1 Temporal attention and oculomotor effects dissociate distinct types of
2 temporal expectation

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10 ABSTRACT

11 Temporal expectation–the ability to predict when events occur–relies on probabilistic information
12 within the environment. Two types of temporal expectation, *temporal precision*, based on the
13 variability of an event's onset, and *hazard rate*, based on the increasing probability of an event
14 with onset delay, interact with temporal attention–ability to prioritize specific moments– at the
15 performance level: Attentional benefits increase with precision but diminish with hazard rate.
16 Both temporal expectation and temporal attention improve fixational stability; however, the
17 distinct oculomotor effects of temporal precision and hazard rate, as well as their interactions
18 with temporal attention, remain unknown. Investigating microsaccade dynamics, we found that
19 hazard-based expectations were reflected in the oculomotor responses, whereas precision-
20 based expectations emerged only when temporal attention was deployed. We also found
21 perception-eye movement dissociations for both types of temporal expectation, yet attentional
22 benefits in performance coincided with microsaccade rate modulations. These findings reveal
23 an interplay among distinct types of temporal expectation and temporal attention in enhancing
24 and recalibrating fixational stability.

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27 **Keywords:** microsaccades, temporal expectation, temporal attention

Introduction

29 The world is dynamic and stochastic, yet it possesses predictable elements that enable us to

30 develop expectations. Temporal expectation, the ability to predict when events occur improves

31 perception in vision (Rohenkohl et al., 2012), audition (Bueti & Macaluso, 2010), and touch

32 (Badde et al., 2020), facilitates actions (Thomaschke & Dreisbach, 2013), alters timing of

33 oculomotor responses (Akdogan & Balci, 2016; Tal-Perry & Yuval-Greenberg, 2021), and

34 modulates neuronal activity at various levels within sensory processing and action pathways

35 (review, Nobre & van Ede, 2023).

36 Our brain utilizes temporal structures within the environment to develop temporal expectations

37 (Nobre et al., 2007, 2018). The passage of time characterizes hazard rate–the increasing

38 probability of an event, conditional on it not yet having occurred (Coull, 2009; Grabenhorst et al.,

39 2021; Duyar, Denison & Carrasco, 2023)–and the variability of an event's onset within a time

40 window characterizes temporal precision (Rolke & Hofmann, 2007). These two types of

41 temporal structures yield expectations with different temporal scales: hazard rate is robust to

42 immediate changes whereas temporal precision is computed over longer timescales (e.g., within

43 experimental trials or blocks, respectively).

44 In the auditory system, hazard rate and temporal precision are partially overlapping and

45 separable at the neural level based on latency, computation, and hierarchical processing stage.

46 The effects of hazard rate on auditory stimulus-evoked MEG responses emerge faster and at

47 lower levels of the hierarchical processing than those of temporal precision. Both effects

48 originate in the early auditory regions, but only temporal precision correlates with activity in the

49 inferior parietal cortices (Todorovic & Auksztulewicz, 2019). Such commonalities and

50 differences are unknown at the oculomotor level.

51 Temporal attention is another fundamental cognitive process, which facilitates allocation of the

52 brain's limited resources across time. It helps us prioritize information at specific moments,

53 regardless of predictability (review, Denison, 2024). Temporal attention mediates the effects of

54 temporal expectation at the neural level (Woldorff, Hackley & Hillyard, 1991; Näätänen, 2011;

55 Todorovic et al., 2015). For example, visual temporal attention modulates anticipatory effects in

56 MEG responses (Denison et al., 2024) and interacts with expectation at the behavioral level. In

57 particular, attentional benefits on performance increase with temporal precision, but decrease

58 with hazard rate (Duyar, Ren & Carrasco, 2024). But it is unknown whether temporal attention

59 distinctly interacts with these two types of expectation at the oculomotor level.

