Susceptibility to social pressure following ventromedial prefrontal cortex damage

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Social pressure influences human behavior including risk taking, but the psychological and neural underpinnings of this process are not well understood. We used the human lesion method to probe the role of ventromedial prefrontal cortex (vmPFC) in resisting adverse social pressure in the presence of risk. Thirty-seven participants (11 with vmPFC damage, 12 with brain damage outside the vmPFC and 14 without brain damage) were tested in driving simulator scenarios requiring left-turn decisions across oncoming traffic with varying time gaps between the oncoming vehicles. Social pressure was applied by a virtual driver who honked aggressively from behind. Participants with vmPFC damage were more likely to select smaller and potentially unsafe gaps under social pressure, while gap selection by the comparison groups did not change under social pressure. Participants with vmPFC damage also showed prolonged elevated skin conductance responses (SCR) under social pressure. Comparison groups showed similar initial elevated SCR, which then declined prior to making left-turn decisions. The findings suggest that the vmPFC plays an important role in resisting explicit and immediately present social pressure with potentially negative consequences. The vmPFC appears to contribute to the regulation of emotional responses and the modulation of decision making to optimize long-term outcomes.

Keywords: social pressure; prefrontal cortex; brain damage; emotion regulation; decision making; driving simulation

INTRODUCTION
Human behavior is highly susceptible to social pressure—the influence exerted by one person or group on another (Asch, 1951; Milgram, 1963). Social pressure may be explicit or implicit, positive or negative, and abrupt (‘immediately present’) or gradual over extended time frames (Markman et al., 2006). In confrontations where one person is trying to alter the behavior of another, explicit, negative and immediately present social pressure can induce stress and negative emotions (e.g. annoyance, anxiety and anger), potentially interfering with decision making and other cognitive processes (Gilligan and Bower, 1984; Lerner and Keltner, 2001; Hemenover and Shen, 2004; Forstmann et al., 2008; Porcelli and Delgado, 2009; Chen et al., 2013). This pressure may bias individuals to behave contrary to their own judgment and self-interest. Young, elderly and brain-injured individuals may be particularly vulnerable to such pressure.

Experiencing social pressure depends on detecting social disparity or conflict as well as recognizing that one’s behavior is contrary to the perceived expectation of others. The disparity or conflict serves as a type of ‘error signal’, associated with feelings or emotions that can alter response biases and prompt implicit or deliberate evaluation of the potential consequences of conforming or not. The neural and cognitive mechanisms underlying the experience of and response to social pressure are not yet well understood, but it is clear that these processes depend on distributed neural systems involving several brain regions. One important region is the caudal anterior cingulate cortex (cACC), which contributes to the detection of social disparity or conflict (Klucharev et al., 2009, 2011; Berns et al., 2010; Shestakova et al., 2012). Neural systems involved in the representation of reward and punishment are also implicated in the experience of social pressure. Individuals conforming to social pressure for social rewards show increased blood-oxygen-level dependent (BOLD) signal in the ventral striatum, while those conforming to social pressure to avoid social punishment show increased BOLD signal in the amygdala (Klucharev et al., 2009; Chein et al., 2011; Edelson et al., 2011; Tomlin et al., 2013).

In this study, we were particularly interested in the role of the ventromedial prefrontal cortex (vmPFC) in resisting ‘explicit’ and ‘immediately present’ social pressure in the presence of personal risk. The vmPFC consists of Brodmann areas 10–12, 25 and 32 (Bechara et al., 2000a) and has strong connections to the cACC, ventral striatum and amygdala (Price, 2006)–areas underpinning the experience of social pressure (Berns et al., 2010; Chein et al., 2011; Edelson et al., 2011). The vmPFC plays a hypothetical role in modulating values of reward and punishment (Damasio, 1994; Anderson et al., 1999; Bechara et al., 2000b) and regulating emotions (Grafman et al., 1996; Levine et al., 1999; Eslinger et al., 2004; Anderson et al., 2006). We and others have observed vulnerability to negative social pressure in the real-world decision making of patients with vmPFC damage. The vmPFC may enable resistance to negative social pressure by modulating values of social conformity/disconformity and downregulating negative emotions such as stress under social pressure.

