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ABSTRACT

Despite changes in the physical structures controlling the eyes, saccades, the rapid eye movements used to explore the visual environment, remain accurate throughout the lifetime. The process underlying this sensorimotor adaptation is studied using a double step paradigm: an intra-saccadic target displacement introduces a systematic position error, which triggers changes in saccadic amplitude or direction across trials. Numerous researches on this saccade adaptation have been conducted, but the level of inter-individual variability and consistency in saccade gain change and how it relates to increase- or decrease-amplitude paradigms is not fully described. We conducted experiments in four groups of 25 participants with 800 trials per participant, including 200 baseline trials and 200 recovery trials. We used four distinct double-step paradigms that differed by the intra-saccadic target-step leading to either a horizontal (Backward or Forward) or vertical (Upward or Downward) gain modulation. Across experiments 95% of the participants exhibited adaptation revealing the consistency of this phenomenon. We observed strong inter-individual differences both in the extent and rate of adaptation, which were not correlated with the individual baseline saccades characteristics. As previously reported, the rates of adaptation were higher for gain decrease than for gain increase experiments but the final extent of adaptation were similar. Our results also support the view that adaptation of oblique saccades occurs where the saccade command is represented as a vector. Finally, at the individual level, we did not observe systematic changes in the saccade metrics in relation to adaptation.

Keywords: Saccadic eye movements; Saccadic adaptation; Learning; Inter-individual differences

INTRODUCTION

Visual acuity is not uniform across the retina, and the fovea, the small region (about 1 degree) located at the center of the macula, is responsible for the high-resolution central vision. Hence accurate and fast voluntary eye movements, termed saccades, are an essential component of visually guided behaviors, allowing one to move the retinal image of an object of interest close to the center of the retina. It has been estimated that we make about three saccades per second (Land, 1999) during our awaken life. One might therefore suppose that we are highly trained and that saccades should be quite stereotyped and unaffected by minute changes in the sensorimotor transformations. Surprisingly, the saccadic system can quickly modulate its metrics: using a double step paradigm in which a post-saccadic position error is introduced by surreptitiously shifting the visual target backward during the saccade, McLaughlin (McLaughlin, 1967) elegantly showed that saccade amplitudes progressively decreased over the course of eight saccades. These results echoed observations in natural settings showing that masking the good eye in patients with paretic eye quickly induced an increase in saccade amplitude in the weak eye (Abel, Schmidt, Dell'Osso, & Daroff, 1978; Kommerell, Olivier, & Theopold, 1976) .

Following these pioneering works, a large number of studies used many variations of the double-step paradigm to probe the conditions under which saccade amplitude would adapt to changes in the post-saccadic target vision. Indeed, the incentive for understanding saccade adaptation is twofold. On the one hand, the ability to maintain saccade accuracy throughout the lifetime is a fundamental component of the saccadic system which should be exhaustively described. On the other hand, saccade adaptation is now widely used to study fundamental process at play in our ability to adapt to changes in environmental contingencies and is often regarded as a good model of flexible and general motor learning (Herman, Blangero, Madelain, Khan, & Harwood, 2013). Much is known about the mechanisms responsible for saccade adaptation and its characteristics both at the neural and behavioral levels (see (Herman, Blangero, et al., 2013; Hopp & Fuchs, 2004; Pelisson, Alahyane, Panouilleres, & Tilikete, 2010) for reviews).

However, as Hopp and Fuchs pointed out in their 2004 review (Hopp & Fuchs, 2004), the amount of adaptation varies considerably within subjects but also between participants. The within-subject variability has been quantified (e.g. (Albano & King, 1989; Erkelens & Hulleman, 1993; Frens & van Opstal, 1994; Fujita, Amagai, Minakawa, & Aoki, 2002) and it is established

that the same participant might exhibit a two-fold difference in the extent of adaptation measured on different days. On the other hand, the level of inter-individual variability in both the amount of adaptation and other saccade parameters affected by a double-step paradigm has not been thoroughly quantified. Moreover, the range of changes in saccade amplitude obtained in a single experiment is often so large that it is difficult to draw definitive conclusions regarding the efficacy of an experimental manipulation: sometimes the amount of observed adaptation shows a four-fold difference across subjects (e.g. (Erkelens & Hulleman, 1993)), while some reported that 3 out of 10 subjects did not adapt at all (Frens & van Opstal, 1994). Unfortunately, the number of participants is often too small to quantify these individual differences. Importantly, despite a large set of experimental data it is still difficult, if not impossible, to assess the extent of these inter-individual differences as one can not simply compile results from the many experimental papers: partly because of theoretical constraints and partly because of the versatility of the paradigm, researchers have used a considerable number of variants of saccade adaptation paradigms to the point that it becomes impractical to compare results originating from different groups. It is noteworthy that to this day one cannot find an estimate of the distribution of changes in amplitude one might observe or even of the proportion of subjects exhibiting significant adaptation in a given double step paradigm.

Describing these differences across participants might be informative for at least two reasons. First, although inter-individual differences in eye movement control are sometimes regarded as uninteresting peculiarities, it has been proposed that they constitute an individual signature that might characterize a particular person (Bargary et al., 2017). More generally, inter-individual differences in behavior may reflect differences in brain functions (Kanai & Rees, 2011; Tomassini et al., 2011). In this view, both the commonalities of saccade adaptation across subjects and the inter-individual differences might be exploited to unravel the neural basis of saccade adaptation. Second, the variations in adaptation across subjects might be correlated with other individual characteristics of saccade control. For instance, it has been shown that individual characteristics of predictive saccades quantified in a baseline phase are well correlated with the adaptation rate (Wong & Shelhamer, 2014). Others have proposed that the inter-individual variability in baseline movements might constitute a central component of motor learning ((Herzfeld & Shadmehr, 2014; Wu, Miyamoto, Gonzalez Castro, Olveczky, & Smith, 2014), see also (Barbado Murillo, Caballero Sanchez, Moreside, Vera-Garcia, & Moreno, 2017)), an

hypothesis that has been particularly investigated in the context of operant conditioning (Neuringer, 2002). It therefore appears that investigating the extents of inter-individual variability might be useful to better understand saccade adaptation as well as other motor learning phenomenon.

Among the variations of the double step paradigm affecting the amount of induced adaptation the spatial parameters were extensively manipulated over the years. The orientation of the first step can be either horizontal (e.g. (Ethier, Zee, & Shadmehr, 2008a; McLaughlin, 1967; Miller, Anstis, & Templeton, 1981)), vertical (e.g. (Kojima, Iwamoto, & Yoshida, 2005; Watanabe, Ogino, Nakamura, & Koizuka, 2003)), or oblique (e.g. (Deubel, 1987; Hopp & Fuchs, 2006; Kojima et al., 2005)). Although the orientation of the first step is often kept unchanged throughout the experiment, some varied it during the experiment (e.g. (Rolfs, Knapen, & Cavanagh, 2010)). Instead of using a first step and an otherwise static target a few studies had the target moving either following a circular (Azadi & Harwood, 2014) or a linear motion (Havermann, Volcic, & Lappe, 2012). The first step amplitude can either be fixed (Ethier et al., 2008a) or variable (Madelain, Harwood, Herman, & Wallman, 2010). The spatial characteristics of the second (intra-saccadic) step are also subject to manipulations. For instance a modulation in saccadic amplitude is observed by manipulating this intra-saccadic step in the axis of the first step (on-axis paradigm) either in the backward or in the forward direction (Noto, Watanabe, & Fuchs, 1999; Scudder, Batourina, & Tunder, 2010; Straube, Fuchs, Usher, & Robinson, 1997) leading to an increase or a decrease in saccade amplitude. It is noteworthy that a forward adaptation is often regarded as more difficult to achieve compared to backward adaptation as evidenced by a longer time course and a more modest amplitude change (Ethier et al., 2008a; Panouilleres et al., 2009; Straube & Deubel, 1995). An adaptation-step perpendicular to the initial direction of the target (cross-axis paradigm) has also been used (Chen-Harris, Joiner, Ethier, Zee, & Shadmehr, 2008; Deubel, 1987; Ethier et al., 2008a). Although differences between "on-axis" and "cross-axis" adaptation have been reported in arm movement experiments (Krakauer et al., 2004; Liu, Mosier, Mussa-Ivaldi, Casadio, & Scheidt, 2011) this is not the case for saccade experiments (see (Bock, 1992) for a discussion regarding the differences between adaptation in saccade versus arm movements). To our best knowledge, there is no theoretical reason to regard the "on-axis" and the "cross-axis" paradigms as inducing different saccade adaptation and the one study directly comparing the two paradigms in the same monkeys did yield similar behavioral changes with

comparable adaption rates and adaptation fields (Noto et al., 1999). The amplitude of the intra-saccadic step (ISS) is most commonly proportional to the first step (Alahyane & Pelisson, 2005; Madelain et al., 2010; Straube & Deubel, 1995) but some used an intra saccadic step based on the amplitude of the saccade (Robinson, Noto, & Bevans, 2003; Eckart Zimmermann & Lappe, 2010). Researchers also manipulated the amplitude of the ISS, usually ranging between 20 and 50% of the first step, although some used a much smaller intra-saccadic step (from 1% to 10%) (Herman, Cloud, & Wallman, 2013). It is most common to keep the intra saccadic step constant throughout an experiment, but some studies explored the effect of the ISS consistency, either by using random noise (Havermann & Lappe, 2010; Srimal, Diedrichsen, Ryklin, & Curtis, 2008), or having the ISS oscillating across trials (Cassanello, Ohl, & Rolfs, 2016).

Temporal parameters of the double step paradigm have also been extensively manipulated, and in particular the duration of the post-saccadic target display. Although the target usually remains visible for some time after the ISS (in the order of 1000ms) a brief backstep paradigm have been used in which the target only stays in the backstepped position for 200-300ms before returning to its original location (i.e. before the ISS) (Wallman & Fuchs, 1998). Some research extinguished the post saccadic target after some delay which might considerably affect the amount of induced adaptation (Bahcall & Kowler, 2000; Panouilleres, Urquizar, Salemme, & Pelisson, 2011; Shafer, Noto, & Fuchs, 2000). Finally, a variant of the double step paradigm consists in flashing the target at the ISS location at different time with respect to saccade onset (Panouilleres et al., 2016). The duration of the inter-trial interval also affects adaptation: it often lasts for about 500 to 2000ms but some used ITI as short as 250ms, allowing one to achieve adaptation in fast paced sessions lasting less than two minutes (Gray, Blangero, Herman, Wallman, & Harwood, 2014) while others manipulated the inter-trial interval by inserting 30 seconds break every 60 adaptation trials (Ethier, Zee, & Shadmehr, 2008b).