60 Microsaccades (MS), the fastest and largest of fixational eye movements, provide an online

61 metric throughout experimental trials. MS rate follows a typical temporal profile (Martinez-Conde

62 et al., 2009, 2013; Rolfs, 2009). Whereas initially MS were considered as reflexive eye

63 movements, Kowler's research revealed that they mirror broader aspects of visual perception

64 (reviews: Collewijn & Kowler, 2008; Kowler & Collewijn, 2010; Kowler, 2011), including cognitive

65 factors such as prediction (He & Kowler, 1989) and attention (Kowler, 2024). Typically, there are

66 three stages of the MS rate within an experimental trial (**Fig 1C**): (1) Pre-stimulus endogenous

67 anticipatory reduction, which occurs before an expected brief visual, auditory, or tactile stimulus

68 (Betta and Massimo, 2006; Dankner et al., 2017; Amit et al., 2019; Abeles et al., 2020; Badde et

69 al., 2020). This reduction becomes more pronounced with temporal attention (Denison, Yuval-

70 Greenberg & Carrasco, 2019; Palmieri, Fernandez & Carrasco, 2023), but becomes weaker

71 following a delayed visual event (Tal-Perry & Yuval-Greenberg, 2023; Duyar & Carrasco, 2024).

72 (2) Post-stimulus reflexive exogenous inhibition (Rolfs, Kliegl, Engbert, 2008), which indexes

73 conscious perception (White & Rolfs, 2016) and temporal expectation (Badde et al., 2020). (3)

74 Rebound to baseline, which depends on endogenous inhibition via expectation (Badde et al.,

75 2020), but effects of temporal attention are lacking (Denison et al., 2019; Palmieri et al., 2023).

76 Precise timing of MS is not only modulated by stimulus physical characteristics (Bonneh, Adini,

77 Polat, 2015), but also by task difficulty (Ezzo et al., 2025) and temporal attention (Denison et al.,

78 2019). Temporal attention shifts the timing of both pre-stimulus and post-stimulus

79 microsaccades earlier for temporally predictable targets. However, how temporal attention and

80   different types of expectation work together to recalibrate oculomotor dynamics and stabilize

81   gaze under temporal uncertainty is yet to be investigated.

82   Although perception and eye movements usually co-occur (Vishwanath & Kowler, 2003; Kowler,

83   2011; Spering & Montagnini, 2011) perceptual reports and eye movements have been

84   dissociated (e.g, Spering, Pomplun & Carrasco, 2010; Spering & Carrasco, 2012; Glasser &

85   Tadin, 2014; Vetter et al. 2019). Recently we found that temporal expectations based on the

86   preceding trial's stimulus timing is reflected in oculomotor dynamics, but it does not affect the

87   specific time when attention benefits performance (Duyar & Carrasco, 2024). To further our

88   understanding of perception-action dissociations (Spering & Carrasco, 2015; Carrasco &

89   Spering, 2024) here we investigate the eye-tracking dataset of a recent study in which we

90   showed that effects of temporal attention on performance interact in an opposite manner with

91   temporal precision and hazard rate (Duyar et al., 2024). Attentional benefits increase with

92   temporal precision, whereas they decrease with hazard rate. Whether these perceptual effects

93   are also reflected in the microsaccade dynamics is unknown.

94   Here, we ask: 1) Do hazard rate and temporal precision differentially modulate MS?; 2) Does

95   temporal attention distinctly modulate hazard rate and temporal precision in MS dynamics?; 3)

96   Are perceptual interactions of attention and expectation reflected in MS dynamics, or is there a

97   perception-eye movement dissociation?

98 **METHODS**

99 **Dataset**

100 We analyzed the eye-tracking data of a previous study in which we investigated behavioral

101 effects on the interaction between temporal attention and expectation (Duyar et al., 2024). The

102 observers, apparatus, stimuli and experimental protocol were identical to that study.

103 **Observers**

104 Sixteen observers (10 females, 6 males, aged 22-34, M=27, SD=3.445) participated in the

105 study. Two observers were removed from the temporal precision analysis due to corrupted raw

106 eye files for at least one of four conditions; thus, we analyzed the data from the remaining 14

107 observers (8 females, 6 males, aged 22-34, M=26.786, SD=3.599). For the hazard rate analysis

108 we included all observers because we had eye files for all conditions relevant to that analysis.

