Journal Club

Editor's Note: These short reviews of a recent paper in the *Journal*, written exclusively by graduate students or postdoctoral fellows, are intended to mimic the journal clubs that exist in your own departments or institutions. For more information on the format and purpose of the Journal Club, please see http://www.jneurosci.org/misc/ifa_features.shtml.

Endogenous Inhibition and the Neural Basis of "Free Won't"

Giovanni Mirabella[1,2]

[1]Department of Human Physiology and Pharmacology, University of Rome "La Sapienza," 00185 Rome, Italy, and [2]IRCCS Neuromed, Pozzilli, Isernia, Italy

Review of Brass and Haggard (http://www.jneurosci.org/cgi/content/full/27/34/9141)

Every day we perform thousands of actions and, although we might pay little attention to them, we take for granted that they are mostly generated by our will. In other words, we seem to believe in the existence of a "free will" as an inner causal agent of our behavior. Furthermore, our feeling of control over actions (i.e., motor awareness) contributes to the building of self-consciousness.

However, the available experimental evidence casts doubt on whether conscious processes cause actions. In fact, as first shown by Libet et al. (1983), a person's conscious experience of intending to act follows the onset of electrical activity that precedes voluntary action [the so-called readiness potential (RP)]. This period ranges from 200 to 500 ms. In particular, Haggard and Eimer (1999) found that awareness of intention correlates better with a later component of the RP, the lateralized readiness potential (LRP). LRP reflects the electrical activity over the motor cortex opposite to the limb that will move. Processes underlying the LRP are closely linked to movement selection. Because the LRP precedes the conscious experience of acting, Haggard and Eimer (1999) proposed that awareness of movement may arise from neural processes linked to the selection of an action to pursue a given goal. Subsequently, a network of cortical regions within the medial frontal cortex has been identified as the neural substrate for intentional actions. In particular, the supplementary motor area (SMA) and the pre-SMA have been implicated in the control of self-initiated acts (Lau et al., 2004; Nachev et al., 2005).

All of these studies lead to the idea that our sense of volition is a percept. In other words, it seems plausible that the motor system generates a movement on the basis of its several inputs and, subsequently, some premotor processes produce the subjective experience of willing to execute that action, which is perceived as being freely chosen (Hallett, 2007). But if free will is a percept, then are we really responsible for what we do? After all, we know that, at least to some extent, we can consciously exert control over our actions by inhibiting those we do not like. This is exactly Libet's (1985) solution to the problem of free will. In his view, because awareness of intention precedes movements by some hundred of milliseconds, there is still time to consciously withhold the upcoming action. This veto power, or "free won't," would therefore be the basis of our freedom. However, Libet (1985) did not find any identifiable neural correlate of this process.

In a study published recently in *The Journal of Neuroscience*, Brass and Haggard (2007) show that a specific area, the left dorsal frontomedian cortex (dFMC; Brodmann's area 9) is specifically associated with endogenous inhibition of intentional action.

To study internally generated inhibition, Brass and Haggard used a variant of the temporal judgment task developed by Libet et al. (1983). Fifteen healthy subjects were required either to make spontaneous key presses while watching a rotating clock hand (action condition) or, in some trials freely selected by the subject, to cancel the pending response at the last possible moment (inhibition condition) [Brass and Haggard (2007), their Fig. 1 (http://www.jneurosci.org/cgi/content/full/27/34/9141/F1)]. After each trial, the clock hand continued to rotate for a random time then stopped, and participants had to report the time when they first felt the will to move by using a trackball to position a cursor at the right time on the clock face. As a control, in separate blocks, participants were asked to indicate the timing of the onset of a tone, which was delivered with a delay similar to the reaction time of key presses measured in action trials. While subjects were performing the task, the blood oxygenation level-dependent (BOLD) signal was recorded using event-related functional magnetic resonance.

As expected, participants reported that, on average, they experienced the will to move 141 ms before pressing the key. The inhibition trials showed great interindividual differences, ranging from 28 to 62%. By contrasting the inhibition with the action condition three areas of the brain showed significant activation: the left dFMC, the left and right anterior ventral insula, and the left superior temporal

Received Nov. 2, 2007; revised Nov. 8, 2007; accepted Nov. 10, 2007.

I thank F. Eusebi for helpful discussions on this manuscript and for his support.

