



‘Just can’t hide it’: a behavioral and lesion study on emotional response modulation after right prefrontal damage

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Abstract

Introduction: Historically, emotion regulation problems have been reported as a common consequence of right prefrontal cortex (rPFC) damage. It has been proposed that the rPFC, particularly the rIFG, has a key role inhibiting prepotent reflexive actions, thus contributing to emotion regulation and self-regulation. This study is the first to directly explore this hypothesis, by testing whether damage to the rIFG compromises the voluntary modulation of emotional responses, and whether performance on inhibition tasks is associated with emotion regulation. **Method:** 10 individuals with unilateral right prefrontal damage and 15 matched healthy controls were compared on a well-known response modulation task. During the task participants had to amplify and suppress their facial emotional expressions, while watching film clips eliciting amusement. Measures of executive control, emotion regulation strategies usage and symptomatology were also collected. **Results:** As a group, individuals with rPFC damage presented a significantly reduced range of response modulation compared with controls. In addition, performance in the suppression task was associated with measures of cognitive inhibition and suppression usage. Interestingly, these effects were driven primarily by a subgroup of individuals with rPFC damage, all of whom also had damage to the right posterior insula, and who presented a marked impairment in suppressing facial emotional expressions

Key words: response modulation; suppression; emotion; emotion regulation; prefrontal cortex; insula; brain injury

Introduction

There is a growing literature documenting emotion regulation (ER) problems after brain damage, and their impact on emotional adjustment and social functioning (Tate, 1999; Bechara, 2004; Lewis et al., 2006; Beer and Lombardo, 2007; Obonsawin et al., 2007; Abreu et al., 2009; McDonald et al., 2010; Salas, 2012;

Salas and Castro, 2014; Salas et al., 2013, 2014a,b). Current experimental evidence appears to suggest that damage to the right hemisphere, and particularly right prefrontal cortex (rPFC), impairs patients’ capacity to modulate emotion. It has been noted, for example, that individuals with rPFC damage present more frequent emotional problems, which may result in

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inappropriate sympathetic responses (Hilz et al., 2006), emotion-based decision-making impairment (Manes et al., 2002; Tranel et al., 2002), delusional pathology (Feinberg et al., 2010; Feinberg, 2011) and neuropsychiatric disorders like mania and alexithymia (Cummings, 1997, for a review). The mechanisms by which ER becomes impaired after rPFC lesions are yet unknown. However, it has been proposed that this cortical area has a special role integrating cognition and affect (Stuss and Alexander, 1999, 2000a,b).

There is a large body of literature suggesting that response inhibition, namely the process of withholding unwanted prepotent reflexive actions (Aron et al., 2004a,b; Dillon and Pizzagalli, 2007) is a key element of ER and self-regulation (e.g. Barkley, 1997, 2001; Thayer and Lane, 2000; Quirk, 2007; Hofmann et al., 2012). Interestingly, a number of studies have linked response inhibition to the rPFC. For example, neuroimaging findings (Rubia et al., 2003; Aron et al., 2004b; Aron, 2007; Dillon and Pizzagalli, 2007; Chambers et al., 2009; Forstmann et al., 2008; Levy and Wagner, 2011) and transcranial magnetic stimulation studies (Chambers et al., 2006, 2007) have suggested that response inhibition is mediated by a right hemisphere lateralized network, centered on the Inferior Frontal Gyrus (rIFG) (for a review see Aron et al., 2014). Neuropsychological studies have found evidence of impaired response inhibition in rPFC lesioned patients, thus confirming the neuroimaging findings. Specifically, damage to the rIFG is associated with increased latencies of inhibitory stop-signal in go/no-go tasks (Aron et al., 2003) and perseverative deficits in switching tasks (Aron et al., 2004a), suggestive a generalized deficit in negotiating rapidly changing behavioral demands.

An important, issue, not addressed by the literature so far, is the role of rPFC in response inhibition when performing emotional, rather than speeded motor tasks. To our knowledge, only one neuroimaging study has used an emotional inhibition paradigm, or interference paradigm, to examine the neural correlates of inhibiting facial emotional expressions in healthy controls (HCs) (Lee et al., 2008). The authors reported that during the inhibition of emotional expressions, activation of right insula, rSTS and rIFG was observed. Additionally, the activation of IFG was also positively correlated with the self-reported frequency of use of suppression. To the best of our knowledge, no neuropsychological study has explored yet whether damage to rPFC compromises the inhibition of emotional responses, despite the well-known association between rPFC damage and emotion regulation problems.

The Process Model of ER (Gross and Thompson, 2007) lists a set of strategies that people use in order to regulate emotional experience. It has been noted that suppression and amplification, both belonging to the family of 'response modulation' strategies, regulate affect by influencing emotion-response tendencies (Gross, 1998; Gross and Thompson, 2007). When suppressing, for example hiding a smile when it is not polite to show that you find something amusing, people voluntarily decreases emotion-expressive behavior while emotionally aroused (Gross and Levenson, 1997; Gross, 2014). In contrast, with amplification, individuals voluntarily augment an already initiated emotion, this, in order to cope with the requirements of the context—smiling in response to a bad joke made by your boss (Demaree et al., 2004; Henry et al., 2009).

Suppression is a complex process, since it requires not only control of facial expressions (Beer and Lombardo, 2007), but also inhibition of other behavioral displays triggered by the felt emotion (Gross, 1999; Gross and John, 2003; Gross and Thompson, 2007; Kühn et al., 2011). In experimental settings, suppression is

commonly assessed by asking participants to inhibit the expression of emotions triggered by external stimuli (Gross and Levenson, 1997). Although neuroimaging studies have suggested that rPFC is activated during suppression (e.g. Goldin et al., 2008; Lévesque et al., 2003), no study has examined whether suppression is compromised in patients with lesions to rPFC.

In this study, we examined the anatomical correlates of response modulation, and in particular, the relationship between response inhibition and suppression. We asked individuals with rPFC lesions, and normotypical controls, to suppress the facial expression of their emotional state, particularly in relation to one intense positive emotion: amusement. In this task, participants were asked to control their facial emotional expressions, while shown standardized film clips that elicited amusement. Prior to each viewing, they were given three different instructions: (i) watch spontaneously, (ii) suppress (hide) and (iii) amplify. Self-report measures were used to assess the effectiveness of the emotional induction procedure, and facial behavior analysis ('FACS', Ekman et al., 2002) was used to measure facial markers of positive emotions.

It was hypothesized that individuals with rPFC lesions would: (i) experience similar levels of induced positive affect as controls ('Emotion reactivity Hypothesis'); (ii) present a decreased range of response modulation compared with controls ('Response modulation range Hypothesis') and (iii) show higher levels of facial expressions when instructed to suppress ('Suppression Hypothesis'). It was also hypothesized that (iv) performance in the suppression condition [hide] would be correlated with measures of inhibition ('Cognitive Control Hypothesis'); and finally that (v) impairment in the suppression condition would be associated with damage to areas previously described in the literature using response inhibition tasks ['Lesion Hypothesis'].

Materials and methods

Participants

Ethical approval was granted by Bangor School of Psychology Ethics Committee and the Betsi Cadwaladr University Health Board. HCs were matched with ten participants with acquired brain injury, referred by neurologists from the Bangor University School of Psychology. A unilateral focal brain lesion, due to stroke, that involved the rPFC was the main inclusion criterion for the neurological group. The lesion could involve exclusively the rPFC or extend to the posterior areas of the RH [rPFC + right posterior]. Several exclusion criteria were considered, such as time since injury [no <6 months] and language ability [no moderate or severe language impairment]. Clinical details and aetiology of the neurological sample are presented in Table 1, and the lesion reconstruction of the rPFC group can be observed in Figure 1.

The overall sample involved a total of 25 participants, including individuals with rPFC lesions (rPFC, $n = 10$, female = 6, male = 4) and HCs ($n = 15$, female = 9, male = 6). Both groups were matched in age [rPFC, $M = 61.78$, $SD = 12.65$; HC, $M = 62.80$, $SD = 4.13$; $t_{(9,03)} = 0.24$, $P = 0.82$] and years of education [rPFC, $M = 14$, $SD = 1.51$; HC, $M = 13.93$, $SD = 1.75$; $t_{(21)} = -0.09$, $P = 0.93$]. The average number of months since the injury in the neurological group was 61.80 ($SD = 40.37$).