109 All observers had normal or corrected-to-normal vision. The research was performed in

110 accordance with the Helsinki declaration and the experimental protocol was approved by the

111 New York University Institutional Review Board.

112 **Apparatus**

113 Stimuli were generated using an Apple iMac (3.06 GHz, Intel Core 2 Duo) and MATLAB 2012b

114 (Mathworks, Natick, MA, USA) using Psychophysics Toolbox (Brainard, 1997; Kleiner et al.,

115 2007), presented on a gamma-corrected CRT monitor (1280 x 960 resolution, 100 Hz refresh

116 rate). The viewing distance was 57 cm, and the observers' head movements were restrained

117 using a chin and headrest. An infrared eye tracker Eyelink 1000 (SR research, Ontario, Canada)

118 was used for recording eye position throughout the experiment. Online eye tracking was

119 implemented to maintain central fixation (1°) throughout the trials. Eye tracking data was

120 recorded at 1000 Hz. Five-point-grid calibration was performed at the beginning of each

121 session, and was repeated within the session when necessary. In case of a fixation break due

122   to blink or ≥1° deviation from the center of the screen, the trial was aborted and repeated at the

123   end of the corresponding experimental block. Observers could blink or make larger saccades

124   during the response window and the intertrial interval.

125   **Stimuli**

126   Stimuli were displayed on a uniform medium gray background. A central fixation circle

127   subtended 0.15°, and four black circles (0.2°) were displayed at corners of a central imaginary

128   square (2.2° side) as placeholders. Two Gaussian-windowed (SD=0.3°), 100% contrast

129   sinusoidal gratings with a random phase were presented at the center sequentially in each trial.

130   Each grating was tilted clockwise or counterclockwise from the horizontal or vertical axis,

131   independently. The amount of tilt was titrated separately for each target interval for each

132   observer.

133   The attentional precues were 200 ms auditory tones, presented through speakers. The valid

134   precue was a sinusoidal wave (T1: 800 Hz, T2: 400 Hz), and the neutral precue was a complex

135   waveform that is a combination of a range of frequencies (50-400 Hz). Response cues were

136   identical to the valid precues to instruct the observers to report the first (T1) or the second (T2)

137   target.

138   **Experimental Procedure**

139   The experimental protocol was reported in detail in Duyar et al. (2024). The experiment was

140   developed by combining a temporal attention (Denison et al., 2017, 2021) and a temporal

141   expectation (Todorovic & Auksztulewicz, 2019) protocol. The experimental protocol is shown in

142   **Fig 1A**.

143   **Expectation Manipulations.** Two types of temporal expectation –temporal precision and

144   hazard rate– were manipulated (**Fig 1B**): Temporal precision via within-session target onset

145   variability. The most expected time point (mean point of each temporal distribution) was kept

146   constant (1400 ms for T1, and 1650 ms for T2 relative to the precue) across precision levels,

147   and the probability of stimulus appearance at the expected moment increased with precision:

148   uniform (33%), wide (42%), narrow (86%), certain (100%) conditions. Hence, the variability of

149   target onset was systematically decreased as the precision increased. The hazard rate was

150   manipulated in the low-precision conditions via within-trial target onset delay (early-expected-

151   late). Within the low precision experimental sessions, the targets could appear earlier or later

152   than the expected moment. In a trial with low temporal precision, the conditional probability of

153   the targets' appearance at each time point increased given that they had not appeared yet,

154   resulting in a lower hazard rate at the earlier than expected moments.

155   **Attention Manipulations.** Temporal attention was manipulated via auditory precues. The

156   precue instructed observers which target to attend, and the response cue which target's

157   orientation to report. The precue either instructed observers to attend to the first (T1) or the

158   second (T2) target, or it was uninformative (neutral/baseline) regarding which targets would be

159   behaviorally relevant. The precues corresponding to T1 or T2 were 100% valid, thus the

160   response cue was identical to the precue in those trials. In the neutral trials, the response cue

161   indicated either target with 50% probability.

162   **Discrimination Task.** The task was to report the orientation (clockwise/counterclockwise) of the

163   target that was indicated by the auditory response cue (T1 or T2). The stimuli orientations were

164   independently selected to have a horizontal or vertical reference axis from which they were

165   slightly offset. Visual feedback (green + or a red -) was presented after each trial based on

166   whether the observers gave a correct or incorrect response. The average accuracy was

167   presented at the end of each block.

