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RESEARCH ARTICLE

Effects of saccade training on express saccade proportions, saccade latencies, and peak velocities: an investigation of nasal/temporal differences

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Abstract
Express saccades have very short latencies and are often considered a special population of saccadic eye movements. Recent evidence suggests that express saccade generation in humans increases with training, and that this training is independent of the actual saccade vector being trained. We assessed the time course of these training-induced increases in express saccade generation and how they differ between the nasal and temporal hemifields, and second whether they transfer from the trained to the untrained eye. We also measured the effects of training on saccade latencies more generally, and upon peak velocities. The training effect transferred between the nasal and temporal hemifields and between the trained and untrained eyes. More surprisingly, we found an asymmetric effect of training on express saccade proportions: Before training, express saccade proportions were higher for saccades made into the nasal hemifield but with training this reversed. This training-induced asymmetry was also observed in overall saccade latencies, showing how training can unmask nasal/temporal asymmetries in saccade latencies. Finally, we report for the first time that saccadic peak velocities increased with training, independently of changes in amplitude.

Keywords Nasal temporal asymmetry · Express saccades · Training effect · Ex-Gaussian analyses · Latency distribution

Introduction

Saccadic eye movements made in response to suddenly appearing stimuli can be used as a model system for understanding how sensory responses translate into motor commands. Saccades are very fast eye movements with peak velocities reaching about 500°/s (Bahill et al. 1975; Collewijn et al. 1988; Jóhannesson and Kristjánsson 2013; Leigh and Zee 2007). Saccades made in response to the sudden appearance of a visual stimulus have been classified as either express saccades, with latencies of less than 120 or 130 msec, or regular saccades, with longer latencies (Delinte et al. 2002; Fischer and Ramsperger 1984; Heeman et al. 2017), but whether they should be considered a distinct population of saccades is debated (Heeman et al. 2017; Jüttner and Wolf 1992; Kingstone and Klein 1993; Wenban-Smith and Findlay 1991). Saccades with latencies shorter than 70–80 ms are generally considered anticipatory and not made in response to an appearing stimulus (Bompas et al. 2008; Delinte et al. 2002; Fischer and Weber 1992).

Neurophysiological work in monkeys suggests that express saccades are initiated when visual signals are directly transformed into motor commands in the superior colliculus (Dorris et al. 1997; Edelman and Keller 1996, 1998). Although express saccade latencies are close to the minimum response time for the shortest pathway leading from the retina to cerebral cortex, and down to the oculomotor plant (Bibi and Edelman 2009; Carpenter 1981; Dorris et al. 1997), they are, nevertheless, not purely stimulus driven, or "bottom–up". They can be influenced by "top–down" processes such as visuomotor set (i.e., a process in the sensorimotor network that converts visual stimulation into motor command; Edelman et al. 2007, 2017), and saccade training (Bibi and Edelman 2009).

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Express saccades are more likely to occur in the so-called gap paradigm, where a fixation point disappears before the target appears (typically ~200 ms; see e.g., Fischer and Ramsperger 1984). The "gap-effect" involves shortened saccade latencies for both humans (Jóhannesson et al. 2013; Kristjánsson et al. 2001; Reuter-Lorenz et al. 1991; Saslow 1967; Takagi et al. 1995) and monkeys (Dorris and Munoz 1995; Fischer and Boch 1983), compared to when the offset of the fixation point and the onset of the target coincide or overlap (Ross and Ross 1980; see; Kristjánsson 2007, 2011 for review). When the fixation point disappears, the activity of neurons encoding fixation or small fixational movements in the rostral superior colliculi (SC) is reduced, potentially increasing express saccade production (Dorris and Munoz 1995; Krauzlis 2003).

Bibi and Edelman (2009) found that long-term saccade training on a gap paradigm increases express saccade production in humans and that the impact of training in one direction (e.g., horizontal) generalizes to other directions (e.g., vertical or oblique). The previous work on monkeys showed that the training of saccades of a particular direction and amplitude increased express saccade frequency (Boch and Fischer 1986; Fischer and Ramsperger 1984; Schiller and Haushofer 2005). If vector-specific motor preparation was necessary for express saccade generation, then training in one direction would not increase express saccade frequency in other directions. Rather, the results of Bibi and Edelman (2009) suggest that increased express saccade frequency results from fixation disengagement or, perhaps, interactions between motor preparation and fixation disengagement.

Visual input to saccade generation systems from the nasal and temporal hemifields

Visual inputs to the retina project to saccade-related areas in the brainstem and cortex via both the geniculostriate pathway through the lateral geniculate nucleus (LGN) as well as the retinotectal pathway through the superficial layers of the SC to the pulvinar and visual cortex (White and Munoz 2011). For each eye, the temporal visual hemifield is projected onto the nasal hemiretina, whereas the nasal hemifield is projected onto the temporal hemiretina. Input from the eye to visual cortex, whether via the geniculostriate or retinotectal pathways, is uncrossed from the temporal retina of each eye to the ipsilateral visual cortex and crossed from the nasal retina of each eye to the contralateral visual cortex. The ratio between projections from the nasal (crossed projections) and temporal (uncrossed projections) retina in the optic nerve is about 1.54 favoring the nasal retina in macaque monkeys (Williams et al. 1995), both for projection to the SC and the LGN. Therefore, crossed connections (from the nasal retina) both in the geniculostriate and the retinotectal pathways are thought to be stronger than uncrossed connections (from the temporal retina), perhaps, especially for the retinotectal pathway, meaning that, for each eye, the temporal hemifield has stronger input to visual cortex and the SC than the nasal hemifield (Hubel et al. 1975; Itaya and Van Hoesen 1983; Sterling 1973; Tigges and Tigges 1981; but see; Williams et al. 1995).