Procedure

Eligible participants were seen twice. Assessment across two sessions was useful to avoid the impact of fatigue on the neurological group. During the first session the goal of the experiment

Table 1. Clinical details and aetiology in the neurological group

Age/Sex	Aetiology	Months since onset	Location
57F	MCA stroke	84	Right prefrontal, insula
50M	MCA and ACA stroke	20	Right prefrontal
73F	MCA stroke	97	Right prefrontal, frontal eye field
45M	ACoA SAH	70	Right prefrontal
74M	MCA Stroke	20	Right ventro-lateral prefrontal cortex, basal ganglia
65M	MCA Stroke	65	Right PFC, right temporoparietal junction
46F	MCA Stroke	114	Right prefrontal, insula
66F	MCA Stroke	120	Right PFC, insula, middle and superior temporal gyrus, TPJ
78F	MCA stroke	13	Right prefrontal insula
68F	MCA stroke	24	Right prefrontal and parietal

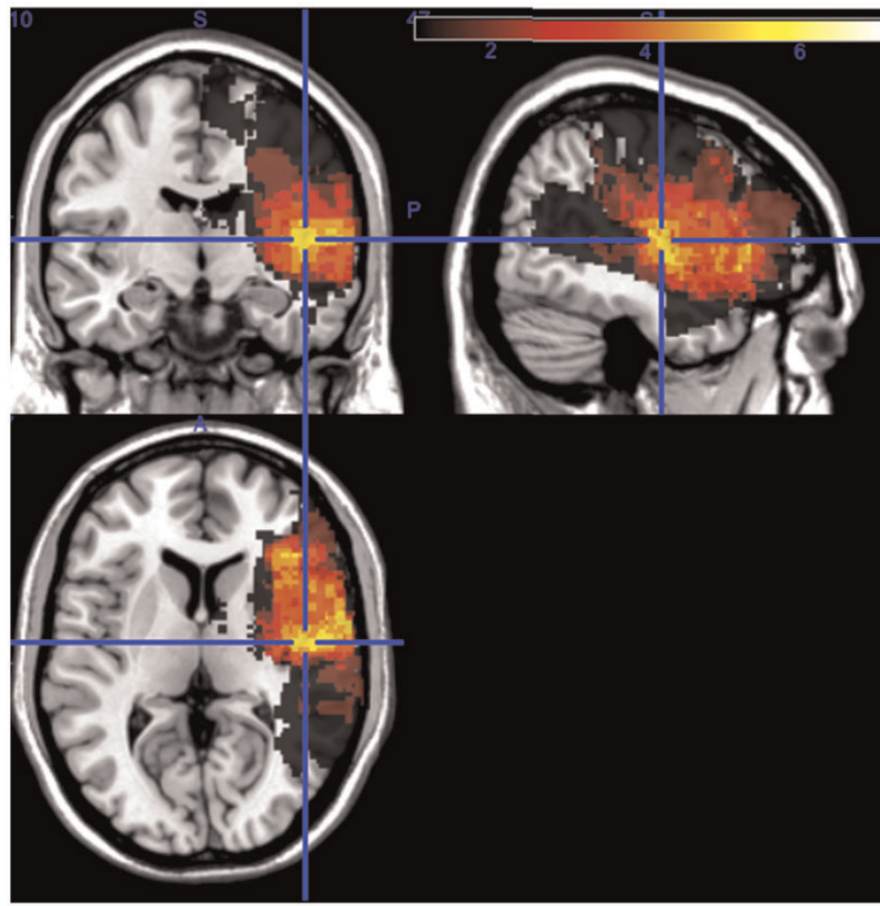


Fig. 1. Neurological group lesion overlapping. The heatmap represents the number of patients who exhibited a lesion at the respective location. At the group level, rPFC patients had overlapping lesion at right insula and rIFG.

was explained to participants and consent was obtained. Participants had been screened by a board certified behavioral neurologist for evidence of dementia using a informant base interview. Measures of overall cognition were also collected. In the second session participants completed a response modulation task, and measures of executive function were obtained. At the end of this session participants were debriefed.

Instruments

Overall cognitive assessment. The ‘Mini Mental State Examination’ (Rovner and Folstein, 1987), ‘Telephone search

and telephone search dual task’ (TEA, Robertson et al., 1994), ‘Token Test’ (De Renzi and Faglioni, 1978), ‘Logic Memory’ (Wechsler, 1987), ‘Rey-Osterrieth Figure’ (Stern et al., 1994) and the ‘Frontal Assessment Battery’ (Dubois et al., 2000) were used in order to obtain an overall profile of cognitive function.

Executive functions assessment. A set of neuropsychological tasks was used to obtain a profile of executive function. Digits Forward and Backward (Wechsler, 1981), Verbal Fluency (Delis et al., 2001) and Similarities (Wechsler, 1981). Measures of inhibition included conflicting instructions (Stuss and Benson, 1986)

inhibitory control (Drewe, 1975) and environmental autonomy (Lhermitte et al., 1986).

Emotional symptomatology and suppression usage. In order to assess the presence of symptomatology, the Hospital Anxiety and Depression scale (HADS, Zigmond and Snaith, 1983), a self-report questionnaire, was employed. The HADS has been shown to be a sensitive tool in assessing depression and anxiety symptoms in acquired brain injury population (Dawkins et al., 2006). To calculate the use of suppression in daily life, the Emotion Regulation Questionnaire (Gross and John, 2003) was applied.

Emotional experience self-report. Emotional experience during the response modulation task (see below) was assessed using a self-report questionnaire adapted from the PANAS-X (Watson and Clark, 1994). Following a previous study (Salas et al., 2011), 12 emotional words were selected from the discrete emotion scales of the PANAS-X. A 5-point likert scale was used to rate each word. Four emotional words were selected for the target emotion, and six words related to negative affect were also considered.

Facial imitation task. In order to determine participants' motoric control of facial expressions, a facial imitation task was adapted from Simons et al. (2003). Participants were asked to imitate facial movements of the researcher, which correspond to Action Units (AUs) described in the Facial Action Coding System (FACS) (Ekman et al., 2002).

Response modulation task. This task is an adaptation from a previous study on response modulation in dementia (Henry et al., 2009), in which subjects are asked to watch short video clips and manipulate their facial expression according to three different instructions: watch spontaneously, suppress or amplify (Figure 2). The spontaneous condition was always performed first. Then, inhibition and amplification conditions were alternated for each participant in order to avoid order effects. Based on the available evidence on emotion elicitation using film clips (Gross and Levenson, 1995; Hewig et al., 2005; Rottenberg et al., 2007), three validated clips were selected to induce amusement ['When Harry met Sally' (155s), 'Robin Williams Live' (205s), 'Whose line is it, Anyway?' (211s)]. On the same premises, three short clips of 60 seconds each were generated from 'Alaska's Wild Denali' (Rottenberg et al., 2007) and used as neutral stimuli.

Participants sat in front of a 16" laptop in a quiet room. Headphones were used to avoid possible distractions. A video-recording camera was placed two meters in front of the subjects. At the beginning of the task participants were told that they would watch a series of clips from movies and they would have to follow some instructions during each clip. Then participants watched the amusement-inducing clips. Neutral clips were viewed before each emotional clip. A self-report questionnaire of experienced emotion, adapted from the PANAS-X (Watson and Clark, 1994), was administered immediately after viewing each emotional clip (see Figure 1).

The instructions for each manipulation were also adapted from Henry and Cols (2009). Before each neutral clip the following instruction was offered: 'now try to relax and clean your mind of any thoughts while you watch the following clip'. In the spontaneous condition participants received the following instruction: 'please watch the following clip spontaneously, as if you were watching TV at home'. In the suppress condition participants were told:

'this time I will ask you to hide any feelings that you might have while watching the following clip. As if later someone looking at the recording of this moment would not have a clue regarding how you were feeling. In other words, use a poker face'.

Finally, in the amplification condition participants were asked:

'this time I will ask you to amplify, to express as much as you can any feelings that you might have while watching the following clip. As if later someone looking at the recording would clearly understand how you were feeling'.

In each condition participants were asked to repeat the main instruction before beginning the task (watch spontaneous, hide or amplify), this, in order to show that they understood what they were expected to do. To control for potential memory confounds, participants were also asked to repeat the main instruction for each condition after watching the clip. All controls were able to accurately state the main instruction for each condition. As for individuals with rPFC damage, they were equally accurate describing the instruction in both 'spontaneous' and 'hide' conditions. Nevertheless, two participants reported that during the 'amplify' condition the instruction was to 'watch the video'. However, when asked to describe what they were expected to do while watching the video, both correctly stated 'to show as much emotion as I can'.