One of the goals of the present study was to quantify the time course and overall extent of gain change in saccade adaptation, and report inter-individual variability in naïve human observers, not only in the gain changes but also in other saccade parameters that might be affected by a double step paradigm. Differences have often been reported in the course of adaptation for amplitude increase and for amplitude decrease. Therefore, we reasoned that it would be informative to assess the inter-individual variability across the two classes of paradigms. Moreover, adaptation is direction specific, an observation that led to the concept of

adaptation fields (Collins, Dore-Mazars, & Lappe, 2007; Frens & van Opstal, 1994) and it is unknown whether subjects adapt in the same way for the horizontal versus vertical saccade component. We chose the intra-saccadic step parameters of our double-step paradigms to probe adaptation exclusively in either the horizontal or vertical saccade component (see (Deubel, 1987) for a similar question) and leading to either an amplitude increase or decrease. However, this creates a situation in which one would typically either have different first target steps for the horizontal and vertical paradigms, or some on-axis conditions (ISS in the direction of the first target step) and some cross-axis conditions (ISS orthogonal to the first target step). This might confound parameters and bias the extent of adaptation in some conditions. To allow a fair assessment of the extent of adaptation obtained in different participants in different paradigms we used a variant of the cross-axis paradigm: we had an oblique first step (always following a 45° vector upward and rightward) such that both saccade components were equally solicited. This permitted to have the ISS direction being systematically different from the first target-step direction. Although one might argue that adaptation in oblique saccades could differ from adaptation in straight saccades, previous results indicate that the two types of paradigms should be regarded as involving identical underlying process (Deubel, 1987; Hopp & Fuchs, 2006; Noto et al., 1999). One added benefit of using our cross-adaptation paradigms is that it allows for specific analysis of saccade peak velocities and curvatures which provide valuable information regarding the possible involvement of a forward model in the observed gain changes (Chen-Harris et al., 2008). Four independent experiments were therefore conducted to probe changes in saccade in gain increase and gain decrease paradigms targeting either the horizontal or vertical saccade component.

To control factors that might induce variations in adaptation across participants we designed our experiments with several important constraints in mind. First, all subjects, regardless of the actual experiment, experienced the exact same sequence of target steps throughout the course of a session thus avoiding possible trial-order effects. Second, we chose to have a fairly large number of trials for each phase of the experiments allowing to measure a sound estimate of steady state saccade amplitude in baseline – from which the amount of adaptation is derived – and giving enough trials to estimate potentially long course of adaptation. Third, we included a long post-adaptation phase, in which the target no longer undergoes an adaptation step. Indeed, it is still unclear whether the course of recovery matches the one of the

original adaptation. Fourth, all data were collected by a single experimenter (one of the authors, SR) to limit a possible experimenter effect which might affect human behavior.

The data collected in the present experiments permitted us to measure the extent of adaptation in naïve participants and quantify the inter-individual variability in the changes in saccade gain. We asked whether the individual extent of adaptation or the adaptation rate might be predicted based on the individual baseline saccade characteristics and their variability. None of the baseline measures we considered were significantly correlated with either the extent or rate of adaptation. When probing whether the specific adaptation paradigms, in particular the gain increase versus gain decrease conditions, would yield different adaptation, we found similar final gain changes but with lower rates in the gain increase conditions. We also quantified the changes in other saccade parameters, most notably the saccade peak velocity, durations and curvatures, and their relation to the amount of adaptation. Although at the group level the pattern of changes in the adapted saccade component were in line with models relying on a forward model, at the individual level these changes were often inconsistent. Overall, these data support the hypothesis stating that adaptation of oblique saccades occurs where the movement is represented as a vector, again with large inter-individual differences.

METHODS

Subjects

One hundred and thirteen subjects participated in the experiment but thirteen were discarded due to the low number of correct trials (see below). For each of our four experimental groups, which differed only by the orientation of the ISS during adaptation trials (see below), we randomly assigned 25 participants. Based on a review of the literature and using our own data previously collected in pilot experiments, we estimated that one could conservatively expect an effect size (Cohen d) of 1.4, leading to a minimum sample size of 12 to reach a statistical power of 1. We chose to have 25 participants per experiments to provide a solid estimate of the extent of adaptation at the group level. As a comparison, the median number of participants is 7 in the 28 relevant papers cited in this article. The final data were collected over one hundred participants, eighty-five females and fifteen males (SD age, 21.45 ±3.98 years, ranging from 18 to 41). 84% of our participants were right-handed and 53% had a right dominant eye. To assess the eye dominance, we used the Miles test (Miles, 1930) in which the observer extends both arms, brings

both hands together to create a small opening, then with both eyes open views a distant object through the opening. The observer then slowly draws the opening back to the head to determine which eye is viewing the object (i.e. the dominant eye). All participants were naïve as to the purpose of the experiments, none but one had previous experience in oculomotor recording - but not in saccadic adaptation, and all had normal or corrected to normal vision. We obtained written consent from all participants. All experimental procedures received approval from the Ethical Committee in behavioral sciences of the University of Lille (Agreement n°2014-3-S24) and conformed to the standards set by the Declaration of Helsinki.

Procedure

Before each training and experimental session, we calibrated the eye tracker by having the subject fixate a set of thirteen fixed locations distributed across the screen.

All subjects performed 800 saccades, starting with 200 baseline trials with a single target step, followed by 400 adaptation trials with a double-step paradigm, and finally 200 post-adaptation trials with a single step. Regardless of the actual experiment, participants were instructed to fixate and follow the target appearing at the beginning of the trial. No further instructions regarding the paradigm were given.

The target was a light gray circle (10 pixels, i.e. about 0.4 deg, in diameter) with an 11.5 cd/m2 luminance displayed against a gray background (luminance 1.78 cd/m2). In all experimental groups, each trial started with a fixation period of random duration ranging from 700 to 1000 ms (drawn from a uniform distribution) during which the target was displayed at P0 [-7.8; -4.9] deg downward and leftward with respect to the center of the screen (Fig. 1.A). The fixation position (P0) was constant throughout the session. The target then stepped by either 7.3, 9.5, 11.7 or 13.9 deg (each amplitude occurred with equal probability in a pseudorandom order) following a 45° vector upward and rightward. In the baseline and post-adaptation trials the target remained visible for 500 ms after saccade onset provided a saccade was detected within 350 ms following the target step. If no saccade was detected within this 350 ms time window the trial was canceled and the target stepped back to the fixation position (P0). The online saccade detection corresponded to the measured eye position crossing an invisible boundary 3 degrees away from the fixation target.

In the adaptation trials, the detection of the saccade within the time window triggered an intra-saccadic target step (ISS) 19 ms after saccade onset on average. The orientation of the ISS depended on the actual experiment (Fig. 1.B). In the Backward - horizontal gain-decrease - experiment, the target stepped leftward along the horizontal axis by 20% of the horizontal component of the original step such that if the horizontal component of the first step vector was 9.8 deg (for a 13.9 deg oblique step) the ISS would displace the target by 1.97 deg leftward while the target vertical position would remain unchanged. In the Forward - horizontal gain-increase - experiment the target stepped rightward by 20% of the horizontal component of the original step. In the Upward - vertical gain-increase - experiment the target stepped upward along the vertical axis by 20% of the vertical component of the original step. Finally, in the Downward - vertical gain-decrease - experiment the target stepped downward by 20% of the vertical component of the original step. The mean inter-trial time was 1761 ms (from 1450 to 2210 ms). We gave subjects short breaks (mean time was 19s) every 50 trials followed by a drift check (and re-calibrated the signal if necessary) before continuing the experiment.

Because our participants had no previous experience with eye tracking, each participant first performed a 100 trials familiarization block (similar to the baseline simple step trials) to accustom to the pacing of the trials, train them on how to correctly fixate the target and how to avoid anticipating the displacement of the target. This training block was repeated one or two times in 32 participants to improve the quality of the eye movements recording. During both the familiarization block and the actual experiment, we used auditory feedbacks (400 Hz; 100 ms) to signal whether the eye was more than 2° away from the target location at the end of the fixation period or they anticipated the displacement of the target (500 Hz; 100 ms).

Apparatus

Subjects sat in a darkened room on an adjustable head and chin rest to minimize head movements, at 60 cm in front of a 22-inch IIYAMA HM204DT screen (1024 × 768 pixels at 100 Hz) CRT screen. Stimuli were generated and the experiment was controlled by a computer using the Psychophysics and Eyelink toolbox (Brainard, 1997; Pelli, 1997) running Matlab® (The Mathworks Inc., Natick, MA). Right eye position was recorded by a Tower Eyelink 1000 sampling at 2000Hz (SR Research Ltd, Osgoode, Ontario, Canada).

Acquisition and data analysis

Eye movements were recorded and measured throughout each trial. For offline data analysis we used the Eyelink saccade detector to identify saccades onset and offset, using 30°/s velocity and 8000°/s^2 acceleration thresholds (Stampe, 1993). An interactive analysis program was then used to analyze each saccade visually; it displayed each saccade from 100 ms before saccade detection to 300 ms after saccade detection and a human observer validated each saccade manually. Saccades with short latencies (<80 ms) or starting further than 2 deg away from the fixation position were excluded. Trials with blinks or in which the saccade gain (i.e. saccade amplitude/target step) was less than 0.4 or larger than 1.5 were discarded as well. An inclusion criterion of a minimum number of correct trials was fixed at 65%; from 66% to 97% of correct trials (mean: 84%) were recorded across our participants. A total of 13 participants, out of 113, were discarded because too few correct saccades were recorded. The correlations between the number of correctly recorded saccades and the level of adaption were not significant in any of the four experiments.

We computed the saccadic gain for each trial using the ratio of the saccade amplitude by the amplitude of the first target step independently for the horizontal and vertical component. We computed the average gain over the last 100 trials of each phase type (i.e. from trial 101 to trial 200 for baseline, trials 501 to 600 for adaptation and trials 701 to 800 for post-adaptation). To estimate the immediate effects of introducing an ISS we also computed the average gain over the last 10 baseline trials (from trial 191 to 200) and the first 10 trials of adaptation (from trial 201 to 210). Saccade sizes were also expressed in terms of percent-change with respect to baseline level by dividing the difference between the saccade gain for each trial and the mean saccade gain for the last 100 baseline trials (from trial 101 to 200) by the mean saccade gain for the last 100 baseline trials. To describe the changes in saccadic gain during the experiment, we computed the mean percentage of gain change for the last 100 trials of each phase (from trial 501 to 600 for adaptation and from trial 701 to 800 for post-adaptation). To show the trends of the percentage of gain change in participants, we smoothed the records of percentage of gain change versus trial number by means of a Lowess iterative nonlinear regression (width: 100 trials) applied separately for each experimental phase.

The rates of adaptation were computed using a piecewise “hinge” function in which a flat line (slope=0, intercept is a free parameter) fitted the last 100 baseline trials (from trial 101 to

trial 200) and a straight line (in which both the slope and the intercept were free) fitted the first 100 adaptation trials (from trial 201 to trial 300) with the constrain that the intercept was the same for the baseline and adaptation trials (Fig. 1.C). The rate of adaptation corresponds to the slope of the model. The same type of model was used to fit the transition from adaptation to post-adaptation.

To estimate the inter-trial variability in saccade amplitude we computed the changes in coefficient of variation by dividing the standard deviation of the gain by the mean gain for the last 100 baseline and last 100 adaptation trials, subtracting and computing the change.

The changes in peak velocities were quantified as follow. To dissociate amplitude-dependent changes from adaptation-dependent changes we first fitted the last 100 baseline trials using a square root fit, in which the peak velocity equals the square root of the amplitude multiplied by a constant (Lebedev, Van Gelder, & Tsui, 1996). This constant, the predicted peak velocity for a one-degree saccade, adequately accounts for the main-sequence, i.e. the saturating relation between the amplitude and peak velocity of saccades (Bahill, Clark, & Stark, 1975). We then computed, for each of the last 100 adaptation trials, the predicted velocity using the individual constant and the actual saccade amplitude. For each saccade, we computed the difference between the measured velocity and the predicted velocity divided by the predicted velocity to obtain a percent change in velocity such that a positive value would indicate an increase in peak velocity for the adaptation trials with respect to the baseline trials. These computations were done, independently, using the vectorial peak velocity as well as the horizontal and vertical peak velocities (in which case we used the horizontal and vertical amplitude, respectively). The same analysis was applied to the saccade duration which also follows the square root relation (Lebedev et al, 1996).

