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Obligatory Adaptation of Saccade Gains

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[2]*Department of Psychology and* [1]*Center for Neural Science, New York University, New York City, New York; and* [3]*School of Psychology, Adeilad Brigantia, University of Wales Bangor, Gwynedd, United Kingdom*

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Srimal R, Diedrichsen J, Ryklin EB, Curtis CE. Obligatory adaptation of saccade gains. *J Neurophysiol* 99: 1554–1558, 2008. First published January 30, 2008; doi:10.1152/jn.01024.2007. We tested the hypothesis that saccade gains adapt to minimize error between the visual target and the saccade endpoint of every saccade we make even when the errors on sequential saccades are not directionally consistent. We utilized a state-space model that estimated the degree to which saccade gains were modified by the magnitude and direction of errors made on the previous trial. Importantly, to show that learning did not depend on the accumulation of directionally consistent errors, we fit the model to saccades made to targets that were displaced in a random direction during the saccade, thereby inducing errors with directions that were not sequentially the same. Saccade gains clearly adapted on a trial-by-trial basis despite that the perturbations were random, and the average amount of learning per trial was of similar magnitude as that found in a constant displacement of the target. These results indicate that saccade adaptation is a rapid and obligatory process that does not require conscious awareness.

INTRODUCTION

Saccades are ballistic eye movements that shift the point of gaze to a new location that is the goal of visual exploration. They are highly accurate despite being too rapid (~20 ms for small saccades) to be influenced by visual feedback (retinal processing time: ~20–30 ms). Therefore the trajectory of the saccade is programmed prior to its initiation. Within the oculomotor system, the motor programs issued given the position of the visual target on the retina remain accurate in the face of fatigue, injury and aging. Such plasticity emerges through motor learning mechanisms that continually adapt the system to new sensorimotor transformations.

Saccade adaptation, as it is called, can be studied in the laboratory by slightly displacing the visual target while the saccade is in flight. Following the saccade, the system perceives an error or mismatch between the position of gaze and the visual target. This error induces new motor learning. Since people are effectively blind while a saccade is in flight, subjects are unaware of the shift but nevertheless do adapt the gain of saccades to match the displaced target over time (McLaughlin 1967). Subjects are presented with blocks of trials where the target is consistently displaced and adaptation follows a characteristic profile depending on whether the target was displaced to a greater (forward-stepped) or lesser (back-stepped) eccentricity. The gain changes slowly and exponentially, reaching its maximum by 30–60 trials in humans (Deubel et al. 1986; Frens and van Opstal 1994; Hopp and Fuchs 2004; Watanabe et al. 2003) and 100–800 trials in monkeys (Hopp and Fuchs 2004; Straube et al. 1997) for back-stepped targets. The amount of adaptation is quantified by comparing the gain values before and after the block of displaced targets. The McLaughlin paradigm has been useful to researchers interested in measuring the final cumulative amount of motor learning. However, the consistent error from trial to trial (e.g., always back-stepped by 10%) confounds our ability to distinguish between competing learning mechanisms. Specifically, learning may operate at a very fast time scale, following every saccade that is inaccurate. Alternatively, learning may occur slowly and only after the system experiences directionally similar errors over a number of instances. To distinguish between these hypotheses, we measured the degree to which saccade gains were modified by errors made on the previous trial. Importantly, to show that learning did not depend on the accumulation of directionally consistent errors, we modified the classic McLaughlin paradigm such that saccades were made to targets that were displaced in a random direction during the saccade, thereby inducing errors with directions that were not sequentially consistent.

We fit a state-space model based on computational models of motor control to estimate the amount of learning on a trial-by-trial basis (Diedrichsen et al. 2005; Donchin et al. 2003; Thoroughman and Shadmehr 2000). These models explain how motor systems maintain fast and accurate movements despite the slow and unreliable nature of biological feedback (Kawato 1999; Wolpert and Kawato 1998). In the case of saccade adaptation, an inverse dynamics model computes the motor command necessary to generate the desired saccade. An efference copy of the motor command is sent to a forward model, which predicts the sensory error given the current state of the system and the perturbation to the system. This error is weighted by a learning parameter to form the teaching signal that trains the inverse dynamics model to produce an updated motor command.

METHODS

Subjects

Twelve healthy individuals (8 female, 4 male; 9 right-handed, 2 left-handed, 1 ambidextrous; ages between 18 and 39) gave written informed consent according to procedures approved by the human subjects Institutional Review Board at New York University, and were paid for participation.