Data analysis

Data reduction

In order to measure facial expressive behavior, the 'FACS' (Ekman et al., 2002) was used to code the appearance and frequency of facial changes while watching the emotional clips. Based on previous research (Rottenberg et al., 2002; Soussignan, 2002; Ekman, 2003; Coan and Gottman, 2007; Ambadar et al., 2009) AUs 12 + 6 was selected as a proxy for 'intense positive affect' (see Figure 2). Several studies have associated AU 12 + 6, also called Duchenne smile, to the felt experience of positive affect and enjoyment and different patterns of autonomic arousal compared with smiles that only involved AU 12 (Ekman and Friesen, 1982; Ekman et al., 1990; Frank et al., 1993; Soussignan, 2002) (Figure 3).

The coding process involved two FACS certified coders who were blind to the participant's group, and to the video's condition (spontaneous, hide, amplify). As a first step, all AUs (facial events) that appeared in each video were coded, following Ekman et al. (2002) guidelines. A new facial event was coded when: (i) an AU appeared from a neutral background; (ii) there was a qualitative increase in intensity (two or more points) in an existing AU; (iii) a new AU was added to an already existing configuration. As a second step, facial events that included the 'Duchenne Smile' (AU 12 + 6), and those that included AU 12 + 6 and 'smile controls', were considered for further analysis. This decision was based on available evidence suggesting that, when smiling, people tend to show a high frequency of lower face actions units (e.g. AU 8 Lips Together; AU 17 Chin raise; AU 26 Jaw Drop), whose muscular action potentially counteract the upward pull of the zygomatic major smile (AU 12), or obscure the smile (Keltner, 1995). Because these smile controls are not related to other emotional configurations, but simply are considered as attempts to microregulate facial displays, Duchenne Smiles that were accompanied by controls were also included in the analysis. There was a high agreement between coders in detecting the occurrence of AU 12 + 6 with and without smile controls ($r_s = 0.80$).



Fig. 2. Response modulation task. Three amusement inducing clips were presented to participants under three different conditions (spontaneous, hide/amplify, amplify/hide). Before each emotional clip a neutral clip was shown, and after each emotional clip a questionnaire to capture emotional experience during the clip was administered.

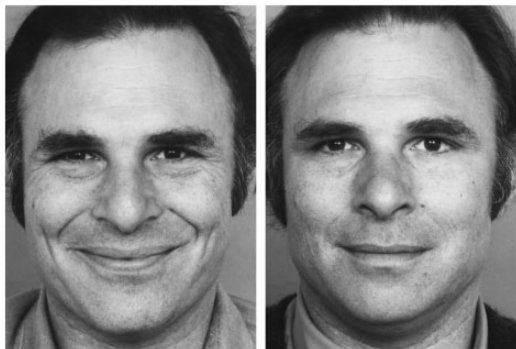


Fig. 3. The Duchenne smile. The Duchenne smile (left) involves activation of the zygomatic major (AU 12, oblique rising of the mouth corners) and the presence of orbicularis oculi activation (AU 6, crow's wrinkles in the eye region). AU 12 alone [right], on the contrary, does not include AU 6 (From Ekman et al., 2002).

Lesion Mapping

In order to examine the lesion anatomy in different subgroups of rPFC participants, we segmented and overlaid brain images obtained on a Phillips 3T Achieva scanner, housed in the School of Psychology at Bangor University. The 3D, T1-weighted images had a spatial resolution equal to or smaller than $1 \times 1 \times 1 \text{ mm}^3$ ($TR = 8.4\text{--}13 \text{ ms}$, $TE = 3\text{--}4.5 \text{ ms}$) and whole brain coverage. Lesions were manually traced using the MRIcron software (Rorden et al., 2000; <http://www.nitrc.org/projects/mricron>). Individual's T1 weighted images were then normalized to the ICBM brain atlas using the unified segmentation routine (SPM12, Wellcome Department of Imaging Neuroscience, University College London). Group and subgroup lesion overlap maps were generated by computing the conjunction of individual lesion maps. To compare within the rPFC participants with and without behavioral deficits in emotional processing, we used the Non-parametric Mapping module of MRIcron to perform voxelwise lesion mapping and contrast the lesion overlap between the impaired and non-impaired groups.

Research hypotheses data analysis

Hypothesis 1 (emotion reactivity). In order to test Hypothesis 1, namely that individuals with rPFC lesions would experience similar level of positive affect as controls, the average scores of

positive affect (PANAS) in each conditions were compared between groups. Because normality and independence of variance assumptions were not met, a non-parametric test for differences between two groups was used (Mann-Whitney U).

Hypothesis 2 (Response modulation range). As noted in the introduction, response modulation refers to both the capacity to inhibit and amplify emotional displays. In order to assess this regulatory capacity, a modulation range was calculated for each participant on the assumption that good regulators would be able to inhibit facial expressions when instructed to suppress (low frequency of AU 12+6), and increase them when instructed to amplify (high frequency of AU 12+6). In other words, 'good regulators' would display a larger difference in the number of AUs 12+6 displayed between the suppression and amplification conditions than 'poor regulators'. Following this logic, a 12+6 'range score' was calculated by subtracting the number of AU 12+6 in the suppression condition to the number of AU 12+6 in the amplifying condition. To assess this variable was normally distributed, we examined profit plots and Kolmogorov-Smirnov tests were performed on the entire sample and on each group separately. These analyses suggested that the distributions did not depart significantly from a normal distribution (Sample: $D(25) = 1.10$, $P = 0.18$; rPFC: $D(10) = 0.70$, $P = 0.71$; HC: $D(15) = 0.80$, $P = 0.54$). The 12+6 range score was compared among groups using one-tailed, independent sample t-tests. When violations of homogeneity of variance assumptions were found, adjusted values were computed.

Hypothesis 3 (suppression hypothesis). Because AU 12+6 reflects the experience and expression of intense positive emotion, no AU 12+6 was expected to be generated in the suppress condition. As a first step in the analysis of this variable, group differences between controls and individuals with rPFC were explored using a non-parametric test (Mann-Whitney U). As a second step, and due to the fact that group analyses can 'wash-out' differences in performance inside each group (Shallice, 1988), the frequencies of appearance of AU 12+6 in the suppress condition were described, both in neurological and HC groups.

Hypothesis 4 (cognitive inhibition). A non-parametric correlation test (Spearman, r_s) was used to assess the associations between

Table 2. Cognitive and Emotional performance of neurological groups and HCs

	Task	RH Group		HC Group	
		M	SD	M	SD
Overall cognition	Mini mental state examination	28.11	1.45	29.00	1.25
	Sustained attention (TEA)	7.88	1.64	11.00	1.57
	Divided attention (TEA)	10.00	4.89	11.00	1.79
	Comprehensive language (token test)	30.40	1.43	31.13	1.36
	Memory (WMS-R) coding	12.50	3.20	13.67	3.43
	Memory (WMS-R) free recall	14.30	3.65	16.33	3.77
	Memory (WMS-R) recognition	13.22	1.39	13.86	1.52
	Executive functions (FAB Total)	14.90	1.91	16.29	2.23
Cognitive Control	Working memory (Digits, WAIS)	8.90	2.61	10.77	2.22
	Inhibition, sensitivity to interference	2.60	0.52	2.92	0.27
	Inhibition, inhibitory control	2.40	0.96	2.38	1.04
	Inhibition, environmental autonomy	2.90	0.31	3.00	0.00
	Verbal fluency (DKEF-S) letter	7.82	2.23	10.71	3.27
	Verbal fluency (DKEF-S) category	7.75	1.91	11.07	2.13
	Abstraction (similarities, WAIS)	9.63	3.29	11.86	3.13
Emotional functioning	Emotional symptoms (HADS) anxiety	6.70	4.73	4.70	3.10
	Emotional symptoms (HADS) depression	6.40	4.62	2.67	1.79
	Emotion regulation (ERQ) suppression	17.33	5.90	17.07	5.20

neuropsychological measures of inhibition and performance in the suppress condition (number of AU 12 + 6).

Hypothesis 5 (lesion hypothesis). In order to examine the relationship between lesion overlap pattern and behavioral deficit, individual patients' lesion was reconstructed on a standard template. We also examined the lesion overlap pattern in a subgroup of rPFC patients who showed marked impairment in the suppression task when compared with the HC.

Results

Cognitive and emotional assessment

The assessment of emotional symptoms indicated that participants with both rPFC lesions and HCs scored below the cut-off point for anxiety and depression subscales of the HADS. Nevertheless, the rPFC presented significantly higher scores on the depression subscale [$F(2,22) = 2.84, P = 0.017$] than controls, but not on the anxiety subscale [$F(2,22) = 1.25, P = 0.11$]. In addition, the neurological group did not differ from the HC group in the self-reported use of suppression, when measured by the Emotion Regulation Questionnaire [$F(2, 22) = 0.11, P = 0.45$]. A detailed description of the average scores of each group on the cognitive and emotional assessment is reported in Table 2.

