Inhibition and the right inferior frontal cortex

Adam R. Aron1,2, Trevor W. Robbins3 and Russell A. Poldrack2

1Department of Psychiatry, University of Cambridge, Cambridge CB2 2QQ, UK
2Department of Psychology, Franz Hall, Box 951563, University of California, Los Angeles, CA 90095, USA
3Department of Experimental Psychology, Downing Street, University of Cambridge, Cambridge CB2 3EB, UK

It is controversial whether different cognitive functions can be mapped to discrete regions of the prefrontal cortex (PFC). The localisationist tradition has associated one cognitive function – inhibition – by turns with dorsolateral prefrontal cortex (DLPFC), inferior frontal cortex (IFC), or orbital frontal cortex (OFC). Inhibition is postulated to be a mechanism by which PFC exerts its effects on subcortical and posterior-cortical regions to implement executive control. We review evidence concerning inhibition of responses and task-sets. Whereas neuroimaging implicates diverse PFC foci, advances in human lesion-mapping support the functional localization of such inhibition to right IFC alone. Future research should investigate the generality of this proposed inhibitory function to other task domains, and its interaction within a wider network.

Many researchers agree that the function of the prefrontal cortex (PFC) is broadly one of ‘executive control’ (i.e. the scheduling and optimizing of subsidiary processes implemented by posterior cortical and subcortical regions; see [1] for a review). There is, however, theoretical controversy over whether subregions of PFC are functionally differentiated. One influential view is that different areas within PFC perform the same operation (i.e. ‘working memory’) but for different sensory inputs [2] (but see [3]). A variant of the ‘working memory’ hypothesis is one which regards the PFC as providing top-down bias of posterior cortical and subcortical regions; see [4]. Accordingly, the PFC acts like the signalman at a railway junction; depending on the context, different incoming traffic gets directed towards different outcomes [1]. Another, complementary, view of PFC function is that it integrates events across time [5].

Meta-analysis of neuroimaging results suggests a localization of function to a network of PFC regions. It appears that, regardless of the particular contrast of tasks, there is regularity of (bilateral) activation of dorsolateral prefrontal cortex (DLPFC), inferior frontal cortex (IFC), and dorsal anterior cingulate cortex (ACC), but not other frontal regions [6]. This indicates a surprising sort of specialization of the PFC: a specific frontal network consistently recruited for solution of diverse cognitive problems. Although it is not disputed that memory is a fundamental function of the PFC, nor that most neuroimaging task comparisons activate the same set of PFC regions (often including bilateral DLPFC, IFC and ACC), recent advances suggest that the IFC, right-lateralized (Figure 1a), can be identified with a particular function. We review recent evidence from behavioural studies of patients with unilateral PFC lesions. Lesion studies, unlike neuroimaging, can establish which brain regions are necessary for cognition, and advances in lesion-mapping technology, using structural MRI, allow better lesion resolution. The evidence supplements classic monkey-lesion work [7,8], by showing that damage to the right IFC impairs independent measures of executive control by disrupting inhibition (specifically of responses and task-sets). This poses a challenge to alternative views concerning the localization of such inhibitory functions to DLPFC [9] or orbital frontal cortex (OFC) [10] (see [11] for a review).

The right IFC and inhibitory control

Historically, an important paradigm for studying executive control has been the Wisconsin Card Sorting Test (WCST). The subject sorts a series of cards on different dimensions such as colour, number and shape. Once the subject has established the currently appropriate rule (e.g. ‘sort successive cards by color’), the experimenter gives negative feedback, and the subject is required to change classification to another dimension. Patients with frontal cortical damage are notoriously bad at the change stage (see [12] for a review) – often explained by ‘perseveration’ of the previously appropriate rule. However, because the WCST is complex, requiring not just shifting – but hypothesis generation, memory, and so on – any component could be affected by lesion damage. Hence, researchers have used executive control paradigms that more effectively decompose cognitive components. Two such influential paradigms are response inhibition (see [13] for a review), and task-set switching (see [14] for a review). Damage to right IFC crucially affects performance in these paradigms, apparently by disrupting inhibition. Additionally, we review studies showing that wider areas of the right PFC are required for the suppression of memories and responses to visual or auditory distractors.

