

## Hypnosis decouples cognitive control from conflict monitoring processes of the frontal lobe

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Hypnosis can profoundly alter sensory awareness and cognitive processing. While the cognitive and behavioral phenomena associated with hypnosis have long been thought to relate to attentional processes, the neural mechanisms underlying susceptibility to hypnotic induction and the hypnotic condition are poorly understood. Here, we tested the proposal that highly hypnotizable individuals are particularly adept at focusing attention at baseline, but that their attentional control is compromised following hypnosis due to a decoupling between conflict monitoring and cognitive control processes of the frontal lobe. Employing event-related fMRI and EEG coherence measures, we compared conflict-related neural activity in the anterior cingulate cortex (ACC) and control-related activity in the lateral frontal cortex (LFC) during Stroop task performance between participants of low and high hypnotic susceptibility, at baseline and after hypnotic induction. The fMRI data revealed that conflict-related ACC activity interacted with hypnosis and hypnotic susceptibility, in that highly susceptible participants displayed increased conflict-related neural activity in the hypnosis condition compared to baseline, as well as with respect to subjects with low susceptibility. Cognitive-control-related LFC activity, on the other hand, did not differ between groups and conditions. These data were complemented by a decrease in functional connectivity (EEG gamma band coherence) between frontal midline and left lateral scalp sites in highly susceptible subjects after hypnosis. These results suggest that individual differences in hypnotic susceptibility are linked with the efficiency of the frontal attention system, and that the hypnotized condition is characterized by a functional dissociation of conflict monitoring and cognitive control processes.

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### Introduction

Within a few minutes of hypnotic induction, some 10–15% of healthy alert individuals are able to demonstrate profound alterations in many aspects of their conscious experience (Hilgard, 1965). In response to suggestion, they may experience a lack of control over their own actions, the inability to recall recent events, the absence of pain and other specific sensations, or conversely the apparent reality of illusory events. These features of hypnosis are utilized by clinicians to facilitate therapeutic interventions in diverse areas of psychological medicine. The effectiveness of incorporating hypnosis in clinical interventions has gained positive empirical support in pain control, anxiety, depression, trauma, weight loss, and eating disorders among other areas (Lynn et al., 2000). The rapid, non-pathological, and reversible changes in conscious awareness and cognitive processing encountered in hypnosis also provide an intriguing domain, as well as a (largely unexploited) tool of research, in the cognitive neurosciences (Raz and Shapiro, 2002). For instance, hypnotic manipulation of subjective experience, such as the processing of pain, in conjunction with current neuroimaging techniques has helped to dissociate the neural bases of distinct aspects of sensory and cognitive processes (Halligan et al., 2000; Rainville et al., 1997). By the same token, advances in neuroimaging have started to facilitate our understanding of specific hypnotic manipulations of sensory experience (Kosslyn et al., 2000; Rainville et al., 1999, 2002; Szechtman et al., 1998). However, the basic neural mechanisms underpinning the phenomena of hypnosis and hypnotic susceptibility are currently not well understood.

Many accounts of hypnosis have proposed that individual differences in hypnotic susceptibility are related to differences in executive attentional control. One traditional view has been that hypnosis itself is characterized by strongly focused attention, and that hypnotic susceptibility is due to individual differences in the ability to engage in such focused attention (Barber, 1960; Spiegel, 2003; Tellegen and Atkinson, 1974; cf. Jamieson and Sheehan, 2002). Another account holds that while highly hypnotizable

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subjects may be exceptionally adept at focusing attention at baseline, their attentional control is largely compromised following hypnotic induction (Crawford and Gruzelier, 1992; Gruzelier, 1990; Hilgard, 1965, 1977; 1998; Woody and Bowers, 1994).

The latter model has received support from a series of studies showing that participants with high, but not low susceptibility exhibit impaired attentional control after hypnotic induction, as evidenced by deteriorated error performance on the Stroop (MacLeod, 1991; Stroop, 1935) paradigm (Jamieson and Sheehan, 2004; Kaiser et al., 1997; see also Nordby et al., 1999), and attenuated orienting responses (Gruzelier and Brow, 1985; Gruzelier et al., 1988). Support has also been forthcoming from neuropsychological evidence which, inter alia, has often suggested that left anterior functions in particular appear to be compromised during hypnosis in more highly susceptible participants; measures have encompassed event-related potentials (Jutai et al., 1993), haptic sorting (Cikurel and Gruzelier, 1990; Gruzelier et al., 1984), and letter versus category word fluency tasks (Gruzelier and Warren, 1993; Kallio et al., 2001; cf. Aikins and Ray, 2001). It should be noted that these observations refer to performance after generic hypnotic inductions that do *not* entail specific instructions aimed at eliminating interference effects. In cases where such instructions are given, Stroop interference in hypnotic as well as post-hypnotic performance may be reduced in highly susceptible individuals (Raz et al., 2002, 2003; Sheehan et al., 1988).

These observations have been tied to neurophysiological hypotheses relating hypnosis in highly susceptible individuals to alterations of anterior brain functions such as selective inhibition, disconnection, and dissociation of the frontal lobe (Gruzelier, 1990, 1998, 2000, 2004), or decoupling between conflict monitoring and cognitive control functions of the frontal attention system (see below) (Jamieson and Sheehan, 2004). The current study aimed at directly testing the merits of the “focused attention” model and the “impaired attention” model of hypnosis at the neural level, by capitalizing on recent advances in the understanding of the neural bases of frontal attention processes.

Much recent work on characterizing the functional neuroanatomy of attentional control suggests the existence of dissociable sub-processes of conflict monitoring and cognitive control, performed by the dorsal anterior cingulate cortex (ACC) and lateral frontal cortex (LFC), respectively (Botvinick et al., 2001; MacDonald et al., 2000). It has been proposed that the ACC performs continuous monitoring of potential response conflict (due to interference or “crosstalk” between different processing streams), and that in the case of high conflict detection, the LFC implements cognitive control, resolving conflict by biasing information processing towards task-relevant properties (Botvinick et al., 2001; for recent reviews, see Botvinick et al., 2004; Ridderinkhof et al., 2004). An ample body of data from neuroimaging studies supports the notion that ACC activation covaries with levels of response conflict in selective attention tasks, such as the Stroop paradigm (e.g., Botvinick et al., 1999; Carter et al., 2000; Casey et al., 2000; MacDonald et al., 2000; Ullsperger and von Cramon, 2001). Varying levels of behavioral interference effects across individuals have been shown to be directly positively correlated with ACC activation (Botvinick et al., 1999; Carter et al., 2000; Casey et al., 2000), making it possible to employ conflict-related ACC activity as a marker of the level of conflict experienced by the individual subject (Richeson et al., 2003).

Support for the view that the LFC implements cognitive control stems from research showing increased left LFC

activation in anticipation of cued high conflict, but not low conflict Stroop trials (MacDonald et al., 2000). MacDonald and colleagues manipulated the expectation of conflict (and by inference the strategic allocation of cognitive control) by switching between cued word-naming (low conflict) and color-naming (high conflict) Stroop trials, and demonstrated a dissociation between preparatory LFC activation and trial-conflict-related ACC activation. In another recent fMRI Stroop study, left LFC activation has been shown to be inversely related to levels of response conflict, correlating positively with reduced interference effects following high conflict trials (Egner and Hirsch, 2005), as would be expected from brain regions underpinning cognitive control (Botvinick et al., 2001).