Saccade curvature in every trial was first quantified by a curve-fitting method using a cubic model (see (Ludwig & Gilchrist, 2002)) and then expressed in terms of proportion to the saccade amplitude. To estimate the changes in curvature between the baseline and the adaptation trials we then computed the difference between the median curvature obtained in the last 100 adaptation trials to the median obtained in the last 100 baseline trials.

We used bootstrapping methods (resampling with replacement, 100 000 times) to estimate all confidence intervals of our statistical parameters (Efron, 1979). Statistics were compared by Fisher's exact test with 100 000 permutations.

RESULTS

We found a difference between the average baseline gain and the average adaptation gain (i.e. individual differences greater than the null hypothesis 99% CIs) in the vast majority - 95 out of 100 - of our participants. To quantify the extent of change in saccade amplitude across subjects in each experiment we estimated the completeness of adaptation by dividing the average percent-change with respect to baseline level by the relative intra-saccadic step imposed in the adaptation trials: a 10% gain decrease in the backward experiment (-20% ISS) would give the same completeness (0.5) as a 10% increase in the upward experiment (+20% ISS). Because adaptation completeness depends on the baseline gain, values higher than unity do not imply that adaptation occurred beyond the target step: a 30% gain increase in the forward experiment (+20% ISS) – and therefore a 1.5 completeness – may be achieved by having a baseline gain of 0.8 and a final - hypometric - gain of 1.1. Comparing the medians completeness (0.69, 0.75, 0.80 and 0.82 for the backward, forward, downward and upward experiments, respectively, Fig. 2) measured in the two gain decrease conditions (backward and downward) to the ones measured in the gain increase conditions (forward and upward) did not reveal a main effect of the adaptation direction (the median completeness reaching 0.75 in both the gain decrease and gain increase experiments, Wilcoxon rank sum, $p=0.98$). Adaptation completeness was also similar in the two horizontal experiments (backward vs forward, Wilcoxon rank sum, $p=0.19$) and in the two vertical experiments (downward vs upward, Wilcoxon rank sum, $p=0.29$). The extent of adaptation (as a proportion of ISS) was quite variable across subjects (Fig. 2). To quantify the extent of inter-individual variability we used the interquartile difference, i.e. the difference between the first and third percentile of the group distributions. The variability in the forward and upward experiments (0.36; 95% CI [0.18, 0.57] and 0.55; 95% CI [0.35, 0.8], respectively) was not different to the one measured in the backward and downward experiments (0.19; 95% CI [0.12, 0.29] and 0.22; 95% CI [0.13, 0.44], respectively) as revealed by Fisher's exact test (observed difference smaller than the null hypothesis 95% CI). Moreover, the interquartile differences were not different when comparing the two horizontal experiments (backward versus forward, Fisher's exact test observed difference smaller than the null hypothesis 95% CI) nor when comparing the two

vertical experiments (downward versus upward, Fisher's exact test observed difference smaller than the null hypothesis 95% CI). We will now present in more detail the data collected in each experiment.

Backward experiment: horizontal gain-decrease

Results from the horizontal gain decrease experiment are plotted on Figure 3. Figure 3.A plots the horizontal and the vertical saccadic gain with respect to trial number for an exemplar subject, participant S63. The two vertical grey lines indicate the first and last trials in which the intra-saccadic step (a horizontal backward step, encompassing 20% of the first target-step horizontal component) was implemented. The 200 first (from trial 1 to trial 200) and 200 last trials (from trial 601 to trial 800) are respectively the baseline and post-adaptation trials with a single step paradigm. In comparison to the mean baseline horizontal gain (mean=0.96; SD=0.07), we observed a progressive decrease in the horizontal gain (-17.85 % with respect to baseline) at the end of the adaptation phase (mean gain=0.79; SD=0.06) followed by a progressive increase during the post-adaptation phase reaching a mean horizontal gain of 0.94 (SD=0.06) such that saccade gain almost completely recovered from the amplitude reduction imposed by the ISS (-1.88% with respect to pre-adaptation). The rate of adaptation (computed across the first 100 ISS trials, Fig 1.C) was -0.14 (in percent change unit per trial) for this participant while the rate of recovery was +0.13. It is noteworthy that the horizontal ISS did specifically affect the horizontal saccade component while leaving the vertical component (gray line on fig. 3.A) essentially unchanged.

Other subjects from the horizontal decrease experiment exhibited similar changes in saccade amplitudes: Figure 3.B plots the smoothed percent change in horizontal gain computed with respect to the mean baseline gain for each participant. We found a systematic horizontal gain decrease during the adaptation phase and a progressive increase during the post-adaptation trials, with some noticeable variability across subjects in the final gain values for each phase. To quantify the horizontal gain change during adaptation and post-adaptation trials we computed the percent change averaged across the 100 last adaptation trials (i.e. from trial 501 to trial 600) and across the last 100 post-adaptation trials (i.e. from trial 701 to trial 800) (fig. 3.C). Across subjects, the mean percent gain change for adaptation was -14.10% (SD=3.01; ranging from -21.59% to -7.94%; Cohen d = 4.68) and the mean percent change post-adaptation was -3.47%

(SD=3.71; ranging from -10.36% to +6.18%). The vertical grey line marks a complete adaptation (-20% gain change with respect to baseline gain) while the horizontal one indicate a full recovery to baseline gain (0% gain change with respect to baseline gain).

To quantify the extent of horizontal gain change, we computed the mean horizontal gain for the 100 last trials of the adaptation phase (mean=0.79; SD=0.06; ranging from 0.69 to 0.94) and plotted it with respect to the mean horizontal gain computed over the last 100 baseline trials (mean=0.92; SD=0.07 ranging from 0.81 to 1.05, Fig. 3.D). The differences in baseline versus adaptation gains were greater than the null hypothesis 99% CIs in all participant. Interestingly, the data points align below the equality line indicating a quite strong correlation between the baseline and adaptation gain across subjects ($R=0.89$; $p<0.01$). We also computed the average gain for the last 100 post-adaptation trials (mean=0.89; SD=0.07; ranging from 0.73 to 1.04) which were systematically greater than the ones in the adaptation phase (all values above the null hypothesis 99% CIs) indicating a gain recovery in all subjects. To probe whether introducing the ISS had an effect on the very first adaptation trials we plotted the mean gain computed across the last 10 baseline trials (mean=0.92; SD=0.08) against the first 10 adaptation trials (mean=0.91; SD=0.08) on Figure 3.E: most of the points fall around the equality line indicating a lack of systematic change across subjects. Finally, Figure 3.F plots the rates of gain change (in percent change unit per trial) computed for the first 100 adaptation trials (mean=-0.13; SD=0.03; ranging from -0.18 to -0.06) versus the ones for the first 100 post-adaptation trials (mean=+0.11; SD=0.03; ranging from 0.05 to 0.18). No correlation was found between these two rates ($R=0.12$; $p=0.58$).

Forward experiment: horizontal gain-increase

Results from the horizontal gain decrease experiments are plotted on Figure 4 (following the same organization as Fig. 3). Figure 4.A plots the raw and smoothed data for participant 34: from a horizontal baseline gain averaging 0.91 (SD=0.08) a significant increase is observed reaching an average gain of 1.14 (SD=0.08) at the end of adaptation, and an average post-adaptation gain of 1 (SD=0.06). The rate of adaptation for this participant was 0.18 while the rate of recovery was -0.19. Interestingly, considerable variability across subjects is visible when examining the smoothed percent change for the whole sessions for all subjects (Fig. 4.B) both during the adaptation phase and during the post-adaptation phase. The percent change at the end

of the adaptation phase was variable, averaging +16.11% (SD=5.77; ranging from +2.82% to+ 29.43%; Cohen d = 2.79) as was the percent change at the end of the post-adaptation phase (mean=+4.65%; SD=5.13; ranging from -4.87% to +17.05%, Fig. 4.C). We observed a general increase in horizontal gain when comparing the average baseline gains (mean=0.92; SD=0.06; ranging from 0.76 to 1.06) to the average adaptation gains (mean=1.07; SD=0.07; ranging from 0.83 to 1.19; Fig. 4.D) except for one participant in whom the difference was less than the null hypothesis 99% CIs. Here again it appears that the baseline and adaptation gains were well correlated (R=0.73: $p<0.01$). The gains generally recovered in the post-adaptation phase (mean=0.97; SD=0.07; ranging from 0.80 to 1.12) as the differences with the adaptation gains were greater than the null hypothesis 99% CIs in all but one participant. Much as in the horizontal gain decrease experiment, introducing the ISS had inconsistent effects on the very first adaptation trials (Fig. 4.E): the mean gains computed across the last 10 baseline trials (mean=0.93; SD=0.08) were close to the mean gains for the first 10 adaptation trials (mean=0.95; SD=0.06). Figure 4.F plots the rates of gain change computed for the first 100 adaptation trials (mean=+0.11; SD=0.04; ranging from +0.05 to +0.18) versus the ones for the first 100 post-adaptation trials (mean=-0.12; SD=0.05; ranging from -0.22 to -0.04). No correlation was found between these two rates (R=-0.21; p=0.31).

Downward experiment: vertical gain-decrease

The saccadic gains measured for one participant (Fig. 5.A) illustrate the evolution of saccade amplitudes observed during the course of a session, starting with an average vertical gain of 0.94 (SD=0.1) followed by a strong decrease in with the vertical gain averaging 0.77 (SD=0.1), and a post-adaptation average gain of 0.82 (SD=0.11). The rate of adaptation for this participant was -0.09 while the rate of recovery was +0.09. A relatively high inter-individual variability in vertical gain percent change can be observed at each stage of the sessions (Fig. 5.B) and particularly during the adaptation and post-adaptation phases. The percent change in vertical gain (Fig. 5.C) during the adaptation phase averaged -16.36% (SD=5.46; ranging from -28.93% to -2.16%; Cohen d = 3.00) while the percent change during the post-adaptation phase averaged -6.27% (SD=5.77 ranging from -+20.23% to +11.13%). Importantly, we also observed a decrease in vertical gain when comparing the average gain in the last 100 baseline trials (mean=0.89; SD=0.07; ranging from 0.73 to 1.01) to the gain measured at the end of the adaptation phase

(mean=0.74; SD=0.07; min=0.58; and max=0.85; Fig. 5.D) except for one participant. These baseline and adaptation gains were positively correlated (R=0.74, p<0.01). The gains systematically increased in the post-adaptation phase (mean=0.83; SD=0.06; ranging from 0.65 to 0.92) as the differences with the adaptation gains were greater than the null hypothesis 99% CIs in all participants. Much as in the horizontal gain decrease experiment, introducing the ISS had limited effects on the very first adaptation trials (Fig. 5.E): the mean gains computed across the 10 last baseline trials (mean=0.88; SD=0.07) were close to the mean gains for the 10 first adaptation trials (mean=0.86; SD=0.09). Figure 5.F plots the rates of gain change computed for the adaptation trials (mean=-0.16; SD=0.05; ranging from -0.24 to -0.02) versus the ones for the post-adaptation trials (mean=+0.11; SD=0.05; ranging from 0 to +0.21). No correlation was found between these two rates (R=-0.18; p=0.39).