168   **Training and titration procedures.** All observers completed a 60-trial training block in the

169   beginning of the first session to familiarize themselves with the attentional precues and the task.

170   In the beginning of each session/day, the observers completed a titration protocol of 128 trials.

171   We used Best PEST procedure (Liberman & Pentland, 1982), which adjusted the orientation tilt,

172   to attain 75% orientation discrimination accuracy separately for each target in the Neutral

173   condition. Neutral performance was monitored throughout the experiment and, if needed,

174   additional adjustments to the tilt were automatically made after each block. Titration procedure

175   was conducted with constant target timing (**Fig 1B**, temporal precision=certain). Observers were

176   explicitly informed regarding the temporal precision of the session.

Eye-tracking data preprocessing and microsaccade detection

178   Each observer completed 3568-4256 trials in total (M=3689, SD=245.773). Due to technical

179   problems the eye data for some blocks was not saved. We recovered ~90% of the eye-tracking

180   data from 16 people for the hazard rate analysis (M=3291.44, SD=425.36), and from 14 people

181   for the precision analysis (M=3368, SD=393.470).

182   We used an established velocity-based algorithm to detect MS within each experimental trial

183   (Engbert & Kliegl, 2003). For each trial, we transformed raw eye position data into a 2D velocity

184   space. A trial-specific 2D threshold was then determined as an ellipse centered on the median

185   velocity in both horizontal and vertical directions. An MS was detected when the eye movement

186   velocity exceeded this threshold for at least 6 ms.

Microsaccade rate timecourse

188   MS rate timecourses were calculated for each observer, separate for each attentional precue

189   and expectation condition. For each experimental condition, we averaged the number of MS per

190   time samples across all trials, multiplied these values by the sampling rate, and then smoothed

191   across time by applying a 50-ms sliding window.

192   We performed a series of nonparametric cluster-based permutation tests to compare MS rate

193   timecourses and identify the intervals when they were significantly different (Maris &

194   Oostenveld, 2007). We used two-tailed dependent samples tests (α = 0.05, 2000 permutations)

195   and implemented Bonferroni correction on the identified clusters to account for multiple

196   comparisons. These tests are widely used in MS research, and provide useful information

197   regarding when rate timecourses differ between two conditions within a time period (e.g., Liu,

198   Nobre & van Ede, 2022, Palmieri et al., 2023).

199   **Microsaccade rate rebound inflection points**

200   As a follow-up to the cluster-based permutation tests, we zoomed into the MS rate window to

201   analyze the timing of MS rate rebound. We estimated the inflection point when the MS rates

202   start to increase –rebound onset– and when they stabilize –rebound offset– by doing the

203   following: First, we focused on the MS rate timecourse between 1500-2700 ms after the precue

204   onset, for each observer and each experimental condition. Then we computed the first

205   derivative of the MS rates within this period, and identified where it crossed zero. These specific

206   points were considered candidate rebound onset and offsets. Three independent raters

207   (including one author A.D. and two research assistants) independently identified the inflection

208   points as the start and end points of the MS rate rebounds for each observer and experimental

209   condition. The raters were blind to these conditions, which were presented in a random order.

210   **Microsaccade timing around the stimulus onset**

211   Precise timings of the MS were analyzed to investigate the effects of temporal attention and

212   expectation on the MS that happen around the stimulus presentation timing. We analyzed the

213   timing of the last MS that occurred before the stimuli presentation (last pre-T1 MS), and the first

214   MS that occurred after the stimuli offset (First post-T1 MS) (Bonneh, Adini, Polat, 2015; Denison

215   et al., 2019). Trials without any MS were excluded from this analysis. We followed the MS timing

216   analysis in the Denison, Yuval-Greenberg and Carrasco (2019) study. In short, for each

217   observer, we first converted all of the MS onsets to z-scores and estimated the kernel density

218   for each precue and expectation condition. Density was evaluated using 300 equally spaced

219   points between -5 and 5, and the median of each kernel was computed to perform further

220   statistical analyses.