This raises the question of whether differences in connection strength may result in a visual, behavioral, or attentional advantage for visual stimuli presented in the temporal hemifield. There is, indeed, considerable evidence for attentional nasal–temporal asymmetries (NTAs) favoring the temporal visual field (Dodds et al. 2002; Rafal et al. 1991; Walker et al. 2000). Similar findings have been observed for saccadic latency in some studies (Kristjánsson et al. 2004; Walker et al. 2000), but not others (Bompas and Sumner 2008; Honda 2002; Jóhannesson et al. 2012). NTAs in latency may, therefore, depend upon specifics of the stimuli. For example, Bompas et al. (2008) found the latency of saccades into the nasal hemifield to be shorter than into the temporal hemifield for S-cone stimuli but not for luminance stimuli. In addition, distractors that appear in the temporal hemifield increase latency more than distractors in the nasal hemifield (Walker et al. 2000). Jóhannesson and Kristjánsson (2013) found higher peak velocities (PV) towards stimuli projecting to the nasal hemiretina, suggesting that NTAs exist for at least some saccadic parameters.

Since training can reduce saccade reaction times (Bibi and Edelman 2009; Knox and Wolohan 2015), this leaves open the possibility that the lack of training acts, in effect, as a limiting step in determining reaction times. It is possible that training could unmask existing differences in saccade reaction times to stimuli in the temporal and nasal hemifields. It is well known that the movement control systems of the two eyes in the brain stem are tightly coupled (Leigh and Zee 2007; Wurtz and Kandel 2000) and the deprivation of visual input from one eye might, therefore, affect the expected training effect. We note that our discussion is heavily influenced by preceding studies of the gap paradigm and express saccades, and cannot be taken out of the context of that literature.

Investigating the time course of reduction of saccade reaction time with training and its possible locus in the monocular visual system

It is possible that training might not just unmask nasal–temporal asymmetries in saccade latency, but eventually lead to floor effects, causing all latencies to decrease to a minimal value and thus, in a sense, *remask* NTAs. Therefore, we analyzed latency data not just before and after training, but at an intermediate stage during training. Moreover, as examining saccadic latency NTAs requires recording eye

movements during monocular viewing, we were in a position to determine whether monocular components of the visual system may contribute to reduction in saccadic latency. It is well known that visual pathways from the retina to V1 in primates are essentially monocular, with cells responding to stimuli from only one eye. If improvement in saccadic reaction time is due to changes in monocular components of the visual-saccadic pathway, then improvement caused by viewing with one eye should not transfer to the other eye.

Current questions

We investigated the effects of training upon saccade generation with the aim of casting light on the following issues: (1) whether differences between latencies of saccades into the nasal and temporal hemifields emerge during and after saccade training. (2) Whether increases in express saccade production due to training transfer from the "trained" eye to the "untrained" eye. (3) Whether these same training manipulations affect three other measures of saccadic performance: amplitude, peak velocity, and overall saccadic latency. In particular, we were interested in corroborating and extending our previous work that showed an NTA in saccade peak velocity (Jóhannesson and Kristjánsson 2013).

We trained observers over several sessions in making saccades to two possible target locations (left and right of fixation: amplitude = 8°) in a gap paradigm (Ross and Ross 1980; Saslow 1967). For consistency across participants, the dominant eye was trained (the non-dominant eye patched; see Fig. 1).

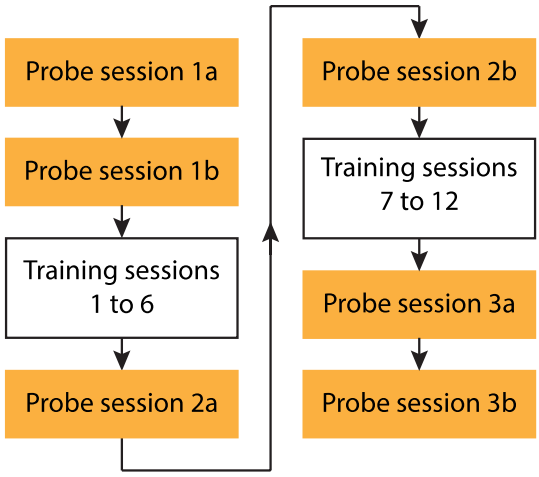

Fig. 1 Training progression through the experiment from probe session 1a to probe session 3b. Each session consisted of 10 blocks of 28 trials. Within the training sessions, the dominant eye was measured for consistency across participants. In probe sessions 1a, 2a, and 3a, the trained eye was measured; and in probe sessions, 1b, 2b, and 3b, the untrained eye was measured. In all cases, the non-measured eye was patched

Methods

Participants

Participants were 9 unpaid volunteers (3 females) who were naïve to the purpose of the study. All had normal, or corrected-to-normal vision. Mean age was 23.8 years (range 22–27 years). All but one had a right dominant eye. Informed consent was obtained from all individual participants included in the study. The study was conducted in accordance with the Declaration of Helsinki and approved by the appropriate Ethics Committee.

Apparatus

A high-speed, video-based eye-tracker from Cambridge Research Systems tracked gaze at 250 Hz (spatial accuracy 0.125°–0.25°). Stimuli were displayed on a 100 Hz 19″ Hansol CRT screen (model: 920D resolution: 1024 × 768) controlled by a 2.33 GHz PC (Windows 7; RAM = 4 Gb). Viewing distance was 60 cm (stability ensured with head rest). The experimental program was written in Matlab. Functions from the Psychtoolbox (Brainard 1997; Kleiner et al. 2007; Pelli 1997) and the Eyetracker Toolbox (Cambridge Research Systems 2006) were used to control stimulus presentation and data collection.

Stimuli

The fixation stimulus was a 1° black (0.22 cd/m^2) dot at screen center. The targets were 1° black (0.22 cd/m^2) dots that appeared after the fixation stimulus disappeared (following a gap, see procedure), randomly 8° either to the left or right. The screen background was light-grey (85.67 cd/m^2).