Oculomotor and stimulus procedures

Subjects were seated in a darkened room 57 cm from a monitor (37 × 30 cm) with their heads stabilized via a chin rest. Eye position

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was recorded at 240 Hz with an infrared videographic camera (ASL 504; Applied Sciences Laboratories, Bedford, MA). Gray dots that subtended 1° of visual angle were used for fixation and target except during inter-trial intervals when the fixation dot was blue. In-house software (Gramalkn, http://www.ryklinsoftware.com) was used to present stimuli, quantify saccade timing and amplitude, and modify visual displays contingent on eye position. Targeting saccades were detected when the eye position left a 1.5 ° radius circle centered on the fixation point; this, on average across subjects, took 262 ± 69 (SD) ms following the presentation of peripheral visual target. Eye-movement data were transformed to degrees of visual angle, calibrated using a third-order polynomial algorithm that fit eye positions to known spatial positions, and scored off-line with in-house software (GRAPES).

Stable adaptation (SA) experiment

Preadapt trials (Fig. 1*A*): subjects maintained fixation (F, gray dot) for 2 s after which a target (T1, gray dot) appeared 16° to the left of fixation. Simultaneous with target onset, the fixation point disappeared, and the subject made a saccade to the target location and then after 2 s another saccade back to the blue fixation dot when it reappeared for the randomly varied intertrial interval (1, 1.5, 2, 2.5, 3 s). The fixation dot reappeared in a new location (1 of 6 possible locations along the horizontal axis – 5, 6, 7, 8, 10, or 11°) and the target location was always 16° to the left of it. *Adapt trials* (Fig. 1*B*): the task was the same as during preadapt trials, except that when the saccade was detected, the target was stepped backward by 2° along the horizontal axis (T2). *Postadapt trials* (Fig. 1*A*): same as preadapt trials.

Random adaptation (RA) experiment

Preadapt trials (Fig. 1*A*): the trial structure was identical to the stable adaptation preadapt trials (see preceding text). *Adapt trials* (Fig. 1*C*): the task was the same as during preadapt except when the saccade was detected, the target was either stepped backward (T2) by 2° or forward (T3) by 1° (to help compensate for inherent hypometricity of saccades) along the horizontal axis in pseudorandom order. *Postadapt trials* (Fig. 1*A*): same as preadapt trials. The random displacement sequence was identical for all subjects.

In both experiments, subjects performed a block of preadapt (100 trials), a block of adapt (200 trials), followed by a block of postadapt trials (100 trials). The same group of 12 subjects performed the RA then SA experiments, and all, but one, were naive to the experiment and the study's goals. All subjects underwent a postadaptation session to reverse saccadic gain adaptation and prevent carry-over effects. Importantly, subjects reported after the experiment that they did not notice the target jumps.

Analysis

Calibrated eye-position data were converted to saccadic gain values (saccadic gain = amplitude of saccade/target eccentricity) for subsequent analysis.

MAGNITUDE AND RATE OF GAIN ADAPTATION. To determine the magnitude of adaptation and whether the gain change between the preadaptation trials and the adapted trials was significant, we calculated the mean of the gains during the last third of the preadaptation trials and the last third of the adapt trials and did a paired sample *t*-test.

For the SA condition, we determined the rate constant of adaptation by fitting an exponential function of the following form (Fujita et al. 2002) to the adaptation data

$$a(t) = a_x + (a_0 - a_x)e^{-t/\tau}$$

where, $a(t)$ = curve fitted gain for trial *t;* a_x = mean adaptive gain value; a_0 = mean preadaptive gain value; t = trial number; τ = time constant for adaptation. τ was calculated using a nonlinear least-squares algorithm.

STATE-SPACE ADAPTATION MODEL. To quantify learning during both our experimental conditions, we fit our data to a state-space model based on a feedforward theory of motor learning. This model comes from control theory and relates a number of input to a number of output variables via a set of state variables. Such state-space models can be used to represent the output of a visuomotor control system as a function of the input and of the hidden state of the system at a particular moment in time, i.e., within a given motor trial. More importantly, they can be used to describe the temporal dynamics of how the hidden state develops over time, i.e., how the visuomotor system learns from trial to trial. The model allows us to accurately quantify learning on a trial-by-trial basis, which cannot be done by simpler percentage gain change calculations. Similar approaches have been used to model learning and generalization in a force-field reaching task (Diedrichsen et al. 2005; Donchin et al. 2003; Smith et al. 2004; Thoroughman and Shadmehr 2000) as well as during sensorimotor adaptation to altered visual feedback (Cheng and Sabes 2006). The model we use is defined by two equations. The output equation states that the saccade gain produced on trial n (y_n) is determined by the adaptive state of the system (z_n) plus some random noise (ε_n)