Generation of facial expressions

Participant's ability to generate facial expressions was explored using data from the motor task (imitation) and considering the productivity of facial displays during the spontaneous condition. In relation to the motor task, and specifically regarding the capacity to generate AU 12 + 6, all participants obtained a score of 1 (77%) or 2 (23%) on a scale of 1–6—where 1 reflects a perfect imitation of long duration and 6 reflects no movement at all (Simmons et al., 2003). When patients and controls were compared, no significant differences in the distribution of these scores were found ($\chi^2 = 0.75, P = 0.39$). In relation to the level of facial productivity during the spontaneous condition, it was

observed that all participants were able to generate facial displays ($M = 46.12$ total number of AUs). These included Duchenne Smiles, or AU 12 + 6 ($M = 6.16; SD = 7.84$), Other Smiles, or AU 12 with no involvement of AU 6 ($M = 13.44; SD = 11.47$), and smile controls ($M = 15.24; SD = 20.56$). When comparing both groups in the spontaneous condition, controls presented a higher frequency of Duchenne Smiles than patients (HC: $M = 7.87; SD = 8.90$; rPFC: $M = 3.60; SD = 5.32$), however, such difference was not found statistically significant [$t(23) = 1.36, P = 0.19$]. Taken together, these data suggest that any difficulty found in manipulating facial behavior during the response modulation task cannot be accounted by a motoric impairment in generating facial expressions.

Emotional reactivity

The three clips were effective eliciting similar levels of positive affect in patients and controls (Figure 4). Both groups did not differ in the levels of self-reported positive affect across all three conditions (rPFC: $X^2(2) = 3.27, P = 0.22$; HC: $X^2(2) = 5.35, P = 0.07$), suggesting that similar levels of positive emotion were experienced when watching the clip spontaneously (HC: $M = 3.28$; rPFC: $M = 2.36$), suppressing (HC: $M = 2.53$; rPFC: $M = 2.14$) or amplifying (HC: $M = 3.28$; rPFC: $M = 2.60$). In relation to differences between groups, individuals with rPFC lesions did not differ significantly from controls in the self-reported experience of positive affect during the three conditions (**Suppress**: $U = 63, Z = -0.68, P = 0.52$; 'Mean Ranking': rPFC = 11.8, HC = 13.8; **Spontaneous**: $U = 45, Z = -1.67, P = 0.10$; 'Mean Ranking': rPFC = 10, HC = 15; **Amplify**: $U = 51, Z = -1.31, P = 0.21$; 'Mean Ranking': rPFC = 14.57, HC = 10.65). Overall, these data suggests that individuals with rPFC lesions experienced levels of positive emotions comparable to those of controls, and therefore response modulation differences between the groups cannot be accounted by abnormal emotional reactivity.

Response modulation range

Participants with rPFC damage were less able to modulate (suppress and amplify) their facial expressions during the

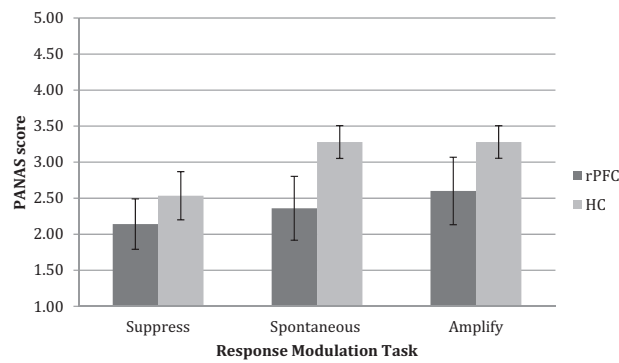


Fig. 4. Levels of positive affect during response modulation task. Averages of self-reported positive affect, and mean ranks across the three conditions are presented in this table. Even though the rPFC group consistently presents lower levels of emotional experience, these are not significantly different from the control group.

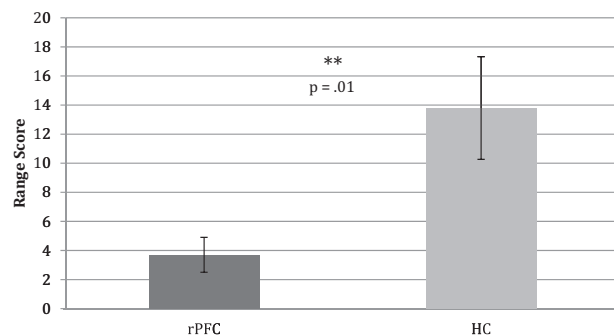


Fig. 5. Response modulation range. Individuals with right PFC lesions (rPFC) are less able than HCs in modulating –suppressing and amplifying– their emotional responses. The range score (12 + 6 suppress – 12 + 6 Amplify) was significantly smaller in subjects with rPFC.

viewing of emotional clips (Figure 5). The comparison between the two groups showed that controls exhibited a greater modulation range ($M = 13.8$, $SE = 3.53$) than rPFC patients ($M = 3.7$, $SE = 1.2$). This difference was statistically significant [$t(21.03) = 2.49$, $P = 0.01$, $r = 0.48$], suggesting that individuals with rPFC damage were less able to voluntarily modulate their facial expressions when instructed.

Suppression of positive affect

Before reporting the results on the suppression of intense positive affect (AU 12 + 6) we would like to comment on the suppression of facial activity in general during this condition. Even though some participants were highly effective completely inhibiting the expression of Duchenne Smiles as instructed, the presence of other AUs, such as Other Smiles, or AU 12 with no involvement of AU 6 (HC: $M = 3.7$, $SD = 6.4$; rPFC: $M = 3.6$, $SD = 4.6$), and smile controls (HC: $M = 8.2$, $SD = 10.5$; rPFC: $M = 4.9$, $SD = 4.8$), was commonly observed. In fact, when all coded AUs were considered, HCs and patients presented an average of 17.2 and 13.6 AUs during the suppression condition respectively (see Figure 6). No significant differences were found between groups in relation to the frequency of appearance of Other Smiles ($U = 83.5$, $Z = 0.49$, $P = 0.62$; ‘Mean Ranking’:

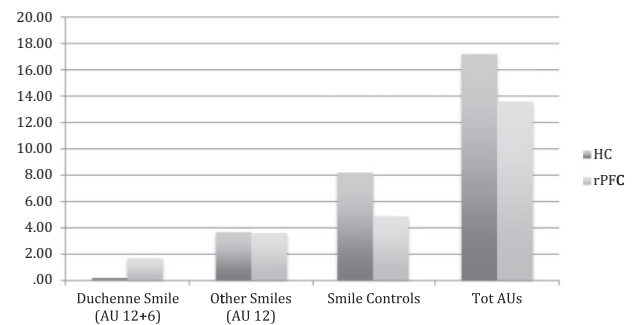


Fig. 6. Facial activity during the suppression condition. Despite the instruction to ‘Hide’, both groups of participants still exhibit levels of facial activity during the suppression condition, mostly, smiles without the involvement of AU 6 and smile controls. Non parametric analyses show there are no differences between groups in any of these variables.

Table 3. Frequency of AU 12 + 6 in the suppress condition

N° AU 12 + 6 Suppress	HC		rPFC	
	Frequency	Percent	Frequency	Percent
0	13	86.7	6	60
1	1	6.7	0	0
2	1	6.7	1	10
3	0	0	0	0
4	0	0	1	10
5	0	0	1	10
6	0	0	1	10

The frequency of AU 12 + 6 on the suppression condition is compared between HCs and individuals with rPFC lesions.

rPFC = 13.85, HC = 12.43), smile controls ($U = 68.5$, $Z = -0.38$, $P = 0.71$; ‘Mean Ranking’: rPFC = 12.35, HC = 13.43), or total AUs ($U = 73$, $Z = -0.11$, $P = 0.91$; ‘Mean Ranking’: rPFC = 12.8, HC = 13.13). Taken together, these data suggest that when instructed to ‘hide’ emotional facial expressions, participants still present facial displays, probably related to the ‘leakage’ of the felt emotion (AU 12) and to its attempted regulation (smile controls).