This study used the human lesion method and applied social pressure in a controlled scenario in a driving simulator. All participants were pressured by a virtual driver while performing a left-turn decision task, allowing us to measure the consequences of controlled ‘explicit’ and ‘immediately present’ social pressure. Resistance to social pressure was defined as the difference in left-turn decision making between the control condition vs the social pressure condition. The task required the participant to decide which gaps were safe for left turns across a stream of oncoming vehicles with various between-vehicle gap sizes (3–9 s; Hancock et al., 1991; Chen et al., 2013; Rusch et al., 2014). Lower social pressure resistance was indexed by choosing smaller (less safe) gaps under social pressure in comparison to the control condition.

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predicted that participants with vmPFC damage would show: (i) lower ability to resist social pressure compared with the participants without vmPFC damage and (ii) poorer regulation of negative emotion under social pressure, based on skin conductance response (SCR), a measure of sympathetic nervous system activity (Dawson et al., 2007).

MATERIALS AND METHODS

Participants

Thirty-nine participants were recruited. One dropped out of the study early and another was excluded due to poor effort. The remaining 37 participants included 11 individuals with vmPFC damage (9 bilateral and 2 unilateral; Figure 1A), 12 with brain damage outside the vmPFC (i.e. brain damaged comparison, BDC) and 14 age-matched individuals with no history of neurologic disease (normal comparison, NC). Brain-damaged participants were recruited from the Patient Registry in the Department of Neurology at the University of Iowa. Etiology of vmPFC lesions included benign tumor resection (n = 6) and hemorrhagic stroke (n = 5). The BDC group included benign tumor resection (n = 3), hemorrhagic stroke (n = 3) and ischemic stroke (n = 6). All participants were studied in a chronic phase, at least 2 years and 3 months after lesion onset (Table 1). All participants were active automobile drivers (except one vmPFC participant who stopped driving after the onset of brain lesion), had normal or corrected-to-normal vision and had no major psychiatric disorders.

Neuroanatomical analysis

The neuroanatomical analysis used magnetic resonance (MR; 1.5 T) scanner, Spoiled Gradient Recalled sequence of 1.5 mm and

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Fig. 1 (A) Lesion overlap of the vmPFC group (n = 11). (B) Driving simulator was a four-door 1994 General Motor Saturn with three front and one back display channels. (C) Schematic design of the social pressure condition. A vehicle honked continuously from behind the participant when the participant contemplated left turns across a stream of oncoming vehicles. The gaps between oncoming vehicles varied and gradually increased from 3 to 9 s.

1 Subject patient registry ID: 297, 318, 1983, 2352, 2391, 2577, 3349, 3350, 3383, 3534, 3535.
### Table 1 Demographic characteristics

|                | vmPFC | BDC | NC | Frt | P   |
|----------------|-------|-----|----|-----|-----|
| N              | 11    | 12  | 14 |     |     |
| Sexb           | 5M, 6F| 8M, 4F| 6M, 8F | 0.46|
| Handednessb    | 9R, 2L| 9R, 1L, 2B| 11R, 3L | 0.47|
| Lesion sideb   | 1L, 9B, 1R| 7L, 3B, 2R | 0.02 |
| Age (years), mean (SE) | 64.85 (2.07) | 63.97 (1.80) | 62.66 (1.68) | 0.37 |
| Years of education (years), mean (SE) | 14.27 (0.74) | 14.83 (0.75) | 16.07 (0.59) | 1.86 |
| Lesion chronicity (years), mean (SE) | 13.52 (3.31) | 9.41 (2.42) | 1.04 (0.32) | 0.02 |
| WAIS III Full Score, mean (SE) | 107.80 (4.88) | 101.92 (3.09) | 11.31 (0.31) | 0.72 |
| Beck anxiety inventory, mean (SE) | 3.82 (1.15) | 3.08 (1.15) | 5.57 (1.95) | 0.50 |
| Current driving status (miles per week), mean (SE) | 102.73 (30.01) | 150.91 (60.91) | 98.93 (20.52) | 0.55 |

*M = male; F = female; *R = right; L = left; b = both; *WAIS = Wechsler Adult Intelligence Scale.