Procedure

Each trial began with the onset of a central fixation spot which was visible for a random period between 800 and 1100 ms. During this period, the system monitored fixation, informed the participant if fixation was outside the boundaries of the fixation spot, and repeated the fixation period. After a successful fixation, a blank screen (the gap) was displayed. The gap had a base time of 200 ms that varied randomly by ± 10, 20, or 30 ms. In a pilot study, we found that a gap time of 200 ms reduced latency and elicited more express saccades than other gap durations, in line with what is typically reported in the literature (see, e.g., Fischer and Ramsperger 1984). After the gap, the peripheral target was displayed (randomly to the left or the right) and participants had to make a saccade to it and fixate it for at least 500 ms.

After the system confirmed the minimal fixation time, the trial ended with a 500 ms blank intertrial interval. Each participant came nine times to the lab and participated in two sessions of 10 blocks (with a short break between sessions) on each occasion, or a total of 18 experimental sessions. Each block consisted of 28 trials. The session sequence is shown in Fig. 1. To assess the effects of saccade training, we used probe sessions where we measured saccade performance for both the trained and untrained eyes. There were three probe sessions (at the start, middle, and end of training, see Fig. 1). The dominant eye was measured first (probe session 1a, 2a, and 3a; the non-dominant eye patched) followed by the non-dominant eye (probe session 1b, 2b, and 3b; the dominant eye patched). There were 12 training sessions, 6 between probe sessions 1a/b and 2a/b, and 6 between probe sessions 2a/b and 3a/b (see Fig. 1). During the training sessions, only the dominant eye was trained (the non-dominant eye patched) and its movements recorded. Participants were trained every other day, with some exceptions where they were not able to attend during weekends. The testing spanned 17 days on average. To determine which eye was dominant, participants pointed with one finger at a vertical, narrow object 4 m away. While pointing, they first closed one eye and then the other and compared the virtual movement of the object. If the virtual movement was less when they closed their left eye than their right eye, we concluded that the right eye was dominant (Greenberg 1960). The eye tracking system was calibrated at the beginning of each session and after five blocks. Participants were instructed to move their eyes as quickly and accurately as possible.

Reaction time curve-fitting procedure

The ex-Gaussian distribution is a convolution of a Gaussian and an exponential distribution that can be expressed as a function of three parameters: μ (mu) and σ (sigma) denote the mean and standard deviation, respectively, of the Gaussian component of the distribution, and τ (tau) which denotes the mean of the exponential component (Dawson 1988; Matzke and Wagenmakers 2009; Ratcliff 1979, 1993). The distribution has a longer tail on the right (long RTs) than the left, fitting latency distributions well (Dawson 1988; Hockley 1984; Hohle 1965; Jóhannesson et al. 2013; Kristjánsson and Jóhannesson 2014; Palmer et al. 2011; Ratcliff 1979; Ratcliff and Murdock 1976). To evaluate the effects of training, we fit the ex-Gaussian distribution (using the mexgauss() function from the retimes package in R; Massidda 2013) to the latencies separately for each session. Antoniades et al. (2013) have argued that it is good practice to present distributional analyses of saccade latencies as they carry more information than measures of central tendency and dispersion on their own. As the tau parameter characterizes the size of the tail of the distribution, characterizing reaction time distribution with an ex-Gaussian fit is particularly useful for understanding the effects of training on reaction time, as it can provide insight into whether one effect of training is to eliminate unusually long reaction times.

Statistical analyses

In all analyses, we used the R statistical program (R Core Team 2014) running within the RStudio environment (RStudio Team 2015). We used linear mixed models (lme4; Bates et al. 2015) to analyze latencies and peak velocities of the saccades. In all models, the effect of participants was random, while we allowed the slope of the relevant factor in each analysis to vary. Furthermore, we used sliding contrasts (the contr.sdif() function of the MASS package; Venables and Ripley 2002) to assess the significance of differences between levels of the relevant factors. When analyzing the results of the ex-Gaussian fits to the latency data, we used repeated-measures ANOVAs (aov; R Core Team 2013).

Saccades with latencies below 70–80 ms are generally considered anticipatory (see e.g. Bompas et al. 2008; Delinte et al. 2002; Fischer and Weber 1992). We, therefore, removed trials with latencies shorter than 75 ms and trials with saccades with amplitude smaller than 5° before any statistical tests. After this step, we ran the ex-Gaussian fitting procedure. However, before running the linear mixed models, we removed trials where latency deviated more than 3 SD from each individual's mean within each session and transformed the data using the Box–Cox method (Box and Cox 1964; the boxcox() function from the MASS package in R; Venables and Ripley 2002). The Box–Cox method moves the distribution closer to a Gaussian distribution and reduces risks related to skewness of the data. For all post-hoc comparisons, we used Tukey's honest significant difference test (Tukey HSD; R Core Team 2014) to adjust the p values with respect to multiple comparisons.

As we wished to address transfer of training, we do not report any separate statistics for the training sessions but only the probe sessions (see Fig. 1). We defined express saccades as saccades with latencies between 75 and 130 ms.

Results

Before analyses, 1591 trials (3.5% of the data) were removed because of signal loss. From the remaining data, we removed 1421 trials (3.3% of the remaining data), either with saccades shorter than 5° or latencies shorter than 75 ms (saccades with shorter latencies are considered anticipatory). For analyses with linear mixed models, we also removed trials where latency deviated more than 3 SD from each individual's mean within each session. We furthermore compared latency and proportion of express saccade generation