221

222 **Statistical analysis and visualization**

223 Repeated-measures ANOVA and paired t-tests were performed with R (version 4.2.3; R Core

224 Team, 2023; ezANOVA package Lawrence, 2016), and the nonparametric cluster-based

225 permutation tests were performed using MATLAB. Categorical data were analyzed and

226 visualized using R, whereas time series and kernel densities were investigated using MATLAB.

227 We implemented Bonferroni correction for the cluster-based permutation tests, and Holm-

228 correction for pairwise t-tests to reduce the likelihood of a Type-I error.

229 **RESULTS**

230 **Microsaccade Rate Dynamics**

231 **Expectation: Hazard Rate vs Temporal Precision**

232 For hazard rate, we analyzed MS rates from the two lowest temporal precision conditions

233 (uniform and wide) as well as the timing of the rate rebound. For temporal precision, we only

234 analyzed the trials where the targets appeared at the Expected moment. These trials had the

235 same target onsets, but were embedded in sessions with different levels of temporal precision

236 (as reported in Duyar et al., 2024).

237 We first compared MS rates in trials in which the stimuli appeared Early, Expected, or Late,

238 regardless of attention (**Fig 2A**). This analysis enabled us to compare prolonged prestimulus

239 inhibition vs stimulus-induced inhibition. The cluster-based permutation tests revealed three

240 significant clusters in the inhibitory period, and two during the rebound. In the inhibitory period,

241 observers made less frequent MS in the Early than the Expected trials (1189-1503 ms,

242 p=0.028), and in the Early than Late trials (1239-1639 ms, p=0.007), and in the Expected than

243 Late trials (1415-1663 ms, p=0.039). Subsequently, there were modulations in the rebound, in

244 which MS rates increase back to baseline level. Observers made less frequent MS when the

245 stimuli appeared Late than Early (1681-2017 ms, p=0.003) and than Expected (1739-2037 ms,

246 p=0.007). Overall, strength and timing of inhibition and rebound followed hazard rate.

247 We then compared MS rates among temporal precision levels (**Fig 2B**). Permutation cluster

248 tests revealed no significant differences among the temporal precision levels. MS rate dynamics

249 were similar regardless of temporal precision; they decreased before the stimulus onset, and

250 rebounded after stimulus offset.

251 We split the data based on attentional precue. For hazard rate (**Fig 3A**), in the Neutral trials (top

252 panel), in the inhibitory period, observers made less frequent MS in the Early than Late (1069-

253 1611 ms, p=0.001) and Expected (1191-1480 ms, p=0.023), and in the Expected than Late

254   (1408-1632 ms, p=0.015) trials. This inhibition was followed by rebound modulations, in which

255   observers made less frequent MS in the Late than Early (1675-2005 ms, p=0.016) and

256   Expected (1729-2011 ms, p=0.002) trials. When T1 was precued (middle panel), in the

257   inhibitory period, observers made less frequent MS in the Early than Late (1249-1568 ms,

258   p=0.020) trials, followed by three clusters during the rebound, and an extra cluster in the post-

259   rebound. During rebound, MS rates were lower in the Expected (1619-1862 ms, p=0.011) and

260   Late (1649-1985 ms, p<0.0001) than Early trials, as well as in the Late than Expected (between

261   1727-2021 ms, p<0.0001) trials. And in the post-rebound, MS rates were lower in Expected than

262   Late trials (2338-2600 ms, p=0.012). When T2 was precued (bottom panel), the MS rates were

263   similar during the inhibitory period, whereas there were clusters after stimulus offset. During

264   rebound, MS rates were lower in the Late than Early (1688-2009 ms, p<0.0001) and Expected

265   (1747-2035 ms, p=0.001) trials. Subsequently, in the post-rebound, MS rates were lower in the

266   Early than Expected (2233-2492 ms, p=0.002), Early than Late (2313-2542 ms, p=0.021), and

267   in then Expected than Late (between 2390-2600 ms, p=0.016) trials.

268   For temporal precision (**Fig 3B**), there were no significant differences among temporal precision

269   levels, in the Neutral (top panel) or T2 (bottom panel) trials. When T1 was precued (middle

270   panel), two clusters emerged. There were less MS (993-1371 ms, p=0.033) during the inhibitory

271   period in the Uniform (33%) than Narrow (86%) trials. And after the rebound (2293-2600 ms,

272   p=0.045) there were less MS in the Wide (42%) than Narrow (86%) condition.