Response inhibition

Response inhibition is the cognitive process required to cancel an intended movement. It is tested using Go/No-Go and stop-signal tasks [13]. The subject is required to
perform speeded responses on Go trials (e.g. pressing a button in response to the letters Q, P, T) and to inhibit responding on (i) No-Go trials (e.g. to the letter X) or (ii) Stop trials (when a beep is sounded). For Go/No-Go tasks the index of inhibitory control is the number of errors a subject makes on No-Go trials (i.e. going when they should not). For stop-signal tasks, the index of inhibitory control is the duration of the stopping process, called the stop-signal reaction time (SSRT) [13]. In neuroimaging studies response inhibition consistently and especially activates a right-lateralized inferior frontal cortex (IFC) region (e.g. [15,16–21]), and this region (but not other regions of right or left PFC) was shown to be crucial by a neuropsychological study of patients with unilateral right-PFC damage [22]. The greater the damage to this region alone, the worse the response inhibition, as indexed by SSRT (Figure 1b,c). Lesions to a homologue of this region (the inferior prefrontal convexity; see Box 1) also impaired No-Go performance in monkeys [8], and it is noteworthy that problems with response inhibition have been widely documented in children and adults with a diagnosis of attention-deficit hyperactivity disorder (ADHD) (e.g. [23,24,25]). Structural MRI (e.g. [26,27]), functional MRI [28,29] and EEG (e.g. [30]) evidence strongly suggests that a right-frontal (especially inferior frontal) deficit underlies impaired response inhibition in this group.

**Task-set switching**

Changing from performing one task to another exercises executive control. A precise measure is given by the task-set switching paradigm (for a review see [14]), which measures switching in terms of the time taken to switch compared with repeating a task (the ‘switch cost’). In brief, subjects perform a series of trials of task A and then switch to performing a series of task B. For each subject, the switch cost is computed by subtracting the average reaction time (RT) of non-switch trials from the average RT of switch trials. Intuitively, it is clear that having to

switch task requires configuring a new attentional and response set (e.g. getting ready to take up your cup once you have finished pouring the coffee). Apart from taking time to load new stimulus–response (S–R) mappings and choosing which attributes to attend to, changing tasks might require the inhibition of competing S–R links specified by the now inappropriate task, or even the inhibition of the entire task [31].

Converging evidence suggests the right frontal cortex might subserve inhibitory processes underlying switching. Neuroimaging studies of the WCST [32–34], reversal learning (e.g. [32,35]) and task-set switching (e.g. [36,37–39]) have especially reported activation of DLPFC and right IFC (although sometimes there is co-activation of left frontal cortex). A direct neuroimaging comparison of a form of switching (the WCST) and response inhibition demonstrated a common locus in the right IFC [18]. A combined EEG/MRI study investigating Go/No-Go and Switch/Repeat factors suggested that the right IFC was responsible for ‘switching into a suppression mode’ [40]. Most persuasively of all, a study of patients with unilateral PFC damage demonstrated that the greater the damage to the right IFC, the greater the switch cost [41] (Figure 2a,b). This was not true for damage to any other region of right or left PFC. The switch deficit of these patients with right frontal damage appeared most consistent with impaired ability to suppress irrelevant responses or irrelevant task-sets on the switch trial relative to non-switch trials. In addition to being reliably correlated with the amount of damage to the right IFC, the switch cost was also reliably correlated with the SSRT measure of response inhibition (Figure 2c). This suggests disruption to a common mechanism underlying performance of the two independent tasks.