Table 1 shows that they considered large enough to make a safe left turn. To select a safe gap, the participant engaged the high-beam lever. Participants were asked to treat the task as realistically as possible and to avoid crashes. Participants selected 10 safe gaps in each condition. Preliminary analyses suggested that the social pressure effect (upon decision making and SCR) between the vmPFC and the comparison groups attenuated toward the end of the task (Supplementary Figure S3). Therefore the last four responses were not included in analyses.

In the social pressure condition, a red Grand Prix pulled up directly behind the participant’s stopped vehicle and began honking aggressively (Figure 1C; Supplementary Video). To make the honking sound realistic, we created eight honking patterns (e.g. 4 consecutive short honks, 1 long honk lasting 4 s), which challenged the participant in a pseudorandom manner. The intervals between consecutive honking patterns varied from 2 to 16 s (Mean = 8.49 s). Every time the participant made a ‘go’ response, the honking stopped for 10 s.

To make oncoming traffic appear realistic and unpredictable, we created three gap schedules (each comprising 141 gaps; Supplementary Table S1). Two of the gap schedules were randomly assigned to the two experimental conditions for each participant. The schedules were based on four rules. First, each gap schedule consisted of 60 target gaps (gap size ≥ 4 s) and 81 filler gaps (gap size ≥ 3 s). Timing definitions were based on the results from a pilot study where we learned that no participant ever deemed the 3 s gap safe for a left turn. Data from this study showed a similar pattern. Second, there was always one to three filler gap(s) between consecutive target gaps. Third, the 60 target gaps of each schedule were further divided into four blocks (i.e. Block 1–4). Early blocks (e.g. Block 1) contained a high frequency of small gaps (e.g. 5, 6, 4, 5, 7, 5, 4, etc.). Conversely, late blocks (e.g. Block 4) contained a high frequency of large gaps (e.g. 8, 9, 8, 5, 9, 9, 9, 4, etc.). Thus, the further the participant got into the block sequence, the larger the gaps became. Fourth, the three schedules were identical by blocks in terms of the composition of gap durations (Supplementary Table S1).

**Procedure**

Participation involved one laboratory visit. Upon arrival, participants provided informed consent in compliance with the local Institutional Review Board. Participants then completed demographic questions and basic visual function tests. Electrodes for psychophysiological recording were attached. Then the participant moved into the simulator cab. Task instructions were projected on the main display channel and spoken by the experimenter. Before beginning experimental task, the participant performed two practice sessions. After completing each left-turn decision task (control condition, social pressure condition), the participant rated emotion pleasantness (1 = very unpleasant, 7 = very pleasant) and intensity (1 = very low, 7 = very high). Immediately after the tasks each participant completed a Motion Sickness Questionnaire, Driving Simulator Experience Questionnaire and Beck Anxiety Inventory. All participants were debriefed before they left the study.

**Decision making**

To examine differences between conditions, we calculated response rates for each gap duration (e.g. 4, 5 and 6 s) during each condition.
RESULTS

Demographic characteristics

Descriptive statistics of demographic characteristics are displayed in Table 1. Note that the vmPFC group had more bilateral lesions and fewer left-side only lesions compared with the BDC group. No other variables indicated any differences among the groups.

Emotion rating

Means and standard errors (SE) of emotion ratings4 are illustrated in Figure 2A. Results from paired t-tests show that the social pressure condition was rated less pleasant than the control condition by the BDC \( t(10) = 2.28, P = 0.046 \) and NC groups \( t(13) = 3.82, P = 0.002 \). A similar but non-significant trend was found in the vmPFC group \( t(10) = 1.79, P = 0.10 \). For the intensity of experienced emotion, only the vmPFC group rated higher emotion intensity in the social pressure condition than in the control condition \( t(10) = 3.32, P = 0.008 \).

Decision making

We examined the smallest gap that participants deemed safe (response rate \( \geq 50\% \)) and illustrated the descriptive statistics and the results from our hypothesis test in Figure 2B left. Only the vmPFC group showed significant decrease on these gaps from the control to the social pressure condition \( t(10) = -2.61, P = 0.03 \). BDC and NC groups did not show this decrease. However, one-way ANOVA showed that the gap-selection change from the control to social pressure conditions was not significantly different between groups, \( F(2, 34) = 1.57, P = 0.22 \) (Figure 2C left).