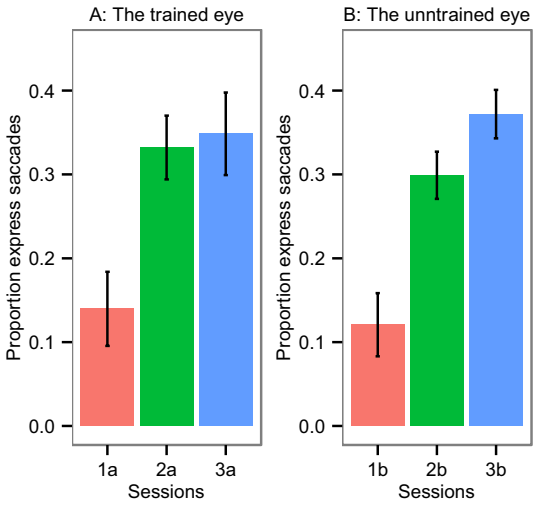

Fig. 2 Changes in the proportion of express saccades (latency < 130 ms) in the probe sessions. **a** Proportion of express saccades as a function of training (measured during probe sessions) for the trained (dominant) eye. **b** Proportion of express saccades as a function of training (measured at probe sessions) for the untrained (non-dominant) eye. Error bars represent 2 × within-condition SEMs

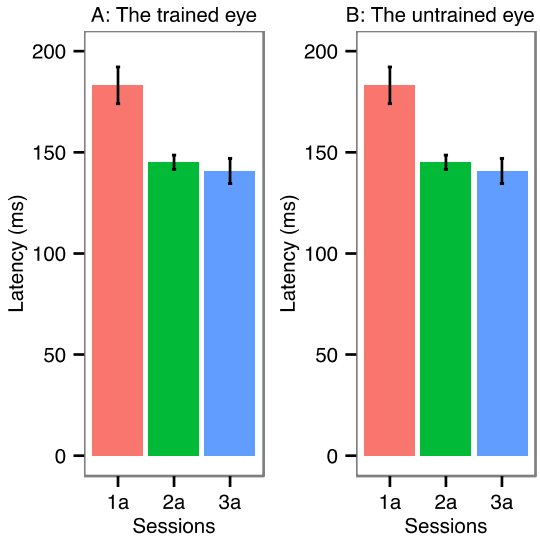

Fig. 3 Saccade latency as a function of training. The figure shows the mean latency of the saccades as a function of training measured in the probe session for the trained, dominant eye (**a**) and the untrained, non-dominant eye (**b**). Error bars represent 2 × within-condition SEMs

Table 1 Mean and standard devitation of express saccade generation as a function of training and the results of the linear mixed models

Probe session	Descriptive statistics		Model		
	Mean	SD	Slope	SE	*t* value
1a Trained eye	0.14	0.35	0.14[a]	0.037	3.75
2a Trained eye	0.33	0.47	0.19	0.060	3.19
3a Trained eye	0.34	0.48	0.21	0.078	2.68
1b Untrained eye	0.12	0.33	0.12[a]	0.026	4.71
2b Untrained eye	0.30	0.46	0.18	0.055	3.45
3b Untrained eye	0.37	0.48	0.25	0.056	4.50

[a]The intercept of the model

Table 2 Mean and standard deviation of saccade latencies as a function of training and the results of the linear mixed models

Probe session	Descriptive statistics		Model		
	Mean	SD	Slope	StErr	*t* value
1a Trained eye	183	59	183[a]	14.40	12.72
2a Trained eye	145	29	−38	11.24	3.38
3a Trained eye	141	25	−42	13.92	3.04
1b Untrained eye	174	47	175[a]	11.03	15.87
2b Untrained eye	146	28	−30	7.10	4.16
3b Untrained eye	140	25	−35	9.39	3.73

[a]The intercept of the model

between the trained and untrained eyes in all probe sessions finding no significant differences between the eyes, neither in latency ($t(8) = 0.62$, $p = 0.551$) nor express saccade generation ($t(8) = 0.53$, $p = 0.613$).

Express saccade proportions

The proportion of express saccades increased with training and this training effect transferred to the untrained eye. A linear mixed model with proportion of express saccades as the dependent variable and probe session as the independent variable showed a main effect of probe session for both the trained (all $t_s > 2.7$) and untrained eye (all $t_s > 3.2$), showing how the express saccade proportions increased with training between all probe sessions (see Fig. 2; Table 1 for detailed results).

Latencies

Linear mixed analyses with latency as the dependent variable and probe session as independent variable revealed that latencies decreased with increased training (see Fig. 3; Table 2 for detailed results). The training effect was significant between all probe sessions for both the trained (all $t_s > 3.0$) and the untrained eye (all $t_s > 3.7$).

Latency distributions

Training also affected latency distributions, reducing the maximum latency, resulting in truncated latency distribution tails, and shifting the distributions to the left (Fig. 4). Histograms for probe sessions 1a and 1b in Fig. 4 show that the distributions have long rightward tails. The remaining panels show how the distribution tails become shorter with increased training and that the peaks are fewer and clearer with the highest peaks between 125 and 145 ms for sessions 2a/b and 3a/b. Changes in mean and maximum latency suggest that both the shape and central tendency of the distributions change with training.

Ex-Gaussian analyses of latency distributions

The ex-Gaussian analyses show that the shifts in distributions, depicted in Fig. 4, are mainly consequences of changes in the sigma and tau parameters of the ex-Gaussian, because these parameters decreased significantly with training, while there was only a non-significant trend for the mu parameter (see below). It is still debated what each of these parameters may represent in latency or RT distributions, but, as argued in Palmer et al. (2011) and Kristjánsson and Jóhannesson (2014), the parameter estimates are useful for describing the distributions (Antoniades et al. 2013). We ran separate ex-Gaussian analyses for the trained and untrained eyes as a function of probe session (Fig. 5). The main effect of probe sessions on *mu* was significant for the trained ($F(2, 16) = 4.03$, $p = 0.038$) but not quite for the untrained eye ($F(2, 16) = 2.98$, $p = 0.079$). A post-hoc test revealed no significant differences (all $ps > 0.14$) for the trained nor for the untrained eye. The main effect of training on *sigma* was significant, both for the trained ($F(2, 16) = 5.53$, $p = 0.015$) and untrained eye ($F(2, 16) = 6.30$, $p = 0.001$). Post-hoc tests revealed a significant difference for the trained eye between probe sessions 1a and 3a ($p = 0.029$) and a marginally significant difference between probe sessions 1a and 2a ($p = 0.069$) but not between 2a and 3a ($p = 0.917$). For the untrained eye, the difference between sessions 1b and 3b was significant ($p = 0.019$) and very close to significant between 1b and 3b ($p = 0.053$) but not between sessions 2b and 3b ($p = 0.884$). The main effect of sessions on tau was significant for both trained ($F(2, 16) = 5.59$, $p = 0.014$) and