**Inhibition during memory retrieval**

In the course of daily life we often try to ‘push out of mind’ unpleasant events or memories. Such blocking of memory retrieval could be like overriding a pre-potent motor
response (for a review see [42]). Using a related paradigm, a recent neuroimaging study identified the neural systems involved in keeping unwanted memories out of mind [43]. Subjects were first trained on cue-target word pairs (e.g., ‘ordeal–roach’). Later they were shown only a cue word on each trial (e.g., ‘ordeal’) and depending on the color in which it was written, they were required to either respond (subvocally) with the target word or else to try to

![Figure 1](image1.png)

**Figure 1.** Cytoarchitectonic maps of the lateral surface of the human (a) and macaque monkey (b) prefrontal cortex. (Adapted from [81] with permission; only left hemisphere available.) In (b), the principal sulcus is demarcated by the mainly horizontal bold line. The arcuate sulcus is shown magnified in the inset. (c) Bilateral inferior frontal functional MRI activations associated with Wisconsin Card Sort shifting for monkey (top row) and humans (bottom row). (d) Same activations displayed on inflated surface reconstructions of human (left) and monkey (right) brains. (Adapted from [33] with permission; only left hemisphere available.) Yellow arrow (left): inferior frontal sulcus; green arrow (right): principal sulcus; blue arrow: arcuate sulcus.

In the monkey brain, the ventralmost part of the anterior bank of the lower limb of the arcuate sulcus (extending onto the adjacent ventrolateral prefrontal convexity, areas 45A and 45B in Figure 1b) has similar architectonic characteristics to human area 45 [81]. An area with architectonic features corresponding to area 44 in the human brain is found in the lower limb of the arcuate sulcus in monkeys (Figure 1b) [81]. Adequate comparison between species requires functional studies, for example, with fMRI. One such study used a modified version of the WCST to investigate cognitive set-shifting [33]. The most prominent shift-related activation of the PFC was found in the bilateral IFC across both species (Figure 1c,d).

The IFC is one of the most heavily connected regions of the PFC, receiving polymodal input from posterior cortical areas, and communicating heavily with other PFC regions [1]. It is one of the last brain regions to develop in both ontogeny and phylogeny [84]. Immature development of the IFC in children versus adults could explain significantly different functional activity for response inhibition [15]. Detailed neuroanatomical knowledge of this region, in tandem with better understanding of innervating catecholamine neurotransmitter systems, is likely to complement future functional studies.

**Box 1. Comparative anatomy and function of IFC in man and monkey**

The IFC (otherwise known as ‘ventrolateral’ PFC) in humans comprises Brodmann Areas 44 (pars opercularis), 45 (pars triangularis) and 47/12 (pars orbitalis) (Figure 1a) [81]. However, relating lesion damage and functional activation to any subregion of IFC must be performed with caution as the correspondence between sulcal landmarks and the underlying cytoarchitectonic areas (i.e. Brodmann Areas) is only approximate [82,83].

In the monkey brain, the ventralmost part of the anterior bank of the lower limb of the arcuate sulcus (extending onto the adjacent ventrolateral prefrontal convexity, areas 45A and 45B in Figure 1b) has similar architectonic characteristics to human area 45 [81]. An area with architectonic features corresponding to area 44 in the human brain is found in the lower limb of the arcuate sulcus in monkeys (Figure 1b) [81].

Adequate comparison between species requires functional studies, for example, with fMRI. One such study used a modified version of the WCST to investigate cognitive set-shifting [33]. The most prominent shift-related activation of the PFC was found in the bilateral IFC across both species (Figure 1c,d).

The IFC is one of the most heavily connected regions of the PFC, receiving polymodal input from posterior cortical areas, and communicating heavily with other PFC regions [1]. It is one of the last brain regions to develop in both ontogeny and phylogeny [84]. Immature development of the IFC in children versus adults could explain significantly different functional activity for response inhibition [15]. Detailed neuroanatomical knowledge of this region, in tandem with better understanding of innervating catecholamine neurotransmitter systems, is likely to complement future functional studies.

