

And motion changes it all

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Demonstrating how specific motor signals modulate sensory processing in the rat vibrissal system, a new study in this issue shows that motor signals first attenuate and then amplify afferent sensory signals.

Although there has been much research into sensory systems and motor systems, these parallel streams of research usually do not intersect. Going to the scientific sessions for one of these topics after seeing the other one in a large neuroscience meeting often feels like moving to a parallel universe. One has to relearn the open questions, the assumptions and sometimes even the language. Of course, large conferences always include sessions with titles such as 'sensory-motor integration' to remind us that these two parallel universes must converge at some point. A paper by Lee *et al.*¹ in this issue is a fine example of what happens when they do.

Lee *et al.*¹ show that sensory processing under an active motor system (during exploration or active touch) is different than sensory processing under an inactive motor system (at rest), and they demonstrate how specific motor signals modulate sensory processing in a specific pathway of the rat vibrissal system¹. The vibrissal system of the rat is responsible for collecting tactile data from the nearby environment via a few tens of vibrissae (whiskers) that are located on each side of the snout. This extremely sensitive system is used for active exploration of the environment and for detection, localization and identification of objects by means of rhythmic fast movements called whisking. As motion and sensation are tightly related in this system, the interactions between motor and sensory signals in the brain are crucially important.

Lee *et al.*¹ studied the effects of motor cortex activation in anesthetized rats and of whisking in awake, behaving rats on one of the major sensory pathways of this system, the lemniscal pathway. The core of the lemniscal pathway ascends via 'barrelettes' of the principal trigeminal nucleus in the brainstem (PrV). This pathway runs along the medial lemniscus and through the core of 'barreloids' in the dorsomedial section of the thalamic ventral posterior medial nucleus (VPMdm). Ultimately, it reaches the 'barrels' in layer 4 of the primary somatosensory cortex (S1). S1 sends topographically aligned feedback connections to VPMdm via its layer 6 (Fig. 1).

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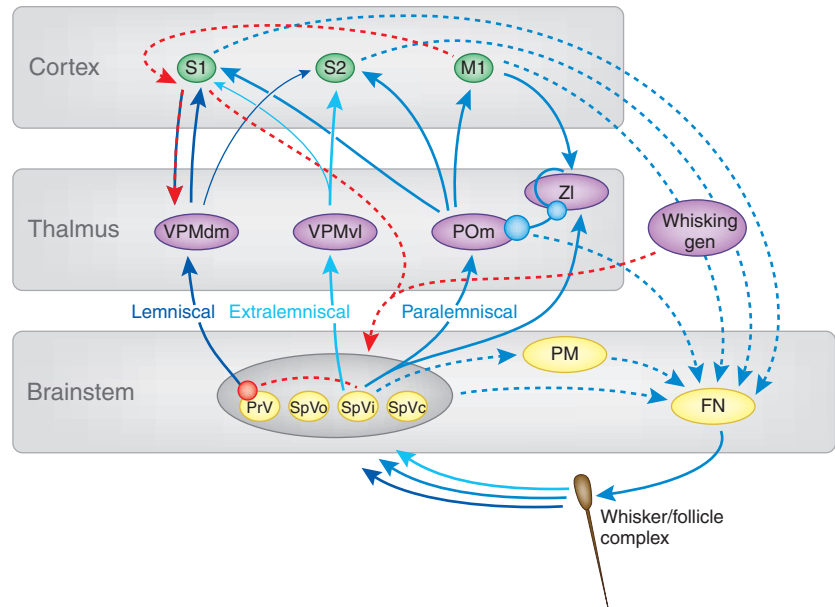


Figure 1 Sensory-motor scheme of the vibrissal system. Sensory pathways are depicted in solid blue colors. Top-down feedback and gating connections and cortico-cortical connections are not depicted, apart from those that were relevant to the study by Lee *et al.*¹. Motor pathways to the facial nucleus (FN) are represented by dashed blue lines (details are not shown and not all pathways are represented). Excitatory connections are depicted by arrows and inhibitory connections by circles. Motor gating pathways implied by the findings of Lee *et al.*¹ are plotted with red. ZI, zona incerta; PM, premotor nuclei; SpVo, nucleus oralis; SpVc, nucleus caudalis.