In relation to the suppression of intense positive affect, or Duchenne Smiles, group comparisons did not show any significant differences between HCs and individuals with rPFC damage ($U = 98.5$, $Z = -1.74$, $P = 0.08$; ‘Mean Ranking’: rPFC = 15.35, HC = 11.43). However, a more detailed analysis of individual performance during the suppression task offered valuable information to explore this research question further (see Table 3). In general, HCs were able to suppress the expression of intense positive affect. Individuals with rPFC lesions, on the contrary, showed a mixed performance, with some successfully inhibiting, while others presented with an abnormally high frequency of AU 12 + 6. Only 2 out of 15 [13.3%] HCs were unable to totally inhibit the expression of AU 12 + 6 in the suppress condition. These participants generated 1 and 2 AU 12 + 6, respectively. Four out of ten individuals with rPFC were unable to successfully inhibit the expression of AU 12 + 6. Three out of the four participants that failed inhibiting AU 12 + 6 had a higher score than the poorest control [2 AU 12 + 6]. Hence not all subjects with rPFC present with suppression impairment. However, an important proportion of the group does exhibit abnormal performance.

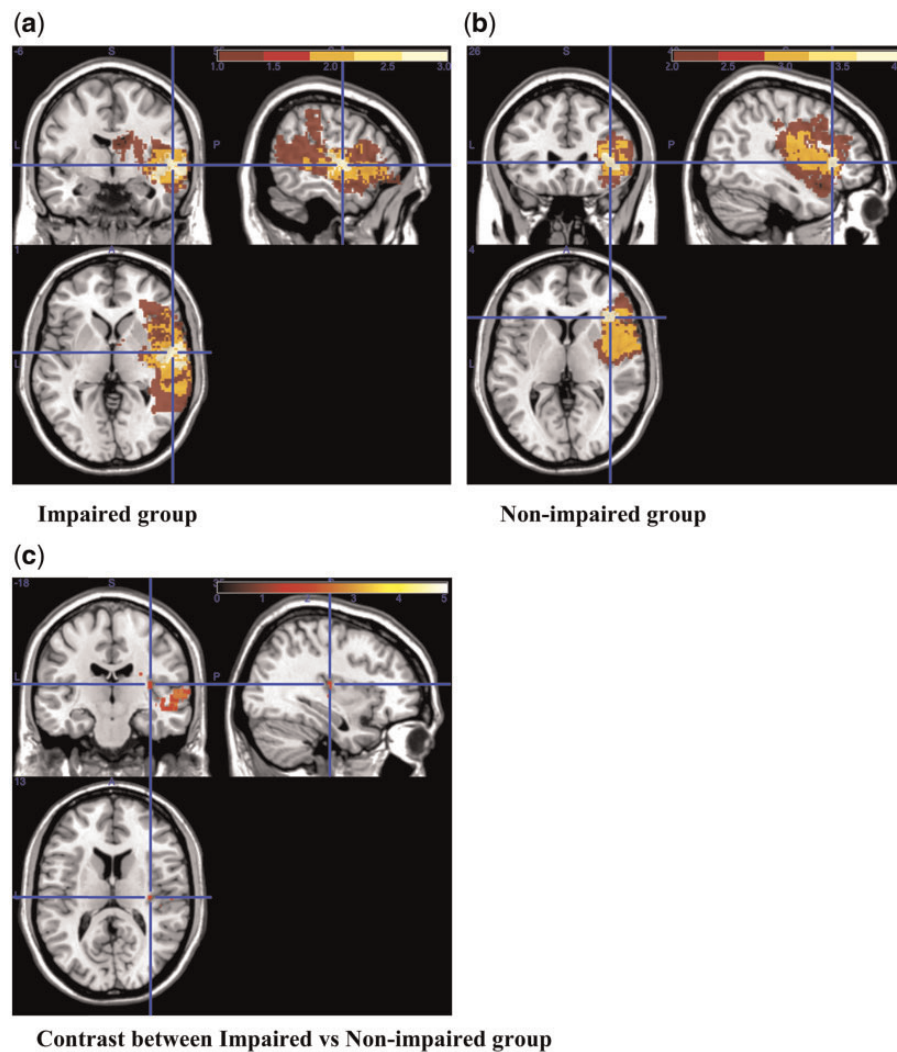


Fig. 7. Patterns of lesion overlap for rPFC patients who presented abnormal performance on the suppression task (a) and those that performed within the normal range (b). The lesion overlap hotspot for the impaired group lies in the right posterior insula, while for the non-impaired group the site of maximum lesion overlap lies in the border between right anterior Insula and right IFG. Contrasting the lesion overlap pattern between the two groups revealed that the neuroanatomical correlates of the behavioral deficit lie in an area of posterior insula extending to superior temporal gyrus.

Suppression and cognitive inhibition

The ability to suppress the expression of positive affect was associated with only one measure of inhibition: inhibitory control. Using non-parametric correlations (Spearman, r_s), an inverse relationship, of medium size, was found between inhibitory control impairment and suppression ability ($r_s = -0.36$, $P = 0.01$). No associations were found between suppression and sensitivity to interference or environmental autonomy. These data suggest that the capacity to withhold a prepotent response is correlated with the ability to suppress emotional facial expressions.

In addition, suppression usage, measured by the subscale of the Emotion Regulation Questionnaire, also presented a positive correlation of medium size with inhibitory control, as measured by cognitive tasks ($r_s = 0.43$, $P = 0.02$). This piece of data is interesting in that it suggests that both behavior (suppression task) and self-reported frequency of use (suppression subscale ERQ) are related to performance on response inhibition tasks.

Neuroanatomical findings on response modulation and suppression

The neuroanatomical analysis suggests that overall our rPFC group had the highest degree of lesion overlap in right insula and rIFG (see Figure 1). Indeed examining neuroanatomical information from the rPFC group revealed that all patients showed various degrees of insula lesion. Interestingly, the subgroup analysis, which separated patients who showed marked impairment in the suppression task from those who performed within the normal range, indicated that damage to the right posterior insula (MNI coordinates: 47, -12, 5) was associated with impairment in the suppression of emotional responses (Figure 7). On the other hand, rPFC patients who showed normal performance in the task had a lesion centered on the anterior Insula (MNI coordinates: 40, 26, 4). Contrasting the pattern of lesion overlap between the two subgroups using the non-parametric Lieberman measure (Rorden *et al.*, 2007) revealed that lesion in the posterior insula extending to the superior temporal gyrus is correlated with the behavioral deficit observed ($z = 1.754$, $P = 0.05$ FDR corrected). We further checked

whether such difference in behavioral deficit could be explained by a general difference in lesion volume. Correlational analyses found no significant associations between lesion volume and performance in the suppression task ($r = -0.3872$, $P = 0.27$), nor the response modulation task ($r = -0.2265$, $P > 0.53$). Thus, we conclude that only a limited area, that is posterior insula, is specifically associated with impaired suppression.

Results summary

This study appears to partially support the idea that the damage to rPFC compromises the ability to modulate the expression of emotional responses. As a group, individuals with rPFC lesions are less able than controls in the voluntary manipulation (amplifying and suppressing) of their emotional facial expressions, thus presenting a more restricted range of modulation. When looking at suppression ability alone, the performance of patients with rPFC damage is strikingly mixed; some participants appear to fall within the normal range, while others present a markedly abnormal performance. Anatomically speaking, results from the lesion analysis suggest that damage to the right posterior insula is associated with markedly abnormal performance on the suppression task. However, due to the small size of subgroups with anterior and posterior insula damage, this result needs to be considered cautiously as preliminary evidence. Data from the PANAS, motor imitation task, and spontaneous condition, suggest that this impairment cannot be explained by an inability to trigger, experience or display positive emotions. Finally, these results also offer supporting evidence for the relationship between suppression and inhibitory control, since behavior in the suppression task, and self-reported suppression frequency of use, are both associated to cognitive measures of inhibition.

Discussion

As reviewed in the introduction, there is a substantial body of literature suggesting that emotion dysregulation is frequent after rPFC lesions. However, one of the main limitations of this line of research has been the lack of an adequate theoretical framework for ER. The present study is the first to explore ER impairment after rPFC lesion, using a well-known model of ER (The Process Model, Gross and Thompson, 2007) and a widely used experimental suppression task. Furthermore, this study is unique, in that it tests whether response modulation, or the capacity to voluntarily manipulate the expression of emotional experience, is compromised after rPFC damage.

The most important finding of this study refers to the relationship between suppression and rPFC damage. The data indicate that suppression impairment after rPFC lesions is not a homogenous deficit. Some participants ($n = 6$) performed similarly to controls, while others ($n = 4$) showed a remarkably abnormal performance. As for the neuroanatomical findings, the data suggest a remarkably specific potential role of the right insula in suppression. As can be seen in Figure 7 all four of the patients who were severely impaired in the suppression task had right posterior insula lesions, whereas this was not true for any of the unimpaired subjects of the patient group. Importantly, differences in performance on the suppression task were not associated to total lesion volume, thus supporting the specificity of our finding.