We employed event-related fMRI in order to assess conflict- and control-related activity in the ACC and LFC at varying levels of response conflict in a Stroop paradigm in participants of high and low hypnotic susceptibility, before and following hypnotic induction (not containing specific Stroop-related instructions). Participants performed a variant of the Stroop task that consisted of four trial types of varying response conflict: word-naming of congruent stimuli (*low conflict*), word-naming of incongruent stimuli and color-naming of congruent stimuli (*moderate conflict*), and color-naming of incongruent stimuli (*high conflict*). Note that congruent trials incur neural conflict (e.g., Bench et al., 1993; Carter et al., 1995) due to simultaneous activation in pathways that compete for response selection, even though the final correct response would be identical. This conflict is expected to be greater in color-naming trials for the same reason that incongruent trials cause greater conflict in color- than in word-naming, namely, the more ballistic nature of the word-naming process. We expected that, across groups and conditions, ACC activity would vary as a function of response conflict, and that cognitive-control-related LFC regions would be more activated in color-naming than in word-naming trials (see MacDonald et al., 2000). Between groups and conditions, however, the focused attention and the impaired attention models of hypnosis make opposing predictions: The focused attention model predicts superior efficiency of executive attention (i.e., *less* conflict-related ACC activation) in highly susceptible subjects both at baseline and after hypnotic induction, relative to subjects with low susceptibility. The impaired attention model, on the other hand, predicts superior efficiency in highly susceptible subjects at baseline, but decreased attentional efficiency (i.e., *more* conflict-related ACC activation) after hypnotic induction, relative to subjects with low susceptibility. Additionally, we explored how the predicted effect of hypnosis on conflict-related ACC activity could be related to control-related processing in the LFC, and/or changes in functional connectivity between medial and lateral frontal cortical sites. To assess functional connectivity between ACC and LFC at a high temporal resolution, we measured EEG coherence between frontal midline and lateral scalp electrodes in the same participants as in the fMRI study, performing the same experimental paradigm.

## Methods

### Subjects

Twenty-two healthy right-handed participants (mean age = 26.5, SD = 5.6; ten females), eleven of low and eleven of high hypnotic susceptibility, gave informed consent to take part in this

study, which received ethical approval by the Riverside Research Ethics Committee, West London (ref. RREC 3193). Participants were derived from a pool of pre-tested subjects, on the basis of their hypnotic susceptibility. Hypnotic susceptibility was determined through pre-screening in group hypnosis sessions with the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS-A) (Shor and Orne, 1962), followed by an individual hypnotic induction with the Stanford Hypnotic Susceptibility Scale Form C (SHSS-C) (Weitzenhoffer and Hilgard, 1962). Participants were classed as having low susceptibility if both their Harvard and SHSS-C scores were  $\leq 3$  (group mean score = 1.7), and as having high susceptibility if both their Harvard and SHSS-C scores were  $\geq 9$  (group mean score = 9.8). Baseline versus post-hypnosis self-report ratings on 10-point Likert-scales of level of hypnosis in this study disclosed a highly significant hypnosis  $\times$  susceptibility interaction ( $P < 0.001$ ), due to increased reported hypnotic depth in the highly susceptible participants only ( $P < 0.001$ ).

#### *Stroop task and procedure*

This variant of the Stroop paradigm was created and presented using Presentation software (Neurobehavioral Systems, <http://www.nbs.neuro-bs.com>). The task consisted of two word stimuli (“red” and “green”, font size 32, Times New Roman) presented in either red or green hue for 1500 ms centrally on a black background, with a stimulus-onset interval of 10480 ms (4 TRs). This represents a “slow” event-related design, approximately replicating the event-timing employed in MacDonald et al. (2000). Stimuli were presented in two runs of eight alternating blocks of word-naming versus color-naming instructions, with six congruent and incongruent stimuli presented in random order in each block. Subjects were instructed to respond as quickly and accurately as possible to each stimulus by pushing either the left (for “red”) or right (for “green”) button on an MRI-compatible response device, with their left or right index finger, respectively. The task was run once at baseline, and once following hypnosis, with order of hypnotic condition counterbalanced across participants and groups. In the MRI scanner, stimuli were presented via a projector to a mirror screen located at the head of the bore, which the participants could view via a mirror attached to the head coil. In the EEG laboratory, stimulus delivery was implemented via a 17-in. screen positioned approximately 1 m away from the participants.

#### *Hypnosis procedure*

Hypnosis induction and Stroop task instructions were administered via headphones from an audiotape after the subject was comfortably placed in the bore of the scanner (or after the EEG electrode cap preparation). These instructions were recorded in the voice of the experimenter by whom subjects had been individually screened (and who dealt with subjects at each subsequent testing session). The hypnotic induction was specifically tailored to the needs of fMRI testing and utilized suggestions which incorporated and reframed potentially uncomfortable aspects of the scanner environment in a non-threatening manner. The induction made no reference to suggestions of relaxation, heaviness, or sleep. Identical instructions were played in both fMRI and EEG testing sessions, except for the exclusion of scanner-specific references in the EEG session. A transcript of taped instructions is available upon request.

#### *fMRI data acquisition*

Images were acquired employing a standard head coil with a Siemens Vision 1.5-T scanner (Erlangen, Germany) at Charing Cross Hospital, London. Functional images were recorded axially along the AC–PC plane with a T2\*-weighted gradient-echo echoplanar imaging (EPI) sequence (echo time TE = 60 ms, acquisition time TA = 2400 ms, repetition time TR = 2620 ms, flip-angle 90°, in-plane resolution 3.5 mm  $\times$  3.5 mm) affording whole brain coverage with 25 contiguous slices of 5 mm thickness. For each run of the Stroop task, there were 229 image acquisitions, with the first 5 scans being discarded to allow the scanner to reach steady state magnetic saturation. T1\*-weighted structural images were acquired with an MP-RAGE sequence (TE = 4 ms, TI = 300 ms, in-plane resolution 1 mm  $\times$  1 mm, effective slice thickness = 2 mm, 128 slices).

#### *fMRI data analysis*

Functional MRI data were analyzed using SPM2 software (Wellcome Department of Cognitive Neurology, London, UK; see <http://www.fil.ion.ucl.ac.uk/spm>). EPI volumes were corrected for differences in acquisition slice-timing, spatially realigned to the first volume of the first session, and a mean EPI image was calculated to which the structural image was co-registered. Transformation parameters were derived from normalizing the co-registered structural image to a template brain within the stereotactic space of the Montreal Neurological Institute (MNI), and the derived parameters were then applied to the EPI volumes. Normalized images were smoothed with a Gaussian kernel of 7  $\times$  7  $\times$  10 mm full-width at half-maximum (twice the size of the original voxel dimensions). Functional data were analyzed using a general linear model (GLM) approach (Friston et al., 1995). Regressors of event-related BOLD responses were modeled in each subject for correct responses from each trial type (word-congruent, word-incongruent, color-congruent, color-incongruent) using a standard hemodynamic response function (canonical HRF), and were used as covariates in GLM analyses for each subject in order to generate voxel-based statistical parametric maps (SPM). Error trials were modeled as a separate regressor. To remove low-frequency temporal confounds, data were high-pass filtered (128 s), and an autoregressive function (AR-1 in SPM2) was employed to estimate for temporal autocorrelation in the data and correct the degrees of freedom accordingly. Contrast SPMs were calculated for each subject individually and these contrasts were then employed for random effects analyses at the group level.