273   **Temporal Attention: Hazard Rate vs Temporal Precision**

274   We compared the attentional conditions by collapsing across hazard rate levels within Wide and

275   Uniform conditions (**Fig 4A**). Cluster-based permutation tests revealed significant differences

276   during the inhibitory period. MS rates were lower for T1 (251-1508 ms, p<0.001) or T2 (599-

277   1303 ms, p=0.003) than Neutral trials. There were similar attentional modulations in the

278   inhibitory period within each hazard rate condition (**Fig 4B**). For Early targets (top panel), MS

279   rates were lower when observers attended to T1 than to Neutral (627-1453 ms, p=0.002), which

280 coincides with the T1 onset. For Expected targets (middle panel), MS rates were lower for T2

281 (604-1036 ms, p=0.010) and T1 (621-1336 ms, p=0.001) than Neutral. For Late targets (bottom

282 panel), MS rates were lower in T2 (970-1264 ms, p=0.021) and in T1 (987-1306 ms, p=0.015)

283 than Neutral trials. When observers attended to the first target, the modulations coincided with

284 Early T1 onsets (between 1200-1350), but never coincided with any T2 onsets when observers

285 attended to the second target.

286 We then compared the attentional conditions by collapsing across temporal precision levels at

287 the expected moment (**Fig 4C**). There were two practically overlapping clusters identified during

288 the inhibitory period. Observers made less frequent MS when attending to T1 (582-1418 ms,

289 p=0.002) or T2 (630-1370 ms, p=0.006) than Neutral. Splitting these data according to temporal

290 precision revealed that similar attentional modulations were present during the inhibitory period

291 within each temporal precision condition (**Fig 4D**). A cluster was identified in the Certain

292 condition (595-886 ms, p=0.022) in which observers made less frequent MS for T1 than Neutral

293 trials. In the Narrow condition, a similar difference emerged between T2 and neutral precue

294 conditions (984-1324 ms, p=0.009). Two clusters practically overlapped in the Wide condition,

295 with lower MS rate in T1 (860-1221 ms, p=0.003) and T2 (828-1247 ms, p=0.006) than in

296 Neutral trials. In the Uniform condition, a similar cluster emerged when attending to T1 than to

297 T2 (994-1330 ms, p=0.010).

298 We also analyzed the timing and the levels of MS rate rebound. The (three) inter-rater

299 consistency for rebound onset was 91% and for offset was 90% of the trials. In the remaining

300 cases, we included consistently chosen points by two out of three raters. We conducted two-

301 way ANOVAs (precue x hazard rate) on the following parameters:

302 Rebound onset was affected by hazard (**Fig 5A,** $F_{(2,30)}=35.387$, p<0.0001, $\eta^2G=0.239$), but not

303 by precue (**Fig 5B,** $F_{(2,30)}=2.290$, p=0.119) or an interaction between these factors

304 (F(4,60)=1.475, p=0.221). Pairwise comparisons revealed that the rebound onset was delayed

305   with stimulus onset (all ps<0.001; Early-Expected: d=0.792, Early-Late: d=1.288, Expected-

306   Late: d=0.659).

307   Rebound offset was affected by hazard (**Fig 5C,** $F_{(2,30)}=19.731$, $p=0.0001$, $\eta_G^2=0.162$) and

308   precue (**Fig 5D** $F_{(2,30)}=11.020$, $p=0.0003$, $\eta_G^2=0.020$). Rebound stopped sooner when the

309   stimulus was Early than Expected, ($p=0.015$, d=0.284) and when it was Expected than Late

310   ($p<0.001$, d=0.754). Moreover, the rebound stopped sooner for T1 ($p<0.001$, d=0.309) or T2

311   ($p=0.001$, d=0.224) than in the Neutral trials.