Upward experiment: vertical gain-increase

For participant 71 (Fig. 6.A) who experienced our upward experiment the baseline vertical gain averaged 0.98 (SD=0.1) and an important increase can be observed with the gain averaging 1.15 (SD=0.1) in the adaptation phase, while the post-adaptation gain decreased to 1 (SD=0.08). The rate of adaptation for this participant was 0.11 while the post-adaptation recovery rate of was -0.14. Across subjects, the percent change in vertical gain (Fig. 6.B) appears very variable throughout the experiment. The percent change during the adaptation phase was strongly variable across subjects (mean=13.89; SD=8.20; ranging from -0.06 to 33.62; Cohen d = 1.69) and also during the post-adaptation phase (mean=2.13; SD=6.99; ranging from -9.42 to 18.55; Fig. 6.C). The gain generally increased from its baseline level (mean=0.88; SD=0.11; ranging from 0.64 to 1.03; Fig. 6.D) when compared to the vertical gain in the adaptation phase (mean=1; SD=0.12 ranging from 0.67 to 1.15) as the differences were greater than the null hypothesis 99% CIs in all but three participants. Interestingly, these baseline and adaptation gains were correlated (R=0.85, p<0.01). The gains decreased in the post-adaptation phase (mean=0.90; SD=0.11; ranging from 0.58 to 1.06) as the differences with the adaptation gains were greater than the null hypothesis 99% CIs in all but three participants. The mean gains computed across the 10 last baseline trials (mean=0.89; SD=0.12) were close to the mean gains for the 10 first adaptation trials (mean=0.91; SD=0.11). Figure 6.F plots the rates of gain change computed for the first 100

adaptation trials (mean=0.08; SD=0.06; ranging from -0.02 to 0.18) versus the ones for the first 100 post-adaptation trials (mean=-0.13; SD=0.12; ranging from -0.58 to 0). No correlation was found between these rates (R=-0.18; p=0.38). It should be noted that one participant had an exceptionally high rate of recovery (-0.58, participant 36). Further inspection of these data revealed that the gain was very unstable for this participant: it increased and decreased back by about 0.2 gain units during the baseline trials, increased quickly during the last 100 adaptation trials and decreased back during the first 50 post-adaptation trials, which explains his high recovery rate. We did not observe a similar behavior in the other 99 participants.

Rates of gain change across experiments

We compared the rates of adaptation (0.14, 0.11, 0.16 and 0.08 for the backward, forward, downward and upward experiments, respectively, Fig. 3-6 panels F) measured in the two gain decrease conditions (backward and downward) to the ones measured in the gain increase conditions (forward and upward). This comparison revealed a main effect of the adaptation direction (0.14 versus 0.1 in the gain decrease and gain increase experiments, respectively, Wilcoxon rank sum, $p<0.01$) such that the rates were faster in the gain decrease than in the gain increase experiments. This difference was also significant when comparing the adaptation rates for the two vertical experiments (downward versus upward, Wilcoxon rank sum, $p<0.01$) but not for the two horizontal experiments (backward versus forward, Wilcoxon rank sum, $p=0.13$). The inter-quartile differences of the rates were not significantly different when comparing the gain decrease versus gain increase conditions as revealed by Fisher's exact test (observed difference smaller than the null hypothesis 95% CI). To further test whether the adaptation rates were different when decreasing the gain and when increasing the saccade gain, we computed the difference between the adaptation and recovery rates for each participant exhibiting a significant gain change (i.e. all but 5 participants). Adaptation rates were larger than the recovery rates in the gain decrease experiments (0.14 versus 0.10, in the adaptation and in the recovery phase, respectively, Wilcoxon rank sum, $p<0.01$) indicating that, at the individual level, the rate of gain decrease (i.e. during adaptation) was larger than the rate of gain increase (i.e. during recovery). This difference between the adaptation and recovery rates was not significant in the gain increase experiment (0.10 versus 0.12, in the adaptation and in the recovery phase, respectively, Wilcoxon rank sum, $p=0.10$). Finally, we compared the adaptation rates in the two horizontal experiments

to the ones obtained in the two vertical experiments and did not find any significant differences (0.12 versus 0.12, Wilcoxon rank sum, p=0.96).

The adaptation rate was computed using a picewise linear model which assumes a linear decrease in gain in the first 100 trials. However, we can not exclude that, at some point during the adaptation trials, participants became conscious of the ISS direction and strategically adjusted their saccade gain to reduce the post-saccadic position error. This sudden change in strategy should result in a sudden decrease in gain for the two gain decrease experiments and in a sudden increase in gain for the two gain increase experiments. To probe for this possibility, we used two 20 trials sliding windows, separated by a single trial, and computed the differences in gain before and after the putative change point for each adaptation trials. We then computed the sign of the largest observed difference. In the gain decrease experiments we found that the difference was positive in 28 participants and negative in 22, while in the gain increase experiments the difference was positive in 20 participants and a decrease in 30 of them. Therefore, these results do not support a strategic change in gain.

Saccade changes across experiments

No consistent changes were observed in the vectorial peak velocities (between the last 100 baseline trials and last 100 adaptation trials): comparing the changes in peak velocities in the two gain decrease experiments versus the two gain increase experiments (Fig. 7.A) did not reveal a main effect of the gain change direction (Wilcoxon rank sum, p=0.15). At the individual level although a majority of participants in the gain increase experiments (27 out of 50) did exhibit different peak velocities in the adaptation trials when compared to the baseline trials (estimated using Fisher's exact test), we found that the peak velocity decreased in 12 of them but increased in 15 of them. Similarly, we found a significant peak velocity increase in 8 participants of the gain increase experiments (out of 50), and a significant decrease in 14 of them (Fig. 7.A). These inconsistent effects of the ISS direction on the saccade main sequence were further confirmed when considering the changes in saccade duration (Fig. 7.B). No significant effect of the ISS direction on saccade duration was observed when comparing the gain decrease to the gain increase experiments (Wilcoxon rank sum, p=0.13). Although there was a change in saccade duration in 20 participants of the gain decrease experiments, these changes were due to either an increase in saccade duration (16 participants) or a decrease (in 4 participants). In the gain

increase experiments, the distribution of changes was similar with 7 participants exhibiting a decrease and 11 exhibiting an increase.

To further probe the extent of changes in saccade velocity induced by adaptation we computed the changes in the peak velocities of the horizontal component and vertical component independently (between the last 100 baseline trials and last 100 adaptation trials) in each participant. This allowed us to consider the changes in peak velocity in the adapted component (i.e. the horizontal velocity for the backward and forward experiments, and the vertical velocity for the downward and upward experiments) on the one hand and the changes in the non-adapted component on the other hand. The changes in peak velocity in the adapted versus non-adapted component in the gain decrease experiments are plotted Figure 7.C. The peak velocity of the adapted component decreased (median change = -2.33%) but this effect was not systematic as 23 participants (out of 50) exhibited a significant decrease while 5 exhibited an increase. On the other hand, the peak velocity of the non-adapted component tended to increase (median change = +0.57%) but this effect was not systematic as 13 participants (out of 50) exhibited a significant increase while 7 exhibited a decrease. When comparing the changes in peak velocity for each component at the group level in the gain decrease experiments we found that the peak velocity changes were significantly smaller in the adapted than in the non-adapted component (Wilcoxon rank sum, $p<0.01$). At the individual level this difference was significant in 21 participants but the opposite effect was true in 2 participants. Finally, we asked whether the changes in peak velocity components were correlated with the adaption completeness (Fig. 2). We found a significant ($p<0.01$) negative correlation ($R=-0.54$) for the peak velocity of the adapted component but not for the non-adapted component ($p=0.19$).

Results from the gain increase experiments are plotted Figure 7.D. The general pattern was in the opposite direction as the peak velocity of the adapted component tended to increase (median change = +0.14%) while the peak velocity of the non-adapted component tended to decrease (median change = -1.93%) but this effect was not systematic as 29 participants (out of 50) exhibited a significant decrease while 7 exhibited an increase. When comparing the changes in peak velocity for each component at the group level in the gain increase experiments we found that the peak velocity changes were significantly larger in the adapted than in the non-adapted component (Wilcoxon rank sum, $p=0.03$). At the individual level this difference was significant in 17 participants but the opposite effect was true in 10 participants. Finally, we asked whether

the changes in peak velocity components were correlated with the adaption completeness (Fig. 2). We found a significant ($p<0.01$) negative correlation ($R=0.53$) for the peak velocity of the adapted component but not for the non-adapted component ($p=0.55$).

When considering the saccade curvature in the baseline trials in our 100 participants we found that the median saccade curvature was significantly negative (median=-0.05; Signtest $p<0.01$), in line with observations of Viviani et al. (1977) showing that oblique saccades usually made a detour directed toward the horizontal axis. Overall, the general velocity pattern was a tendency to decrease the peak velocity in the adapted component associated with an increase in the other component in the gain decrease experiments, and the opposite pattern in the gain increase experiments. These changes in peak velocity should induce some specific changes in the saccade curvatures during adaptation. However, the relation between the changes in the components velocity profiles and the changes in saccade curvatures depends on the timing of the two velocity signals. Indeed, it has been established that the velocity in the adapted component tend to change later in the movement compared to the velocity in the other component (Chen-Harris et al., 2008). As a result, one should expect that, in a gain increase experiment, adapted saccade should first curve in the direction of the non-adapted component and toward the end of the saccade steer the trajectory in the direction of the adapted component. This process, resulting in curved saccades, has been quantified for cross-axis adaptation in which a horizontal movement was adapted upward by introducing an upward ISS: researchers found negative curvatures (i.e. the saccade curved below the straight line joining the starting and end point of rightward movements, (Chen-Harris et al., 2008), their figure 3). In the case of the present experiments, one would therefore predict that saccade curvatures should become more negative for the upward adaptation experiment, because of the late velocity increase in the vertical component, and less negative in the forward experiment, because of the late velocity increase in the horizontal component. Based on these results, one should also expect changes in saccade curvatures in the gain decrease experiments. However, because the peak velocity of the adapted component is now decreasing compared to the peak velocity of the non-adapted component, predictions regarding the direction of curvature changes should be different: curvatures should become more negative in the backward experiment and less negative in the downward experiment. To test this possibility, we computed the difference in curvature (between the last 100 adaptation trials and the last 100 baseline trials) in each participant, expressed in proportion of the saccade amplitudes

(Fig 7.E). Comparing the changes in the backward and upward experiments against the ones in the forward and downward experiments revealed a significant difference which goes in the predicted direction (Wilcoxon rank sum, $p<0.01$). This pattern of differences was however not systematic as it was observed in 36 participants (out of 100) while 8 participants exhibited significant changes in the opposite direction. Moreover, these changes in curvature were not significantly correlated with the adaptation completeness either when considering the backward and upward experiments ($p=0.7$) or when considering the forward and upward experiments ($p=0.89$).