$$y_n = z_n + \varepsilon_n$$

Second, the state-update equation states that the adaptive state of the next trial z_{n+1}, is calculated by updating the current predicted gain, z_n, by a certain proportion (B) of the difference between the predicted saccade gain on the current trial and the target perturbation (u_n). Therefore the learning rate parameter B determines how fast the system adapts to a new saccade gain

$$z_{n+1} = z_n - B(z_n - u_n)$$

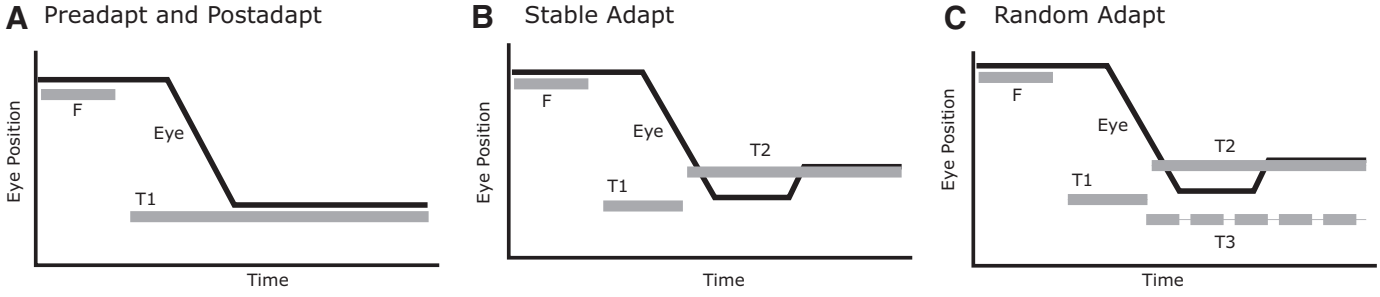

FIG. 1. Experimental paradigms. *A*: pre- and postadapt trial structure for both conditions were the same. A fixation dot, F, appeared for 2 s followed by a target, T1. *B*: during stable adapt (SA) trials, the target, T1, was back-stepped to T2. *C*: during random adapt (RA) trials, the target, T1, was randomly back-stepped to T2 or forward-stepped to T3 during the saccade.

For each subject's data, we used a nonlinear least squares algorithm to solve for the B parameter, an index of the amount of learning. To account for individual differences in inherent hypometricity, we set the starting value of z_n to be the mean gain of the last third of each subject's preadaptive trials.

RESULTS

Magnitude and rate of adaptation during SA

As expected, during the SA paradigm the gain of saccades reduced exponentially over the course of the adapt block. Figure 2A shows the adaptive gain change for a representative subject. During the preadapt block, saccade gains were slightly hypometric, reaching 99% of the visual target's eccentricity on average. By the end of the adapt block, the average saccade amplitude shortened to 58% of the back-stepped distance or ~1.2°, $t(56) = 7.38$, $P < 10^{-9}$. To quantify the rate of adaptation, we fit an exponential function to the saccade gains (green trace) and estimated a rate constant (τ) for the adaptation process. The example subject shown in Fig. 2*A* adapted at a rate defined by $\tau = 84$ trials. We found similar results for the others subjects, where on average they adapted to ~52% of the gain of the back-step and the mean rate constant for the adaptation (τ) was 58 ± 16.3 trials. Overall the mean saccade gains across subjects were significantly smaller when we compared the last third of late-adapt trials to the last third of preadapt trials in the SA condition, $t(11) = 8.66$, $P < 10^{-5}$.

State-space model quantification of learning during SA and RA

Next, we fit the state-space model to each subject's data from the SA and RA experiments and solved for the learning rate parameter, B. Again, this parameter estimates how much weight was given to the error experienced on a current trial to predict the gain on the next trial. During SA, subjects significantly adapted their saccade gains by the end of the adapt block, mean B value = 0.02 ± 0.01, $t(11) = 2.21$, $P < 0.05$, which corresponds to an exponential learning rate of 44 trials, thereby corroborating the previous analysis of the amount of adaptation in the SA condition. Moreover, the individual subjects' B values had a trend toward negative correlation with the τ values estimated in the preceding text from the exponential fits, $r(10) = -0.42$, $P = 0.18$, and a positive correlation with the percent amount of adaptation, $r(10) = 0.69$, $P < 0.05$. This correlation is critical because it demonstrates that the B parameter is sensitive to learning. Figure 2*A* shows a plot of predicted gain derived from the model for all three stages of the SA experiment for a representative subject. The predicted gain (red trace) has an exponential form as the system changes gain during the adapt block to minimize the error between the saccade endpoint and the back-stepped target.