A priori analyses of conflict-related ACC activation were carried out in the following way: First, event-related activations to moderate and high conflict stimuli were assessed across groups and conditions within the ACC, as defined by an anatomical ROI mask consisting of Brodmann areas BA 24 and BA 32, applied with the WFU Pick Atlas (see <http://www.rad.wfubmc.edu/fmri>) (Maldjian et al., 2003). Activation foci within the ACC that significantly activated above a false discovery rate (FDR) (Genovese et al., 2002) threshold of 5% (i.e.,  $P \leq 0.05$ , corrected) and a cluster-extent threshold of a minimum of 10 contiguous voxels to both moderate and high conflict stimuli were then employed as a functional ROI, created with Marsbar software (Brett et al., 2002; <http://marsbar.sourceforge.net/>). From this functionally defined ROI, mean conflict-related activation data were extracted for each

subject and entered into a  $2 \times 2$  condition (baseline vs. hypnosis) by group (low vs. high susceptibility) analysis of variance (ANOVA). Subsequently, we employed the same analysis strategy to assess activation in putative cognitive control regions of the LFC, namely, by defining functional ROIs exhibiting more activation on color-naming than on word-naming trials within an anatomical ROI comprising BAs 8, 9, 10, and 44–46.

#### EEG acquisition and analysis

EEG was recorded in the Cognitive Neuroscience laboratory, Imperial College London, Charing Cross campus on a Neuroscan Synamps system (Compumedics Inc.) in an electrically shielded chamber via a 28-channel Electro-cap (ECI International). Signal was acquired and digitized at a sampling rate of 500 Hz and passed through a 0- to 100-Hz bandpass filter (24 dB/octave roll-off). Electrodes were placed in accordance with the international 10–20 system, with a ground electrode placed 1.5 cm anterior to the central frontal electrode (FZ), and referenced off-line to linked earlobe electrodes. All electrode impedances were kept below 10 k $\Omega$ . The electrooculogram (EOG) was recorded with tin cup electrodes placed on the orbis ocularis muscle above and below the left eye, and on the left and right outer canthi, approximately 1 cm lateral to either eye. Eye-blinks, horizontal, and vertical eye-

movements were removed by applying an offline artifact correction algorithm (Croft and Barry, 2000). All data processing and analyses were carried out with Neuroscan software (version 4.2). EOG-corrected data were epoched into 1024-ms intervals around each stimulus presentation (–100 ms–924 ms) and baseline-corrected with respect to a 100-ms pre-stimulus interval. Epochs containing amplitude fluctuations exceeding  $\pm 100\mu\text{V}$  were rejected as artifact-contaminated. The EEG epochs were grouped according to the four trial categories (word-congruent, word-incongruent, color-congruent, color-incongruent) for the wakeful and hypnotic task conditions separately, excluding error trials. Average coherence values were then calculated for delta (0–3.9 Hz), theta (4–7.9 Hz), alpha (8–12.9 Hz), beta (13–29.9 Hz), and gamma (30–49.9 Hz) frequency bands and used in the statistical analyses (see Results). The EEG recordings were carried out approximately 2 to 4 weeks after the fMRI recordings.

## Results

#### fMRI behavioral data

Subjects exhibiting outlier values ( $>2$  SD from the mean) were excluded from the behavioral data analyses, and error trials were excluded from RT analyses. Significant Stroop interference effects

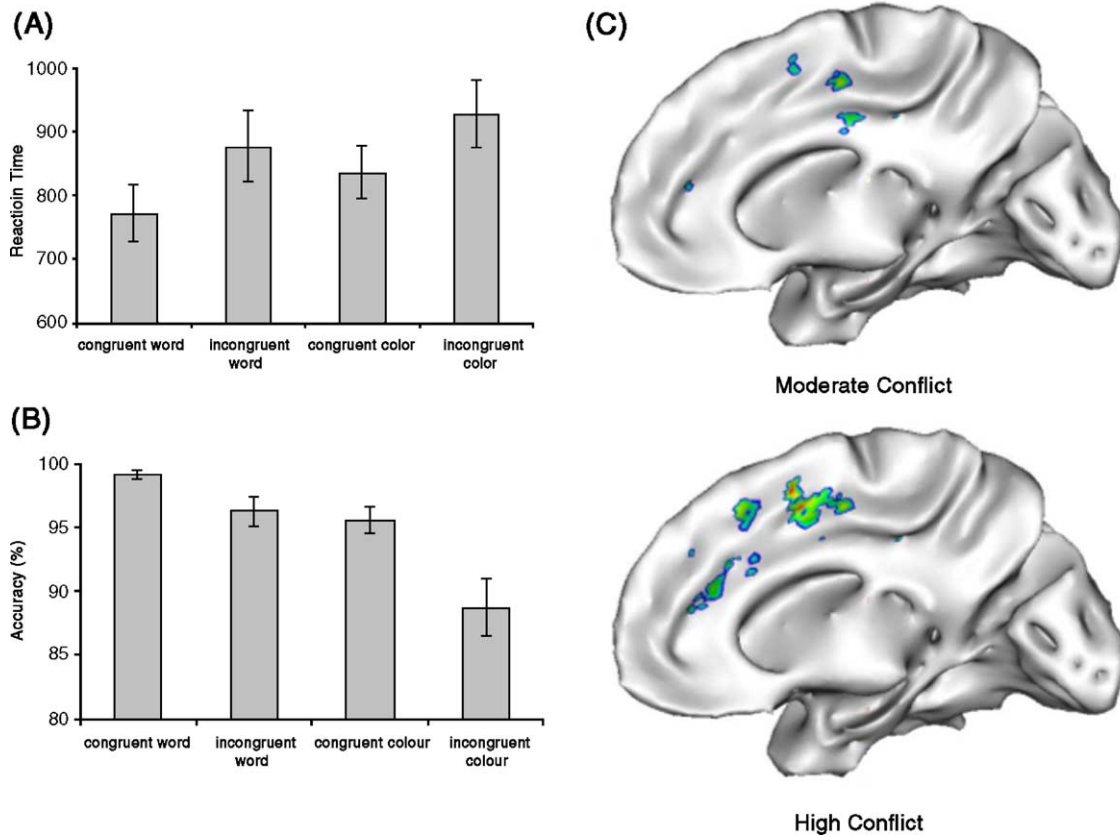


Fig. 1. Stroop performance and ACC BOLD responses at varying levels of response conflict. (A) Mean reaction times ( $\pm$ SEM) for the different Stroop trial types. (B) Mean accuracy rates ( $\pm$ SEM) for the different Stroop trial types. (C) Significant activations to moderate response conflict (top panel: incongruent word-naming/congruent color-naming > congruent word-naming) and the high conflict contrast (bottom panel: incongruent color-naming > congruent word-naming) in the (right) ACC. Activity is displayed at a false discovery rate (FDR) of  $P = 0.05$  with an extent threshold of at least ten contiguous voxels. For display purposes, activations are superimposed on a partially inflated right hemisphere medial wall of a normalized single subject T1 scan (segmentation and inflation was carried out with Brain VISA software; <http://brainvisa.info/index.html>).

were observed in the RT data (Fig. 1A) for both congruency ( $F[1,18] = 18.25$ ;  $P < 0.001$ ) and type of instruction ( $F[1,18] = 7.02$ ;  $P < 0.05$ ). In line with expectations, RTs to high conflict trials were largest, significantly larger than in moderate conflict trials ( $t[19] = 3.64$ ;  $P < 0.005$ ;  $t[19] = 1.75$ ;  $P = 0.097$ ) and in low conflict trials ( $t[19] = 5.64$ ;  $P < 0.001$ ). Compared to the low conflict trials, RTs were significantly larger in both moderate conflict conditions ( $t[19] = 4.44$ ;  $P < 0.001$ ;  $t[19] = 2.59$ ;  $P < 0.05$ ), with no difference in RTs between moderate conflict trials. The accuracy data mirrored the interference effects of the RT results (Fig. 1B), main effects being detected for congruency ( $F[1,18] = 24.84$ ;  $P < 0.001$ ), and instruction ( $F[1,18] = 10.89$ ;  $P = 0.005$ ). As expected, high conflict trials produced most errors, significantly more than the moderate conflict ( $t[19] = 2.83$ ;  $P < 0.05$ ;  $t[19] = 3.59$ ;  $P < 0.005$ ), and low conflict trials ( $t[19] = 4.55$ ;  $P < 0.001$ ). Both moderate conflict trial types induced significantly higher error rates than the low conflict condition ( $t[19] = 2.75$ ;  $P < 0.05$ ;  $t[19] = 3.22$ ;  $P = 0.005$ ), with no difference in accuracy between the moderate conflict conditions. There were no effects involving hypnotic induction or susceptibility variables.