**Figure 2.** Disruption of task-switching by right inferior frontal cortex (IFC) damage. (a) Extent of damage to right IFC, but not other regions, correlated significantly with a reaction-time measure of the switch cost for 18 patients with right-frontal damage ($r = 0.82$, $P < 0.0001$ [41]). (b) An even stronger correlation was apparent between extent of damage to pars opercularis of IFC and switch cost ($r = 0.84$, $P < 0.0001$). (c) A statistically reliable correlation ($r = 0.59$, $P < 0.005$) between the same measure of switch cost and the response inhibition measure (SSRT), was reported for the same right frontal patients [22].

![Figure 2](image2.png)

TRENDS in Cognitive Sciences

www.sciencedirect.com
suppress the target word. Activation was compared during suppression and respond trials. Although the authors emphasized a DLPFC focus associated with inhibition of unwanted memories, activation foci were also found in bilateral IFC. Investigating the DLPFC focus alone, the authors found evidence that it interacts with the medial temporal lobe (MTL), a region crucial for memory, during attempts to suppress recollection.

Future lesion studies are required to establish which PFC regions are necessary for the inhibition of unwanted memories, and whether these might in fact overlap with the right IFC region. A recent lesion study [44] provided some support for this hypothesis using a ‘directed forgetting’ procedure. On each trial, the subject was given a single word, followed by an instruction to remember or forget. This led to a high level of recall for to-be-remembered items and a low level of recall for to-be-forgotten items. Patients with right (as opposed to left) frontal-lobe damage showed impairments for directed forgetting [44]; however the locus of lesion damage was not specified at greater resolution.

The above studies concern inhibitory mechanisms in long-term memory. Evidence also exists for right IFC involvement in working memory retrieval [45,46], perhaps also related to inhibition. There is also evidence for left IFC recruitment related to inhibitory mechanisms in working memory, specifically for resolving interference from previous trials [47,48]. In a common manipulation, subjects perform a test of item recognition: target letters are presented for storage followed, after a brief interval, by a probe letter that could match a target letter or not. On some trials, when the probe did not match a target letter, and required a ‘no’ response, the probe had matched a target letter of the previous trial, so on these trials a ‘yes’ response was prepotent and supposedly had to be inhibited. A patient with damage restricted to the left inferior and middle frontal gyrus showed a particularly large effect, reflecting increased interference in the prepotent response condition [49].

**Interference tasks and negative priming**

There exists other evidence for the role of right frontal cortex in cognitive inhibition. In a selective attention task requiring subjects to reach and touch targets but not distractors, an index of distractor suppression correlated reliably with lateral PFC damage in both hemispheres (albeit a region more diffuse than IFC alone) [50]. Increased distractibility as a consequence of lateral PFC damage has been demonstrated in auditory and antisaccade tasks in monkeys and humans [51–53]. Other neuropsychological studies have explored the phenomenon of negative priming — ostensibly reflecting the after-effects of inhibition: if a subject suppresses a response to a location or object on trial t, then responding to that object or location on trial t+1 is slower relative to responding to a novel object or location (for a review see [54]). Negative priming is reduced in patients with RF damage [55,56]. The requirement to overcome distraction or interference is also necessary for the Eriksen Flanker paradigm: comparison of incongruent trials (affording two potential responses) with congruent trials (affording one potential response) produces right IFC activation [15,57,58]. Finally, right-IFC activation is also reliably greater as a consequence of dual-task interference; that is, when the interval between one task and the next is short relative to when it is long [59]. One interpretation of this latter finding is that the IFC is recruited to suppress the second task until processing resources are liberated.

**Neurophysiological evidence**

Although we have argued from the above evidence that functional activations in the right IFC reflect a cognitive inhibitory mechanism and that lesions to this region in non-human primates and humans alike disrupt this mechanism, it is unclear how cognitive inhibition relates to inhibition in a neural sense (by ‘neural’ we mean the systems level rather than that of single neurons).

