



## Right frontal cortical lesions disrupt anger mimicry

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### ABSTRACT

The current study investigates the neural substrates of facial expression mimicry by assessing individuals with right and left lateralised frontal cortical lesions. Electromyography was used to measure spontaneous changes in electrical activity over the *corrugator supercilii* (brow) and *zygomaticus major* (cheek) muscle regions in response to happy and angry facial expressions. Individuals with right ( $n=4$ ) and left ( $n=5$ ) frontal cortical lesions and demographically matched controls ( $n=9$ ) were compared. It was shown that while all three groups mimic happy facial expressions, only controls and individuals with left frontal lesions mimic angry expressions. These data are consistent with evidence for right frontal cortical specialisation for the processing of anger.

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### 1. Introduction

Viewing a facial expression spontaneously induces a congruent facial reaction in the observer (Dimberg, 1982; Dimberg & Lundquist, 1988), even when the observed face is not perceived consciously (Bailey & Henry, 2009; Dimberg, Thunberg, & Elmehed, 2000). Most investigations of the human neural substrates of this mimicry phenomenon have focused on the imitation of hand and finger movements, and only a handful have assessed facial expression mimicry. The first of these studies found that explicit instructions to imitate facial expressions result in predominantly right lateralised inferior frontal gyrus activation (Lee, Josephs, Dolan, & Critchley, 2006). However, it is of particular interest to understand the neural mechanisms of *spontaneous*, as opposed to intentional, facial expression mimicry since it has been suggested that this type of behaviour facilitates successful social interaction, including interpersonal rapport (Lakin & Chartrand, 2003) and emotion recognition (Goldman & Sripada, 2005; Oberman, Winkielman, & Ramachandran, 2007). Some studies do not find that this type of mimicry is associated with emotion recognition (Bogart & Matsumoto, 2010), but they do show that it facilitates

congruent emotions between interaction partners (Blairy, Herrera, & Hess, 1999).

Each of these potential functions of spontaneous facial expression mimicry is consistent with a motor theory of empathy (Leslie, Johnson-Frey, & Grafton, 2004). According to this theory, perceiving another's facial emotion spontaneously activates one's own motor representation of that emotion, which in turn facilitates recognition of, and empathy for, the observed emotion (Lipps, 1903; Preston & de Waal, 2002). This form of motor mimetic responding is also considered a form of empathy in its own right (Blair, 2005), with studies showing that stronger facial expression mimicry is associated with higher levels of empathy (Dimberg, Andreasson, & Thunberg, 2011; Sonnby-Borgstrom, 2002), in particular when mimicry occurs via the *corrugator* (i.e., brow) region in the upper half of the face (Harrison, Morgan, & Critchley, 2010). Disruptions of mimicry in groups with reduced capacity for empathising, such as autism (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006) and schizophrenia (Varcin, Bailey, & Henry, 2010) provides further evidence for the link between mimicry and empathy.

In a brain imaging study, Leslie et al. (2004) found that whereas passive viewing of a facial expression activates the right frontal cortex, explicit instructions to imitate results in bilateral frontal activation. This suggests dissociation between conscious left frontal control of facial musculature and right frontal involvement in spontaneous empathic mimicry. It has also been shown that observation of facial expression stimuli activates the right premotor cortex (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003), contributing to the suggestion that the right, but not left, frontal region may play an

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**Table 1**  
Individual patient characteristics.

Patient	Age/sex	Localisation	Pathology	Years post surgery/(diagnosis)	GDS	NART
ME	62/F	R frontal	Meningioma	3	0	122
CJ	61/F	R frontal	Meningioma	13	8	100
DB	45/M	R posterior frontal	AVM	7	14	98
KM	62/M	R posterior frontal	AVM	(3)	18	111
DE	75/F	L frontal	AVM	6	2	91
LH	56/F	L frontal	Cavernous malformation	(5)	5	102
VS	49/F	L frontal	Meningioma	(7)	15	–
DC	68/F	L frontal	Meningioma	2	6	107
SP	54/F	L frontal	Unspecified autoimmune	2	4	112

AVM = arteriovenous malformation; GDS = Geriatric Depression Scale score; NART = National Adult Reading Test Full Scale IQ; F = female; L = left; M = male; R = right.

important role in spontaneous facial expression mimicry. The only other study to assess the neural substrates of spontaneous facial expression mimicry suggests that the frontal regions may be particularly important for the mimicry of angry, but not happy, facial expressions. Specifically, individuals with traumatic brain injury did not mimic angry facial expressions, and this was attributed to the involvement of ventral frontal damage in this type of injury (McDonald et al., 2011).

However, there have been no lesion studies to date that have specifically investigated the role of the right frontal cortex in facial expression mimicry, which clearly seems an important next step in delineating the neural substrates of this behaviour. Indeed, Dimberg and Petterson (2000) show that mimicry of angry and happy facial expressions produces stronger muscle activity on the left than the right side of the face, suggesting right hemisphere dominance in imitative behaviour. In addition, individuals with injury to the right hemisphere are less able to produce positive and negative facial expressions (Borod, Koff, Lorch, & Nicholas, 1985; Borod, Koff, Lorch, & Nicholas, 1986), and have difficulty recognising basic emotional expressions (Adolphs, Damasio, Tranel, & Damasio, 1996). Further evidence for the potential importance of the right hemisphere in processing facial expressions comes from a study showing improved recognition of emotional valence when expressions are presented to the left rather than the right visual field (Natale, Gur, & Gur, 1983).