#### fMRI imaging data

In order to verify that ACC activation was susceptible to the manipulation of conflict levels in the current study, blood oxygenation level-dependent (BOLD) responses to different levels of response conflict were assessed within the ACC by comparing event-related activation in moderate versus low conflict trials (moderate conflict contrast), and in high versus low conflict trials (high conflict contrast), excluding error trials. As hypothesized, the extent of ACC activation increased with conflict level across groups and conditions, as can be seen in Fig. 1C (see Table 1 for summary of activations). ACC regions that were susceptible to both moderate and high conflict contrasts served as a functional ROI for comparing activations between groups and conditions (see Fig. 2A and Table 1). A  $2 \times 2$  condition (baseline vs. hypnosis) by

group (low vs. high susceptibility) ANOVA on conflict-related activation revealed a significant condition  $\times$  group crossover interaction effect ( $F[1,20] = 6.27$ ;  $P < 0.05$ ), as shown in Fig. 2B. This interaction effect was characterized by significantly higher ACC activation in highly susceptible subjects compared to subjects with low susceptibility in the hypnotic condition ( $t[20] = 2.25$ ;  $P < 0.05$ ), but not at baseline ( $t[20] = 1.58$ ;  $P = 0.13$ ). Furthermore, conflict-related ACC activation in the highly susceptible subjects showed a significant increase from baseline to hypnosis ( $t[10] = 2.67$ ;  $P < 0.05$ ), whereas there was a non-significant decrease in the subjects with low susceptibility ( $t[10] = 1.10$ ;  $P = 0.29$ ). In order to corroborate these results in the more traditional framework of comparing incongruent to congruent trials, these trials were contrasted irrespective of color- or word-naming instructions. This contrast identified a conflict-related activation cluster (26 voxels) in the right medial frontal gyrus (BA 32;  $x = 6$ ,  $y = 8$ ,  $z = 44$ ). This cluster showed the same pattern of condition  $\times$  group interaction as the previous conflict-responsive ROIs ( $F[1,20] = 5.10$ ;  $P < 0.05$ ), characterized by a significant increase in activity from baseline to hypnosis in the highly susceptible subjects only ( $t[10] = 2.37$ ;  $P < 0.05$ ).

LFC regions of cognitive control were identified by contrasting color-naming with word-naming trials across groups and conditions. Within bilateral LFC, no region displayed significant activation for this contrast at an FDR of  $P < 0.05$ . However, when adjusting the statistical threshold to an uncorrected voxelwise level of  $P < 0.001$ , a single significant activation cluster (27 voxels) was obtained in the left LFC (see Fig. 3A), namely, in the left inferior frontal gyrus (GFi) in BA45 ( $x = -56$ ,  $y = 14$ ,  $z = 5$ ). A  $2 \times 2$  condition (baseline vs. hypnosis) by group (low vs. high susceptibility) ANOVA on control-related activity in this ROI revealed no main or interaction effects (Fig. 3B), indicating that there were no differences in the allocation of cognitive control resources between groups and conditions. Finally, an exploratory analysis of condition by group effects across the whole brain as well as regions of interest (bilateral

Table 1  
Anterior cingulate foci activated by moderate response conflict, high response conflict, or both

	Talairach		Talairach coordinates			Cluster size	Voxelwise $P$ (FDR-corrected)
	Label	BA	$x$	$y$	$z$		
Moderate conflict	CG	24	4	-22	34	24	0.004
	CG	24	12	8	48	11	0.012
	CG	24	-2	-22	36	12	0.012
	CG	24	16	-4	46	42	0.012
	AC	32	8	43	13	25	0.016
	CG	32	-2	8	40	18	0.025
High conflict	AC	32	4	40	16	421	0.016
	AC	32	-2	36	24	102	0.017
	CG	24	4	22	34	20	0.017
	CG	32	-2	16	42	38	0.019
	CG	24	-2	-22	34	16	0.020
	AC	32	18	34	24	10	0.023
	CG	24	-4	4	28	20	0.028
	CG	24	4	-22	34	12	0.001
Overlap	CG	24	12	8	48	11	0.001
	CG	24	-2	-22	36	11	0.001
	CG	24	16	-4	46	37	0.001
	AC	32	8	43	13	24	0.001
	CG	32	-2	8	40	10	0.001
	CG	32	-2	8	40	10	0.001

Note. BA = Brodmann Area; Talairach labels: CG = Cingulate Gyrus, AC = Anterior Cingulate.

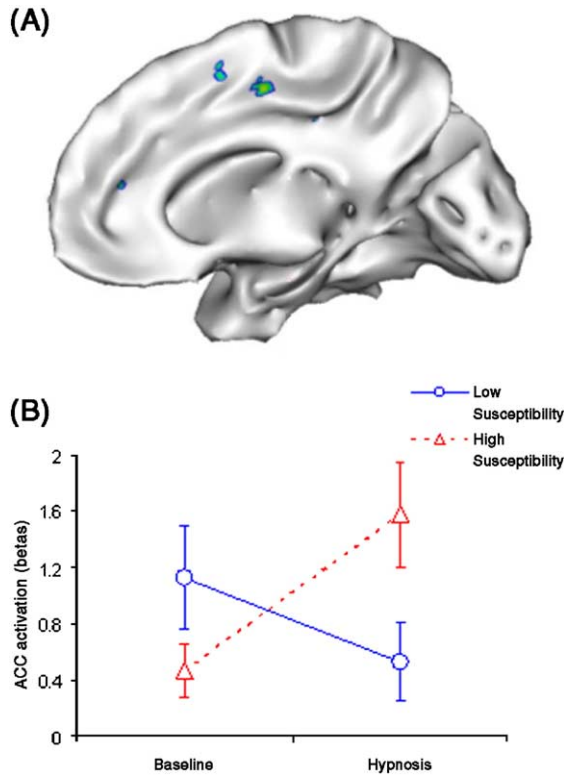


Fig. 2. Conflict-susceptible regions of the ACC and their response magnitudes in subjects of low and high hypnotic susceptibility, at baseline and following hypnosis. (A) ACC ROIs displaying significant activation ( $FDR < 0.05$ ) to both moderate and high response conflict, superimposed on a partially inflated right hemisphere medial wall of a normalized single subject T1 scan (segmentation and inflation was carried out with Brain VISA software; <http://brainvisa.info/index.html>). (B) Mean conflict-related ACC activity (beta weights  $\pm$  SEM) from the ROIs in panel A for baseline and hypnotic conditions, in participants with low and high hypnotic susceptibility.

prefrontal and parietal cortices) did not yield any significant effects after correction for multiple comparisons. When lowering the statistical threshold to uncorrected voxelwise  $P < 0.001$ , a single cluster (18 voxels) was found in the left parahippocampal gyrus (BA 28;  $x = -20$ ,  $y = -20$ ,  $z = -22$ ), where highly susceptible subjects displayed a hypnosis-specific increase in activation, compared to subjects with low susceptibility. In summary, in accordance with the impaired attention model hypotheses, highly susceptible participants were characterized by a significantly decreased efficiency of conflict resolution in the hypnotic condition, relative to baseline as well as with respect to participants with low susceptibility. The significant increase in conflict-related ACC activity in highly susceptible subjects in the hypnosis condition was not accompanied by a commensurate increase in cognitive-control-related LFC activation.

