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Anne Hillairet de Boisferon, Céline Paeye

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Saccadic "Adaptation" at Late Target Reappearance

Introduction

Oculomotor plasticity allows constant adjustments of saccade amplitudes to a changing environment. Saccadic adaptation in humans is however disrupted when the presentation of the post-saccadic target is delayed (100-400ms), and disappears at delays around 600ms. This generally accepted result is however based on two small research samples (6 subjects in total)^[1,2]. In this study, we examined the effect of various temporal delays of target displacements on the amount of adaptation to establish **the critical time window for saccade adaptation**.

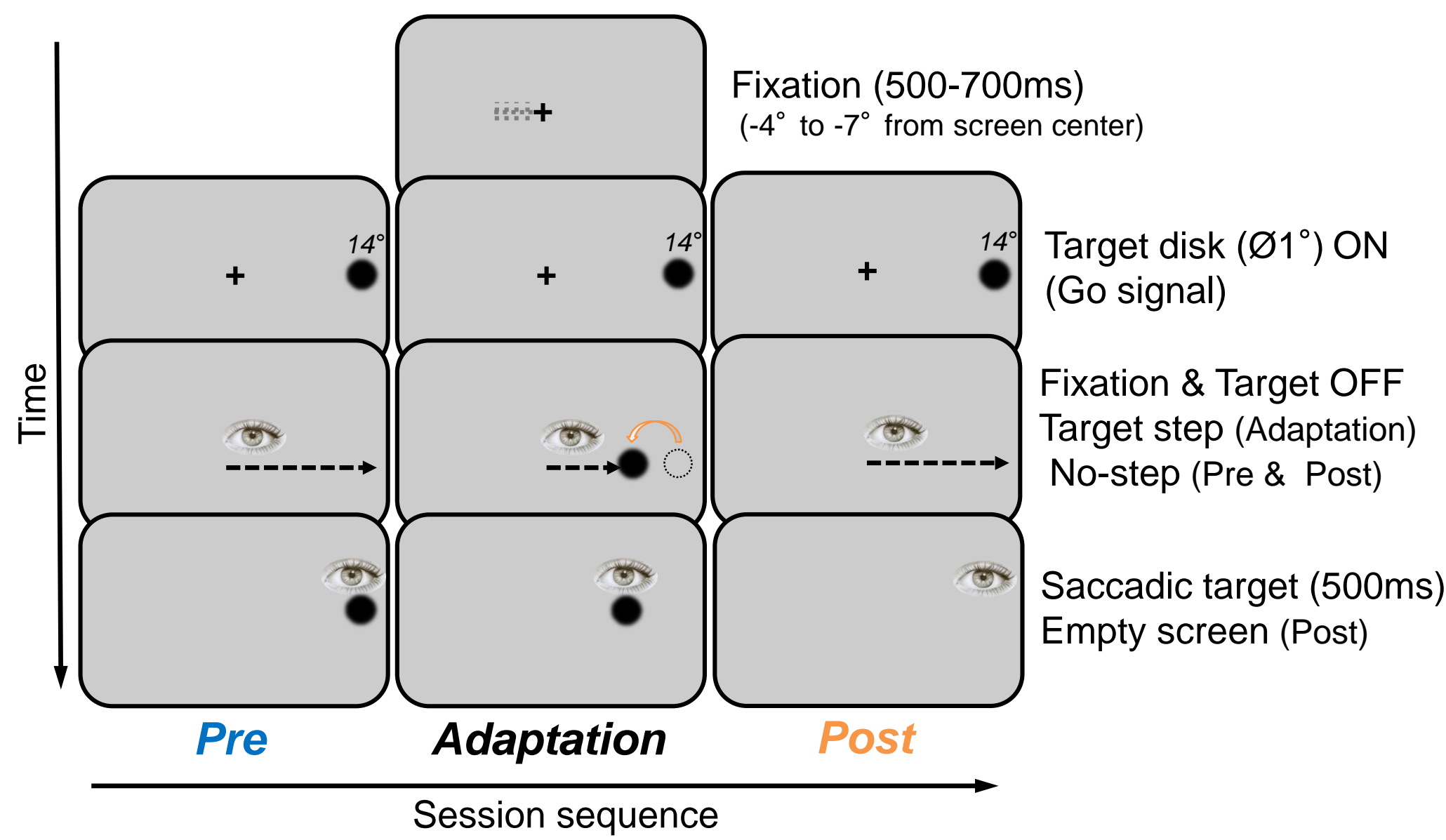
When a long delay is introduced, participants are likely to perceive the target displacement and the modification of saccadic amplitude could involve more explicit learning (voluntary and fast) than implicit learning (automatic and slow). In this study, we then evaluated both **components of motor adaptation** at each delay.

