Editor's Note: These short, critical reviews of recent papers in the *Journal*, written exclusively by graduate students or postdoctoral fellows, are intended to summarize the important findings of the paper and provide additional insight and commentary. For more information on the format and purpose of the Journal Club, please see http://www.jneurosci.org/misc/ifa_features.shtml.

Occipital–Parietal Network Prepares Reflexive Saccades

Masayuki Watanabe, Masahiro Hirai, Robert A. Marino, and Ian G. M. Cameron

Centre for Neuroscience Studies, Queen's University, Kingston, Ontario K7L 3N6, Canada

Review of Hamm et al.

The reaction times of saccadic eye movements vary considerably even when they are initiated in response to identical visual stimuli. This variability of saccade reaction times has been studied extensively as a probe for cognitive brain functions because it depends on cognitive demands imposed by psychophysical paradigms. The distribution of saccade reaction times is usually unimodal. However, in some behavioral situations, it becomes bimodal, with the faster component (90–130 ms) approaching the minimum efferent and afferent conduction delays in the saccade control system. Triggered saccades with such short reaction times are called “express” saccades (Fischer and Weber, 1993). In a recent report published in *The Journal of Neuroscience*, Hamm et al. (2010) addressed the cortical mechanism of express saccade generation in humans. They suggest that the occipital–parietal network may be an important player in express saccade preparation.

Express saccades can be observed frequently using a simple visually guided saccade task (generating a saccade in response to the appearance of a peripheral visual target) with an important manipulation: a central fixation point, on which subjects maintain their eyes, disappears 200 ms before target appearance (gap paradigm). The neural mechanisms of ex-

press saccade generation have been studied extensively in behaving monkeys using the gap paradigm. The superior colliculus (SC) is critical for express saccade generation (Schiller et al., 1987). The activity of SC saccade neurons during the gap period is higher before express saccades than before saccades with longer reaction times (regular saccades) (Dorris et al., 1997). Several findings suggest that the enhanced activity of SC saccade neurons before express saccades is controlled by cortical and subcortical mechanisms. First, saccade-related neurons in the frontal eye field (FEF) project to the SC and have enhanced activity during the gap period before express saccades (Munoz and Everling, 2004). Second, neurons in the caudate nucleus, a major input stage of the oculomotor basal ganglia (BG), have higher activity before target appearance on trials with a fixation gap than on trials on which the fixation point remains visible throughout the trial, which results in prolonged saccade reaction time (Watanabe and Munoz, 2010). Finally, the hypothesis that the frontal cortex (FEF) and BG play roles in express saccade generation is consistent with a clinical study in which patients with Parkinson's disease, which influences the frontal–BG neural circuits by dopamine degeneration, generate express saccades more frequently than control subjects (Chan et al., 2005).

Complementing these previous studies, Hamm et al. (2010) show evidence suggesting that the occipital–parietal network is important for express saccade generation. They used dense-array elec-

troencephalography (EEG) to evaluate the dynamics of signals in a distributed cortical region before saccade initiation. This cannot be achieved either by single-neuron recordings that focus only on a single structure (e.g., SC and FEF) or by functional magnetic resonance imaging, because its temporal resolution (a few seconds) is too low to resolve events occurring within a few hundreds of milliseconds in the gap paradigm. Furthermore, EEG can quantify cortical oscillations correlated with cognitive processes in humans.

The authors show several activity patterns that differ between express and regular saccades in the occipital–parietal region. Enhanced activation (global field power amplitude) occurred before express saccades compared to regular saccades during the gap period [Hamm et al. (2010), their Fig. 4]. Interestingly, the enhancement was biphasic with peaks at ~100 ms after fixation point disappearance and 20 ms before target appearance. The signal source of the initial component lay in the superior dorsal medial parietal lobe (Brodmann's area 7), while that of the later component lay in the right posterior cortex at the junction of the parietal and occipital cortices (at the junction of Brodmann's area 7 and 19) as well as in the bilateral calcarine fissure in the primary visual cortex (V1).

The authors suggest that enhanced activation in the occipital–parietal network facilitates express saccade generation by priming saccade generator circuits in the brainstem. Such priming could include the enhanced activity of SC saccade neurons before target appearances (Dorris et

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Correspondence should be addressed to Masayuki Watanabe, Centre for Neuroscience Studies, Queen's University, Room 234, Botterell Hall, 18 Stuart Street, Kingston, ON K7L 3N6, Canada. E-mail: masayuki@biomed.queensu.ca.