We tested if the smallest gap that participants deemed safe to go more often than not (response rate \( \geq 50\% \)) changed between conditions (Figure 2B middle). Paired t-tests showed that these gaps became significantly smaller under social pressure in the vmPFC group \( t(10) = 4.50, P = 0.001 \), but not in the BDC or NC groups. When comparing the three groups in terms of gap-selection changes, a one-way ANOVA showed a significant group effect \( F(2, 34) = 5.00, P = 0.01 \). Results from post hoc comparisons showed the change was larger in the vmPFC group than in the BDC \( (P = 0.01) \). There was a trend toward larger change in the vmPFC group compared with the NC group \( (P = 0.08) \). No significant difference was found between the two comparison groups (Figure 2C middle).

We also examined if the smallest gap that participants always deemed safe to go (response rate \( = 100\% \)) changed between conditions (Figure 2B right). Paired t-tests showed significant decrease in these gaps only in the vmPFC group \( t(10) = 3.07, P = 0.01 \). When comparing the three groups in terms of the changes between conditions, a one-way ANOVA showed significant effect of group \( F(2, 34) = 3.68, P = 0.04 \). Post hoc comparisons showed that the change was larger in the vmPFC group than in the NC group \( (P = 0.049) \). There was a trend toward larger change in the vmPFC group than in the BDC group \( (P = 0.099) \). No significant difference was found between the two comparison groups (Figure 2C right).

Results, described earlier, suggest that the vmPFC group selected significantly smaller gaps under social pressure than in the control condition. This effect was not evident in the two comparison groups. Additional one-way ANOVAs were performed to test if gap selection in the control condition varied significantly across groups, as might occur if participants with vmPFC damage were less apt to wait (for large gaps) or judge that oncoming vehicles would slow down and not hit them. Results for all different response rates

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4 Emotion rating data from one BDC participant was incomplete due to procedural error.
(≥ 0%, ≥ 50%, = 100%) showed no significant effect of group \(F(2, 34) < 0.70, P > 0.50\).

**Skin conductance response**

Figure 3A–C shows SCR traces from (i) the first 10 s of each task/condition (i.e. initial SCR) and (ii) the 10 s before each gap selection (i.e. pre-response SCR). Figure 3D shows the differences between conditions (social pressure—control) for each group.

As shown in Figure 3A–C left, the ‘initial SCR’ was significantly higher in the social pressure condition than in the control condition in all three groups [vmPFC: \(t(10) = 2.67, P = 0.02\); BDC: \(t(11) = 2.20, P = 0.049\); NC: \(t(13) = 2.72, P = 0.02\)]. One-way ANOVA showed no significant group difference in changes in initial SCR from control to social pressure conditions \(F(2, 34) = 1.92, P = 0.16\), which suggests that social pressure triggered similar initial SCR (measured in the first 10 s of the condition) in all three groups.

To examine SCR prior to gap selection, we analyzed the average of the six ‘pre-response SCRs’ (measured 10 s before each gap selection). Paired t-tests showed that only the vmPFC group exhibited significantly higher pre-response SCR under social pressure than in the control condition \(t(10) = 2.95, P = 0.01\); Figure 3A right). When comparing the three groups in terms of pre-response SCR changes between conditions, a one-way ANOVA showed a significant group effect \(F(2, 34) = 5.15, P = 0.011\). Post hoc comparisons showed larger pre-response SCR change in the vmPFC group compared with the NC group.
group \( (P = 0.013) \). There was a trend toward larger pre-response SCR change in the vmPFC compared with the BDC group \( (P = 0.055) \). No significant difference was found between the two comparison groups (Figure 3D right).