Methods

Backward Adaptation

Three phases during a same session:

- Pre-adaptation (100 trials) / Adaptation (4×100 trials) / Post-adaptation-Retention (100 trials)



Five time delays for target reappearance:

- -20, +60, +300, +600 and +1200ms by reference to saccade landing

Progressive modification of target step size during Adaptation:

- Trials 101-200 = 11%
 - Trials 201-300 = 22%
 - Trials 301-500 = 33%
- of target initial eccentricity (14°)

Participants

58 adults (11 males, 47 females), $M_{age} = 22 \pm 5$ year-olds

- Delay -20, $n = 10$; Delay +60, $n = 11$; Delay +300, $n = 14$;
- Delay +600, $n = 12$; Delay +1200, $n = 11$

Apparatus

- EyeLink 1000+, SR Research®, sampled at 1000 Hz, monocular recording.
- Visual stimuli projected on a screen (598 × 344 inches; 1280 × 720 pixels) by a digital micromirror video projector (PROPixx Full, VPixx Technologies, 244 Hz).

Data analyses

Saccade Amplitude = Distance covered by the eyes to reach the target (in degrees of visual angle)

Latency = Delay between target appearance and saccade onset (in ms)

Saccadic amplitude variability:

$$U = \frac{-\sum(\rho_b \log_2(\rho_b))}{\log_2(B)}$$

ρ represents the relative frequency of a bin b and B the number of bins ($B=11$). U -value reflects the likelihood of a saccade amplitude falling in each bin. $U = 0$, there is no uncertainty; $U = 1$, indicates the maximum entropy.

Percent Change in saccade amplitude:

$$PC = \frac{\text{Amplitude}_{Ada \text{ or } Post} - \text{Amplitude}_{Pre}}{\text{Amplitude}_{Pre}} \times 100$$

Mean amplitudes are based on the last 40 valid trials of the Pre- and Adaptation phases, and on the first 40 valid trials of the Post-adaptation phase.

Rate Constant of amplitude change:

τ = the number of saccades to achieve 66% of the total change in amplitude (asymptote)

The relation between amplitude and the number of saccades was fitted with an exponential function.

Resistance Index:

$$R_{index} = \text{Intercept}_{Post} - \text{Asymptote}_{Ada}$$

Exploration Index:

