclear the role that these regions played in responses to provocation.

The dorsal-ventral distinction in the MPFC. Using a difference score computed by subtracting dorsal MPFC responses to provocation from ventral MPFC

responses, we observed that IPV perpetrators exhibited a significant dorsal bias in their

MPFC reactivity to provocation from a stranger (not an intimate partner). Further, the

more that participants exhibited this dorsal MPFC bias, the more likely they were to

have committed any act of IPV and the more severe their IPV perpetration was. These

findings mesh well with a wealth of research implicating the dorsal MPFC and the

underlying dorsal anterior cingulate cortex in aggressive responses to provocation from

strangers (Chester & DeWall, 2016; Denson et al., 2011; Krämer et al., 2007; Lotze et

al., 2007). Our results extend this work into the realm of aggression directed at *intimate*

partners, not at strangers. However, it is crucial to note that our study's provocateur was

a stranger and not an intimate partner. As such, it is uncertain whether our observed

MPFC mechanism would generalize to provocations from intimate partners.

Dorsal bias in provocation-related MPFC activity may then serve as a potential

biomarker of aggressive intent. Conversely, a ventral bias in MPFC reactivity to

provocation may serve as a biomarker for reduced aggression. These findings were

obtained while statistically covarying for gender. However, we employed a largely-

female sample and were substantially underpowered to detect any moderation effects

for gender. As such, it remains to be seen whether the putative antagonistic role of the

dorsal MPFC and affiliative role of the ventral MPFC are not specific to males or

females (as observed by Denson et al., 2009).

Retaliatory versus displaced aggression. Prior research has linked DMPFC

responses to provocation with both retaliatory (Chester & DeWall, 2016) and displaced

(Denson et al., 2011) forms of aggression. To help clarify whether the dorsal bias in

MPFC and its association with IPV perpetration reflected a tendency to retaliate against

provocation from one's intimate partner, to displace aggression onto one's intimate

partner from another individual's provocation, or both, we included a measure of

displaced aggression. This measure was unassociated with any bias in MPFC reactivity

to provocation or IPV in our study. Null results are difficult to interpret and the absence

of these effects may simply be a product of our relatively small sample size. However,

these null effects may imply that the links we observed between IPV perpetration and a

dorsal MPFC bias may reflect retaliatory, and not displaced, aggressive tendencies

against the source of individuals' perceived provocation. It could also be that the MPFC

bias' effect on IPV perpetration reflects a proactive, and not reactive, aggressive

tendency, in which individuals seek to harm others for instrumental reasons that do not

originate in perceived provocation. Our data are unable to tease these two forms apart,

but future research that experimentally manipulated whether participants' intimate

partners provoked them or not would be able to do so.

Clinical and forensic populations. IPV perpetration is a pervasive behavioral

outcome among individuals with psychopathology and among criminal offenders. If

additional support is obtained for the generalizability of the dorsally-biased MPFC

response to provocation as a precursor to IPV perpetration, then this biological indicator

might have substantial clinical and forensic utility. A simple fMRI assessment could be

developed to identify individuals most prone to IPV perpetration and interventions could

be tailored to these individuals, seeking to drive MPFC reactivity in a ventral direction.

As one example, fear extinction treatments for anxiety disorders are effective in shifting

MPFC reactivity towards ventral regions (Hofmann, 2008). Neurally-informed

interventions hold much promise for the reduction of IPV perpetration.

Limitations and Future Directions

Our sample consisted of mostly-female undergraduate students. Although

undergraduates do tend to commit IPV at moderate rates (Smith, White, & Holland,

2003), most of the severe cases of IPV perpetration occur in low socioeconomic strata

populations, such as substance dependent individuals (Field & Caetano, 2004).

Research should seek to replicate and extend our findings with more diverse samples,

with a greater emphasis on recruiting male participants who tend to commit IPV at

higher rates (Black et al., 2011). Further, the provocation scenario employed by our

study occurred in a controlled MRI scanning environment and with a same-sex stranger.

It remains unknown whether our findings would replicate in a 'real-world' scenario with

participants' actual romantic partners. Future research would benefit from comparing

the neural correlates of provocation from strangers to provocation from romantic

partners, and their relations with IPV perpetration. Further, experimental manipulations

of the perceived familiarity and closeness with a given provocateur would provide a

compelling test for whether our proposed neural mechanism is influenced by the

intimacy of a given relationship. The use of same-sex strangers as the source of

provocation also leaves the role of gender uncertain in the response to provocation.

Experimentally crossing the gender of provocateurs with familiarity would serve to

identify the respective roles of these variables. Another demographic variable that was

unexamined in the present research was that of socioeconomic status. We did not

measure socioeconomic status or include a proxy for it in this study, which leaves the

socioeconomic status of our sample unknown and the effects of this variable untested.

Given socioeconomic status' established effects on violent human behavior (Yuma-

Guerrero, Orsi, Lee, & Cubbin, in press), it is critical for future research to examine the

effects of this variable on the neural mechanisms of intimate partner violence and

related phenomena.

       The provocation paradigm was situated within a competitive context, in which

participants sought to 'beat' their 'opponents' in a competitive task. This competitive

environment may have influenced our results. Future research might induce provocation

in a more affiliative or at least innocuous context in order to assess the impact of

context on our results. Our measure of IPV does not disentangle whether the acts of

IPV were offensive or defensive in nature. Therefore, it is possible that our results

reflect the tendency for our, mostly-female, participants to be in relationships in which

IPV is perpetrated initially by their partner and they retaliate in self-defense (Black et al.,

2011). Future research should seek to replicate our findings within and without the

context of self-defense.

       These findings are purely correlational and thus prevent any claims of causality.

Future research should implement brain stimulation approaches, such as repeated

transcranial magnetic stimulation (rTMS), to experimentally manipulate brain activity in

the dorsal and ventral MPFC during provocation and then test the effect of these

manipulations on inclinations to perpetrate IPV. Finally, larger samples are needed in

future work to replicate our findings with more statistical power, and to further test for

the moderating role of gender, which we did not possess a large enough sample to

examine.

Conclusion

Why do people hurt the ones they love? The answer is a complicated one, spanning from microbiological processes to macrostructural societal and evolutionary legacies. We have provided preliminary evidence for a proximate, neural mechanism through which provocation can lead to IPV perpetration: a dorsal bias in the MPFC response to provocation. Such evidence can advance our theoretical understanding and practical intervention upon aggressive acts, hopefully yielding a more peaceful world.

Acknowledgments

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