To further estimate whether saccade triggering was affected by the introduction of an ISS, we computed the difference in the median saccade latency between the last 100 baseline trials and last 100 adaptation trials. No consistent changes in the latencies were observed: we found some increase (greater than the null hypothesis 99% CIs) in 3 to 8 participants in each experiment and some decrease in 2 to 4 participants (Fig. 7.F).

To estimate whether the saccade gains were more variable at the end of the adaptation trials than in the baseline, we computed the differences in the coefficient of variation of the gains (i.e. the ratio of the standard deviation to the mean). Although we found some differences in 10 participants, these data did not reveal a systematic change in the saccade gain variability between the baseline and adaptation trials (Fig. 7.G).

So far, we reported results regarding the saccade component specifically targeted by the ISS (except when considering the main sequence). To probe the effect of the ISS on the other saccade component (i.e. vertical for the backward and downward experiments and horizontal for the downward and upward experiments) we computed the proportion of gain changes by subtracting the mean baseline gain to each saccade gain and averaging these proportional changes for the last 100 adaptation trials (i.e. from trial 501 to trial 600) for each participant in every experiment. Results were similar across experiments in that we did not observe a consistent effect: changes distributed mostly around zero (averaging -0.21%, 3.77%, -0.02% and 0.95% for the backward, forward, downward and upward experiment, respectively; see Fig. 7.H). We found changes in the adaptation gains outside the null hypothesis 99% CIs in 34 participants often reflecting a slight increase (22 participants). It is worth noting that the dispersions were again larger in the vertical saccade component than in the horizontal one.

Finally, we computed the autocorrelation in the saccade gains (i.e. the correlation between the gains with a 1 trial lag) for all baseline trials, both for the horizontal and vertical components. Importantly we found very limited positive correlations ($p<0.05$) in only a fraction of our participants averaging 0.13 in 14 participants for the horizontal saccade component and 0.25 in 27 participants for the vertical component.

Correlation with baseline saccade characteristics

To explore the relation between the characteristics of the baseline saccades and the extent of adaptation we first selected three saccade parameters that might reflect the pre-adapted state of the saccadic system, i.e. the saccade gain, the saccade vectorial peak velocity and the saccade latency. For each of these parameters we measured both the central tendency and the dispersion, measured by the interquartile difference, in the 200 baseline trials for each participant. We then computed the correlation between the percent change in gain and each of these measures in the gain decrease and in the gain increase experiments (Table 1). No correlation was significant.

Mean Gain	Gain dispersion	Main sequence	Velocity dispersion	Median latency	Latency dispersion
Gain decrease experiments					
-0.19	0.18	-0.1	0.12	0.1	0.02
p=0.18	*p=0.21*	*p=0.51*	*p=0.39*	*p=0.48*	*p=0.88*
Gain increase experiments					
-0.24	0.23	-0.21	-0.11	0.21	0.14
p=0.09	*p=0.11*	*p=0.14*	*p=0.47*	*p=0.14*	*p=0.34*

Table 1: Correlations and associated probabilities (in italics) between the signed percent gain change and 6 saccade parameters measured over the 200 baseline trials, for the gain decrease and gain increase experiments.

We also computed the correlation between the adaptation rate and each of the individual baseline measures in the gain decrease and in the gain increase experiments (Table 2). No correlation was significant.

Mean Gain	Gain dispersion	Main sequence	Velocity dispersion	Median latency	Latency dispersion
Gain decrease experiments					
0.11	0.01	0.1	-0.13	-0.03	-0.16
p=0.46	*p=0.96*	*p=0.48*	*p=0.38*	*p=0.81*	*p=0.26*

Gain increase experiments					
0.15	-0.16	0.01	0.05	0.01	-0.04
p=0.31	*p=0.28*	*p=0.96*	*p=0.75*	*p=0.95*	*p=0.77*

Table 2: Correlations and associated probabilities (in italics) between the signed adaptation rate and 6 saccade parameters measured over the 200 baseline trials, for the gain decrease and gain increase experiments.

DISCUSSION

In this study, we collected data in naïve adult participants in four distinct double step paradigms to probe the consistency of saccadic adaptation across subjects. Strikingly, all paradigms elicited clear saccadic adaptation and we observed a change in gain in the direction of the ISS in 95 out of our 100 participants. However, it appears that one should look past this large figure to adequately describe the phenomenon at hand: most of our measures quantifying saccade changes exhibits a two- to three-fold difference, further confirming that although the saccadic system's flexibility in response to changes in environmental contingencies is certainly a general learning phenomenon, substantial variations both in the amount and time course of adaptation should be expected at the group as well as at the inter-individual level. Importantly, neither the extents nor the rates of adaptation were correlated with individual baseline saccade characteristics. Before discussing this result we will first present the implications of the behavioral changes we observed for saccade adaptation.

General features of saccade adaptation across paradigms

Our data exhibit a number of features previously reported in the saccade adaptation literature (see (Herman, Blangero, et al., 2013; Hopp & Fuchs, 2006; Pelisson et al., 2010) for reviews) that we will now discuss.

The paradigms we implemented had strong effects on the saccade amplitudes in response to the post-saccadic errors induced by the ISS, but these changes in saccade amplitude most often do not compensate fully for the ISS. This is apparent when computing the proportion of gain with respect to the ISS (Fig. 2). This incompleteness of adaptation has been previously reported and it has been proposed that two underlying components with different timescales are actually responsible for adaptation: a rapid one correcting only a fraction of the current retinal error and a second, slower one, retaining these changes of longer period of time and responsible for accumulating gain changes across time (Ethier et al., 2008b; Miller et al., 1981; Robinson et al., 2003; Scudder et al., 2010). We therefore cannot rule out that, had we conducted these experiments for longer periods of time or even across several days, that adaptation would have been complete and the gain would have reached a level equivalent to its baseline value with respect to the post-saccadic target position. However, we think that, even within the relatively short time of our experiments, both the short- and the long-timescale mechanisms were

instrumental in the gain change for at least two reasons. First, the changes in saccade gain did not rapidly wash out at the beginning of the post-adaptation phase. On the contrary, recovery was incomplete (see panels C of Fig. 3-6), revealing some retention of adaptation despite the absence of ISS for 200 post-adaptation trials. It should be noted that, given that we found uncorrelated but comparable rates in gain change for the adaptation phases and the recovery phases (panels F of figures 3-6), one should expect an incomplete recovery as we had twice as many adaptation trials as recovery trials. Second, it has been previously demonstrated that the gain changes induced by double step paradigms with a number of adaptation trials similar to ours might lead to long term (at least five days) modifications of saccade amplitudes (Alahyane & Pelisson, 2005).

We found a systematic relation between the baseline gains and the adapted gains as evidenced by the positive correlation we observed in all four experiments (see also panels D for Fig. 3-6), as others have reported before (e.g., (Bahcall & Kowler, 2000; Wong & Shelhamer, 2011)). This relation is consistent with the conventional view stating that saccadic adaptation tends to maintain a gain close to its baseline value despite changes in the environmental contingencies rather than fully canceling out the post-saccadic position-error, which served as the basis for the prediction error hypothesis (Alahyane & Pelisson, 2005; Chen-Harris et al., 2008; Wong & Shelhamer, 2011, 2014) postulating that adaptation is driven by the difference between the expected and actual visual error after the saccade. This is also consistent with the observation that the baseline gain is generally hypometric (Becker, 1989; Henson, 1978) – a feature present in our data with baseline gains close to 0.9 in all experiments – as the mechanisms at play during adaptation would otherwise lead to a gain of 1 in all saccades.

When considering the post-adaptation trials, we observed consistent recovery from adaptation in all experiments: saccadic amplitudes gradually changed in the direction of the baseline values. Recovery was generally incomplete as the post-adaptation gain changes did not reach zero (see panels C of Fig. 3-6). Interestingly the rates of change for recovery was not correlated to the adaptation rates. Moreover, we did not find that the recovery rates were higher than the adaptation rates but rather that they depended whether the amplitude had to increase or to decrease to reach the baseline level (see panels F of Fig. 3-6) as we further discuss below.

Saccade adaptation results from an accumulation across trials following the introduction of an ISS. However, it has been proposed that an immediate, and possibly strategic, adjustment of amplitude might occur leading to a sudden change in gain within the first adaptation trials as

observed, for instance, when using paradigms with fixed target positions combined a perceptual task rather than an ISS (Schutz, Kerzel, & Souto, 2014; Schutz & Souto, 2015). Perceiving the ISS might be responsible for a strategic adaptation and, although we did not measure the rate of ISS detection, previous researches indicate that it should range around 10-25% in our case (Deubel, Wolf, & Hauske, 1986). However, several effects favor an absence of strategic gain change in our experiments. The differences between the early adaptation gain (computed across the first 10 adaptation trials) and the late baseline gain (computed across the last 10 baseline trials) were inconsistent across subjects and also across paradigms (see panels E of Fig. 3-6). We also established the absence of systematic sudden changes in saccade gain during adaptation in relation to gain decrease or gain increase demands. This should not be the case if adaptation was strategic. Moreover, we did not observe a systematic difference in saccade latencies between the baseline and adaptation trials (Fig. 7.F). Finally, a strategic adaptation should be followed by a very rapid, if not instantaneous, recovery. In our experiments, recovery from adaptation developed over time, as revealed by the fact that recovery rates were comparable to the adaptation rates (see panels F of Fig. 3-6). When considering these effects one might conclude that the adaptation we observed resulted from a true learning process rather than being strategic.

We did not explicitly probe the specificity of adaptation, which requires introducing test trials with target-step directions different from the one used to induce adaptation. However, we were able to confirm the lack of transfer of amplitude change across the saccade components by comparing the effects on the vertical and horizontal components of the saccades: changes in the non-adapted component (i.e. vertical in the backward and forward experiments and horizontal in the downward and upward experiments) were inconsistent across subjects (Fig. 7.H). Previous results are consistent with our finding (Watanabe et al., 2003). This reveals that the ISS specifically targeted the amplitude of one saccade component, an observation consistent with the well-established characteristics of adaptation-transfer across saccades (Collins, Dore-Mazars, et al., 2007; Frens & van Opstal, 1994).

All in all, we confirmed with our cross-axis paradigms some of the main features of adaptation induced with on-axis paradigms, a similarity that has been proposed before (Hopp & Fuchs, 2004). Indeed, we found that both adaptation and recovery were incomplete, that the adapted gain is well correlated with the baseline gain, that the adaptation rate is higher for gain decrease than for gain increase conditions, and that a similar amount of adaptation may be

obtained in gain decrease and gain increase adaptation, if enough adaptation trials are used. Moreover, as previously discussed, we find that the changes we report should not be attributed to a voluntary – possibly strategic – adjustment in saccade gain but instead should be regarded as the outcome of a true learning process. Although more experimental work is needed, the similarities in behavioral changes in on-axis and in cross-axis experiments has been established before either by intra-individual comparisons (Noto et al., 1999) or by comparing different experiments (Deubel, 1987; Hopp & Fuchs, 2006). The existence of similar adaptation fields in the two paradigms has also been demonstrated (Deubel, 1987; Noto et al., 1999). These results indicate that in both class of paradigms, the underlying process involved in saccade adaptation might be considered as identical. We are therefore confident that our observations might generalize to other double-step paradigms rather than being specific to cross-axis adaptation of oblique saccades.