Correspondence should be addressed to Dr. Giovanni Mirabella, Department of Human Physiology and Pharmacology, University of Rome "La Sapienza," Piazzale Aldo Moro 5, 00185 Rome, Italy. E-mail: giovanni.mirabella@uniroma1.it.

DOI:10.1523/JNEUROSCI.4943-07.2007

 0270-6474/07/2713919-02$15.00/0

sulcus [Brass and Haggard (2007), their Fig. 2 (http://www.jneurosci.org/cgi/content/full/27/34/9141/F2), Table 1 (http://www.jneurosci.org/cgi/content/full/27/34/9141/T1)]. Crucially, the reverse contrast, that is the difference between action and inhibition trials, showed an increase in the BOLD signal in motor-related regions (e.g., the primary sensorimotor cortex), but not in those areas involved in the preparation of spontaneous actions, such as SMA and the pre-SMA (Lau et al., 2004; Nachev et al., 2005). Vice versa, by separately comparing action and inhibition conditions with the control condition a significant activation of both SMA and pre-SMA was revealed [Brass and Haggard (2007), their Fig. 3 (http://www.jneurosci.org/cgi/content/full/27/34/9141/F3)]. Because in the control condition subjects did not prepare or execute any voluntary movements, these results indicate that in both the action and inhibition trials they were generating self-initiating actions. Then, in just the latter condition, the intention to act was stopped. Brass and Haggard (2007) argued that the core of this process lies in the activation of dFMC. Two findings support this interpretation. First of all, the degree of activation of the dFMC in the inhibition condition was positively correlated with the proportion of inhibition trials of each subject. Second, activity in dFMC was negatively correlated with the activation of primary motor cortex. This last finding, plus the fact that dFMC is anatomically well segregated from brain regions involved in internally driven actions, supports the view that the role of dFMC in endogenous inhibition is to produce a specific top-down control signal which halts the neural processes translating intentions into acts.

But is the dFMC the only neural substrate involved in the inhibitory control of voluntary actions? The likely answer is no. Recently, Sumner et al. (2007) demonstrated that the supplementary eye field and the SMA are critically involved in automatic and unconscious effector-specific suppression of unwanted responses elicited by the surrounding context (of the eyes and the hand, respectively). Such inhibitory mechanisms prevent us from inappropriately and automatically reacting to environmental stimuli and therefore they represent a fundamental requisite for voluntary behavior. Perhaps the inhibitory control of volitional acts can be decomposed in several components and the dFMC might be specifically involved in the conscious suppression of self-initiated actions. Another topic that deserve study is the lateralization of the dFMC activity to the left hemisphere. Several studies have ascribed the inhibitory function to a network of regions belonging to the right and not to the left hemisphere (Aron et al., 2007). A possible explanation for this divergence comes form the fact that most such studies focused on inhibition triggered by external stimuli rather than inner volition.

In any case the study by Brass and Haggard (2007) provided the first clear evidence of the existence of a neural correlate of the "free won't," a key component of what we define as "self-control."

References

Aron AR, Behrens TE, Smith S, Frank MJ, Poldrack RA (2007) Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI J Neurosci 27:3743–3752.

Brass M, Haggard P (2007) To do or not to do: the neural signature of self-control. J Neurosci 27:9141–9145.

Haggard P, Eimer M (1999) On the relation between brain potentials and the awareness of voluntary movements. Exp Brain Res 126:128–133.

Hallett M (2007) Volitional control of movement: the physiology of free will. Clin Neurophysiol 118:1179–1192.

Lau HC, Rogers RD, Haggard P, Passingham RE (2004) Attention to intention. Science 303:1208–1210.

Libet B (1985) Unconscious cerebral initiative and the role of conscious will in voluntary action. Behav Brain Sci 8:529–566.

Libet B, Gleason CA, Wright EW, Pearl DK (1983) Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). The unconscious initiation of a freely voluntary act. Brain 106:623–642.

Nachev P, Rees G, Parton A, Kennard C, Husain M (2005) Volition and conflict in human medial frontal cortex. Curr Biol 15:122–128.

Sumner P, Nachev P, Morris P, Peters AM, Jackson SR, Kennard C, Husain M (2007) Human medial frontal cortex mediates unconscious inhibition of voluntary action. Neuron 54:697–711.