Evidence for systems-level inhibition underlying cognitive inhibition comes from monkey neurophysiology [60]. Electrical stimulation of PPC No-Go foci produced reduced electrical activity in motor cortex, concomitant with a cancelled manual response (Figure 3). One No-Go focus was within the principal sulcus, whereas another was in the ‘rostroventral corner’ of the PFC (in both hemispheres). Although this latter region is anterior to the prefrontal cortex in non-human primates and humans alike disrupt this mechanism, it is unclear how cognitive inhibition relates to inhibition in a neural sense (by ‘neural’ we mean the systems level rather than that of single neurons).

Other research has shown that electrical stimulation of frontal eye field neurons causes inhibition of saccade production, possibly through suppression of brainstem eye movement generators [61]. Most of the suppression sites were located deep within the anterior bank of the arcuate sulcus (i.e. partly overlapping with IFC; see Box 1, and figure 1 in [62]). Saccade inhibition has also been explored with the stop-signal paradigm (for a review see [62]). When a stop-signal is given, activity in frontal eye field saccade-generating neurons rapidly decays, within the SSRT, whereas that for fixation neurons rapidly increases. It is possible that some foci within monkey frontal eye field affect the balance between fixation and saccade production neurons in such a way as to cancel movement [61].

In humans, as noted above, PFC apparently interacts with posterior-cortical regions such as MTL during cognitive inhibition of unwanted memories [43]. It is moreover possible in the case of response inhibition, that the right PFC suppresses basal-ganglia output, perhaps via the subthalamic nucleus (STN). Recent research has shown that patients with deep-brain stimulation of the STN had significantly improved response inhibition relative to a group with stimulation of the thalamus (W. van den Wildenberg, PhD thesis, University of Amsterdam, 2003). Subthalamic nucleus stimulation increases firing of STN output neurons, which increases inhibition of thalamocortical projections [63].

The emerging picture suggests an interaction between right PFC and (i) basal-ganglia, (ii) primary motor regions and (iii) memory-related MTL, in implementing cognitive inhibition. It remains to be elaborated whether the PFC source of such cognitive inhibition is specifically the right IFC, and whether neural activity in this region is itself...
Some caveats should be mentioned. First, our review concerns mainly a specific sort of inhibition – that of responses and task-sets – measured using reaction-time methodology in humans. Although we predict that this right-IFC implemented inhibitory function might also apply in such domains as memory retrieval [74], affective-shifting [75,76] and attentional-set shifting [75], this remains to be demonstrated empirically. Although prior research [75] established a double dissociation in the marmoset between attentional-set shifting in lateral PFC and affective-shifting in the OFC, it is unclear whether any inhibitory component in those monkey tasks (having high learning requirements) is really analogous to stop-signal inhibition or inhibition in task-set switching. It is also unclear whether the cortex left undamaged in that study (lying between the lateral and orbital regions) could represent the marmoset homologue of the IFC: if so, that blocking of memory retrieval might also depend on this same region, and a wider prediction is that any task requiring cognitive suppression of responses, task-sets or memories will be affected by damage or momentary deactivation of this region. ‘Inhibition’, as we therefore define it means the ‘suppression of inappropriate responses, S-R mappings or task-sets when the context changes, and suppression of interfering memories during retrieval’. Future research could establish to what extent this usage overlaps with other mentions of frontal ‘inhibition’ such as the inhibition of psychomotor representations in the parietal lobe [65], inputs to the sensory cortices [66], motor channels of the basal-ganglia [67], reflexes [68], orienting of attention (inhibition of return) [69], perseveration in WCST [70] (and see [11,66,71] for a review).