#### EEG behavioral data

Subjects exhibiting outlier values ( $>2$  SD from the mean) were excluded from the behavioral data analyses, and error trials were excluded from RT analyses. Stroop reaction time data revealed the familiar main effects of stimulus congruency ( $F[1,18] = 37.5$ ;  $P < 0.001$ ) and instruction ( $F[1,18] = 12.0$ ;  $P < 0.005$ ). High conflict trials were associated with slower reaction times than moderate

conflict trials ( $t[19] = 6.58$ ;  $P < 0.001$ ;  $t[19] = 3.26$ ;  $P < 0.005$ ), and low conflict trials ( $t[19] = 8.70$ ;  $P < 0.001$ ). The two moderate conflict conditions did not differ from each other, but both induced significantly slower responses than the low conflict trials ( $t[19] = 4.22$ ;  $P < 0.001$ ;  $t[19] = 2.9$ ;  $P < 0.01$ ). No hypnosis or hypnotic susceptibility effects were obtained. Response accuracy again was affected by stimulus congruency ( $F[1,18] = 16.35$ ;  $P < 0.005$ ) and instruction ( $F[1,18] = 5.44$ ;  $P < 0.05$ ). High conflict trials displayed the lowest accuracy rate, inducing significantly more errors than moderate conflict trials ( $t[19] = 2.86$ ;  $P < 0.05$ ;  $t[19] = 1.93$ ;  $P = 0.069$ ), and low conflict trials ( $t[19] = 3.51$ ;  $P < 0.005$ ). Furthermore, moderate conflict trials induced higher error rates than low conflict trials ( $t[19] = 4.20$ ;  $P < 0.001$ ;  $t[19] = 2.57$ ;  $P < 0.05$ ). However, these accuracy data were qualified by a hypnosis  $\times$  susceptibility interaction ( $F[1,18] = 4.79$ ;  $P < 0.05$ ), as participants with low susceptibility improved their accuracy in the hypnotic condition ( $t[19] = 2.60$ ;  $P < 0.05$ ), while highly susceptible participants did not (see Fig. 4A).

#### EEG coherence data

Functional connectivity (EEG coherence) between neural processes in frontal midline (Fz electrode) and left dorsolateral frontal sites (F3 electrode) was calculated for delta, theta, alpha, beta, and gamma frequency bands (see Methods), and employed

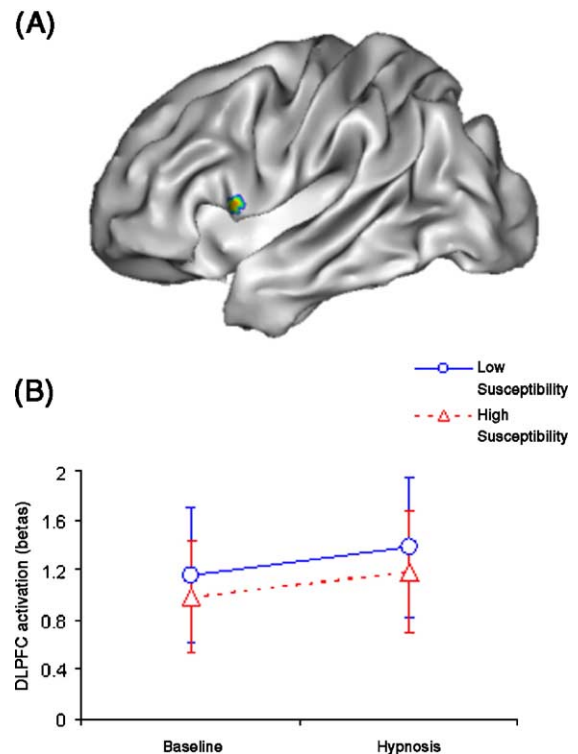


Fig. 3. Cognitive-control-related area in left LFC and its response magnitudes in subjects of low and high hypnotic susceptibility, at baseline and following hypnosis. (A) LFC ROI displaying significantly higher activation (uncorrected  $P < 0.001$ ) to color-naming than to word-naming trials, superimposed on a partially inflated left hemisphere of a normalized single subject T1 scan (segmentation and inflation was carried out with Brain VISA software; <http://brainvisa.info/index.html>). (B) Mean control-related LFC ROI activity (beta weights  $\pm$  SEM) for baseline and hypnotic conditions, in participants with low and high hypnotic susceptibility.

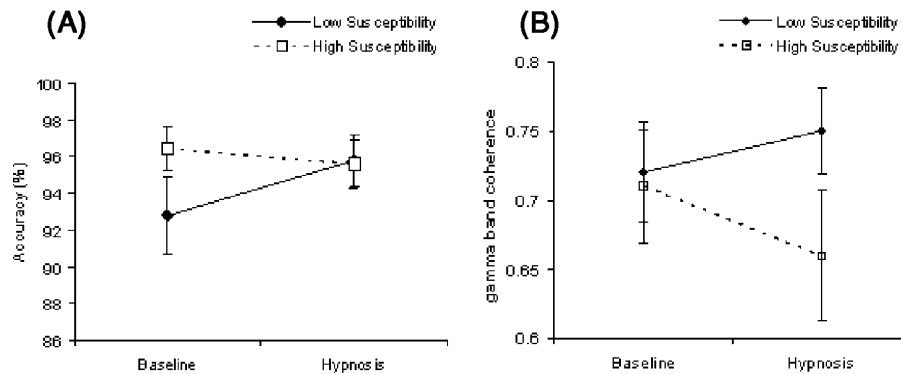


Fig. 4. Stroop accuracy data and EEG gamma band coherences. (A) Performance accuracy ( $\pm$ SEM) on the Stroop task for low and high susceptible hypnotic subjects, at baseline and in hypnosis. (B) Mid- to left frontal EEG gamma band coherence values following high conflict trials, in low and high susceptible hypnotic participants, at baseline and in hypnosis.

in  $2 \times 2 \times 5$  condition by group by frequency band ANOVAs. While there were no effects on coherence for low conflict, or moderate conflict conditions, in the high conflict condition, a hypnosis  $\times$  band  $\times$  susceptibility interaction effect was evident ( $F[4,15] = 2.51$ ;  $P < 0.05$ ). Inspection of the data revealed that the interaction stemmed from the fact that gamma band coherence decreased in highly susceptible subjects, but increased in subjects with low susceptibility between baseline and hypnotic states (see Fig. 4B). In order to confirm that these results were specific to midline–left frontal connectivity, the same analyses were carried out for the right frontal lateral site (F4 electrode). There were no significant results obtained from this analysis. In summary, the EEG coherence analyses have supported the suggestion of a decreased functional connectivity between frontal midline and left frontal regions in highly susceptible subjects after hypnotic induction.