During the RA condition, the overall saccade gains reduced slightly during the adaptation block compared with the preadapt block, $t(11) = 4.36$, $P < 0.005$. However the gain reduction during the RA compared with the SA condition was significantly less, about half [~1° gain reduction during SA vs. ~0.5° during RA, $t(11) = 3.87$, $P < 0.005$]. Moreover, this measure of learning is not valid during RA because learning took place in opposing directions, and the true amount of learning is underestimated. To correct for this, we fit the RA data with our state-space model and estimated the true amount of trial-by-trial learning, mean B value = 0.03 ± 0.001, $t(11) = 4.22$, $P < 0.01$. Recall that B estimates the degree to which the gain of a given saccade was influenced by the experienced error on the past trial. Figure 2*B* shows a plot of predicted gain (red line) calculated by the state-space model for the course of an entire RA experiment for a representative subject.

Next, we compared the amount of learning during the SA and RA conditions with a paired t-test across individuals. The

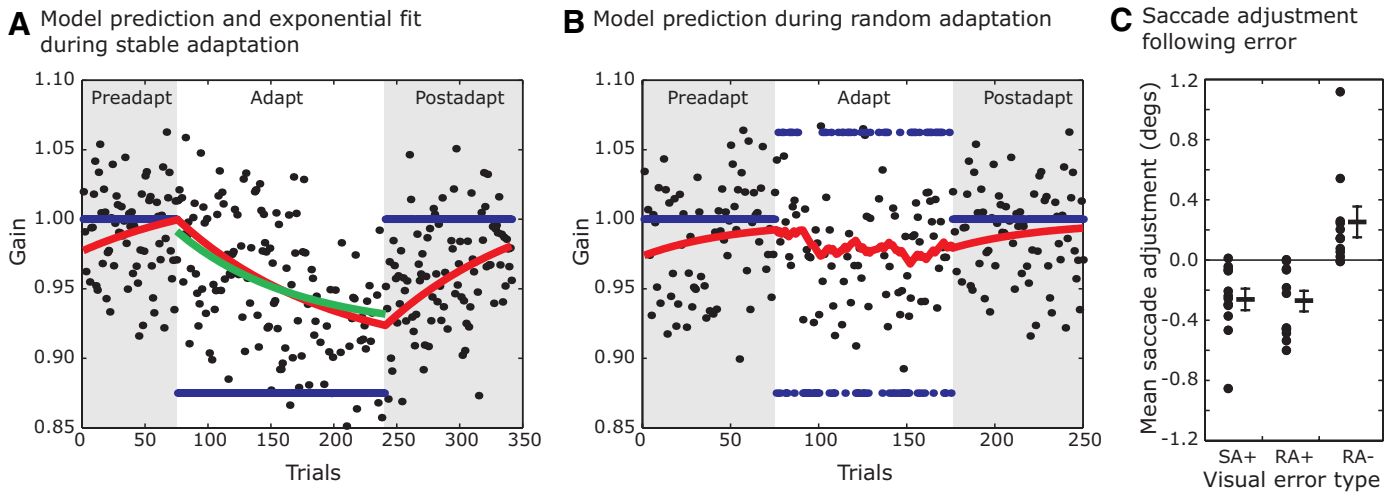

FIG. 2. Saccade gains before, during, and after adaptation from an example subject. *A*: during the SA block, the target (blue trace) was back-stepped during the saccade. The saccade gain (black dots) steadily adapted to match the new target location at a characteristic exponential rate (green trace). State-space model fit for the predicted gain (z; red trace) reached an asymptote during SA as the saccadic system adapted to constant error signals. *B*: during the RA block, the target gain was randomly stepped to 2° backward or 1° forward (blue trace). The predicted gain (z; red trace) fluctuated between the 2 target perturbations as the saccadic system learned from random error signals on each trial. *C*: mean saccade amplitude adjustment for each subject in response to positive visual errors (overshooting target) during SA and RA learning and in response to negative visual errors (undershooting target) during RA learning. In response to positive visual errors, saccades became more hypometric (negative saccadic adjustment). In response to negative visual errors, saccades became more hypermetric. Each dot is an individual subject's mean saccade amplitude adjustment. Lines and error bars represent sample mean ± SE for each condition.