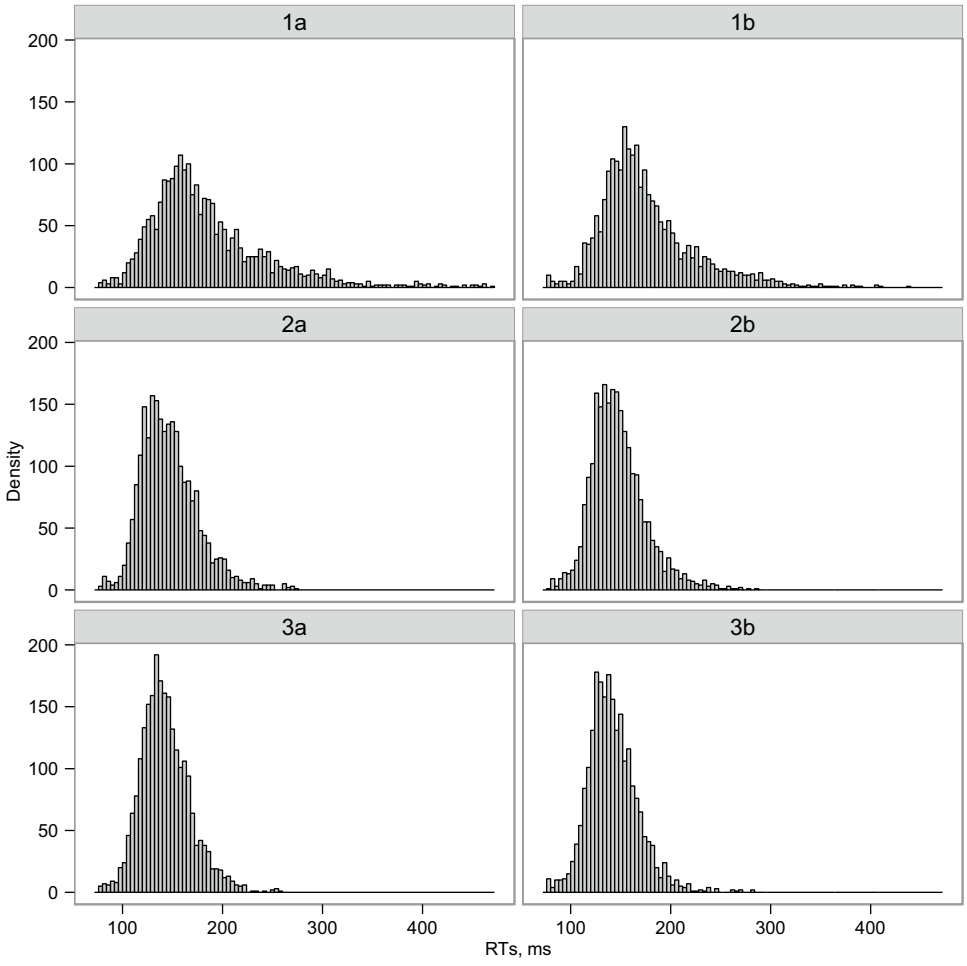

Fig. 4 Latency distributions for all participants from the three probe sessions (1–3) for the trained (**a**) and untrained (**b**) eyes. Panels 1a, 2a, and 3a show the distributions for the trained, eye and panels 1b, 2b, and 3b for the untrained eye for probe sessions 1, 2, and 3