Effects on saccade metrics

It is still debated whether the changes in amplitude induced by introducing an ISS might be systematically related to changes in other saccade parameters. However, we did not find consistent changes in the parameters we tested. Specifically, the variability in saccade amplitude was not consistently different in the baseline and in the adaptation phase as evidenced when contrasting the coefficient of variations in gains for the baseline and adaptation trials (Fig. 7.G), contrary to what has been previously reported (Erkelens & Hulleman, 1993). Moreover, the changes in saccade latencies when comparing the baseline trials to the adaptation trials were often absent or inconsistent (Fig. 7.F). This lack of systematic effect is interesting as, if adaptation was voluntary, one might expect to see increased latencies when the ISS is implemented.

When probing the effects of adaptation on the saccade main sequence we did not observe any systematic effect on the vectorial peak velocity at the individual level and the changes were similar when comparing the gain decrease and gain increase experiments (Fig. 7.A). This lack of systematic effect was also true when considering the saccade duration (Fig. 7.B). Taken together, these results indicate that the main sequence was mostly preserved during saccade adaptation. However, when considering independently the horizontal and vertical peak velocities we found significant changes at the group level: peak velocities in the adapted component tend to decrease

while peak velocities in the non-adapted component tend to increase in the gain decrease experiments (Fig 7.C); the opposite pattern was found in the gain increase experiments (Fig 7.D). It is important to point out that, although these effects were significant at the group level, at the individual level only a fraction of our participants exhibited these particular patterns (21 out of 50 in the gain decrease experiments, 17 out of 50 in the gain increase experiments). Adaptation completeness was significantly correlated with these changes in the peak velocity of the adapted component (negative correlation in the gain decrease experiments, positive correlation in the gain increase experiments) but not with the ones in the non-adapted components.

This overall pattern of effects is consistent with the hypothesis that saccade adaptation occurs at a stage where the saccade command is represented as a vector rather than where it is represented as its components (Hopp & Fuchs, 2006). Indeed, if adaptation occurred after the decomposition of the saccade command into components one would expect changes in the peak velocity affecting specifically the adapted component, leaving the non-adapted component unchanged. This would induce changes in the vectorial peak velocity as well, given that the saccade duration is preserved. That the adaptation site affects the vectorial command has been demonstrated by testing whether saccade adaptation of a component transferred to oblique saccades (Hopp & Fuchs, 2006; Noto et al., 1999; Wallman & Fuchs, 1998): the transfer of component adaptation is at best very limited, indicating that adaptation affects the vectorial saccade command. Our results further support the view of an adaptation site upstream from the components decomposition. Another argument favoring this possibility comes from van Gisbergen and collaborators (van Gisbergen, van Opstal, & Schoenmakers, 1985) who proposed a common-source model followed by a decomposition stage generating the two velocity-command signals which accounts for the deviations from the main sequence at the component level in oblique saccades. They found that the main-sequence relation was not fixed for each component but depended on the relative size of the orthogonal component (their Fig. 7): the peak velocity decreases in the smaller component while the saccade duration is preserved resulting in a stretching that depends on the saccade vector. This effect was further confirmed (Becker & Jurgens, 1990; Smit, Van Opstal, & Van Gisbergen, 1990). The peak velocity changes we observed are in line with this prediction. Taken together our data therefore support the possibility that saccade adaptation occurs before saccade component decomposition, where the motor signal

is represented as a vector. That these effects were significant in only a fraction of our participants has also been observed in adaptation-transfer experiments (Hopp & Fuchs, 2006).

Finding that the peak velocity changes were different in the adapted and non-adapted component prompted us to quantify the changes in saccade curvature. Two opposite patterns in the changes of saccade curvature might be expected. On the one hand, in regular oblique saccades in which one component is shorter than the other, one should expect a peak velocity decrease in the smaller component while the saccade duration should be preserved, resulting in a stretching that depends on the saccade vector. This should induce curvatures along the longer component. Applying this view to our experiments results in predicting more negative curvatures in experiments in which the horizontal component becomes longer than the vertical component (i.e. our downward and forward experiments) and more positive curvatures in experiments in which the vertical component becomes longer than the horizontal component (i.e. our upward and backward experiments). On the other hand, as it has been noted before (Smit & Van Gisbergen, 1990), curvatures depend on the ratio of the components-velocity profile at any moment during the movement. This implies that if the dynamics of the two velocity profiles were to be differentially affected by the adaptation process a different pattern of curvatures might be expected. Chen-harris and collegues (Chen-Harris et al., 2008) established that, during cross-axis adaptation, the changes in the velocity signals arrive late in the saccade's trajectory, an effect that has been regarded as the signature of an internal feedback monitoring process correcting the saccade trajectories as they are generated. Based on this hypothesis, one should expect more negative curvatures in the backward (because of the late velocity decrease in the horizontal component) and upward (because of the late velocity increase in the vertical component) experiments and more positive curvatures in the downward (because of the late velocity decrease in the vertical component) and forward (because of the late velocity increase in the horizontal component) experiments, i.e. the opposite pattern that is predicted for regular oblique saccades. The changes we observed in the saccade curvatures depended on the adaptation paradigm (Fig. 7.E) and were consistent with the hypothesis of forward models driving adaptation (Chen-Harris et al., 2008; Ethier et al., 2008a; Schubert & Zee, 2010). It is however noteworthy that these changes in saccade curvature were not correlated with adaptation completeness.

Changes in vectorial peak velocity during saccade adaptation have been previously reported (Catz, Dicke, & Thier, 2008; Ethier et al., 2008a; Prsa, Dicke, & Thier, 2010; Sun,

Smilgin, Junker, Dicke, & Thier, 2017) and they have sometimes been regarded as evidence supporting the role of fatigue in saccade adaptation (Prsa et al., 2010). For instance monkeys exhibited a decrease in saccade peak velocity together with increased latencies over the course of the first 300 trials of a fatigue experiment ((Prsa et al., 2010), their Figure 1B). However, we did not find a consistent effect on the vectorial saccade peak velocity during adaptation, a lack of effect that has been previously reported by others (Collins, Semroud, Orriols, & Dore-Mazars, 2008; Frens & van Opstal, 1994) (see also (Hopp & Fuchs, 2004; Pelisson et al., 2010) for further discussion). We also did not observe any consistent changes in saccade latencies. Regardless of the actual cause of these results, our data certainly cast doubts on the causal role sometime attributed to fatigue on saccade adaptation (Sun et al., 2017) because the measured changes in vectorial peak velocities were at best limited, inconsistent across participants, and inconsistent across experiments.

Differences across paradigms: gain decrease versus gain increase

It is generally proposed that the adaptation associated with amplitude increase differs from that of amplitude decrease as revealed by its larger amplitude modification and higher rate of gain change (see (Hopp & Fuchs, 2004; Pelisson et al., 2010) for further discussion). This proposition is particularly appealing because it is consistent with the observation that the saccadic system is usually hypometric (Henson, 1978), possibly to minimize the saccadic flight time and improve vision (Harris, 1995; Harris & Wolpert, 2006). Maintaining hypometria would require more gain change in gain decrease than in gain increase experiments as, in the latter, saccades become more hypometric when the ISS is introduced. It has been proposed that gain increases and decreases employ different behavioral adaptation processes, suggesting that amplitude increases rely on a remapping of the desired end position of saccades, whereas amplitude decreases are best described by an overall reduction in gain (Semmlow, Gauthier, & Vercher, 1989). Our data offer a more contrasted view on the differences between gain increase and gain decrease paradigms. First, the number of participants in whom we found a change in gain between the baseline and adaptation phase was only slightly lower in the gain increase experiments than in the gain decrease ones (46 vs. 49 out of 50). Second, when comparing the adaptation completeness, we found very similar values in the gain decrease and in the gain increase experiments (see Fig. 2 and panels C of Fig. 3-6). Third, when considering the

adaptation rates our results are nicely in line with the ones from previous studies as we observed significantly faster absolute rates in the gain decrease experiments than for the gain increase ones indicating that learning in a gain increase paradigm does indeed require more trials to accumulate (compare panels B and F of Fig. 3-6). It is also noteworthy that higher adaptation rates when decreasing than when increasing saccade amplitudes were further confirmed when comparing the recovery to the adaptation rates within experiments (see (Deubel et al., 1986) for a similar conclusion): for the two gain decrease experiments the rates in the post-adaptation phase, i.e. when the gain had to increase to return to baseline level, were significantly higher than the ones in the adaptation phase. The opposite pattern was true for the gain increase experiments although the difference was not significant. This effect is also apparent when plotting the recovery rates versus adaptation rates (panels F of Fig. 3-6): most data fall below the equality line. Overall, similar amounts of adaptation across experiments combined with different rates when decreasing or increasing the amplitude reconcile our present data with previous observation as, in our design, we had a larger number of adaptation trials (400) than what has often been used before. One could therefore speculate that others would have also found similar amounts of adaptation across paradigms had they use more adaptation trials. Finally, quantifying the differences in inter-individual variability in adaptation completeness between the gain decrease and gain increase experiments did not yield any significant differences (Fig. 2). Although the present data do not allow one to rule out the possibility of different underlying process for gain decrease and gain increase adaptation, our results strongly suggest that if there are indeed different processes involved they specifically affect the adaptation rates. Alternatively, one could postulate that the observed difference in adaptation rates is well accounted for by the hypometria hypothesis (Henson, 1978) as it is compatible with a higher rate for gain decrease paradigms (which initially decrease hypometria) and the lack of difference in the final adaptation completeness which might be attributed to the overall maintenance of a default hypometric state.

Differences across paradigms: vertical versus horizontal adapted components

One of the features of our experiments is that we used a variation of the “cross axis” paradigm to probe adaptation either in the horizontal or vertical saccade component independently using identical first target-steps. To our best knowledge our study is the first using this specific implementation of the double step paradigm. Importantly, the differences observed

between the horizontal and vertical experiments were not significant when considering the central tendencies of our group results: the mean change in gain were not significantly different in the two horizontal experiments when compared to the ones obtained in the two vertical experiments, nor were the mean rates of adaptation. Although others used oblique target step to induce adaptation (e.g. (Deubel, 1987; Hopp & Fuchs, 2006)) or probed adaptation in the horizontal or vertical saccade component independently (e.g. (Watanabe et al., 2003)) we could not find a quantitative comparison of variability in adaptation in each component. It is tempting to conclude from our data (see Fig.2) that the adaptation of the vertical component of an oblique saccade is more variable than the one of its horizontal components. However, when comparing the interquartile differences measured in the horizontal experiments versus the ones measured in the vertical experiments there were no significant differences. This is particularly interesting because we found an overall greater inter-individual variability in the vertical component. We quantified the inter-individual variability (in all 100 participants) in the mean horizontal gains for the last 100 baseline trials using the interquartile difference. When comparing this dispersion to the one in the mean vertical gains, we found that the dispersion was larger for the vertical than for the horizontal component (0.11 vs. 0.07, $p=0.03$), although the two gains were well correlated ($R=0.59$; $p<0.01$). The dependence of the horizontal and vertical saccade components has been previously quantified and modeled at the level of the individual movement (Becker & Jurgens, 1990; Deubel, 1987; Quaia & Optican, 1997; Vitu, Casteau, Adeli, Zelinsky, & Castet, 2017). However, to our best knowledge, there is no report of individual differences in the average horizontal versus vertical saccade gain for oblique saccades. Specific experiments are therefore needed to disentangle the respective contributions of the adaptation process and of the component-specific individual variations to the variability in adaptation.