## Discussion

Investigating executive attention processes in relation to hypnosis and hypnotic susceptibility, we employed a Stroop paradigm that successfully produced interference effects on reaction time and accuracy rates, resulting in significantly different levels of low, moderate, and high response conflict, replicated across fMRI and EEG recording sessions. In lieu of these behavioral effects, we demonstrated that frontal midline activation of the dorsal anterior cingulate cortex covaried positively with the level of response conflict, confirming results from previous studies (Botvinick et al., 1999; Carter et al., 2000; Casey et al., 2000). Importantly, the identification of conflict-responsive ACC loci allowed us to compare neural efficiency in conflict resolution at baseline and following hypnosis, between groups of subjects with low or high hypnotic susceptibility. Conflict-related ACC activation displayed an interaction effect (see Fig. 2), as this activation increased significantly from baseline to hypnosis in the highly susceptible subjects only, leading to significantly more conflict-related ACC activation in highly susceptible subjects than in subjects with low susceptibility after hypnotic induction. These data were obtained under conditions of equal performance levels between the experimental groups, and can therefore not be accounted for by differential task difficulty. This lack of behavioral effects can likely be attributed to the much longer inter-stimulus intervals employed in the current experiment in comparison to

previous studies that have documented such effects (e.g., Jamieson and Sheehan, 2004; Kaiser et al., 1997; see also Nordby et al., 1999). These results are directly supportive of both the proposition that differences in executive attention processes mediate the stable trait variable of hypnotic susceptibility, and that executive function is impaired in highly susceptible individuals following hypnosis (Crawford and Gruzelier, 1992; Gruzelier, 1990, 1998, 2004; Hilgard, 1965, 1977; Jamieson and Sheehan, 2004; Woody and Bowers, 1994). The results do not support the notion that hypnosis in highly susceptible subjects represents a state of highly focused attention (Barber, 1960; Spiegel, 2003; Tellegen and Atkinson, 1974).

But what exactly happened to the highly susceptible participants, at the neural level? Control-related activation in the inferior frontal gyrus of the left LFC did not distinguish between baseline and hypnotic conditions or low and high hypnotic susceptibility (see Fig. 3). Thus, it appears that the increased conflict-related ACC activation in highly susceptible subjects in the hypnosis condition was not accompanied by a concurrent strategic increment in cognitive control, as would be expected under normal circumstances (Botvinick et al., 2001, 2004; Kerns et al., 2004). These fMRI data suggest the possibility of a decoupling of conflict-monitoring and cognitive control function in highly susceptible subjects after hypnotic induction, corresponding to a breakdown in the functional integration of two key components of the frontal attentional control system. It should be noted that the control-related focus identified in the present study was located more caudal and inferior to regions reported in previous studies that explicitly dissociated control-related from conflict-related activity (Egner and Hirsch, 2005; MacDonald et al., 2000). The current control-related activation is likely to be reflective of sustained implementation of task-specific processing requirements, as compared to adaptation to changes in conflict on a trial-by-trial basis (Egner and Hirsch, 2005; MacDonald et al., 2000). Its locus in the GF<sub>i</sub> indeed corresponds very closely to previous studies that have interpreted this region as imposing an attentional set geared at selecting task-relevant information at a conceptual/semantic level rather than the response-level (Brass and von Cramon, 2004; Milham et al., 2001; Zysset et al., 2001). Similarly, this left inferior frontal region has been conceptualized as underlying the top-down modulation of contention scheduling (Shallice, 2002) and contextual control processes (Koechlin et al., 2003), such as associating external cues with the appropriate choice of action (Passingham et al., 2000).

In support of the notion of a decoupling between conflict monitoring and control processes in hypnosis, the results obtained from analyzing functional connectivity, by means of EEG coherence measures, show that low and high susceptibility groups were distinguished by baseline-to-hypnosis changes in gamma band coherence between frontal midline and left lateral scalp sites. In participants with low susceptibility, gamma band coherence showed an increase, whereas in highly susceptible participants, it was found to be decreased, and this pattern was mirrored in the behavioral accuracy data (see Fig. 4), recalling previous findings of occasional performance improvement in subjects with low susceptibility following hypnotic induction (see Gruzelier, 1998, 2000; Gruzelier et al., 2002). Coherence in the gamma frequency range has frequently been associated with functional connectivity processes, such as the binding of various features of a stimulus in primary sensory cortices (Tallon-Baudry and Bertrand, 1999) or of different stimulus classes paired during associative learning tasks (Miltner et al., 1999). Furthermore, it has been previously reported that hypnosis interfered with a (non-hypnotic) relationship between subjective ratings of pain and the amplitude of gamma oscillations localized to the ACC with low-resolution tomography (LORETA) (Croft et al., 2002).

Thus, together these data support the proposal that hypnosis in highly susceptible individuals may be underpinned by a profound alteration of frontal lobe functions (Gruzelier, 1998, 2000). We note that this was specific to regions engaged by the cognitive challenges posed by the Stroop task, and no background accompaniment of hypnosis was detected elsewhere in the brain. More specifically we found a fractionation between ACC conflict monitoring and left LFC cognitive control processes (Jamieson and Sheehan, 2004). This complements previous reports of altered frontal EEG gamma coherence following hypnosis in highly susceptible subjects (Jamieson et al., 2003). Interestingly, Kaiser and colleagues (reported in Gruzelier, 1998) have also found that EEG alpha coherence was altered during hypnosis specifically within the left frontal lobe during a Stroop-like task. However, in that experiment (Kaiser et al., 1997), a further dissociation between ACC processes measured by error-related negativity (*NE*, conflict monitoring) and error-related positivity (*PE*, context updating) was disclosed in the more highly susceptible participants. The *NE* was unaffected by hypnosis whereas hypnosis abolished the *PE*. This was interpreted as hypnosis influencing the impact of motivational factors and so compromising Stroop performance (Gruzelier, 1998; Kaiser et al., 1997).

In conclusion, hypnotic susceptibility interacted with hypnotic induction in the effects on conflict-related ACC activity during a Stroop task. Following hypnosis, highly susceptible subjects exhibited significantly increased conflict-related ACC activation, indicative of decreased attentional efficiency, as compared to baseline, and as compared to subjects with low hypnotic susceptibility. This increased conflict, however, was not accompanied by corresponding adjustments in the allocation of cognitive control. High conflict events in the hypnotic condition in highly susceptible participants were furthermore characterized by a relatively decreased functional connectivity between midline and left lateral frontal lobe sites. These data are compatible with the proposal that trait differences in response to hypnotic suggestions (hypnotic susceptibility) are mediated by differences in attentional processing, and that hypnosis in highly susceptible subjects is underpinned by a functional decoupling of response conflict monitoring and cognitive control processes. These results contra-

dict the assumption that hypnosis is directly associated with highly focused attention in highly susceptible hypnotic subjects. However, we suggest that the hypnosis-related decoupling of executive functions is highly malleable, as in the presence of strategic attentional instructions, superior conflict resolution can be achieved by highly susceptible participants (Raz et al., 2002, 2003; Sheehan et al., 1988). The challenge of these results is now to delineate the mechanism(s) underlying the fractionation of executive functions, which may offer profound insights into attentional processes in the normal and disordered brain. The results contribute to a growing body of evidence from other investigators implicating the ACC in the hypnotic process and importantly in its analgesic actions (Crawford et al., 1998; De Pascalis et al., 2001; Derbyshire et al., 2004; Faymonville et al., 2000; Horton et al., 2004; Rainville et al., 1999; Ray et al., 2002; Wik et al., 1999).