$$E_{index} = \frac{\sum(\text{amp}_{n-1} - \text{amp}_n)^2}{|PC_{Ada}|}$$

References

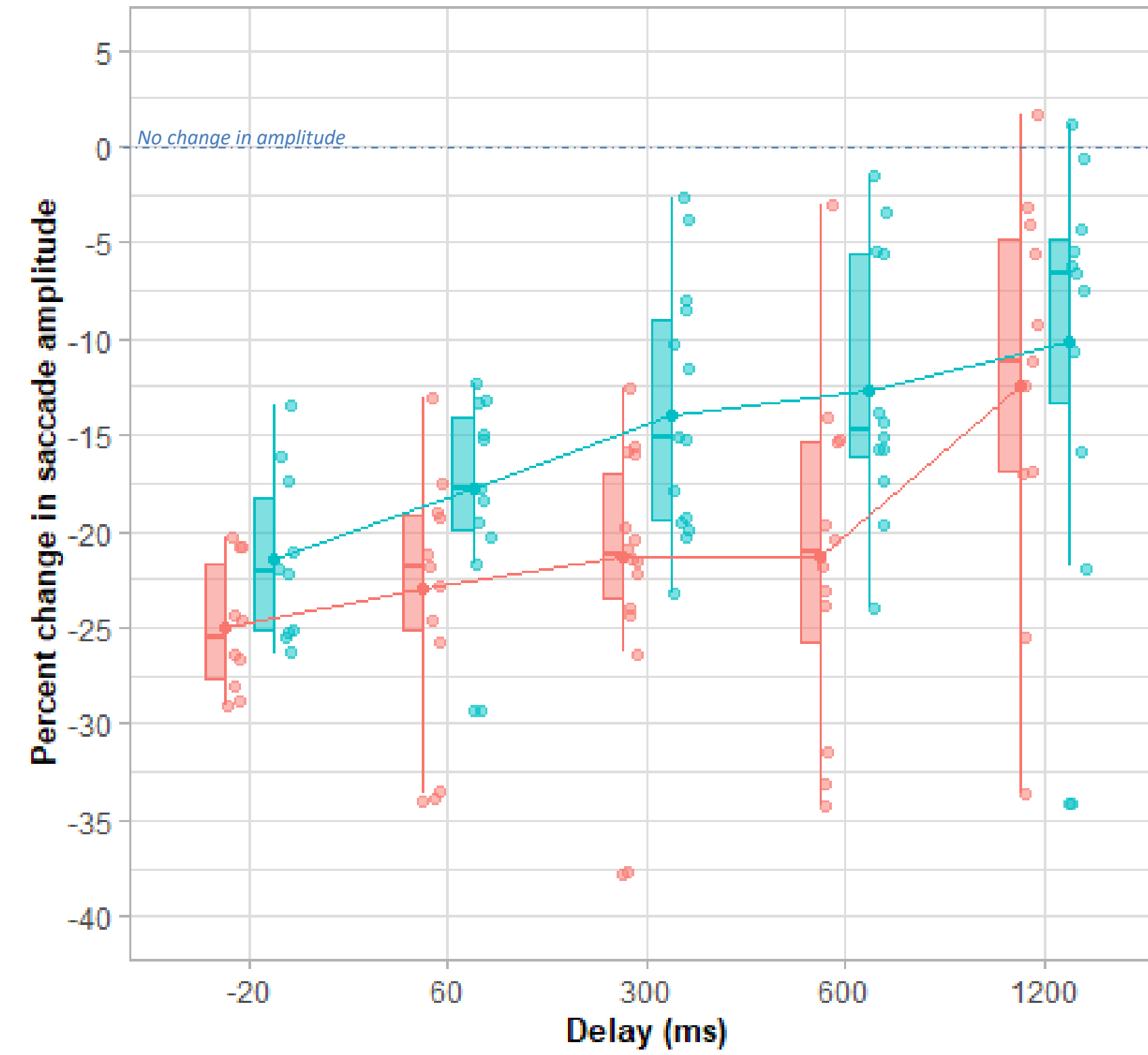
- [1] Fujita, M., Amagai, A., Minakawa, F., & Aoki, M. (2002). Selective and delay adaptation of human saccades. *Cognitive Brain Research*, 13(1), 41-52.
 [2] Bahcall, D. O., & Kowler, E. (2000). The control of saccadic adaptation: implications for the scanning of natural visual scenes. *Vision Research*, 40(20), 2779-2796.

Preliminary results

Time window for saccadic adaptation

Pourcent Change ~ Delay * Phase + (1 | participant)

$\chi^2(4)=10.6, p=.031, R^2_{adj}=.58$



Delay: $F(4,53) = 4.55, p = .003$; Phase: $F(1,53) = 67.5, p < .001$;
 Delay*Phase: $F(4,53) = 3.30, p = .017$ (Satterthwaite's method)