B values estimated during the SA condition were not significantly different from those estimated during the RA condition, $t(11) = 0.61$, $P > 0.4$. This indicates that on a trial-by-trial basis the saccade gains adapted to a similar degree during the SA and RA conditions. However, the *B* values from SA and RA did not significantly correlate across subjects, $r(10) = -0.26$, $P > 0.4$, indicating that the strongest learners in SA were not necessarily the strongest learners in RA. Although this could indicate that different mechanisms support learning in SA and RA, several issues preclude this interpretation. One would expect an individual's SA and RA learning parameters to be correlated only if they measure stable trait-like aptitudes that were consistently expressed over the two different testing sessions. Additionally, the RA session always preceded the SA session leaving open the possibility that an order effect masked a potential correlation. Finally, it may be the case that a good learner is one that is not only sensitive to error feedback, but one that also discounts feedback when it is unreliable as was the case in the RA condition. All of these uncontrolled factors could have affected the correlation between SA and RA learning.

Trial-by-trial gain changes following induced errors

The results of our model suggest that the saccade gain for any given saccade is influenced by the direction of the error experienced even when the error signal is not consistent. An earlier study found evidence in favor of this idea (Desmurget et al. 2000). They reported that saccades had greater amplitudes on trials following random forward-steps compared with saccades on trials following random back-steps. However, they did not calculate the actual visual error on each trial. They assumed that on trials with forward jumps, the saccades always undershot the target, thus driving saccade lengthening on the subsequent trial and the opposite phenomenon on trials with back-steps. Although rare, this may not always be the case as saccades can overshoot forward-stepped targets and undershoot back-stepped targets due to noise in the system. In our data, subjects undershot the back-stepped target 14% of the trials (range: 0–40% of trials) and overshot the forward-stepped target 8% of the trials (range: 0–38%). Thus we used the difference between the stepped target and the actual saccade endpoint as a metric of visual error. Visual error, in the form of a feedback signal, is thought to drive learning in the system (although see Bahcall and Kowler 2000; Bonnetblanc and Baraduc 2007).

We divided RA trials into those in which subjects experienced positive and negative visual errors due to overshooting or undershooting the stepped target, respectively. The average saccade adjustment (i.e., difference between the gain on the current trial and the gain on the next trial) following positive errors (i.e., overshot the stepped target) was $-0.26 \pm 0.06°$, and the average adjustment following negative errors (i.e., undershot the stepped target) was $0.21 \pm 0.09°$ in the opposite direction. Although the difference in these saccade adjustments ($0.47 \pm 0.05°$) was significant, $t(11) = 3.53$, $P < 0.005$, the amplitude of adjustment in each direction was not different from one another. These results support and extend the observation of Desmurget et al. (2000). More importantly, they corroborate our state-space model; the significant *B* values of which indicate that learning occurred during the RA condition. To compare the average saccade adjustment following a positive visual error during RA ($-0.26 \pm 0.06°$) to that during SA, where the magnitude of errors exponentially decreased as the saccade gains reduced, we focused on the trials during the dynamic portions of the exponential fits when the gains were adjusting. Otherwise the errors and subsequent gain adjustments were negligible and could not be compared with RA. We therefore limited our analyses to the dynamic portion of the learning, specifically, between the first adapt trial to the trial in which the exponential fit reached asymptote (i.e., maximum gain reduction). Following positive visual errors, saccade gains reduced by an average of $-0.23 \pm 0.09°$ during SA, which was not significantly different from that observed during RA, $t(11) = 0.63$, $P = 0.54$. In Fig. 2*C*, for each subject, we show the mean saccade amplitude adjustment following positive errors during the SA and RA conditions and following negative errors during the RA condition. In response to positive errors during SA and RA, the subjects shortened their saccade amplitudes on the next trial. In response to negative errors during RA, they lengthened their saccade amplitudes on the next trial. Neither the condition (SA vs. RA) nor the direction of visual error (overshoot vs. undershoot) affected the size of the saccade adjustment on the next trial; both adjustments were around 1/4 to 1/5 of a degree. Moreover, the amount of saccade adjustment following errors during SA and RA showed a trend of positive correlation across subjects, $r(10) = 0.51$, $P = 0.09$. Together, these data suggest that following a saccade error, the amplitude of the next saccade is adjusted by about 1/4 to 1/5 of a degree to compensate for the error. Desmurget et al. (2000) reported a slightly larger average gain adjustment of about 1/3 of a degree during RA. If the adjustments were consistent across trials, subjects should have adapted to the 2° back-step during SA in no more than ~20 trials. However, subjects took on average 58 trials for their gains to stabilize and only adapted to about half of the full target back-step. This apparent overestimate of the trial-by-trial adjustment in saccade gains can be explained. Following the few SA trials in which the saccade undershot the back-stepped target, subjects made large positive corrections to their next saccade (i.e., $1.12 \pm 0.48°$, mean ± SD), which had the effect of slowing the rate of adaptation.

