particularly when it is reduced, adjustments become necessary. Either global, reactive control is recruited, possibly by the ACC, allowing the organism to be responsive to any potentially relevant stimulus, or a proactive mode is engaged, possibly by the pre-SMA, which selectively primes appropriate task sets in a preparatory manner. As Hikosaka and Isoda acknowledge, ACC and pre-SMA coding for different modes of control can be complementary processes which ultimately produce a balance between effort and efficiency.

An important question is how, i.e., by which interactions, anatomical structures within the pMFC drive the above-mentioned variations in adaptive behavior. Recent evidence on close interactions with basal ganglia and lateral prefrontal cortex [2] provides suggestions on possible mechanisms. Validation of our extension of the concepts by Hikosaka and Isoda and those offered by the Dual Control Mechanisms theory [4] will require testing relationships between pMFC-mediated control recruitment and subsequent changes in task-specific processing.

**Letters**

**Action and the fallacy of the ‘internal’: Comment on Passingham et al**

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Passingham et al. [1] argue that the conceptual distinction between actions seemingly guided by events in the ‘external world’ (e.g. a flash of light) and the ‘internal world’ (e.g. a pang of hunger) must be reflected in an anatomical division in the brain that conventional neurophysiological experiments can capture. Although that could be the case, the problem is that the data that do not contradict their claim arguably cannot be said either to support or undermine it.

Focusing for reasons of economy and inferential power on disruptive studies, the finding of profound and selective modulation of externally-guided action when the medial frontal cortex is targeted [2–5] makes the authors’ thesis that this region is specialized for internally-guided action difficult to sustain. That their proposal is consistent with a great deal of other data is beside the point: so is a myriad of alternative explanations that the brain is complex enough to accommodate [6].

Be that as it may, the fundamental issue is that the contrast between internally- and externally-guided actions is empirically intractable (as discussed at length in [7]). This is because the full gamut of relevant internal factors is actually impossible to parameterize. Experimenters might say that it is this internal factor that determines the action in a given case, but they cannot know what other internal factors come into play because they are – by definition – hidden from view.

When the monkey in one of Passingham et al.’s elegant studies [8] learned to raise its arm for a reward the investigators concluded that this was an example of an ‘internally’ generated action. However, any combination of internal and external factors might have been integrated in the process of deciding to act, as directed by the animal’s past experience, whether in the experiment or outside it. Even the absence of a sensory cue, where the animal would normally expect it, is itself no less a factor than its presence. The lack of such a visual signal, under such circumstances, is logically as much an external cue as a sensory event.

If both the number and the nature of the relevant internal factors are indeterminate, then a difference between the neural dependents of ‘externally’- and ‘internally-guided’ actions becomes uninterpretable: any aspect of the contrast, including differences in number and nature of the factors, could be the explanation. Thus the ‘externally-guided’ equivalent of the supposedly internal action in Passingham et al.’s study [8] is not, as the authors assert, a single sensory cue, but a multitude of sensory cues of unknown number, interdependence and strength of association with the action in question. Just as performance on a visual search task depends on the number and nature of the distractors as well as

**References**


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the target, so performance here cannot be indifferent to this wholly unquantifiable background. That it is invisible – being ‘internal’ – does not mean it can be simply ignored.

One objection to our argument here might be that the absence of other relevant internal factors could be confirmed by the subject’s report (although monkeys, of course, report nothing). Such an objection, however, presupposes an ‘internal world’ whose properties the subject can directly survey and report on, as one would survey and report on the ‘external world’.

Indeed, the authors have previously used such surveillance as a tool for brain cartography, purportedly identifying the neural substrate of a ‘mental object’ – the intention to act – by asking the subject to attend to his or her intention [9]. Attempting to identify the neural correlates of ‘mental objects’ in this manner presupposes that a purely conceptual separation of the ‘subject’ and ‘object’ of attention – here the ‘self’ and ‘intention’ – must also apply to the underlying neural organization. This conception of the relation between the mental and the physical is derived from Cartesian mind/body dualism whose faults – familiar since the time of Wittgenstein [10] – are sufficiently well known to make any neuroscientific theory built on it very difficult to defend (see [11,12]).

That our main criticisms are conceptual makes them no less relevant to empirical neuroscience: the value of data is limited if the conceptions in which they are couched are flawed.

References

Letters Response

Is it fallacious to talk of self-generated action?:
Response to Nachev and Husain

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Nachev and Husain present several objections [1] to our paper [2]. We reply first to the empirical objections. They argue that several papers are inconsistent with our proposal that medial frontal cortex is especially involved in self-generated action.

One of these papers [3] is on reaction times (RTs), showing that these are increased if the supplementary motor area (SMA) is cooled. We have previously reported that, so long as the timing of the cues is relatively predictable, there is activation of the SMA because subjects can learn to prepare [4]. In our paper we suggest that the effect of disruption of SMA activity is to interfere with this endogenous preparation, thus leading to longer RTs.

The other three papers are on response conflict [5–7]. Although conflict resolution is typically considered to be an endogenous process, Nachev and Husain’s point is that in these studies the response conflict or change of action is triggered by visual cues. However, the issues are: (i) whether the cues fully specify the new action and (ii) whether the subjects fail because of disruption of an endogenous process, namely, the resolution of response conflict. In fact, there is more medial activation when a switch cue does not fully specify the new response [8]; and, after an anterior cingulate lesion, there is a much greater increase in switching errors, if the switch is signalled by reduced reward rather than by an arrow [9]. Thus, we believe that the results of these studies are not inconsistent with our hypothesis that medial frontal cortex is especially involved in self-generated action.

We take our thesis to have greater explanatory power than the alternative offered by Nachev and Husain [1,10], although we accept that this is not the same as showing

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