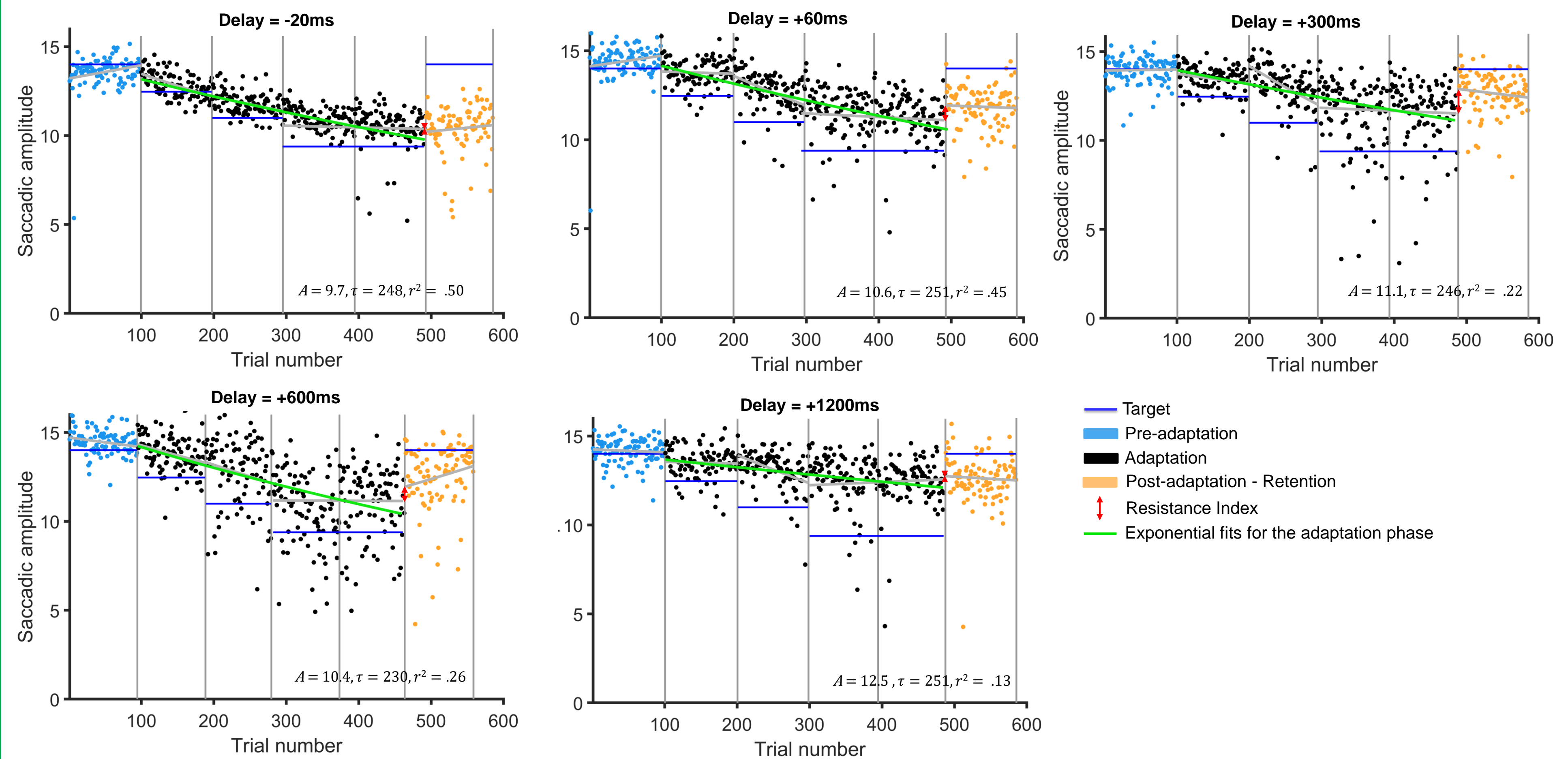
At the end of the Adaptation phase, the percent change in saccadic amplitude is less important for the +1200ms delay. (Tukey HSD, all $p_s < .05$)

Delay	Adaptation	One-sample t-test vs. 0% change	Post-adaptation	Δ Ada-Post	Simple effects of Phase
-20	-25 ± 3.4%	$p < .001$	-21.4 ± 4.4%	+3.6%	$p = .029$
+60	-23 ± 6.3%	$p < .001$	-17.8 ± 4.9%	+5.2%	$p = .001$
+300	-21.3 ± 6.1%	$p < .001$	-14 ± 6.5%	+7.3%	$p = .001$
+600	-21.3 ± 9%	$p < .001$	-12.6 ± 7%	+8.7%	$p < .001$
+1200	-12.4 ± 10.4%	$p = .003$	-10.2 ± 10.3%	+2.2%	$p = .14$