**DISCUSSION**

This study examined the mechanism underlying the resistance to explicit and immediately present social pressure, and particularly the role of the vmPFC in this mechanism. Individuals with vmPFC damage selected smaller, potentially unsafe gaps in traffic under the influence of social pressure. This pattern was not evident in two comparison groups (BDC and NC). All three groups showed high SCRs on the first exposure to social pressure (i.e. initial SCR). Participants with vmPFC damage showed significantly higher SCR under social pressure than in the baseline (control condition) during the time period of left-turn decision making (i.e. pre-response SCR). In contrast, during the same time period, the two comparison groups showed similar degrees of SCR under social pressure as in the control condition.

The social pressure in this study elicited unpleasant emotions in the participants. Both comparison groups rated the social pressure condition less pleasant than the control condition. A similar trend was obtained in the vmPFC group, although the effect was not statistically significant. We suspect that this might reflect poor self-awareness of emotion after vmPFC damage (Barrash et al., 2000). Another possible explanation for this could be a deficit in affect labeling, although prior research has primarily linked this function to the right ventrolateral prefrontal sectors (Lieberman et al., 2007; Burklund et al., 2014).

Participants with vmPFC damage showed sustained high SCR and reported experiencing more intensive emotion in the social pressure than in the control conditions, suggesting lower ability to regulate negative emotions associated with social pressure. This result is consistent with previous studies showing the involvement of the vmPFC in emotion regulation (Grafman et al., 1996; Levine et al., 1999; Barrash et al., 2000; Eslinger et al., 2004; Anderson et al., 2006). In this study, sustained emotional disturbance in participants with vmPFC damage may have disrupted their ability to conduct an objective and balanced evaluation of the contingencies and hazards presented in the traffic scenario. This emotional disturbance may increase attentional focus on the aggressive driver (rather than on the task of making safe left turns), with increasing bias to comply with the stressor to mitigate unpleasant feelings.

The vmPFC may contribute to emotion regulation and thereafter the resistance to social pressure by modulating the sensitivity to immediately present stimuli at the expense of long-term goals and future outcomes (Damasio, 1994; Anderson et al., 1999; Bechara et al., 2000b). In the left-turn decision task, the experience of social pressure is immediately present and emotionally salient, which triggers the urge to conform to the aggressive driver. The immediately present goal of avoiding social pressure conflicts with the long-term goal of making safe decisions for left turns. Participants with vmPFC damage may have failed to reduce their sensitivity to the immediately present goal, resulting in a higher compliance with, or lower resistance to, social pressure.

Our finding that damage to the vmPFC increases susceptibility to social pressure adds to previous reports that (i) vmPFC damage decreases tendency to conform to the expectations of another ‘person’ in the Trust Game (Kraibich et al., 2009) and (ii) vmPFC BOLD signals in normal individuals increase with behavioral changes after persuasion (Falk et al., 2010). The vmPFC may bias subjective values of choices and goals (especially appetitive; Bartra et al., 2013), and we hypothesize...
that vmPFC also contributes to a re-evaluation process when multiple choices/goals conflict with each other (i.e. appetitive vs aversive, short-term vs long-term, implicit vs explicit). Damage to the vmPFC does not inherently increase or decrease social conformity. Rather, as mentioned earlier, it enhances the tendency to respond to explicit, emotionally salient and immediate-present goals (e.g. to terminate social pressure) and diminishes the ability to consider implicit and long-term outcomes (e.g. to drive safely). In this vein, vmPFC damage affects Iowa Gambling Task performance, with hyper-responsiveness to explicit and immediate outcomes whether they are gains or losses (Bechara et al., 2000b).

The findings of this study may have practical implications. Susceptibility to social pressure was evaluated in a left-turn scenario, the third most common setting for single- and two-vehicle crashes (resulting in ~877,000 crashes, with 32% associated with fatality or injury in 2012 in USA; National Highway Traffic Safety Administration, 2014). Our findings suggest an increased risk of unsafe driving in this situation (making left turns under social pressure) for drivers with vmPFC dysfunction, such as aging drivers (Tisserand et al., 2002; Resnick et al., 2003; Raz et al., 2004; Lemaître et al., 2005) or drivers with neurological disorders affecting the vmPFC (Rosen et al., 2002; Salmon et al., 2003). Increased risk can also be implied for young drivers with immature vmPFC functions (Paus, 2005; Blakemore and Choudhury, 2006). With better knowledge about the effect of social pressure on driving, these drivers may benefit from advanced driver assist systems to mitigate the risks of driving under social pressure. For example, such systems can automatically detect safe gaps and provide go/no-go suggestions (Caird et al., 2008; Parasuraman and Wilson, 2008; Rusch et al., 2014) or provide warning signals to delay the immediate yet maladaptive reactions to social pressure. As the preliminary data showed, the adverse effect of social pressure was strongest when it first occurred (Supplementary Figure S3). Specific counseling and intervention for patients and families regarding potentially increased vulnerability to social pressure may also be warranted (e.g. learn how to inhibit the immediate reactions to social pressure).