More specifically, it has been shown that a patient with focal right frontal trauma demonstrated impairment in recognition of, and autonomic responding to, angry expressions, as well as high levels of aggressive behaviour and difficulty understanding others' anger (Blair & Cipolotti, 2000). Together with evidence that viewing angry facial expressions activates the right orbitofrontal cortex (Blair, Morris, Frith, Perrett, & Dolan, 1999), it has been suggested that the right frontal region represents a specialised system for responding to, and generating expectations of, others' anger (Blair & Cipolotti, 2000). In contrast, activations in non-frontal regions have been associated with smiling to happy expressions (Lee et al., 2006), as well as with viewing happy faces (Phan, Wager, Taylor, & Liberzon, 2002). A further study shows that processing negative expressions results in greater orbitofrontal activity relative to happy expressions (Iidaka et al., 2001). These studies suggest that damage to the right frontal region may disrupt the mimicry of angry, but not happy, facial expressions.

Spontaneous facial mimicry responses are generally detected in experimental settings using facial electromyography (EMG; measurement of changes in the electrical activity of muscles), which is sensitive to even subtle changes in facial muscle activity. Numerous EMG studies have shown that images of angry facial expressions evoke increased *corrugator supercilii* (i.e., brow) activity relative to images of other types of expressions. In addition, happy facial expressions elicit greater *zygomaticus major* (i.e., cheek) activity relative to other types of expressions. Using this technique, the present

study was the first to assess spontaneous facial expression mimicry among individuals with lesions localised to either the right or left frontal cortex. It was predicted that right frontal cortical lesions would disrupt the spontaneous mimicry of angry, but not happy, facial expressions.

## 2. Method

### 2.1. Participants

Five adults with lesions localised to the left frontal cortex (5 female), and four with lesions localised to the right frontal cortex (2 female, 2 male), participated in the current study. Patient information is given in Table 1. All were recruited from the Prince of Wales Hospital, Sydney, based on clinical reports from neurosurgeons at the hospital. Only patients whose medical records indicated no prior neurological or psychiatric disorders and no evidence of aphasia or agnosia, and were deemed by treating clinical staff to have sufficient cognitive capacity to understand and comply with instructions were recruited. The experimenter also independently determined throughout testing that all had sufficient cognitive and motor capacity to understand and comply with instructions. Nine demographically matched controls (7 female) also participated in the current study. Exclusion criteria for all control participants included a self-reported psychological or neurological disorder, alcohol/drug abuse and experimenter-determined inability to communicate adequately. All participants received \$10 Australian (~10 USD) per hour for participation. Patients and controls did not differ in age ( $M = 59.1$  years,  $SD = 9.28$ ;  $M = 59.4$  years,  $SD = 9.65$ , respectively;  $t(16) = .08$ ,  $p = .94$ ,  $d = .03$ ), years of education ( $M = 13.0$  years,  $SD = 3.20$ ;  $M = 13.7$  years,  $SD = 2.60$ , respectively;  $t(16) = .49$ ,  $p = .63$ ,  $d = .24$ ), or premorbid intelligence as measured by Full Scale IQ on the National Adult Reading Test (Nelson & Willison, 1991;  $M = 105.4$ ,  $SD = 9.68$ ;  $M = 110.3$ ,  $SD = 9.80$ , respectively;  $t(15) = 1.05$ ,  $p = .31$ ,  $d = .50$ ). Note that one patient did not complete the NART since English was their second language. In addition, there was a non-significant trend for depression scores to be higher for patients ( $M = 8.0$ ,  $SD = 6.27$ ) than controls ( $M = 4.1$ ,  $SD = 4.14$ ;  $t(16) = 1.55$ ,  $p = .14$ ,  $d = .73$ ), as measured by the 30-item Geriatric Depression Scale (Yesavage et al., 1983). Importantly, however, no participants scored within the severely depressed range on this scale (i.e., 20–30), and only four scored within the mildly depressed range (i.e., 10–19; see Table 1 for individual depression and NART scores). All participants provided informed consent according to the Declaration of Helsinki. Ethics approval for this study was obtained from the South-Eastern Sydney Illawarra Area Health Service – Eastern Section and the University of New South Wales ethics committee.

### 2.2. Passive viewing paradigm

Surface EMG was used to record changes in the level of muscle activity over the left *corrugator supercilii* region, which knits the brow, and the left *zygomaticus major*, which lifts the corners of the lips into a smile. Site preparation and electrode placement followed the standard procedure set out by Tassinari and Cacioppo (2000). The skin over the regions of interest was cleansed with an alcohol wipe and then gently abraded with NuPrep gel. The electrodes were gold-plated, hat-shaped discs with 9 mm housings that were placed in pairs, with an inter-electrode distance of approximately 1.25 cm, and with an additional electrode approximately in the centre of the forehead acting as an earth. The electrodes were attached with Ten20 conductive paste. Muscle activity was continuously recorded with a PowerLab 4/30 Data Acquisition System (AD Instruments, Castle Hill, Australia) at a sampling rate of 2000 Hz with a 10–500 Hz bandpass filter, a 50 Hz notch filter, and an amplification factor of 20,000. The PowerLab was triggered by DMDX (Version 3.2.3.0; Forster & Forster, 2003), which synchronised timing of the facial expression presentations with the recording of the data acquisition system.

Participants were asked to sit in a comfortable chair and watch a series of stimuli on a monitor. To disguise the true purpose of the study participants were also told that the EMG sensors were measuring changes in the sweat gland activity of their

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