To look at the effects of active motor system in behaving rats, the authors measured multi-unit activity and local field potentials in VPMdm while electrically stimulating single whisker follicles. To do this in animals without their whiskers actually touching any objects, the authors trained water-deprived rats to stand on a small platform with their forelimbs on a bar, whisking in air before they were allowed to drink. Lee *et al.*¹ saw a suppression of VPMdm's responses when whisker follicles were stimulated during active whisking compared with stimulations during nonwhisking periods. However, when follicle stimulations were replaced by stimulations of the axons projecting from PrV to VPMdm, at the medial lemniscus, the effect of whisking on VPMdm's responses was reversed, and these responses were then facilitated during whisking.

These observations suggest that PrV is suppressed during whisking, while VPMdm

is facilitated. The primary suspect in suppressing PrV is a neighboring brainstem nucleus, the nucleus interpositus (SpVi). A set of direct inhibitory connections from the caudal part of SpVi to the PrV have also been reported recently². Lee *et al.*¹ tested whether SpVi indeed mediates PrV suppression by pharmacologically inactivating it while stimulating the follicle. Inactivating SpVi reversed the effects of whisking and facilitated VPMdm responses. These results suggest that the motor system suppresses PrV during whisking via neurons in SpVi.

SpVi receives direct projections from the somatosensory cortex but is unlikely to receive direct projections from the primary motor cortex (M1)³. Thus, M1-induced modulations ('gating') of PrV are probably mediated via the large pyramidal cells of layer 5 of S1 or S2, which also project to many motor stations and to the paralemniscal thalamic nucleus (the posteromedial nucleus, POM)⁴. Alternatively,

SpVi is modulated during whisking via connections from a whisking-generating system, a system whose components are not yet clear (Fig. 1). It is not known whether the cortico-SpVi projections specifically target the rostral (SpVir) or caudal (SpVic) part of SpVi. Moreover, the roles of nucleus caudalis and the cholinergic projections onto the trigeminal nuclei in mediating motor gating in the brainstem² are also unclear.

Clarifying the exact connections will help elucidate the relationships between motor gating of the lemniscal pathway and that of the palelemniscal pathway, which seems to take part in whisking control. In the latter, M1 possesses control of signal transfer through the POm via a disinhibition circuit in zona incerta⁵ (Fig. 1). POm and zona incerta are known to receive whisking-related sensory input from SpVir^{6,7}. SpVic projects via the extralemniscal pathway to the thalamus and conveys contact-time signals during active touch^{6,7}. Thus, if motor gating of PrV does not involve SpVir at all, then the modulations of lemniscal and extralemniscal inputs during active touch would seem largely independent of modulations of whisking-related palelemniscal inputs. In any case, the modulations of lemniscal and extralemniscal inputs appear to be coordinated in one way or another.

Reversing the effect of whisking on VPMdm responses by bypassing the PrV or inactivating SpVi strongly suggests that VPMdm is facilitated by motor activity. To reveal the nature of this facilitation, Lee *et al.*¹ measured neuronal responses of corticothalamic neurons in S1 layer 6 and thalamo-cortical neurons in VPMdm in lightly anesthetized rats. They found that focal and location-specific stimulation in M1 enhanced thalamocortical transmission in topographically aligned circuits (that is, circuits containing motor and sensory neurons that were affiliated with the same whiskers) but not across nonaligned circuits. Thus, the strong spatial specificity of the lemniscal pathway not only keeps sensory channels separated but facilitates independent control of entire single-whisker motor-sensory loops.