Specificity of IFC inhibitory function and lateralization

Some caveats should be mentioned. First, our review concerns mainly a specific sort of inhibition – that of responses and task-sets – measured using reaction-time methodology in humans. Although we predict that this right-IFC implemented inhibitory function might also apply in such domains as memory retrieval [74], affective-shifting [75,76] and attentional-set shifting [75], this remains to be demonstrated empirically. Although prior research [75] established a double dissociation in the marmoset between attentional-set shifting in lateral PFC and affective-shifting in the OFC, it is unclear whether any inhibitory component in those monkey tasks (having high learning requirements) is really analogous to stop-signal inhibition or inhibition in task-set switching. It is also unclear whether the cortex left undamaged in that study (lying between the lateral and orbital regions) could represent the marmoset homologue of the IFC: if so, that blocking of memory retrieval might also depend on this same region, and a wider prediction is that any task requiring cognitive suppression of responses, task-sets or memories will be affected by damage or momentary deactivation of this region. ‘Inhibition’, as we therefore define it means the ‘suppression of inappropriate responses, S-R mappings or task-sets when the context changes, and suppression of interfering memories during retrieval’. Future research could establish to what extent this usage overlaps with other mentions of frontal ‘inhibition’ such as the inhibition of psychomotor representations in the parietal lobe [65], inputs to the sensory cortices [66], motor channels of the basal-ganglia [67], reflexes [68], orienting of attention (inhibition of return) [69], perseveration in WCST [70] (and see [11,66,71] for a review).

Specificity of IFC inhibitory function and lateralization

Some caveats should be mentioned. First, our review concerns mainly a specific sort of inhibition – that of responses and task-sets – measured using reaction-time methodology in humans. Although we predict that this right-IFC implemented inhibitory function might also apply in such domains as memory retrieval [74], affective-shifting [75,76] and attentional-set shifting [75], this remains to be demonstrated empirically. Although prior research [75] established a double dissociation in the marmoset between attentional-set shifting in lateral PFC and affective-shifting in the OFC, it is unclear whether any inhibitory component in those monkey tasks (having high learning requirements) is really analogous to stop-signal inhibition or inhibition in task-set switching. It is also unclear whether the cortex left undamaged in that study (lying between the lateral and orbital regions) could represent the marmoset homologue of the IFC: if so, that blocking of memory retrieval might also depend on this same region, and a wider prediction is that any task requiring cognitive suppression of responses, task-sets or memories will be affected by damage or momentary deactivation of this region. ‘Inhibition’, as we therefore define it means the ‘suppression of inappropriate responses, S-R mappings or task-sets when the context changes, and suppression of interfering memories during retrieval’. Future research could establish to what extent this usage overlaps with other mentions of frontal ‘inhibition’ such as the inhibition of psychomotor representations in the parietal lobe [65], inputs to the sensory cortices [66], motor channels of the basal-ganglia [67], reflexes [68], orienting of attention (inhibition of return) [69], perseveration in WCST [70] (and see [11,66,71] for a review).
region could implement an inhibitory mechanism that allows normal performance of attentional-shifts when OFC is damaged and normal performance of affective-shifts when lateral PFC is damaged. Second, we do not claim that the right IFC plays only an inhibitory role. This region is implicated in category learning [77], visuomotor conditional learning (reviewed in [78]), memory retrieval [46], and memory encoding [79]. Although some of these results could be interpreted in terms of inhibition (e.g. suppression of competing memories [46], or inhibition in selective attention [80]), it is likely that multiple other functions are implemented there. Third, although right-IFC (but not left-IFC) damage in humans crucially affects stop-signal inhibition [22] and task-switching [41] it is apparent that left IFC might play some role related to inhibition too. Neuroimaging studies of No-Go inhibition sometimes find bilateral IFC activation (e.g. [15,18,20]), as do studies of switching or shifting (e.g. [33,34,37]). Interference (inhibitory) effects in one working memory paradigm consistently activate left IFC [47,48], and the left IFC might even be crucial [49]. It remains to be established whether left IFC interacts with right IFC in inhibitory control or whether either hemisphere can implement inhibition, depending on the context (e.g. semantic load).