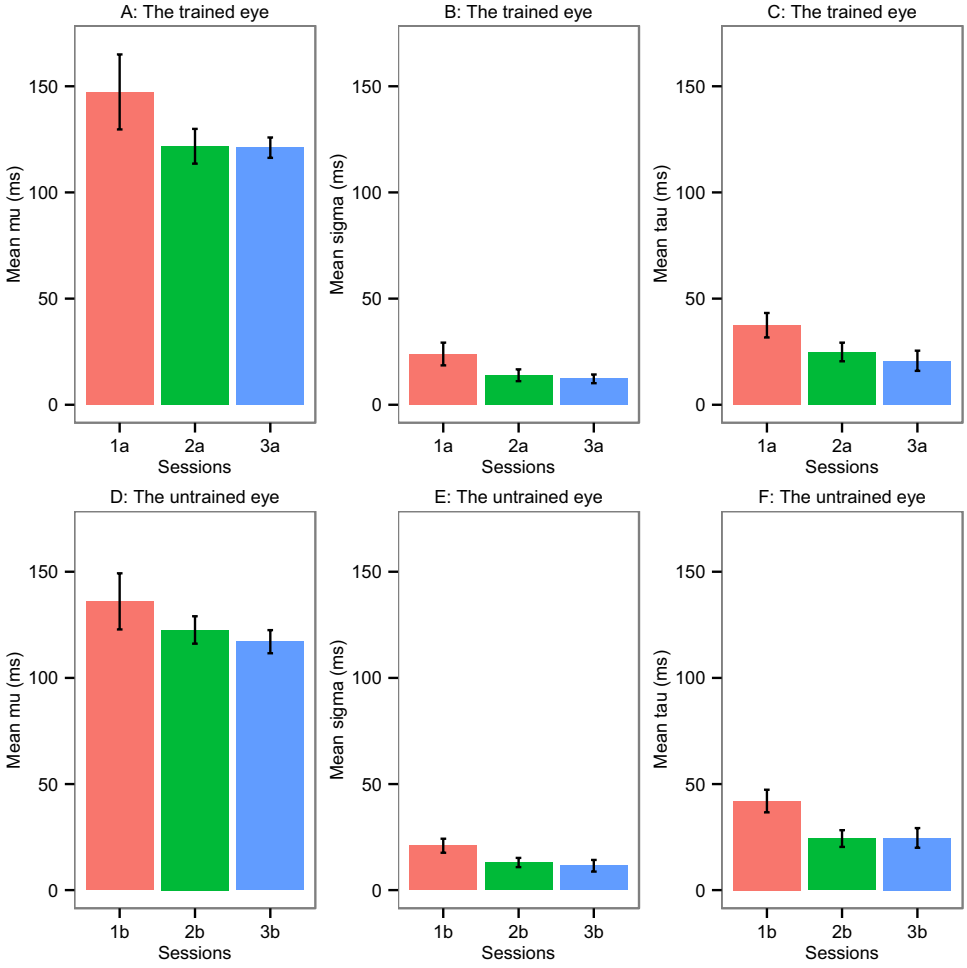

Fig. 5 Relationship between training and the values of all the three parameter estimates from the ex-Gaussian analyses (mu, sigma and tau) for the trained eye (**a**–**c**) and the untrained eye (**d**–**f**). Error bars represent 2 × within-participants' SEMs

untrained eyes ($F(2, 16) = 9.41$, $p = 0.002$). A post-hoc test revealed a significant difference between sessions 1a and 3a for the trained eye ($p = 0.021$) but neither between sessions 1a and 2a ($p = 0.096$) nor sessions 2a and 3a ($p = 0.759$). Post-hoc tests for the untrained eye revealed significant differences between sessions 1b and 2b and 1b and 3b ($p = 0.009$ and $p = 0.01$, respectively) but not between sessions 2b and 3b ($p \approx 1$).

Nasal–temporal asymmetries in express saccade generation and latency

The proportions of express saccades by hemifield and probe session are shown in Fig. 6. Saccade training affected express saccade proportion differentially for the different hemifields. Initially, the express saccade proportion was higher towards stimuli in the nasal hemifield, but with practice, this proportion became higher towards stimuli in the temporal hemifield. The increase was roughly threefold for the temporal hemifield but only twofold for the nasal hemifield. Logistic mixed regression analyses showed that the slopes of probe sessions 2a, 2b, 3a, and

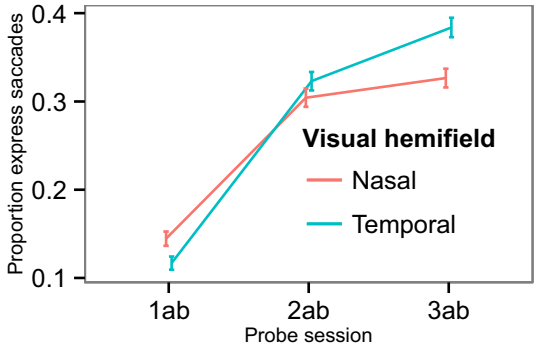

Fig. 6 Effects of training upon express saccade generation by hemifield. The figure shows the proportions of express saccades by hemifield and probe session

3b were significant (all $ps < 0.01$) but not of probe sessions 1a and 1b. The main effect of hemifield was not significant ($p = 0.67$). The interaction between probe session 1b and hemifield was significant ($p = 0.021$) and the interaction between probe session 3b and hemifield

was also significant ($p = 0.002$), confirming the patterns seen in Fig. 6. No other interactions were significant (all $ps > 0.13$).

We also separately analyzed latencies for regular and express saccades as a function of hemifield. In the whole data set, the average latency towards the nasal hemifield was 152 ms (SD = 77 ms) and 151 ms towards the temporal hemifield (SD = 81 ms). The mean latency of regular saccades towards the nasal and temporal hemifields was 164 ms (SD = 31 ms) and 163 ms (SD = 32 ms), respectively. The latencies of express saccades towards the nasal hemifield were 117 ms (SD = 11 ms) and 116 ms (SD = 11 ms) into the temporal hemifield (all $ts < 0.63$, n.s.).

However, this is not the whole story, since we also analyzed latencies for all saccades in the probe sessions as function of hemifield but now also as a function of probe session (see Fig. 7). While the main effect of hemifield was not significant (estimated difference: temporal–nasal = 0.4 ms, SE = 3.22, $t = 0.13$), there was a significant reduction of latency between probe sessions 1ab and 2ab (estimated difference (1ab–2ab) = 34 ms, SE = 9.101, $t = 3.75$). The interaction was between hemifield and probe sessions 1ab and 2ab (estimated effect—7.03 ms, SE = 1.254, $t = 5.61$) but not between hemifield and probe sessions 2ab and 3ab (estimated effect—1.45 ms, SE = 1.244, $t = 1.16$). Most notably, in probe sessions 1a and 1b, latencies towards the temporal hemifield were longer than towards the nasal hemifield, while in the later probe sessions, this was reversed. This result raises the intriguing possibility that latency differences between saccades into the nasal and temporal hemifields may have been masked by different levels of saccade training in the previous studies where no differences between saccade latencies into the two hemifields were found.