Why is the insula important for suppression? A substantial literature now agrees (for review, see Craig, 2008) that the insula has a fundamental role in interoception, or the perception of

the internal state of the body. It has been proposed by authors from the ER field that interoception has a role in suppression since, in order to manipulate emotional expressive behavior, interoceptive awareness is required to adjust the external body in relation to internal emotional experience (Giuliani et al., 2011). This idea has been predominantly investigated using a range of neuroimaging techniques in non-brain injured patients (Goldin et al., 2008; Lee et al., 2008; Hayes et al., 2010; Giuliani et al., 2011). Our findings confirm the potential role of the insula in suppression, more importantly, using a completely different methodological approach and patient population. However, data from this study differ in that it is the posterior insula that has a key role in suppressing emotional expressions. Notably, the posterior insula has been largely associated to interoceptive awareness, due to its reciprocal connectivity with the somatosensory cortex (Simmons et al., 2012). These evidence, thus, offer further support to the idea that suppression requires interoceptive awareness to adjust behavioral emotional displays (Giuliani et al., 2011).

These data also open the interesting question of the relationship between cognitive control and the suppression of facial emotional experience. Thus far, the only cognitive control process associated with suppression has been verbal fluency (Gyurak et al., 2009; Goodkind et al., 2010). It is intriguing that associations between suppression and inhibition have not been found, especially considering that suppression, by definition, involves the process of inhibiting the behavioral emotional display of an already triggered emotional response (Gross and Levenson, 1997). The present study is the first to offer support for this theoretical assumption, by reporting an association between the capacity to withhold prepotent responses (response inhibition task) and the ability to suppress or hide the expression of feelings.

Why previous studies have not found a relationship between inhibition and suppression? It is interesting that studies addressing the role of cognitive control in suppression have commonly used a Stroop paradigm as an inhibition task (Gyurak et al., 2009, 2012). Notably, the inhibitory effort demanded by this task is that a verbal command conflicts with sensory information, thus requiring to inhibit an automatic response (reading the word). One of our inhibition measures ('conflicting instructions') which is similar in form to the Stroop task, did not produce significant correlations, thus replicating previous negative findings. However, another inhibition task used in this study (inhibitory control), which follows a go-no go logic, by requiring the inhibition of newly acquired responses, did show a correlation with suppression. It would be interesting to explore in the future what makes the inhibitory control task more sensitive to suppression impairment than the conflictive instructions task. Perhaps the use of several measures of inhibition in studies interested in suppression would allow a better understanding of this topic.

The results presented in this study are also of relevance for neuropsychological rehabilitation, for they suggest that some individuals with rPFC lesions are particularly impaired in suppressing and amplifying their feelings according to contextual demands. These deficits may impact patients' social life, compromising the interpersonal regulation of emotions (Niven et al., 2009) and the maintenance intimate interpersonal relationships (Bowen et al., 2010). For example, when experiencing joy at other's people misfortune (the Schadenfreude phenomenon, Smith et al., 1996; van Dijk et al., 2011) people 'may need' to inhibit their expression of joy, in order to show socially appropriate concern or sympathy. In this case, inhibition of facial

expression would allow suppression of the prepotent feeling, and thus tuning to the negative emotions experienced by the other. Loss of this ability may have devastating effects on patient's personal life. In a similar way, the ability to amplify positive emotions when witnessing someone else's accomplishment (a process sometimes termed 'synhedonia', Royzman and Rozin, 2006) may also have an important role in fostering social bonds. In this case, the amplification of joy, would allow connecting to others, thus triggering a spiral of positive affect and increasing mutual well-being (Gable et al., 2004). In the future, studies should address this problem and explore how specific forms of ER impairment, as the inability to suppress facial emotional expressions, can impact socio-emotional functioning as well as patients and relatives' quality of life.

There are some limitations of this study that need to be considered in order to interpret the data and improve the design of future experiments on this subject. First, the analysis of facial expressions only included as a variable the frequency of Duchenne Smiles but not its duration. Future studies exploring the suppression of positive affect should address this issue, since there is some evidence suggesting that the duration of facial actions appears to offer a more accurate index of emotion than frequency alone (Ekman et al., 1990). As a consequence, enjoyment smiles have a more 'consistent duration' than non-enjoyment smiles (Frank et al., 1993). However, it has also been noted that capturing the temporal unfolding of a smile—e.g. duration, velocity of beginning and ending—is extremely difficult when facial behavior is coded manually. This has led several authors to suggest the use of EEG, or automated facial analysis, in order to facilitate capturing these dynamic variables (Ekman et al., 1990; Ambadar et al., 2009). A second limitation of this study resides in the lack of ecological measures that target changes in emotional suppression as a direct consequence of the injury. Future studies should consider this point and incorporate other sources of data that can offer convergent validity to laboratory findings. The use of personality change questionnaires (e.g. Iowa Scale of Personality Change, Barrash et al., 2011) and in-depth interviews, to both brain injured survivors and relatives, are potentially useful tools to gather this type of data.

Conclusions

Management of our emotional life is clearly of enormous scientific and clinical relevance. Even though the study of ER has showed an exponential growth in experimental psychology during the last decade (Gross, 2014), such knowledge has not been transferred yet to neuropsychology (for some exceptions see Salas et al., 2013, 2014a,b). This is surprising in view that emotion dysregulation is a common problem after brain injury, presenting clinicians with important challenges in the assessment and treatment of patients. Lesion studies could contribute greatly to the understanding of the neural basis of ER, and ER strategies, by complementing neuroimaging findings.

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References

- Abreu, B., Zgaljardic, D.J., Borod, J.C., et al. (2009). Emotion regulation, processing, and recovery after acquired brain injury. Contributors to life balance. In: Matuska, K., Christiansen, C., editors. *Life Balance: Multidisciplinary Theories and Research*, pp. 223–40. Bethesda: SLACK Incorporated and AOTA Press.
- Ambadar, Z., Cohn, J.F., Reed, L.I. (2009). All smiles are not created equal: morphology and timing of smiles perceived as amused, polite, and embarrassed/nervous. *Journal of Nonverbal Behavior*, **33**, 17–34.
- Aron, A.R. (2007). The neural basis of inhibition in cognitive control. *The Neuroscientist: A Review Journal Bringing Neurobiology, Neurology and Psychiatry*, **13**(3), 214–28.
- Aron, A.R., Fletcher, P.C., Bullmore, E.T., Sahakian, B.J., Robbins, T.W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience*, **6**(2), 115–6.
- Aron, A.R., Monsell, S., Sahakian, B.J., Robbins, T.W. (2004a). A componential analysis of task-switching deficits associated with lesions of left and right frontal cortex. *Brain: A Journal of Neurology*, **127**(Pt 7), 1561–73.
- Aron, A.R., Robbins, T.W., Poldrack, R.A. (2004b). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences*, **8**(4), 170–7.
- Aron, A.R., Robbins, T.W., Poldrack, R.A. (2014). Inhibition and the right inferior frontal cortex: one decade on. *Trends in cognitive sciences*, **18**(4), 177–85.
- Barkley, R. a. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, **121**(1), 65–94. Available :<http://www.ncbi.nlm.nih.gov/pubmed/9000892>
- Barkley, R. a. (2001). The executive functions and self-regulation: an evolutionary neuropsychological perspective. *Neuropsychology Review*, **11**(1), 1–29. Available: <http://www.ncbi.nlm.nih.gov/pubmed/11392560>.
- Barrash, J., Asp, E., Markon, K., Manzel, K., Anderson, S.W., Tranel, D. (2011). Dimensions of personality disturbance after focal brain damage: Investigation with the Iowa Scales of Personality Change. *Journal of Clinical and Experimental Neuropsychology*, **33**(8), 1–20.
- Bechara, A. (2004). Disturbances of emotion regulation after focal brain lesions. *International Review of Neuroscience*, **62**, 159–93.
- Beer, J., Lombardo, M.V. (2007). Insights into emotion regulation from neuropsychology. In: Gross, J., editor. *Handbook of Emotion Regulation*, pp. 69–86. New York: The Guilford Press.
- Bowen, C., Yeates, G., Palmer, S. (2010). *A Relational Approach to Rehabilitation: Thinking about Relationships after Brain Injury*. London: Karnac Books.
- Chambers, C.D., Bellgrove, M. A., Stokes, M.G., et al. (2006). Executive “brake failure” following deactivation of human frontal lobe. *Journal of Cognitive Neuroscience*, **18**(3), 444–55.
- Chambers, C.D., Bellgrove, M.A., Gould, I.C., et al. (2007). Dissociable mechanisms of cognitive control in prefrontal and

