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A neuropsychological investigation of decisional certainty

Aaron M. Scherer [a,*], Bradley C. Taber-Thomas [b,d], Daniel Tranel [c,d]

[a] Center for Bioethics and Social Sciences in Medicine, University of Michigan, Ann Arbor, MI, USA
[b] Department of Psychology, Penn State University, University Park, PA, USA
[c] Department of Psychology, University of Iowa, Iowa City, IA, USA
[d] Department of Neurology, University of Iowa, Iowa City, IA, USA

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ABSTRACT

The certainty that one feels following a decision increases decision-making efficiency, but can also result in decreased decision accuracy. In the current study, a neuropsychological approach was used to examine the impact of damage to the ventromedial prefrontal cortex (vmPFC) on core psychological processes promoting decision certainty: selective exposure, overconfidence, and decisiveness. Given previous research demonstrating that vmPFC damage disrupts the generation of negative emotional (somatic) states that have been associated with selective exposure and overconfidence, it was hypothesized that damage to the vmPFC would disrupt engagement in selective exposure, decrease overconfidence, and increase indecision. Individuals with vmPFC damage exhibited increased indecision, but contrary to our hypothesis, engaged in similar levels of selective exposure and overconfidence as the comparison groups. These results indicate that indecision may be an important psychological mechanism involved in decision-making impairments associated with vmPFC injury. The results also suggest that the vmPFC may not be critical for selective exposure or overconfidence, which provides support for a recent "desirability" account of selective exposure.

1. Introduction

Feeling certain about the decisions we make is a double-edged sword. On the one hand, decision certainty can increase decision-making efficiency and reduce the negative, uncomfortable arousal that results from uncertainty or from considering that we might have made the wrong decision (Harmon-Jones et al., 2009). On the other hand, decision certainty can lead us to engage in increased confirmatory information seeking (selective exposure; Hart et al., 2009); to think that our decisions are correct and that outcomes linked to our decisions are more likely than is warranted (overconfidence; Moore and Healy, 2008); and to spend less time evaluating new decision-relevant information (decisiveness; Webster and Kruglanski, 1994); all of which undermine our ability to objectively reassess the quality of our decisions to guide future decision making (e.g., Kray and Galinsky, 2003; Schulz-Hardt et al., 2002). Despite the literature examining these core psychological processes involved in decision certainty and the implications of decision certainty in a wide variety of contexts—from business (e.g., Karlsson et al., 2009) to politics (e.g., Knobloch-Westerwick, 2012) to medicine (e.g., Kostopoulou et al., 2009)—the underlying neural mechanisms are unknown.

The current study tested whether the ventromedial prefrontal cortex (vmPFC) is a key neural substrate underlying decision certainty. The vmPFC, which includes Brodmann areas 10, 14, 25, 32, and sections of Brodmann areas 11–13, is extensively connected to structures in the limbic system (i.e., amygdala, hippocampus, and insula), hypothalamus, and brain stem (Öngür and Price, 2000; Rolls, 2000). Individuals who sustain damage to the vmPFC are remarkable in that they exhibit relatively intact intellectual functioning, yet have deficits in complex decision making, resulting in atypical decisions and judgments in the economic, social, and moral domains (e.g., Anderson et al., 1999; Bechara et al., 1997). These decision-making deficits have been posited to result from disruption in the integration of emotional responses (somatic markers) into decision making (Damasio, 1994).

It is this disruption in the integration of emotional responses into decision making that underlies the hypothesis that the vmPFC may be a critical neural substrate for in decision certainty. Experimental psychologists have argued that defense motivation, a motivation to avoid the negative emotional (somatic) state that results from being wrong or seeing indications that one is wrong, plays a key role in processes involved with decision certainty, such as selective exposure (Hart et al., 2009; Jonas et al., 2006; Smith

* Correspondence to: University of Michigan, Center for Bioethics and Social Sciences in Medicine, 2800 Plymouth Rd, Building 16, Rm 430W, Ann Arbor, MI 48109, USA.
E-mail addresses: aaschere@med.umich.edu, aaron-scherer@uiowa.edu (A.M. Scherer).

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et al., 2008). Negative somatic states associated with defense motivation include anticipated regret (e.g., Zeelenberg, 1999), embarrassment or guilt (e.g., Higuchi and Fukada, 2008), and cognitive dissonance (e.g., Zanna and Cooper, 1974). Consequently, if damage to the vmPFC disrupts the negative emotional states that drive processes that increase decision certainty, like selective exposure, then damage to the vmPFC should lead to decreased decision certainty.

Several lines of evidence from neuroimaging and lesion studies suggest that the vmPFC is involved in processes related to decision certainty, such as affective states related to defense motivation, processing belief-inconsistent information, and decisiveness.

First, lesion and neuroimaging studies support the involvement of the vmPFC in experiencing the negative affective states associated with defense motivation, such as guilt and embarrassment (Krajbich et al., 2009; Takahashi et al., 2004) or anticipated regret (Anderson et al., 1999; Camille et al., 2004). Defense motivation is hypothesized to drive engagement in selective exposure and subsequent feelings of overconfidence (e.g., Smith et al., 2008). Consequently, this research would be consistent with a role for the vmPFC in selective exposure and overconfidence, decision processes that, when intact, serve to solidify one's decision certainty.