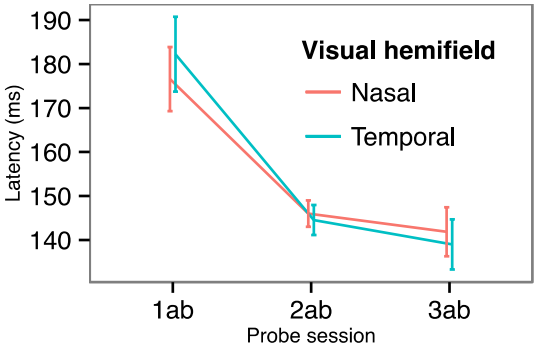

Fig. 7 Interaction between training and hemifield for saccade latencies. The figure shows how training differently affected latency with respect to visual hemifield. Error bars represent 2×within-condition SEMs

Amplitude gain

Amplitude gain (AG) is the ratio of the amplitude of the saccade to the distance from the center of the fixation point to the center of the target (Table 3). The average AG (for all saccades) for both the trained and untrained eyes was 0.91. The results from a linear mixed analysis with AG as the dependent variable and probe session as factor showed that the AG was significantly higher in probe session 3a than in probe session 1a for the trained eye but no significant effects of training were found for the untrained eye, see Table 3. The moderate correlation between peak velocity and AG was neither significant for the trained ($r = 0.42$, $t(7) = 1.21$, $p = 0.263$) nor the untrained eye ($r = 0.41$, $t(7) = 1.18$, $p = 0.277$). Furthermore, we found no significant difference in AG between the hemifields (paired $t(8) = 0.792$, $p = 0.792$).

Peak velocities

While we found no significant differences in peak velocities (for all saccades) between the trained and untrained eyes ($t = 0.6$; trained: M = 324°/s, SD = 78°/s; untrained: M = 322°/s, SD = 78°/s), the effect of training was significant for the trained eye. In probe session 1a, the peak velocity was 312°/s (SD = 73°/s), while the peak velocity was 334°/s in probe session 3a (SD = 81°/s). The training effect transferred to the untrained eye as the peak velocity in probe session 1b was 315°/s (SD = 78°/s) but 327°/s (SD = 79°/s) in probe session 3b. Linear mixed models with peak velocity as the dependent variable and probe sessions as factor showed that the differences between training sessions were always significant (all $t_s > 2.1$), see Fig. 8. We know of no other demonstration in the literature of training-induced increases in saccadic peak velocity. This modulation of saccadic peak velocities is in contrast with the classic assumption that peak velocities are constant as a function of saccade amplitude (Bahill et al.

Table 3 Mean and standard devitation of amplitude gain as a function of training and the results of the linear mixed models

Probe session	Descriptive statistics		Model		
	Mean	SD	Slope	StErr	*t* value
1a Trained eye	0.89	0.18	0.884[a]	0.023	37.76
2a Trained eye	0.90	0.18	0.016	0.013	1.29
3a Trained eye	0.93	0.16	0.046	0.014	3.37
1b Untrained eye	0.90	0.17	0.903[a]	0.024	38.29
2b Untrained eye	0.91	0.18	0.002	0.019	0.12
3b Untrained eye	0.92	0.17	0.012	0.011	1.09

[a]The intercept of the model

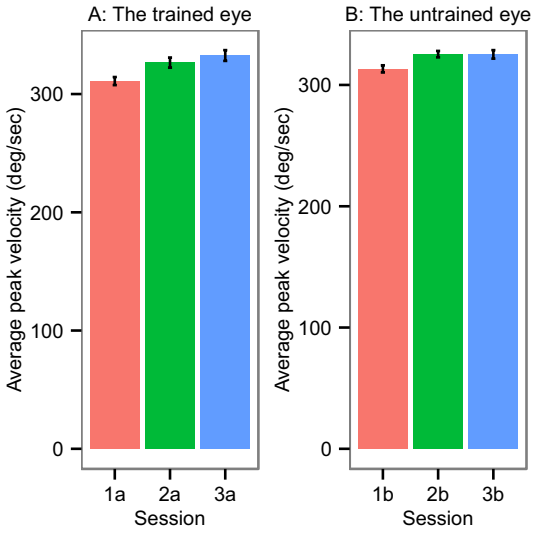

Fig. 8 Peak velocity and training. The figure shows the effect of training on the peak velocity of saccades during the probe sessions for the trained (**a**) and the untrained (**b**) eyes. Error bars represent 2 × within-participants' SEMs

1975; Collewijn et al. 1988). Given that the SC drives both latency and peak velocities, we measured whether there was a correlation between peak velocities and latencies, finding a significant negative correlation (− 0.78, $t(7) = 3.35$, $p = 0.012$) showing that with lower latencies, peak velocities increase (see, e.g., Edelman et al. 2006). Note that the small training effects on amplitude gain (described above) are unlikely to explain this difference.

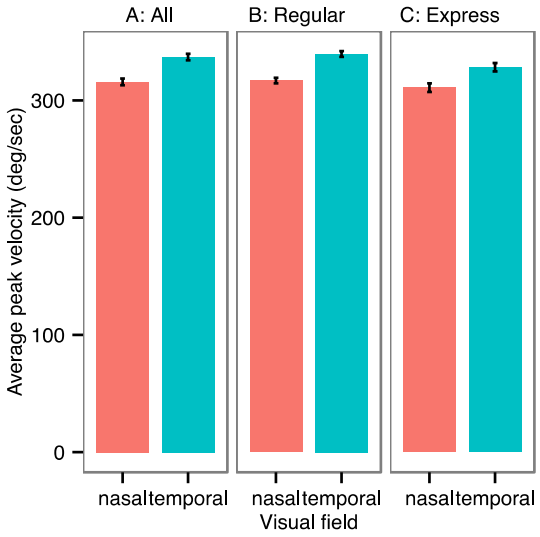

Fig. 9 Nasal–temporal asymmetry in peak velocity. The figure shows the peak velocities of the saccades as a function of type and visual field. Error bars represent 2 × within-participants' SEMs

Nasal–temporal asymmetries in peak velocity

Peak velocities into the temporal visual field were significantly ($t = 5.4$) higher than into the nasal visual field or 338°/s (SD = 80°/s) vs. 316°/s (SD = 77°/s). The same pattern was found for both express saccades (temporal: PV = 357°/s, SD = 77°/s; nasal: PV = 330°/s, SD = 71°/s; $t = 3.3$) and regular saccades (temporal: PV = 331°/s, SD = 81°/s; nasal: PV = 311°/s, SD = 78°/s; $t = 6.6$), see Fig. 9. These findings are in accordance with Jóhannesson and Kristjánsson (2013) who observed similar NTAs in peak velocities. In Fig. 10, we plot differences in peak velocity by hemifield for individual participants, subtracting the nasal hemifield PV´s from the temporal hemifield PV's, showing how the peak velocities of saccades into the temporal visual field are higher for all participants.