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### References

- Aikins, D., Ray, W.J., 2001. Frontal lobe contributions to hypnotic susceptibility. *Int. J. Clin. Exp. Hypn.* 49, 320–329.
- Barber, T.X., 1960. The necessary and sufficient conditions for hypnotic behavior. *Am. J. Clin. Hypn.* 3, 31–42.
- Bench, C.J., Frith, C.D., Grasby, P.M., Friston, K.J., Paulesu, E., Frackowiak, R.S.J., Dolan, R.J., 1993. Investigations of the functional anatomy of attention using the Stroop test. *Neuropsychologia* 31, 907–922.
- Botvinick, M., Nystrom, L.E., Fissell, L., Carter, C.S., Cohen, J.D., 1999. Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature* 402, 179–181.
- Botvinick, M., Braver, T.S., Barch, D.M., Carter, C.S., Cohen, J.D., 2001. Conflict monitoring and cognitive control. *Psychol. Rev.* 108, 624–652.
- Botvinick, M.M., Cohen, J.D., Carter, C.S., 2004. Conflict monitoring and anterior cingulate cortex: an update. *Trends Cogn. Sci.* 8, 539–546.
- Brass, M., von Cramon, D.Y., 2004. Selection for cognitive control: a functional magnetic resonance imaging study on the selection of task-relevant information. *J. Neurosci.* 24, 8847–8852.
- Brett, M., Anton, J.L., Valabregue, R., Poline, J.B., 2002. Region of interest analysis using an SPM toolbox. 8th International Conference on Functional Mapping of the Human Brain, June 2–6, 2002, Sendai, Japan. Available on CD-ROM in *NeuroImage*, Vol 16, No 2.
- Carter, C.S., Mintun, M., Cohen, J.D., 1995. Interference and facilitation effects during selective attention: an H215O PET study of Stroop task—Performance. *Neuroimage* 2, 264–272.
- Carter, C.S., MacDonald, A.M., Botvinick, M., Ross, L.L., Stenger, V.A., Noll, D., Cohen, J.D., 2000. Parsing executive processes: strategic vs. evaluative functions of the anterior cingulate cortex. *Proc. Natl. Acad. Sci. U. S. A.* 97, 1944–1948.
- Casey, B.J., Thomas, K.M., Welsh, T.F., Badgaiyan, R.D., Eccard, C.H., Jennings, J.R., Crone, E.A., 2000. Dissociation of response conflict, attentional selection, and expectancy with functional magnetic resonance imaging. *Proc. Natl. Acad. Sci. U. S. A.* 97, 8728–8733.



- Cikurel, K., Gruzelier, J., 1990. The effect of an active-alert hypnotic induction on lateral asymmetry in haptic processing. *Br. J. Exp. Clin. Hypn.* 7, 17–25.
- Crawford, H.J., Gruzelier, J.H., 1992. A midstream view of the neurophysiology of hypnosis: recent research and future directions. In: Fromm, E., Nash, M. (Eds.), *Contemporary Hypnosis Research*. Guilford Press, New York, USA, pp. 227–266.
- Crawford, H.J., Horton, J.E., Harrington, G.C., Vendemia, J.M.C., Plantec, M.B., Jung, S., et al., 1998. Hypnotic analgesia (disattending pain) impacts neuronal network activation: an fMRI study of noxious somatosensory TENS stimuli. *Neuroimage* 7, S436.
- Croft, R.J., Barry, R.J., 2000. EOG-correction of blinks with saccade coefficients: a test and revision of the aligned-artefact average solution. *Clin. Neurophysiol.* 111, 440–443.
- Croft, R.J., Williams, J.D., Haenschel, C., Gruzelier, J.H., 2002. Pain perception, hypnosis and 40 Hz oscillations. *Int. J. Psychophysiol.* 46, 101–108.
- De Pascalis, V., Magurano, M.R., Bellusci, A., Chen, A.C., 2001. Somatosensory event-related potential and autonomic activity to varying pain reduction cognitive strategies in hypnosis. *Clin. Neurophysiol.* 112, 1475–1485.
- Derbyshire, S.W., Whalley, M.G., Stenger, V.A., Oakley, D.A., 2004. Cerebral activation during hypnotically induced and imagined pain. *Neuroimage* 23, 392–401.
- Egner, T., Hirsch, J., 2005. The neural correlates and functional integration of cognitive control in a Stroop task. *Neuroimage* 24, 539–547.
- Faymonville, M.E., Laureys, S., Degueldre, C., DelFiore, G., Luxen, A., Franck, G., Lamy, M., Maquet, P., 2000. Neural mechanisms of antinociceptive effects of hypnosis. *Anesthesiology* 92, 1257–1267.
- Friston, K.J., Holmes, A.P., Worsley, K.J., Poline, J.-B., Frith, C.D., Frackowiak, R.S.J., 1995. Statistical parametric maps in functional imaging: a general linear approach. *Hum. Brain Mapp.* 2, 189–210.
- Genovese, C.R., Lazar, N.A., Nichols, T., 2002. Thresholding of statistical maps in functional neuroimaging using the false discovery rate. *Neuroimage* 15, 870–878.
- Gruzelier, J.H., 1990. Neurophysiological investigations of hypnosis: cerebral laterality, and beyond. In: Van Dyck, R., Spinhoven, P.H., Van der Does, A.J.W. (Eds.), *Hypnosis: Theory, Research, and Clinical Practice*. Free University Press, pp. 38–51.
- Gruzelier, J.H., 1998. A working model of the neurophysiology of hypnosis: a review of the evidence. *Cont. Hypn.* 15, 3–21.
- Gruzelier, J.H., 2000. Redefining hypnosis: theory, methods and integration. *Cont. Hypn.* 17, 51–70.
- Gruzelier, J.H., 2004. Neurophysiologische Erörterung der ungenügenden Effekte der Hypnose unter besonderer Berücksichtigung der Bühnen-Hypnose. *Hypn. Kognit.* 21, 225–259.
- Gruzelier, J.H., Brow, T.D., 1985. Psychophysiological evidence for a state theory of hypnosis and susceptibility. *J. Psychosom. Res.* 29, 287–302.
- Gruzelier, J.H., Warren, K., 1993. Neuropsychological evidence for reductions on left frontal tests with hypnosis. *Psychol. Med.* 23, 93–101.
- Gruzelier, J.H., Brow, T.D., Perry, A., Rhonder, J., Thomas, M., 1984. Hypnotic susceptibility: a lateral predisposition and altered cerebral asymmetry under hypnosis. *Int. J. Psychophysiol.* 2, 131–139.
- Gruzelier, J.H., Allison, J., Conway, A., 1988. A psychophysiological differentiation between hypnosis and the simulation of hypnosis. *Int. J. Psychophysiol.* 6, 331–338.
- Gruzelier, J.H., Gray, M., Horn, P., 2002. The involvement of frontally modulated attention in hypnosis and hypnotic susceptibility: cortical evoked potential evidence. *Cont. Hypn.* 19, 179–189.
- Halligan, P.W., Athwal, B.S., Oakley, D.A., Frackowiak, R.S.J., 2000. Imaging hypnotic paralysis: implications for conversion hysteria. *Lancet* 355, 986–987.
- Hilgard, E.R., 1965. *Hypnotic Susceptibility*. Harcourt, Brace, and World, New York, USA.
- Hilgard, E.R., 1977. *Divided Consciousness: Multiple Controls in Human Thought and Action*. Wiley Interscience, New York, USA.
- Horton, J.E., Crawford, H.J., Harrington, G., Downs III, J.H., 2004. Increased anterior corpus callosum size associated positively with hypnotizability and the ability to control pain. *Brain* 127, 1741–1747.
- Jamieson, G.A., Sheehan, P.W., 2002. A critical evaluation of the relationship between sustained attentional abilities and hypnotic susceptibility. *Cont. Hypn.* 19, 62–74.
- Jamieson, G.A., Sheehan, P.W., 2004. An empirical test of Woody and Bower's dissociated control theory of hypnosis. *Int. J. Clin. Exp. Hypn.* 52, 232–249.
- Jamieson, G.A., Zech, T.F., Hasegawa, H., Burgess, A.P., Bennion, A., Gruzelier, J.H., 2003. Dissociated control in hypnosis: evidence from changes in cortical gamma band coherence. 33rd Annual Congress of the Australian Society for Hypnosis September 11–September 16 2003, Gold Coast, Australia.
- Jutai, J., Gruzelier, J.H., Golds, J., Thomas, M., 1993. Bilateral auditory-evoked potentials in conditions of hypnosis and focused attention. *Int. J. Psychophysiol.* 15, 167–176.
- Kaiser, J., Barker, R., Haenschel, C., Baldeweg, T., Gruzelier, J.H., 1997. Hypnosis and event-related potential correlates of error processing in a Stroop-type paradigm: a test of the frontal hypothesis. *Int. J. Psychophysiol.* 27, 215–222.
- Kallio, S., Revonsuo, A., Hamalainen, H., Markela, J., Gruzelier, J.H., 2001. Anterior brain functions and hypnosis: a test of the frontal hypothesis. *Int. J. Clin. Exp. Hypn.* 49, 95–108.
- Kerns, J.G., Cohen, J.D., MacDonald, A.W., Cho, R.Y., Stenger, V.A., Carter, C.S., 2004. Anterior cingulate conflict monitoring and adjustments in control. *Science* 303, 1023–1026.
- Koechlin, E., Ody, C., Kouneiher, F., 2003. The architecture of cognitive control in the human prefrontal cortex. *Science* 302, 1181–1185.
- Kosslyn, S.M., Thompson, W.L., Costantini-Ferrando, M.F., Alpert, N.M., Spiegel, D., 2000. Hypnotic visual illusion alters color processing in the brain. *Am. J. Psychiatry* 157, 1279–1284.
- Lynn, S.J., Kirsch, I., Barabasz, A., Cardena, E., Patterson, D., 2000. Hypnosis as an empirically supported clinical intervention: the state of the evidence and a look at the future. *Int. J. Clin. Exp. Hypn.* 48, 239–259.
- MacDonald, A.W., Cohen, J.D., Stenger, V.A., Carter, C.S., 2000. Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science* 288, 1835–1838.
- MacLeod, C.M., 1991. Half a century of research on the stroop effect: an integrative review. *Psychol. Bull.* 109, 163–203.
- Maldjian, J.A., Laurienti, P.J., Kraft, R.A., Burdette, J.H., 2003. An automated method for neuroanatomic and cytoarchitectonic atlas-based interrogation of fMRI data sets. *Neuroimage* 19, 1233–1239.
- Milham, M.P., Banich, M.T., Webb, A., Barad, V., Cohen, N.J., Wszalek, T., Kramer, A.F., 2001. The relative involvement of anterior cingulate and prefrontal cortex in attentional control depends on the nature of the conflict. *Brain Res. Cog. Brain Res.* 12, 467–473.
- Miltner, W.H., Braun, C., Arnold, M., Witte, H., Taub, E., 1999. Coherence of gamma-band EEG activity as a basis for associative learning. *Nature* 397, 434–436.
- Nordby, H., Hugdahl, K., Jasiukaitis, P., Spiegel, D., 1999. Effects of hypnotizability on performance of a Stroop task and event-related potentials. *Percept. Mot. Skills* 88, 819–830.
- Passingham, R.E., Toni, I., Rushworth, M.F.S., 2000. Specialisation within the prefrontal cortex: the ventral prefrontal cortex and associative learning. *Exp. Brain Res.* 133, 103–113.
- Rainville, P., Duncan, G.H., Price, D.D., Carrier, B., Bushnell, M.C., 1997. Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science* 277, 968–971.
- Rainville, P., Hofbauer, R.K., Paus, T., Duncan, G.H., Bushnell, M.C., Price, D.D., 1999. Cerebral mechanisms of hypnotic induction and suggestion. *J. Cogn. Neurosci.* 11, 110–125.
- Rainville, P., Hofbauer, R.K., Bushnell, M.C., Duncan, G.H., Price, D.D., 2002. Hypnosis modulates activity in brain structures involved in the regulation of consciousness. *J. Cogn. Neurosci.* 14, 887–901.