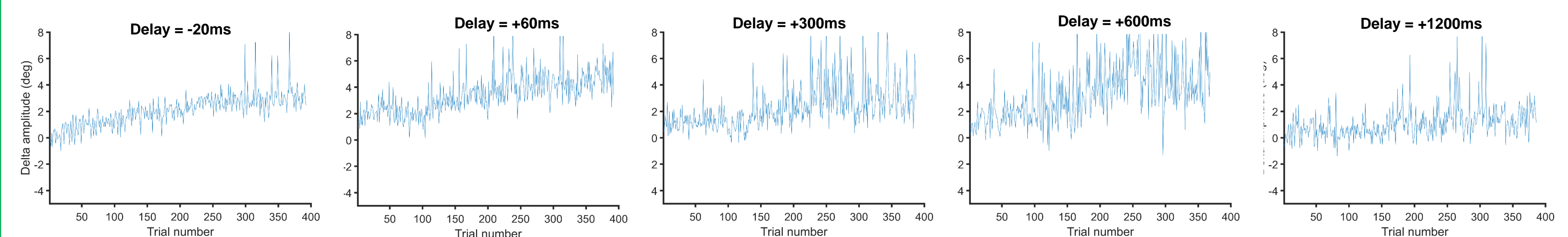
Phase
 Adaptation
 Post-adaptation

Time course and learning mechanisms

Examples of individual data



Exponential functions with Adaptation asymptotes (A), rate constants (τ), and correlation coefficients (r^2)



Cumulative saccade amplitude change (in deg) between the current trial and the previous trial

Delay	Latency (ms)	U-Value	Rate Constant τ	Resistance Index	Exploration Index	Adapter*
-20	177 ± 18	0.52 ± .05	249 ± 2	1.19 ± 0.60	29.6 ± 15.2	10/10
+60	186 ± 34	0.63 ± .10	245 ± 11	1.35 ± 0.70	54.0 ± 20.6	11/11
+300	176 ± 38	0.69 ± .05	242 ± 13	1.66 ± 0.80	86.5 ± 52.6	13/14
+600	211 ± 36	0.69 ± .07	233 ± 29	1.86 ± 0.77	91.2 ± 55.9	11/12
+1200	212 ± 33	0.62 ± .10	246 ± 12	1.05 ± 0.93	136.5 ± 91.1	7/11