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al., 1997). However, the authors did not suggest explanations for biphasic cortical activation during the gap period, which has never been reported in studies using single-neuron recordings in behaving monkeys. This biphasic cortical activation is potentially important because it might be related to the fundamental mechanisms of the saccade control system as described below.

It has been suggested that saccades are controlled by two distinct commands: reflexive (sensory driven) and volitional (internally driven) (Munoz and Everling, 2004). In the gap paradigm, reflexive commands are programmed by the abrupt appearance of a target, while volitional commands are programmed depending on task demands (look toward a target). Although reflexive and volitional commands direct the eyes toward the same target in the gap paradigm, their temporal dynamics are different; reflexive commands are programmed earlier than volitional commands. Therefore, the latency difference between reflexive and volitional commands could be one of the mechanisms that produce the bimodal distribution of saccade reaction times (Fischer and Weber, 1993; Munoz and Everling, 2004).

The above hypothesis has been developed for motor commands triggering saccades. However, it can be extended to neural mechanisms controlling eye fixation. That is, eye fixation also can be controlled by reflexive signals driven by foveal visual stimulation as well as volitional signals depending on task demands (maintain fixation until target appearance). In the gap paradigm, reflexive fixation signals should be diminished immediately after fixation point disappearance. In contrast, volitional fixation signals should also be attenuated during the gap period, but only immediately before target appearance for two reasons: first, subjects are required to maintain their eye position on the blank screen during the gap period until target appearance; and second, subjects can anticipate target appearance because the gap duration is constant (200 ms). It has been suggested that inhibitory interactions exist between eye fixation and saccade signals (Munoz and Everling, 2004). Accordingly, we suggest the following hypothesis to explain the biphasic activation: the earlier and later components

reflect saccade preparatory signals released by the attenuation of reflexive and volitional fixation signals, respectively. Because saccade preparatory signals are variable (Dorris et al., 1997), express saccades might be generated only when they are enhanced strongly after the attenuation of the two fixation signals. This hypothesis can be tested easily by several task manipulations. For instance, randomizing gap durations can disrupt anticipations for stimulus appearance, which should change the temporal dynamics of volitional fixation signals without influencing reflexive fixation signals.

The authors also describe two additional differences between express and regular saccades that would benefit by additional exploration to help interpret the underlying mechanisms. First, during visual fixation before the gap period, the phase synchrony of occipital cortex oscillations in the 8–9 Hz (low α) frequency range was lower for express saccades than for regular saccades [Hamm et al. (2010), their Fig. 3]. The authors suggest that the α synchronization might enhance eye fixation signals and/or suppress the perceptual detection of fixation point disappearance. The former possibility is inconsistent with the fact that SC fixation neurons, which reside in the rostral SC and show tonic activity during active fixation, have the same firing rates for express and regular saccades (Dorris et al., 1997). We therefore favor the latter possibility, although it needs to be tested directly in future research. Second, EEG signals recorded from the parietal cortex immediately before saccade initiation (presaccade period: –45 to –15 ms from saccade initiation) were weaker for express saccades than for regular saccades [Hamm et al. (2010), their Fig. 5]. This might be explained simply by the following mechanisms. On trials with regular saccades, visual responses evoked by target appearance precede saccade responses. Therefore, saccade activity quantified during the presaccade period is likely contaminated by visual responses. In contrast, on trials with express saccades, visual and saccade responses occur almost at the same time (Dorris et al., 1997). Accordingly, contamination by visual responses is less likely before express saccades because the presaccade period could mainly quantify baseline activity preceding visual responses. Therefore, the amount of contamination by visual

responses might explain the weaker presaccade activity for express saccades compared to regular saccades.

In summary, by using dense-array EEG, this study has revealed the involvement of the occipital–parietal network in the preparation of express saccades by showing that different EEG signals occur depending on the type of upcoming saccades (express or regular) before target appearance. The experimental design adopted in this study can be extended further by manipulating the behavioral paradigm to test our hypothesis regarding the two types of fixation signals (reflexive and volitional). This will stimulate future research in basic studies in behaving monkeys as well as clinical studies in patients with neurological and psychiatric disorders. In particular, examining EEG signals from the occipital–parietal network in patients with Parkinson's disease will be valuable for the better understanding of their paradoxical saccadic behavior (more express saccades than normal subjects) (Chan et al., 2005) despite their degraded abilities of visual processing (Uc et al., 2005) and action preparation (Cunnington et al., 1997).

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