- Ray, W.J., Keil, A., Mikuteit, A., Bongartz, W., Elbert, T., 2002. High resolution EEG indicators of pain responses in relation to hypnotic susceptibility and suggestion. *Biol. Psychol.* 60, 17–36.
- Raz, A., Shapiro, T., 2002. Hypnosis and neuroscience: a crosstalk between clinical and cognitive research. *Arch. Gen. Psychiatry* 59, 85–90.
- Raz, A., Shapiro, T., Fan, J., Posner, M.I., 2002. Hypnotic suggestion and the modulation of Stroop interference. *Arch. Gen. Psychiatry* 59, 1155–1161.
- Raz, A., Landzberg, K.S., Schweizer, H.R., Zephrani, Z., Shapiro, T., Fan, J., Posner, M.I., 2003. Posthypnotic suggestion and the modulation of Stroop interference under cycloplegia. *Conscious. Cogn.* 12, 332–346.
- Richeson, J.A., Baird, A.A., Gordon, H.L., Heatherton, T.F., Wyland, C.L., Trawalter, S., Shelton, J.N., 2003. An fMRI investigation of the impact of interracial contact on executive function. *Nat. Neurosci.* 6, 1323–1328.
- Ridderinkhof, K.R., Ullsperger, M., Crone, E.A., Nieuwenhuis, S., 2004. The role of the medial frontal cortex in cognitive control. *Science* 306, 443–447.
- Shallice, T., 2002. Fractionation of the supervisory system. In: Stuss, D.T., Knight, R.T. (Eds.), *Principles of Frontal Lobe Function*. Oxford University Press, pp. 261–277.
- Sheehan, P.W., Donovan, P.B., MacLeod, C.M., 1988. Strategy manipulation and the Stroop effect in hypnosis. *J. Abnorm. Psychol.* 97, 455–460.
- Shor, R.E., Orne, E.C., 1962. *The Harvard Group Scale of Hypnotic Susceptibility, Form A*. Consulting Psychologists Press, Palo Alto, USA.
- Spiegel, D., 2003. Negative and positive visual hypnotic hallucinations: attending inside and outside. *Int. J. Clin. Exp. Hypn.* 51, 130–146.
- Stroop, J.R., 1935. Studies of interference in serial verbal reactions. *J. Exp. Psychol.* 18, 643–662.
- Szechtman, H., Woody, E., Bowers, K.S., Nahmias, C., 1998. Where the imaginal appears real: a positron emission topography study of auditory hallucinations. *Proc. Natl. Acad. Sci. U. S. A.* 95, 1956–1960.
- Tallon-Baudry, C., Bertrand, O., 1999. Oscillatory gamma activity in humans and its role in object representation. *Trends Cogn. Sci.* 3, 151–162.
- Tellegen, A., Atkinson, G., 1974. Openness to absorbing and self-altering experiences: absorption, a trait related to hypnotic susceptibility. *J. Abnorm. Psychology* 83, 268–277.
- Ullsperger, M., von Cramon, D.Y., 2001. Subprocesses of performance monitoring: a dissociation of error processing and response competition revealed by event-related fMRI and ERPs. *NeuroImage* 14, 1387–1401.
- Weitzenhoffer, A.M., Hilgard, E.R., 1962. *Stanford Hypnotic Susceptibility Scale, Form C*. Consulting Psychologists Press, Palo Alto, USA.
- Wik, G., Fischer, H., Bragee, B., Finer, B., Fredrikson, M., 1999. Functional anatomy of hypnotic analgesia: a PET study of patients with fibromyalgia. *Eur. J. Pain* 3, 7–12.
- Woody, E., Bowers, K., 1994. A frontal assault on dissociated control. In: Lynn, S.J., Rhue, J.W. (Eds.), *Dissociation: Clinical and Theoretical Perspectives*. Guilford Press, New York, USA, pp. 52–79.
- Zysset, S., Muller, K., Lohmann, G., von Cramon, D.Y., 2001. Color–word matching Stroop task: separating interference and response conflict. *Neuroimage* 13, 29–36.