In such loops, firing of thalamo-cortical neurons in VPMdm appears to be continuously modulated by aligned feedback connections from layer 6 of S1 (ref. 8). Typically, layer 6 corticothalamic neurons show little or no activity. Lee *et al.*'s¹ results suggest that the corticothalamic neurons in S1 are under the continuous control of M1 neurons and that VPMdm neurons are indirectly affected by M1 via S1's corticothalamic feedback connections (Fig. 1). Although this scheme requires more direct experimental validation, its theoretical implications can already be considered. The

effectiveness of controlling the feedback, rather than the feedforward, branch in a closed loop (such as the thalamocortical one) depends on the specific architecture of the loop and on the magnitude of the modulations, and these can be directly tested in the lemniscal thalamocortical system. Furthermore, if M1-PrV gating is indeed mediated via S1 layer 5 corticobulbar neurons, then a hierarchical scheme of motor gating should be considered, in which a single signal from M1 to S1 is translated to multiple gating signals (to thalamus and brainstem) according to S1 processing requirements.

Thus, motor gating in the vibrissal system first attenuates and then amplifies sensory responses. What is the rationale behind this modulation? Lee *et al.*¹ suggest that such modulation, acting on the inherently nonlinear transfer functions in the sensory pathway, can filter out nonrelevant signals. Assuming that nonrelevant signals are of lower amplitudes than relevant signals, the attenuation of all afferent signals at the PrV can render nonrelevant signals below response threshold while leaving relevant signals above that threshold. Thalamocortical amplification is then used to amplify the relevant signals for further processing.

What are these nonrelevant signals? At a superficial level, re-afferent signals carrying information about whisking itself^{9,10} and signals related to nonaligned circuits might be considered as irrelevant for lemniscal processing. However, these signals are often relevant to lemniscal processing and thus should not always be filtered out. The characteristics of the lemniscal system make it ideal for processing of object details during active touch. Such details are encoded by complex spatiotemporal relationships of receptor activations when whiskers scan an object. The decoding of these signals must therefore involve signals reporting whisker motion and should often compare signals picked up by neighboring whiskers. Thus, motor gating in this system must be more sophisticated than a single operation, such as attenuation or amplification, and should probably depend on the context and the stimulus. The circuitry in the brainstem allows implementations of more complex gating signals via inhibitory and disinhibitory circuits across and in nuclei². Accordingly, it is conceivable that motor gating, as part of the processing loop, exerts different control during consequent palpation cycles according to the dynamics of the relevant sensory-motor loops.

This study by Lee *et al.*¹ joins two major lines of studies that collectively address the ways that a motion of sensory organs affects

sensory processing. Studies of motor-control signals and their effects on sensory processing inside of the brain^{11–13} form one line of inquiry, whereas studies on how sensor motion affects sensory acquisition outside of the brain via active sensing (reviewed in ref. 14) form another. The paper by Lee *et al.*¹ joins the first group and adds substantially to its exploration. Together, these two lines of research indicate that perception is not a sensory story but is instead a motor-sensory story. Moreover, given the fact that perception is usually not achieved by one motor-sensory cycle, but rather by a sequence of such cycles, perception would be an ongoing story of motor-sensory-motor loops. Sensor motion often makes sensory acquisition happen. Without eye movement, the world becomes uniformly gray; without sniffing, only the initial changes in the odor environment are sensed; and without finger or whisker motion, objects cannot be identified. Granted, relative changes in sensation and perception can be caused by external changes being applied over passive stationary sensory organs; we can see flashed images, smell a sudden puff of odor or feel a tennis ball hitting our head. However, the richness of perception lies in continuous 'palpation' of objects with our sensory organs. Moreover, the most flexible factor in sensory acquisition is the motor component, adapting to meet new challenges by modifying its scanning patterns in fractions of a second until optimal conditions for sensory processing are created. As understanding that motor control is an integral part of sensation is accelerated, it is hoped that sensation-targeted motor control¹⁵ will be subjected to theoretical and experimental studies in the future.

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