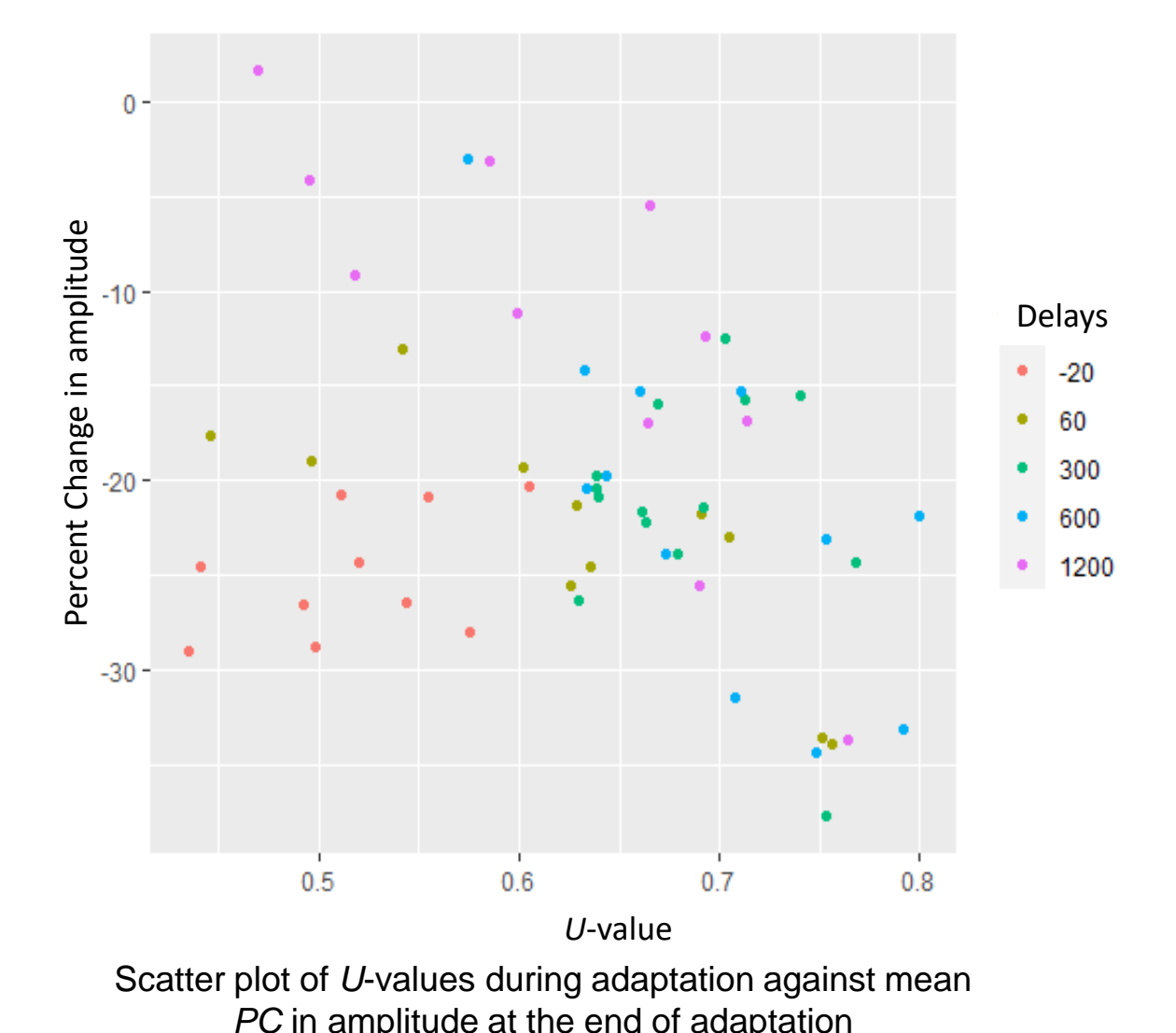
*Adapter = Individual PC_{Ada} in saccade amplitude significantly differs from Pre-adaptation natural variability in saccade amplitude

One-way ANOVAs

- **U-Value ~ Delay** for Adaptation (trials 301 to 500); $F(4,53) = 9.56, p < .001, \eta_p^2 = .42$
 Saccadic amplitude varies less for the -20ms delay (Tukey HSD, all $p_s < .02$)
- **Log Latency ~ Delay** for Adaptation; $F(4,53) = 3.71, p = .01, \eta_p^2 = .22$
 (Tukey HSD post-hoc tests indicate no consistent results)
- **Resistance Index ~ Delay** for « adapters »; $F(4,47) = 1.89, p = .13$
- **Rate Constant ~ Delay** for « adapters »; Delay: $F(4,47) = 1.52, p = .21$

Pearson's Correlations

- **U-Value** and PC_{Ada} : $r(58) = -.33, p = .012$
- $\sum(\text{amp}_{n-1} - \text{amp}_n)^2$ and PC_{Ada} : $r(58) = -.73, p < .001$



Conclusion

Preliminary results show that saccades' amplitude is modified up to a 1200ms delay in the appearance of the post-saccadic target, and that this amplitude reduction is still visible in large proportion (~21%) after a 600ms delay. Our visual system continues to "adapt" eye movements even when target displacements are likely perceived by participants. We observed however that variability in saccadic amplitude increased when the displacements were post-saccadic in comparison to intra-saccadic. We suggest that such variability could reflect different attributions to target displacements (change in the environment or saccadic errors) and different exploration strategies. More research is still needed to determine the exact nature of the mechanisms at play in oculomotor learning.