Discussion

The effect of training on hemifield dependence of saccade latencies

The central goal of this work was to measure saccade performance towards targets that appear either in the nasal or temporal hemifields before and after saccade training. We found that, with respect to the visual hemifields, training had an asymmetric effect on saccade latency. Whether there are latency differences between saccades to nasal and temporal hemifields has been controversial, with some studies reporting differences (Kristjánsson et al. 2004; Walker et al. 2000), but others reporting no difference (Bompas and Sumner 2008; Honda 2002; Jóhannesson et al. 2012). Our results here showed that training affects general saccadic latencies into the two hemifields differently. While before training, saccade latencies to the nasal hemifield were lower, this reversed following saccade training, with a lower latency

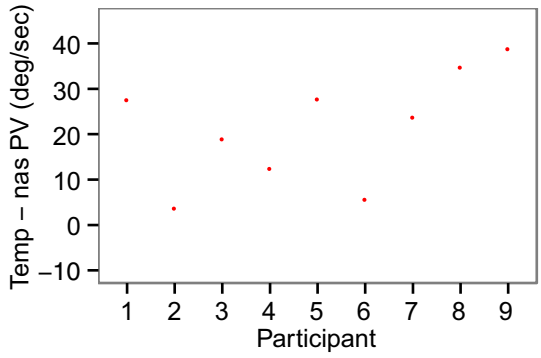

Fig. 10 Temporal–nasal asymmetry in peak velocity and individual differences. The data points show peak velocities to temporal hemifield stimuli minus the peak velocities to nasal hemifield targets

for saccades towards the temporal hemifield (in addition to an overall decrease in saccade latency). This suggests that, initially, there is a nasal–temporal asymmetry in latency that then reverses with training. Importantly, this also suggests that latency differences between saccades into the nasal and temporal hemifields have been masked by different levels of saccade training in the previous studies, if observers' performance at different levels of training was grouped. Different amounts of training may, therefore, explain why the results in the literature on whether there are nasal–temporal asymmetries in saccadic latency have been so inconclusive. Furthermore, our curve-fitting analysis indicated that these changes were not simply due to the elimination of unusually long latency saccades, but to a decrease in latency across the entire latency distribution.

This asymmetric effect of training had a corresponding effect on express saccade production. Express saccade proportion tripled for saccades to the temporal hemifield, but only doubled for saccades to the nasal hemifield. These results raise the intriguing possibility that mechanisms responsible for saccade generation into the temporal hemifield are more amenable to training. This may reflect differences in neural density in the retina and asymmetric projections from the retina to brainstem control sites. This asymmetry in express saccade proportion between hemifields is yet another example of differential processing in the nasal and temporal hemifields (see, e.g., review in Jóhannesson et al. 2012; Jóhannesson and Kristjánsson 2013).

Transfer of training from saccades made with monocular viewing to the non-viewing eye

Our results also show that with increased training latency decreases and express saccade frequency increases for saccades made by the uncovered, trained eye, and that these effects transfer to the untrained eye. These results localize the training effects to binocular sites in the visual pathways, and show that they cannot easily be attributed to mechanisms incorporating the monocular pathway from the retina to V1, since then, incomplete transfer between the nasal and temporal hemifields would have been expected. As Bibi and Edelman (2009) speculated, since express saccade training is not vector-specific, we believe that enhanced performance after training is a result of some combination of an enhanced visual response, increased low-level, pre-motor activity in the SC and other saccade-related structures, and tighter control of omnipause neurons in the pontine brain stem, which have to be silenced before a saccade can be initiated (Leigh and Zee 2007).

Training not only shortened latency but also changed the shape of the latency distribution (Fig. 4). The results from the ex-Gaussian analyses show that the training influenced the sigma and tau parameters (denoting, respectively, the standard deviation of the Gaussian part and the mean of the exponential part; Matzke and Wagenmakers 2009; Ratcliff 1993). The value of these parameters decreased more than the value of the mu parameter (denoting the mean of the Gaussian part, see Fig. 5). We speculate that the observed changes in sigma and tau reflect that saccadic generation becomes more automatic and less effortful with training.

The influence of saccade training on peak velocity

We also found that training increased saccadic peak velocity (see Fig. 8). This increase in peak velocity is not a consequence of increased saccadic amplitude, because there was no significant correlation between amplitude and peak velocity, neither for the trained nor the untrained eye. However, training significantly increased amplitude for the trained eye between probe sessions 1a and 3a (see Table 3), while training did not affect the amplitude for the untrained eye. In both the probe and training sessions, visual information about target location was projected from the trained eye to the brain. However, the untrained eye only projected visual information to the brain in the probe sessions. This difference in projection of visual information might account for the observed difference in training effects between the eyes. In addition, peak velocities were higher to targets in the temporal hemifield (projecting to the nasal hemiretina) than to nasal hemifield targets, replicating the pattern observed in Jóhannesson and Kristjánsson (2013).

Conclusions

Training-induced increases in express saccade proportion transfer between the nasal and temporal hemifields and from the trained (dominant) to the untrained (non-dominant) eye. Notably, our findings also reveal that training increases peak velocities and decreases saccadic latencies. Finally, a highly interesting pattern emerged where express saccade proportion was higher towards nasal hemifield stimuli before training, but that this then reverses, so that a higher percentage of express saccades is made towards temporal hemifield stimuli following training. This was also the case for overall saccade latencies, which suggests that different levels of training on saccade tasks may have masked hemifield differences in latency in the previous studies.

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