- premotor cortex. *Journal of Neurophysiology*, **98**(6), 3638–47. Available: <http://dx.doi.org/10.1152/jn.00685.2007>.
- Chambers, C.D., Garavan, H., Bellgrove, M.A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience and Biobehavioral Reviews*, **33**(5), 631–46.
- Coan, J., Gottman, J. (2007). The specific affect coding system (SPAFF). In Coan, J., Allen, J., editors. *Handbook of Emotion Elicitation and Assessment*, pp. 267–285. New York: Oxford University Press.
- Craig, A. (2008). Interoception and emotion: a neuroanatomical perspective. *Handbook of Emotions*, **3**(602), 272–88.
- Cummings, J.L. (1997). Neuropsychiatric manifestations of right hemisphere lesions. *Brain and Language*, **57**(1), 22–37. doi:10.1006/brln.1997.1832.
- De Renzi, E., Faglioni, P. (1978). Normative data and screening power of a shortened version of the Token Test. *Cortex*, **14**(1), 41–9. Available: <http://www.ncbi.nlm.nih.gov/pubmed/16295108>.
- Delis, D., Kaplan, E., Kramer, J. (2001). *Delis-Kaplan Executive Function System (D-KEFS)*. Texas: The Psychological Corporation.
- Dillon, D., Pizzagalli, D. (2007). Inhibition of action, thought, and emotion: a selective neurobiological review. *Applied and Preventive Psychology*, **12**(3), 99–114. Available: <http://www.sciencedirect.com/science/article/pii/S0962184907000327>.
- Demaree, H., Schmeichel, B., Robinson, J., Everhart, D.E. (2004). Behavioural, affective, and physiological effects of negative and positive emotional exaggeration. *Cognition & Emotion*, **18**(8), 1079–97.
- Drewe, E.A. (1975). Go - no go learning after frontal lobe lesions in humans. *Cortex*, **11**(1), 8–16. Available: <http://www.ncbi.nlm.nih.gov/pubmed/1149469>.
- Dubois, B., Slachevsky, A., Litvan, I., Pillon, B. (2000). The FAB: a Frontal Assessment Battery at bedside. *Neurology*, **57**(3), 1621–6. Available: <http://www.ncbi.nlm.nih.gov/pubmed/11113214>.
- Dawkins, N., Cloherty, M.E., Gracey, F., Evans, J.J. (2006). The factor structure of the Hospital Anxiety and Depression Scale in acquired brain injury. *Brain Injury [BI]*, **20**, 1235–9.
- Ekman, P., Davidson, R.J., Friesen, W.V. (1990). The Duchenne smile: emotional expression and brain physiology. II. *Journal of Personality and Social Psychology*, **58**(2), 342–53. Available: <http://www.ncbi.nlm.nih.gov/pubmed/2319446>.
- Ekman, P., Friesen, W.V. (1982). Felt, false, and miserable smiles. *Journal of Nonverbal Behavior*, **6**(4), 238–52.
- Ekman, P., Friesen, W., Hager, J.C. (2002). *Facial Action Coding System*. Salt Lake City: Research Nexus.
- Feinberg, T.E. (2011). Neuropathologies of the self: clinical and anatomical features. *Consciousness and Cognition*, **20**(1), 75–81.
- Feinberg, T.E., Venneri, A., Simone, A., Fan, Y., Northoff, G. (2010). The neuroanatomy of asomatognosia and somatoparaphrenia. *Journal of Neurology, Neurosurgery and Psychiatry*, **81**, 276–81.
- Forstmann, B.U., Jahfari, S., Scholte, H.S., Wolfensteller, U., van den Wildenberg, W.P.M., Ridderinkhof, K.R. (2008). Function and structure of the right inferior frontal cortex predict individual differences in response inhibition: a model-based approach. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, **28**(39), 9790–6.
- Frank, M.G., Ekman, P., Friesen, W.V. (1993). Behavioral markers and recognizability of the smile of enjoyment. *Journal of Personality and Social Psychology*, **64**(1), 83–93.
- Gable, S.L., Reis, H.T., Impett, E.A., Asher, E.R. (2004). What do you do when things go right? The intrapersonal and interpersonal benefits of sharing positive events. *Journal of Personality and Social Psychology*, **87**(2), 228–45.
- Giuliani, N.R., Drabant, E.M., Bhatnagar, R., Gross, J.J. (2011). Emotion regulation and brain plasticity: expressive suppression use predicts anterior insula volume. *NeuroImage*, **58**(1), 10–5.
- Goldin, P.R., McRae, K., Ramel, W., Gross, J.J. (2008). The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biological Psychiatry*, **63**(6), 577–86. doi:10.1016/j.biopsych.2007.05.031.
- Goodkind, M.S., Gyurak, A., McCarthy, M., Miller, B.L., Levenson, R.W. (2010). Emotion regulation deficits in frontotemporal lobar degeneration and Alzheimer's disease. *Psychology and Aging*, **25**(1), 30–7.
- Gross, J.J. (1998). The emerging field of emotion regulation: an integrative review. *Review of general psychology*, **2**(3), 271.
- Gross, J. (1999). Emotion regulation: past, present, future. *Cognition and Emotion*, **13**(5), 551–73.
- Gross, J.J., John, O.P. (2003). Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, **85**(2), 348–62.
- Gross, J.J., Levenson, R.W. (1995). Emotion elicitation using films. *Cognition and Emotion*, **9**(1), 87–108.
- Gross, J.J., Levenson, R.W. (1997). Hiding feelings: the acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology*, **106**(1), 95–103. Available: <http://www.ncbi.nlm.nih.gov/pubmed/9103721>.
- Gross, J., Thompson, R. (2007). Emotion regulation: conceptual foundations. In: Gross, J.J., editor, *Handbook of Emotion Regulation*, vol. 50, pp. 3–24. New York: The Guilford Press. doi:10.1080/00140130600971135.
- Gross, J. (2014). *Handbook of Emotion Regulation*. New York: The Guilford Press.
- Gyurak, A., Goodkind, M.S., Kramer, J.H., Bruce, L., Levenson, R.W. (2012). Executive functions and the down-regulation and up-regulation of emotion. *Cognition and Emotion*, **26**(1), 37–41.
- Gyurak, A., Goodkind, M.S., Madan, A., Kramer, J.H., Miller, B.L., Levenson, R.W. (2009). Do tests of executive functioning predict ability to downregulate emotions spontaneously and when instructed to suppress? *Cognitive, Affective and Behavioral Neuroscience*, **9**(2), 144–52.
- Hayes, J.P., Morey, R.A., Petty, C.M., et al. (2010). Staying cool when things get hot: emotion regulation modulates neural mechanisms of memory encoding. *Frontiers in Human Neuroscience*, **4**, 1–10.
- Henry, J.D., Rendell, P.G., Scicluna, A., Jackson, M., Phillips, L.H. (2009). Emotion experience, expression, and regulation in Alzheimer's disease. *Psychology and Aging*, **24**(1), 252–7.
- Hewig, J., Hagemann, D., Seifert, J., Naumann, E., Bartussek, D. (2005). A revised set for the induction of basic emotions. *Cognition and Emotion*, **19**, 1095–109.
- Hilz, M.J., Devinsky, O., Szczepanska, H., Borod, J.C., Marthol, H., Tutaj, M. (2006). Right ventromedial prefrontal lesions result in paradoxical cardiovascular activation with emotional stimuli. *Brain*, **129**(Pt 12), 3343–55.
- Hofmann, W., Schmeichel, B.J., Baddeley, A.D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences*, **16**(3), 174–80.
- Keltner, D. (1995). Signs of Appeasement?: Evidence for the Distinct Displays of Embarrassment, Amusement, and Shame. *Journal of Personality and Social Psychology*, **68**(3).
- Kühn, S., Gallinat, J., Brass, M. (2011). Keep Calm and Carry On": structural correlates of expressive suppression of emotions. *PLoS One*, **6**(1), 4.