Lesion mapping methodology supports the localisationist hypothesis of PFC function

Demonstrating the crucial importance of the right IFC to cognitive inhibition represents an advance in the fractionation of PFC function and poses a challenge to such global hypotheses as ‘working memory function’ [2] or a ‘specialized frontal network’ [6]. It is unclear how those views could account for the specificity of the human neuropsychological evidence implicating the right IFC, and not other PFC regions, in two independent tasks measuring cognitive inhibition (Figures 1 and 2). Furthermore, it is difficult to reconcile those views with primate neurophysiology showing that stimulation of No-Go foci during Go trials suppresses electrical activity in the motor cortex concomitant with canceling a manual response (Figure 3) [60], or that stimulation of a monkey homologue of IFC during a saccade task canceled saccade production [61].

Recent advances in human lesion-mapping methodology, combined with cross-task comparisons, show that a common component – inhibition – is specifically implemented by the right IFC. Progress in understanding PFC function depends on converging neuroimaging, neuropsychological and electrophysiological cross-species methods, and on properly interpreting imaging activations/deactivations in terms of the underlying systems level (see also Box 2). Extending these methods should enable a richer understanding of executive control as a set of interacting psychological processes instantiated by discrete PFC regions.

Acknowledgements

The authors thank Luke Clark and Anthony Wagner for helpful comments on an earlier version of this manuscript.

References

3 Rushworth, M.F. et al. (1997) Ventral prefrontal cortex is not essential for working memory. J. Neurosci. 17, 4829–4838
21 Rubia, K. et al. (2003) Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. NeuroImage 20, 351–358


Guitton, D. et al. (1985) Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal-directed saccades. Exp. Brain Res. 58, 455–472


Sasaki, K. et al. (1989) Suppression of visually initiated hand movement by stimulation of the prefrontal cortex in the monkey. Brain Res. 495, 100–107


Milner, B. (1963) Effects of different brain lesions on card sorting. Arch. Neurol. 9, 90–100


78 Passingham, R.E. et al. (2000) Specialisation within the prefrontal cortex: the ventral prefrontal cortex and associative learning. Exp. Brain Res. 133, 103–113

---

**Endeavour**

the quarterly magazine for the history and philosophy of science

You can access Endeavour online either through your BioMedNet Reviews subscription or via ScienceDirect, where you’ll find a collection of beautifully illustrated articles on the history of science, book reviews and editorial comment.

featuring

‘Dr. Steinach coming to make old young!’: sex glands, vasectomy and the quest for rejuvenation in the roaring twenties by C. Sengoopta
Cheese mites and other delicacies: the introduction of test objects into microscopy by J. Schickore
An herbal El Dorado: the quest for botanical wealth in the Spanish Empire by P. De Vos
Zones of inhibition: interactions between art and science by K. Davies
Global science: the eruption of Krakatau by M. Döerries
Two pills, two paths: a tale of gender bias by M. Potts
Joseph Banks: Pacific pictures by P. Fara
and coming soon

Mr Blandowski misses out: discovery and loss of fish species in 19th century Australia by P. Humphries
The traffic and display of body parts in the early 19th century by S. Alberti and S. Chaplin
Exhibiting monstrosity: Chang and Eng, the ‘original’ Siamese twins by S. Mitchell
The ancient mariner and the transit of Venus at Hudson Bay by R. Griffin-Short
‘I got rhythm’: Gershwin and birth control in the 1930s by P. Viterbo
The market for Julia Pastrana by J. Browne and S. Messenger
Race mixing and science in the United States by P. Farber
Continental drift under the Third Reich by E. Buffetaut
The first president of the Royal Society by P. Fara
and much, much more . . .

Locate Endeavour in the BioMedNet Reviews collection (http://reviews.bmn.com) or on ScienceDirect (http://www.sciencedirect.com)