DISCUSSION

Learning is traditionally viewed as an adaptive change in behavior. For instance, in the stable saccade adaptation experiment when the target was back-stepped on every trial, the error distance between the saccade endpoints and the back-stepped visual targets got smaller over time. In essence, the subjects learned. Their performance improved as their saccade gains adapted to the new sensorimotor transformation. We also found evidence that subjects learned even when their performance could not improve because the error on any given trial did not predict the direction with which the target would be displaced on the next trial. To quantify learning under such circumstances when it cannot be inferred by an exponential decrease in saccade gains over the course of many trials, we adopted a model based on linear dynamical systems that has been used to model trial-by-trial learning of reaching movements (Diedrichsen et al. 2005; Donchin et al. 2003; Thoroughman and Shadmehr 2000). Fitting the model to data from the random saccade adaptation

experiment, we estimated the degree to which any given saccade gain was influenced by the visual error experienced on the previous trial. Our findings clearly demonstrate that such learning takes place even when the visual error feedback cannot be used to improve overall behavior. This finding, combined with the fact that subjects were not aware of the transaccadic displacements of the targets, indicates that saccade gain adaptation occurs without conscious awareness and is obligatory.

These data have two important implications for theories of motor control and learning (Kawato 1999; Wolpert and Kawato 1998). The first implication is that trial-by-trial learning is of an obligatory nature. It occurs even under conditions when subjects' behavioral performance does not or cannot improve, as when we randomly displaced the visual target. Moreover this type of learning does not require conscious awareness. A fast, obligatory, and automatic learning mechanism could continually calibrate the registration between incoming sensory information and outgoing motor commands free of the need for volitional resources and unencumbered by the need to track errors over longer time scales.

The second implication is that motor learning is not gated by whether the change in behavior is adaptive. Learning in the motor system does not begin when there is something to learn nor does it end when adaptation is complete. Rather adaptation occurs with every error that is experienced. This has an important implication especially for functional neuroimaging studies that rely on a random perturbation condition as a control condition for *true* learning (Desmurget et al. 1998, 2000). We need to be aware that neural processes that lead to adaptive changes are active when perturbations are random.

This raises the important question of whether the processes that lead to adaptation during random perturbations are the same as those during stable perturbations. It is tempting to speculate that the learning that took place over many trials when the perturbation was constant, as measured during the SA condition, was simply the accumulation of small gain adjustments on single trials, as measured during the RA condition. Indeed the most parsimonious explanation of our data are consistent with a single learning mechanism that operates on a fast trial-by-trial time scale. This is based on the fact that the *B* values and average gain adjustments were not significantly different for the SA and RA conditions. Even if the fast adaptive processes for random and stable conditions are the same, it is likely that additional mechanisms that work on slower time scales become important in stable adaptation paradigms (Kojima et al. 2004; Kording et al. 2007; Smith et al. 2006). For example, a learner is more flexible and efficient across a wide range of contexts when she can employ a system that is more sensitive to errors but forgets quickly and another system that is less sensitive to errors but forgets slowly (Kording et al. 2007; Smith et al. 2006). Nonetheless we cannot rule out the alternative hypothesis that fast trial-by-trial learning during stable and random adaptation is governed by independent mechanisms (Desmurget et al. 2000). We did not find that the amount of learning in the SA condition correlated with the amount of learning in the RA condition, leaving open the possibility that two independent mechanisms may have been operating.

We conclude that the oculomotor system adapts to random perturbations in motor control via a mechanism that is rapid, obligatory, and does not require conscious awareness. Moreover, these features fit well within emerging theories of motor control composed of dynamic internal models used to compute and anticipate the sensory consequences of motor actions.

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