Second, neuroimaging studies have shown increased vmPFC activation during the comparison of different options (Boorman et al., 2009; Chau et al., 2014) and when participants passively view belief-inconsistent information (Kato et al., 2009; Westen et al., 2006). Increased vmPFC activation is also associated with attitude perseverance in the face of conflicting information (Kato et al., 2009). These studies suggest that the vmPFC may be involved in evaluation of relative evidence and devaluing of disconfirmatory information in order to maintain decision certainty, a process which has been repeatedly demonstrated in the psychological literature (e.g., Fischer et al., 2010).

Third, it has been observed that individuals exhibit increased indecisiveness following injury to the vmPFC (Anderson, et al., 2006; Barrash et al., 2000). For example, in the classic report of patient E.V.R., Eslinger and Damasio (1985) note that "Deciding where to dine might take hours, as he discussed each restaurant's seating plan, particulars of menu, atmosphere, and management" (p. 1732). However, these observations have not been consistently replicated in the few experimental studies of decision timing in vmPFC patients (Ciaramelli et al., 2007; Fellows, 2006; Rogers et al., 1999; Young et al., 2010). One study to find increased decision times in patients with orbitofrontal damage (Rogers et al., 1999) used a task in which participants indicated decision certainty by betting on their choice. Given the predictable structure of the task (choice followed by a bet with the same betting options each time), prior to entering choices participants may have already been evaluating their post-decisional certainty (i.e., "deliberating" their bet), suggesting reduced decision-certainty may account for the increased response time. Decision certainty is also readily observed in the real world, where one may hem-and-haw, making a choice and re-evaluating it, precisely where increased indecision has been reported in vmPFC patients. If reports of indecisiveness due to vmPFC injury are indicative of diminished decision certainty, as predicted here, this could be captured in longer reaction times in the post-decisional (but not pre-decisional) phase.

To summarize, the vmPFC plays a critical role in generating the negative somatic markers associated with defense motivation in response to decision-inconsistent information, which is thought to drive selective exposure and overconfidence. Additionally, evidence that vmPFC damage increases indecisiveness suggests that the length of time for deciding which information to read may be affected by vmPFC damage. We predict that damage to the vmPFC will diminish processes of post-decisional certainty: selective exposure, overconfidence, and decisiveness.

2. Material and methods

2.1. Participants

A group of nine individuals with bilateral vmPFC damage (vmPFC group) and 10 brain-damaged comparison individuals (BDC group) were recruited from the Patient Registry of the University of Iowa's Division of Behavioral Neurology and Cognitive Neuroscience. The BDC group contained only individuals with brain lesions located outside the vmPFC and other emotion-related areas, such as the limbic system and insula. The two groups were comparable on demographic variables and chronicity (time since lesion onset), and all patients were tested in the chronic epoch, three or more months post-lesion onset (Table 1). A group of 15 neurologically normal comparison participants (NC group) was recruited from the local community (Table 1). The only significant difference between the three groups was in education, $F(2,31)=6.34$, $p=.005$. Post-hoc comparisons revealed that the vmPFC group had overall lower education than the two comparison groups, $ps<.02$. However, there was no main effect or significant interactions with education on the main dependent variables, so it will not be discussed further, $Fs<1.5$. The neuropsychological profiles of individuals in the vmPFC group revealed generally intact performances on standard intelligence and neuropsychological tests (Table 2). This research was approved by the University of Iowa Internal Review Board.

Table 1
Demographic and clinical data.

Patient	*Gender*	*Age*	*Education*	*Handedness*	*Chronicity*	*Etiology*
0318	M	72	14	R	36	Meningioma resection
1983	F	49	13	R	16	SAH
2352	F	64	14	R	13	SAH; ACoA aneurysm
2391	F	66	13	R	12	Meningioma resection
2577	M	72	12	R	13	SAH; ACoA aneurysm
2990	M	23	12	R	18	Focal brain injury from trauma
3350	M	60	18	R	8	Meningioma resection
3534	F	73	12	R	2	Meningioma resection
3591	F	70	12	R	3	SAH
vmPFC, mean (*SD*)	4M;5F	60.9 (16.1)	13.3 (1.9)	9R	13.4 (10.1)	
BDC, mean (*SD*)	6M;4F	59.1 (13.8)	16.0 (2.3)	8R;1L;1M	11.7 (8.9)	
NC, mean (*SD*)	4M;11F	62.6 (8.1)	16.6 (2.4)	11R;1M;3 unknown		

Individual participants are in the vmPFC group. Group mean and *SD* values for all participants are reported below the individual vmPFC patient data. Age is in years at time of testing. Education is in years of formal schooling. Chronicity is the time between lesion onset and completion of the present experiment, in years. Handedness reports dominant hand preference. Etiology describes the cause of neurological lesion (SAH=subarachnoid hemorrhage; ACoA=anterior communicating artery).