Inter-individual differences

As we previously pointed out, an important feature of our data is that one finds great variations across subjects. For instance, the percent change in gain showed an eight-fold difference in the vertical gain increase experiments and a three-fold difference in the other ones. These strong inter-individual differences are also apparent in the other measures we reported (Fig. 2-7) albeit with various extents, and it is noteworthy that these differences across subjects should not be attributed to the implementation of our paradigms or the actual data collection: all

participants experienced the exact same first target-step in the same order, and all the data were collected on the same experimental setup, by the same experimenter. Although variations in adaptation should certainly be expected given the large individual differences found in eye movements as revealed by a recent study across 1000 young adults (Bargary et al., 2017), it is worth trying to connect the variability in adaptation to variability in other individual saccade features. To this end we reasoned that computing the degree of correlation between the amount of adaptation, in the form of percent change in gain, to some individual saccade characteristics collected in the 200 baseline trials might help understanding the underlying factors responsible for the individual differences in saccade adaptation. We therefore identified some candidate parameters that might be indicative of crucial individual differences and predict the amount of final adaptation such as (1) the average baseline gain, as it might relate to the individual tolerance for position error, (2) the variability in the baseline gain (expressed by the interquartile difference), as it has been proposed that some degree of variability might favor learning (Neuringer, 2002; Wu et al., 2014), (3) the peak velocity of baseline saccades, expressed using the main sequence parameter, (4) the variability of the vectorial peak velocity, measured by the interquartile difference of the residuals, (5) the baseline median saccade latency and (6) the variability in baseline saccade latency, measured by the interquartile difference, as these variables might be indicative of individual differences in saccade control. Autocorrelation in the saccade gains (i.e. the correlation between the gains with a one trial lag) for the baseline trials might also be very informative in that it reflects the tendency to correct the actual saccade based on the previous trial position-error and possibly the sensitivity to this error signal. However, we had to exclude the autocorrelation for it most often did not reach significance, as we previously reported, and was therefore a meaningless measure in this context.

None of the baseline saccade parameters we considered was correlated with either the amount of adaptation (Table 1) or the adaptation rate (Table 2). Although negative, this result is interesting in that it might point to a fundamental aspect of saccade adaptation. Indeed, one could regard this lack of correlation as pointing to other factors, unrelated to the fundamental characteristics of the saccadic system itself, to account, at least in part, for some of these individual differences in saccade adaptation. In support of this view there is ample evidence demonstrating that the significance of saccade adaptation extends beyond a simple recalibration process of the saccadic system. First, saccade adaptation depends on target selection as evidenced

by (1) the ability to selectively adapt in response to a target post-saccadic position while ignoring the position error associated with a similar distractor (Madelain et al., 2010), (2) the ISS causing adaptation when saccades are made between objects but not within objects (Collins, Vergilino-Perez, Beauvillain, & Dore-Mazars, 2007), (3) the displacements of the saccade target against a structured static background inducing gain changes while displacements of the background in the presence of a static target do not (Deubel, 1995; Madelain, Herman, & Harwood, 2013). Second, saccade adaptation is specific to the adapted direction and eccentricity (Frens & van Opstal, 1994) unless a specific paradigm is used to generalize adaptation (Rolfs et al., 2010). Moreover, saccade adaptation is sensitive to contextual features such as target eccentricity and depth (Chaturvedi & van Gisbergen, 1997), horizontal and vertical orbital eye position (Alahyane & Pelisson, 2004; Shelhamer & Clendaniel, 2002; E. Zimmermann & Lappe, 2011), head orientation (Shelhamer, Peng, Ramat, & Patel, 2002) or target motion (Azadi & Harwood, 2014). Finally, depending on the specific contingencies in force, saccadic adaptation might be triggered by visual information gain (Meermeier, Gremmler, & Lappe, 2017; Meermeier, Gremmler, Richert, Eckermann, & Lappe, 2017; Schutz et al., 2014; Schutz & Souto, 2015), or even non-visual reinforcement (Madelain, Paeye, & Wallman, 2011). In our view these results demonstrate that saccade adaptation is more general than previously thought (see (Herman, Blangero, et al., 2013) for similar discussion), relying on the individual learning abilities rather than on the specifics properties of the saccadic system. It is therefore possible that the individual differences we report here are attributable to general properties of the individual at the time of the experiment as, for instance, an ability to maintain accurate prediction (see (Wong & Shelhamer, 2014), a sensitivity to changes in environmental contingencies, the value attributed to the post-saccadic target (Chen-Harris et al., 2008; Meermeier, Gremmler, & Lappe, 2016, 2017; Meermeier, Gremmler, Richert, et al., 2017) or the ability to solve the credit assignment problem, i.e. to connect causes and effects (Staddon, 2001). Although our present data do not allow to disentangle between the respective contributions of the specific properties of the saccadic system on the one hand, and of more general learning abilities on the other hand, this question certainly constitutes an exciting new avenue for future research.

Conclusions

Our results clearly demonstrate that saccadic adaptation is highly consistent across individuals as we observed a change in saccade amplitude in response to an intra-saccadic target displacement in the vast majority of our participants, regardless of whether a gain increase or a gain decrease was induced, or whether the paradigm targeted the horizontal or vertical saccade component. However, there are also strong inter-individual differences that might not be entirely attributable to the individual characteristics of the saccadic system but might reflect more general learning abilities. Unraveling the precise origins of these individual differences constitutes a challenge that should shed a new light on this form of motor learning and further reveal the sensitivity of the saccadic system to the current state of the environment.

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Captions

Figure 1: General methods. A) Schematic diagram of the temporal sequence of trials used in our experiments. After a fixation period the target (white disk) steps (first step) at a 45° angle upward and to the right (vector amplitudes of 7.3, 9.5, 11.7 or 13.9°). As soon as a saccade onset is detected, the target steps again (here backward, by 20% of the first step horizontal component) and remains visible for 550 ms. The dark disks illustrate the previous target positions but were not visible in the actual experiments. B) Schematic diagram of the four intra-saccadic target steps implemented in separate experiments, using a between-participants design with 25 different naïve participants in each adaptation direction. P0 (i.e. the target position at fixation) was fixed throughout all experiments. C) Illustration of the piecewise model used to estimate the adaptation rate (starting at trial 200) and the recovery rate (starting at trial 600), and the last 100 trials of baseline and adaptation sessions that were used for calculating the adaptation magnitude (the last 100 trials of the recovery were also used for measuring the recovery magnitude). The gray dots plot the saccade amplitude for all trials expressed in percent gain change with respect to the baseline gain (see Methods section).

Figure 2: Adaptation completeness for each experiment. Each circle plots the individual adaptation magnitude as a relative proportion of the imposed ISS. Filled symbols indicate a change in gain greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions.

Figure 3: Results for the Backward experiment. A) Horizontal saccade gain for each trial in a single participant (black dots), and smoothed values (Lowess) for the horizontal (black) and vertical (grey) saccade component for the three experimental phases. The horizontal grey lines mark the first and last adaptation trials. B) Smoothed estimate of the percent change in horizontal gain with respect to baseline for all 25 subjects. C) Individual percent change in horizontal gain for the adaptation and recovery trials (means and 99% CIs). The vertical grey line marks complete adaptation, the horizontal grey line marks complete recovery. D) Individual baseline and adaptation horizontal gain for all subjects (means and 99% CIs). The oblique grey line marks equal values. Filled symbols indicate a change in gain greater than the null hypothesis 99% CIs.

E) Individual late baseline (last 10 baseline trials) and early adaptation (first 10 trials) horizontal gain for all subjects (means and 99% CIs). The oblique grey line marks equal values. F) Individual adaptation and recovery rate for all subjects. The oblique grey line marks identical rates.

Figure 4: Results for the Forward experiment. Panels same as figure 3.

Figure 5: Results for the Downward experiment. Panels same as figure 3 except for: A) Vertical saccade gains (black dots) and smoothed values (Lowess) for the vertical (black) and horizontal (grey) saccade component. B) Smoothed estimate of the percent change in vertical gain. C) Individual percent change in vertical gain. D) Individual baseline and adaptation vertical gain. E) Individual late baseline (last 10 baseline trials) and early adaptation (first 10 trials) vertical gain F) Individual adaptation and recovery rate for all subjects.

Figure 6: Results for the Upward experiment. Panels same as figure 5.

Figure 7: Saccade changes across experiments. A) Difference between the actual and predicted vectorial peak velocity in adaptation trials based on the vectorial peak velocity-amplitude fits obtained in the baseline trials for all subjects in all experiments (see Methods). Filled symbols indicate a change in velocity greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. B) Difference between the actual and predicted saccade durations in adaptation trials based on the duration-amplitude fits obtained in the baseline trials for all subjects in all experiments (see Methods). Filled symbols indicate a change in duration greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. C) Difference between the actual adapted and predicted component peak velocity in adaptation trials based one the adapted component peak velocity-amplitude fits obtained in the baseline trials as a function of the difference between the actual and predicted non-adapted component peak velocity in adaptation trials based on the non-adapted component peak velocity-amplitude fits obtained in the baseline trials for all subjects in the two gain decrease experiments (see Methods). Filled symbols indicate a change in velocity greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. D) Same as panel C for the two

gain increase experiments (see Methods). E) Difference in median saccade curvatures between the adaptation and baseline trials for all subjects in all experiments. Filled symbols indicate a change in curvature greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. F) Difference in median saccade latencies across the adaptation and baseline trials for all subjects in all experiments. Filled symbols indicate a change in latency greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. G) Difference in coefficient of variation in gain across the adaptation and baseline trials for all subjects in all experiments. Filled symbols indicate a change greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions. H) Difference in saccade gain for the saccade component untargeted by the ISS, across the adaptation and baseline trials for all subjects in all experiments. Filled symbols indicate a change in gain greater than the null hypothesis 99% CIs. Boxplots mark the three quartiles of the group distributions.