In this study, we used driving simulation to simulate a common scenario in real-world driving—a traffic conflict with another driver. This particular scenario involved (i) a driver waiting behind, (ii) the intent of that driver and (iii) a manner to deliver the intent, which usually is honking. In most cases, social information on the driver waiting behind is unknown. We attempted to capture those factors in the context of a realistic simulated environment. Because social pressure was applied via honking, the observed effect might reflect reactions to loud noises rather than feeling socially pressured. This seems unlikely, as participants reported feeling the scenarios were highly realistic. If the effects simply reflected a reaction to noise, SCR would drop with the cessation of honking. In Supplementary Figure S5, participants with vmPFC damage still show significantly higher SCR in the social pressure condition (compared with the control condition) even after honking was paused for 10 s after ‘go’ responses.

Other alternative accounts of our findings include that participants with vmPFC damage may be more generally less willing to wait for larger gaps, and/or they may have greater belief that drivers in the oncoming traffic lane will slow down to avoid collision, and therefore feel more comfortable taking smaller gaps for left turns. We consider these explanations unlikely because if the vmPFC-damaged participants were generally less willing to wait or had increased tendency to trust drivers in the opposite lane, they would have taken smaller gaps not only in the social pressure but also in the control condition. As the results showed, this was not the case. In addition, the speed of the oncoming vehicles remained constant throughout the entire study, providing no indication that the oncoming vehicles might slow for the participant to cross.

The vmPFC group had a higher proportion of bilateral lesions relative to the BDC group (9/11 = 82% vs 3/12 = 25%; Fisher’s exact test of P = 0.012). We cannot rule out the possibility that this may have contributed to the severity of the observed impairments. However, the vmPFC group included one participant with unilateral damage to the right side and one participant with unilateral damage to the left side, and their behavioral and SCR patterns were well within the range of the others with bilateral vmPFC damage. Similarly, three BDC participants had unilateral brain damage. Their behavioral and SCR patterns were also within the range of the other BDC participants with unilateral lesion (except one bilateral BDC participant who showed lower SCR in the social pressure condition than in the control condition). When comparing participants with bilateral (n = 12) vs unilateral lesions (n = 11) regardless the lesion sites, no statistical difference was found (Mann–Whitney tests, P > 0.23). In the BDC group, there appears to be more left-side lesion than right-side (7/12 = 58% vs 2/12 = 17%). We consider laterality would unlikely confound our findings. Mann–Whitney tests indicated that damage to either the right (n = 5) 6 or left hemispheres (n = 10) 6 outside the vmPFC did not increase the susceptibility to social pressure (right-side damaged vs right-side spared: P > 0.59; left-side damaged vs left-side spared, P > 0.14).

In conclusion, this study provides novel evidence that focal damage to the vmPFC is associated with diminished ability to resist explicit and immediately present social pressure with potentially negative consequences. We suggest that the vmPFC contributes to this function by regulating negative emotions associated with social pressure, and modulating sensitivity to immediately present stimuli in connection with future outcomes.

SUPPLEMENTARY DATA
Supplementary data are available at SCAN online.

Conflict of Interest
None declared.

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5 Our data indicated that participants considered our scenarios highly realistic compared to their real-world experience. As shown in Supplementary Figure S4, participants rated ‘the sounds of horns’ and ‘the behaviors of other vehicles’ 5.67 and 5.06, respectively, on a ‘realistic’ scale from 1 (not realistic) to 7 (realistic).

6 Including BDC participants with bilateral brain damage.