- Lee, T.W., Dolan, R.J., Critchley, H.D. (2008). Controlling emotional expression: behavioral and neural correlates of nonimitative emotional responses. *Cerebral Cortex* (New York, N.Y.: 1991), 18(1), 104–13.
- Lévesque, J., Eugène, F., Joanette, Y., et al. (2003). Neural circuitry underlying voluntary suppression of sadness. *Biological Psychiatry*, 53(6), 502–10.
- Levy, B.J., Wagner, A.D. (2011). Cognitive control and right ventrolateral prefrontal cortex: reflexive reorienting, motor inhibition, and action updating. *Annals of the New York Academy of Sciences*, 1224(1), 40–62.
- Lewis, M.D., Lamm, C., Segalowitz, S.J., Stieben, J., Zelazo, P.D. (2006). Neurophysiological correlates of emotion regulation in children and adolescents. *Journal of Cognitive Neuroscience*, 18(3), 430–43.
- Lhermitte, F., Pillon, B., Serdaru, M. (1986). Human autonomy and the frontal lobes. Part I: imitation and utilization behavior: a neuropsychological study of 75 patients. *Annals of Neurology*, 19(4), 326–34.
- Manes, F., Sahakian, B., Clark, L., et al. (2002). Decision-making processes following damage to the prefrontal cortex. *Brain: A Journal of Neurology*, 125(Pt 3), 624–39. Available: <http://www.ncbi.nlm.nih.gov/pubmed/11872618>.
- McDonald, S., Hunt, C., Henry, J.D., Dimoska, A., Bornhofen, C. (2010). Angry responses to emotional events: the role of impaired control and drive in people with severe traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 32(8), 855–64.
- Niven, K., Totterdell, P., Holman, D. (2009). A classification of controlled interpersonal affect regulation strategies. *Emotion* Washington DC, 9(4), 498–509. Available: <http://dx.doi.org/10.1037/a0015962>.
- Obonsawin, M.C., Jefferis, S., Lowe, R., et al. (2007). A model of personality change after traumatic brain injury and the development of the Brain Injury Personality Scales. *Journal of Neurology, Neurosurgery, and Psychiatry*, 78(11), 1239–47.
- Quirk, G.J. (2007). Prefrontal-Amygdala interactions in the regulation of fear. In Gross, J.J., editor. *Handbook of Emotion Regulation*, pp. 27–46. New York: The Guilford Press.
- Robertson, I.H., Ward, T., Ridgeway, V., Nimmo-Smith, I. (1994). *The Test of Everyday Attention (TEA)*. Bury: Thames Valley Test Company.
- Rorden, C., Brett, M. (2000). Stereotaxic display of brain lesions. *Behavioural neurology*, 12(4), 191–200.
- Rorden, C., Karnath, H.O., Bonilha, L. (2007). Improving lesion-symptom mapping. *Journal of cognitive neuroscience*, 19(7), 1081–8.
- Rottenberg, J., Kasch, K.L., Gross, J.J., Gotlib, I.H. (2002). Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. *Emotion*, 2(2), 135–46.
- Rottenberg, J., Ray, R.R., Gross, J. (2007). Emotion elicitation using films. In Coan, J., Allen, J., editors. *Handbook of Emotion Elicitation and Assessment*, pp. 9–28. New York: Oxford University Press.
- Rovner, B., Folstein, M. (1987). Mini-mental state exam in clinical practice. *Hospital Practice*, (22), 103–10.
- Royzman, E.B., Rozin, P. (2006). Limits of symhedonia: the differential role of prior emotional attachment in sympathy and sympathetic joy. *Emotion*, 6(1), 82–93.
- Rubia, K., Smith, A.B., Brammer, M.J., Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *NeuroImage*, 20(1), 351–8.
- Salas, C., Castro, O. (2014). Mente desorganizada y reaccion catastrófica: regulación emocional intrínseca y extrínseca en sobrevivientes de lesión cerebral adquirida. *Revista Chilena De Neuropsicología*, 9(1), 38–45.
- Salas, C.E. (2012). Surviving Catastrophic Reaction after Brain Injury: The Use of Self- Regulation and Self – Other Regulation. *Neuropsychanalysis*, 14(1), 77–92.
- Salas, C.E., Gross, J.J., Rafal, R.D., Viñas-Guasch, N., Turnbull, O.H. (2013). Concrete behaviour and reappraisal deficits after a left frontal stroke: a case study. *Neuropsychological Rehabilitation*, 23(4), 467–500.
- Salas, C.E., Gross, J.J., Turnbull, O.H. (2014a). Reappraisal generation after acquired brain damage: The role of laterality and cognitive control. *Frontiers in Psychology*, 5, 242.
- Salas, C.E., Radovic, D., Yuen, K.S.L., Yeates, G.N., Castro, O., Turnbull, O.H. (2014b). “Opening an emotional dimension in me”: Changes in emotional reactivity and emotion regulation in a case of executive impairment after left fronto-parietal damage. *Bulletin of the Menninger Clinic*, 78(4), 301–34.
- Salas, C.E., Radovic, D., Turnbull, O.H. (2011). Inside-out: comparing internally generated and externally generated basic emotions. *Emotion*, 12(3), 568.
- Shallice, T. (1988). *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.
- Simons, G., Ellgring, H., Smith Pasqualini, M. (2003). Disturbance of spontaneous and posed facial expressions in Parkinson's disease. *Cognition & Emotion*, 17(5), 759–78.
- Simmons, W.K., Avery, J.A., Barcalow, J.C., Bodurka, J., Drevets, W.C., Bellgowan, P. (2012). Keeping the body in mind: Insula functional organization and functional connectivity integrate interoceptive, exteroceptive, and emotional awareness. *Human Brain Mapping*, 34(11), 2944–58.
- Smith, R., Turner, T., Garonzik, R., Leach, C., Urch-Druskat, V., Weston, C. (1996). Envy and schadenfreude. *Personality and Social Psychology Bulletin*, 22(2), 158–68.
- Soussignan, R. (2002). Duchenne smile, emotional experience, and autonomic reactivity: a test of the facial feedback hypothesis. *Emotion*, 2(1), 52–74.
- Stern, R.A., Singer, E.A., Duke, L.M., Singer, N.G. (1994). The Boston qualitative scoring system for the Rey-Osterrieth complex figure: description and interrater reliability. *Clinical Neuropsychologist*, 8(3), 309–22.
- Stuss, D., Alexander, M. (1999). Affectively burnt in: a proposed role of the right frontal lobe. In: Tulving, E., editor, *Memory, Consciousness and the Brain: The Tallin Conference*, pp. 215–27.
- Stuss, D.T., Alexander, M.P. (2000a). Executive functions and the frontal lobes: a conceptual view. *Psychological Research*, 63(3–4), 289–98. Available: <http://www.ncbi.nlm.nih.gov/pubmed/11004882>.
- Stuss, D.T., Alexander, M.P. (2000b). The anatomical basis of affective behavior, emotion and self-awareness: a specific role of the right frontal lobe. In: Hatano, G., Okada, K., Tanabe, H., editors. *Affective Minds*, pp. 13–25. Amsterdam: Elsevier.
- Stuss, D., Benson, D. (1986). *The Frontal Lobes*. New York: Raven Press.
- Tate, R.L. (1999). Executive dysfunction and characterological changes after traumatic brain injury: two sides of the same coin? *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 35(1), 39–55. Available: <http://www.ncbi.nlm.nih.gov/pubmed/10213533>.
- Thayer, J.F., Lane, R.D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, 61(3), 201–16. Available: <http://www.ncbi.nlm.nih.gov/pubmed/11163422>.
- Tranel, D., Bechara, A., Denburg, N.L. (2002). Asymmetric functional roles of right and left ventromedial prefrontal cortices in

- social conduct, decision-making, and emotional processing. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, **38**(4), 589–612. Available: <http://www.ncbi.nlm.nih.gov/pubmed/12465670>.
- Van Dijk, W.W., Ouwerkerk, J.W., Wesseling, Y.M., van Koningsbruggen, G.M. (2011). Towards understanding pleasure at the misfortunes of others: the impact of self-evaluation threat on schadenfreude. *Cognition and Emotion*, **25**(2), 360–8.
- Watson, D., Clark, L.A. (1994). The PANAS-X Manual for the Positive and Negative Affect Schedule - Expanded Form. *Unpublished Manuscript, University of Iowa*. Available: <http://scholar.google.com/scholar?hl=en&btnG=Search&q=intitle:The+PANAS-X+Manual+for+the+Positive+and+Negative+Affect+Schedule+-+Expanded+Form#0>
- Weschler, D. (1981). *Wechsler Adult Intelligence Scale Revised (Wais-R)*. New York: The Psychological Corporation.
- Weschler, D. (1987). *Wechsler Memory Scale-Revised (WMS-R) Manual*. New York: The Psychological Corporation.
- Zigmond, A.S., Snaith, R. P. (1983). The hospital anxiety and depression scale (HADS). *Acta Psychiatrica Scandinavica*, **67**, 361–70.