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Figure 1
Methods

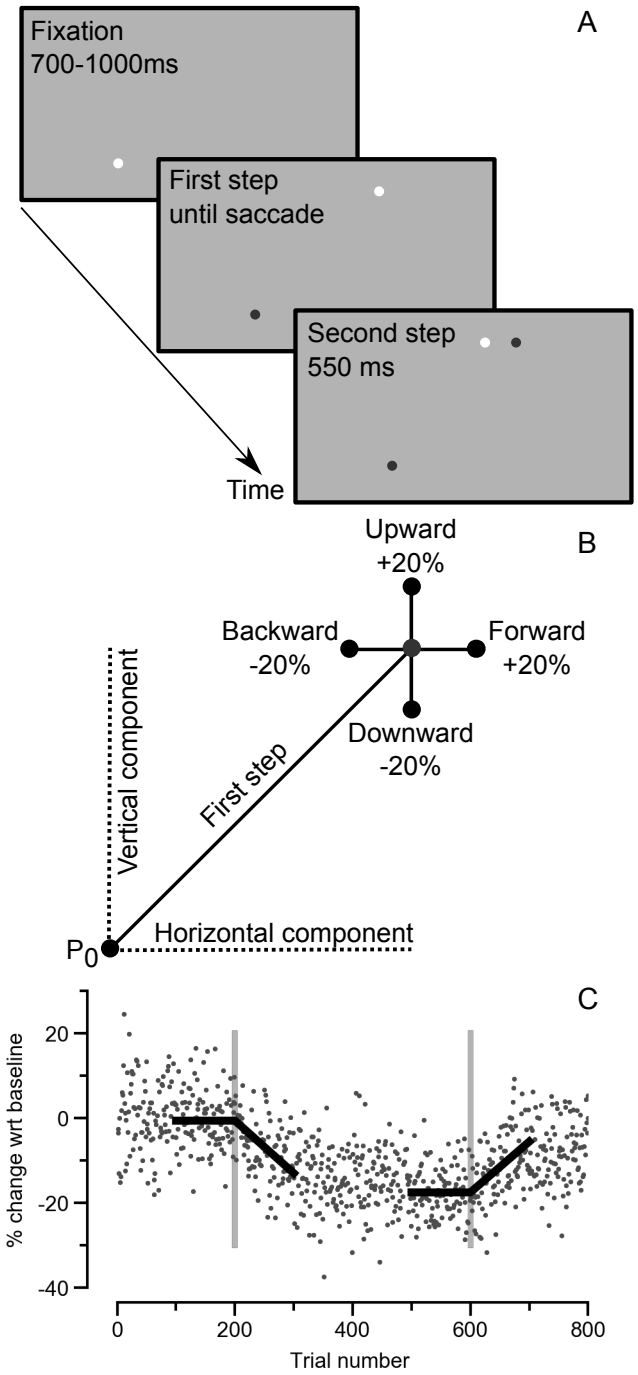

Figure 2
Adaptation completness

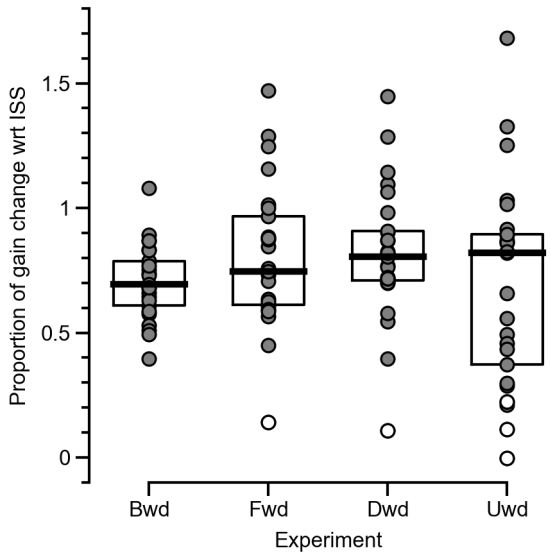

Figure 3
Backward experiment

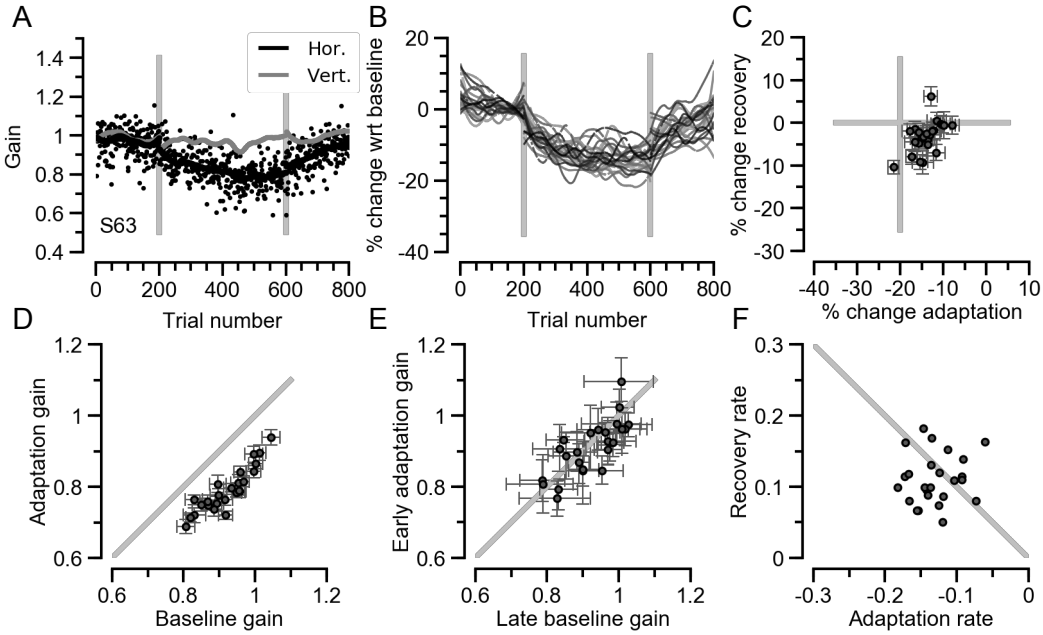

Figure 4
Forward experiment

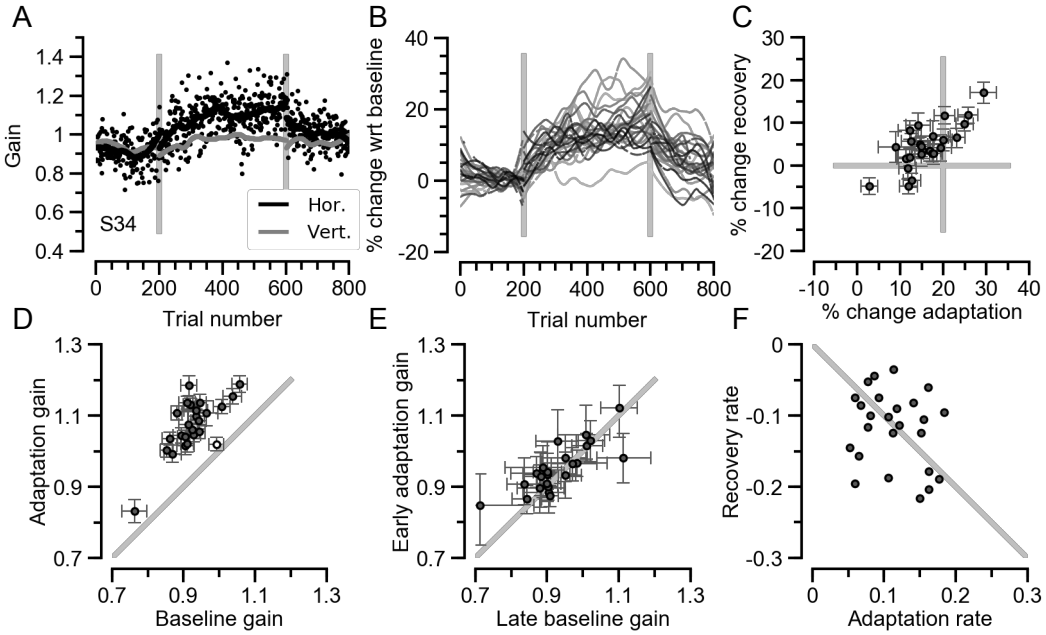

Figure 5
Downward experiment

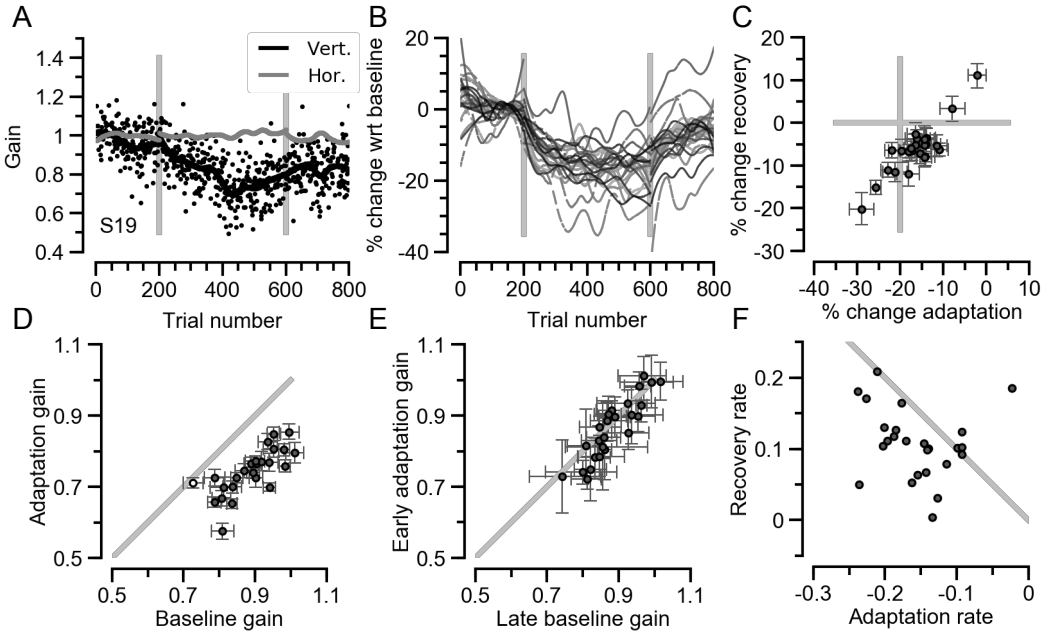

Figure 6
Upward experiment

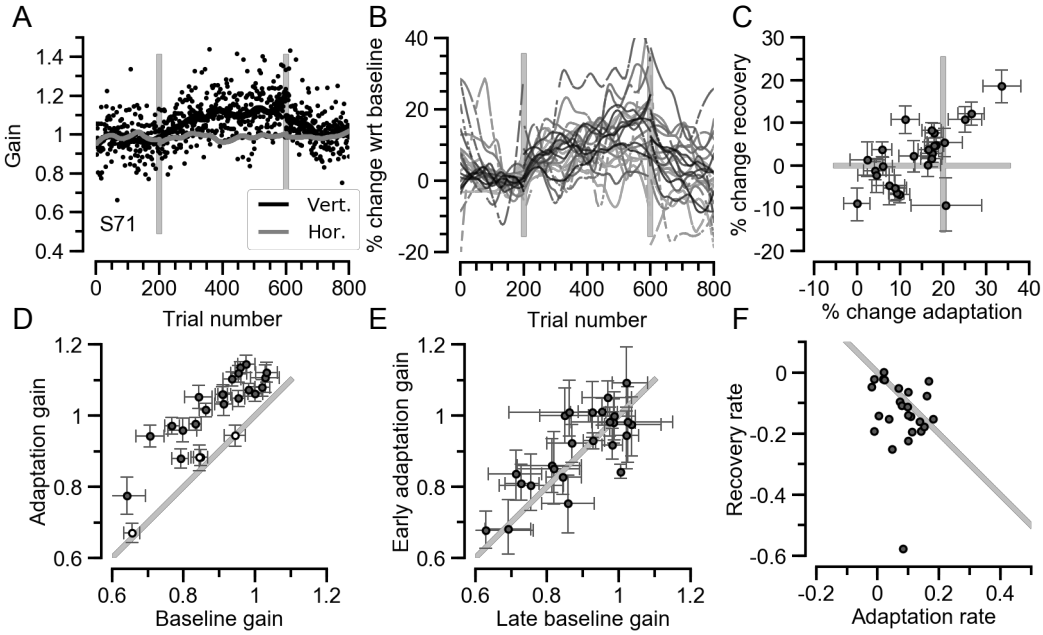

Figure 7
Saccade changes