312   Rebound magnitude was affected by hazard (**Fig 5E,** $F_{(2,30)}=9.095$, $p=0.0008$, $\eta_G^2=0.006$) and

313   precue (**Fig 5F**, $F_{(2,30)}=3.969$, $p=0.0295$, $\eta_G^2=0.006$). The rebound was smaller for Early than

314   Late targets ($p=0.0023$, d=0.186). Moreover, the rebound was larger when observers attended

315   to T1 ($p=0.019$, d=0.152) or T2 ($p=0.018$, d=0.175) than in the Neutral trials.

316   Rebound slope was affected by hazard ($F_{(2, 30)}=4.036$, $p=0.028$, $\eta_G^2=0.018$) and precue

317   ($F_{(2,30)}=7.443$, $p=0.002$, $\eta_G^2=0.012$). The slope was steeper at Expected than Early ($p=0.005$,

318   d=0.305) targets, and for T1 ($p=0.028$, d=0.189) or T2 ($p=0.009$, d=0.249) than in Neutral trials.

319   Rebound offset MS rate was affected by hazard rate ($F_{(2,30)}=4.351$, $p=0.018$, $\eta_G^2=0.002$). MS

320   rate was lower at the rebound offset for Early than Late targets ($p=0.043$, d=0.127). Lastly, there

321   was no main effect or interaction of precue and hazard rate on MS rate for rebound onset (all

322   Fs<1, ps>0.05) or duration (hazard: $F_{(2,30)}=2.112$, all Fs<1, all ps>0.1).

323   In sum, with increasing hazard: (i) MS rate inhibition was delayed when collapsing across

324   attentional conditions. This effect was more pronounced in the Neutral than in Attended trials,

325   with the effect being present in T1 but not in T2; (ii) MS rate rebound was delayed and its

326   magnitude increased. But regardless of hazard rate, attention: (i) facilitated MS inhibition; (ii)

327   increased the rebound magnitude, steepened its slope, and hastened its offset.

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329

330   **Microsaccade Latency Dynamics**

331   To investigate whether the precise timing of the MS are affected by the interaction between

332   temporal attention and expectation, we analyzed the timing of the MS that occurred around the

333   stimulus onset. We analyzed the timing of the last MS that occurred before the stimuli (last pre-

334   T1 MS), and the timing of the first MS that occurred after the stimuli (first post-T1 MS) (Bonneh,

335   Adini, & Polat, 2015; Denison, Yuval-Greenberg & Carrasco, 2019). Analyzing first post-T2 MS

336   yielded the same results because very few MS occurred between T1 and T2.

337   **Hazard Rate**

338   Given that target timings varied among trials, we analyzed last pre-T1 and first post-T1 MS

339   latency both relative to precue, as well as to the target onset. We performed two-way (3 hazard

340   rate x 3 precue) ANOVAs on the median timings of the estimated kernel densities.

341   Relative to precue onset, ANOVA on the last pre-T1 MS latency revealed main effects of the

342   hazard rate ($F_{(2,30)}=26.012$, $p<0.0001$, $\eta_G^2=0.112$) and attention ($F_{(2,30)}=11.875$, $p=0.002$,

343    $\eta_G^2=0.104$), but no interaction ($F_{(4,60)}=1.284$, $p=0.293$). Pairwise t-tests showed that the last

344   pre-T1 MS was sooner for Early than Expected ($p<0.0001$, $d=0.478$), which in turn was sooner

345   than Late ($p<0.001$, $d=0.339$) targets (**Fig 6A**), and when observers attended to T1 than to T2

346   ($p=0.017$, $d=0.216$), which in turn was sooner than Neutral ($p=0.0001$, $d=0.542$) (**Fig 6B**).

347   ANOVA on the first post-T1 MS rebound locked to the precue yielded main effects of hazard

348   rate ($F_{(2,30)}=202.788$, $p<0.0001$, $\eta_G^2=0.513$) and attention ($F_{(2,30)}=4.212$, $p=0.024$,

349    $\eta_G^2=0.009$). Pairwise comparisons revealed that the first post-T1 MS were delayed with hazard

350   (**Fig 6C**; all $p<0.0001$; Early-Expected: $d=1.303$, Early-Late: $d=2.596$, Expected-Late: $d=1.131$).

351   Moreover, the first post-T1 MS were earlier when T1 was precued than in Neutral trials

352   ($p=0.008$, $d=0.158$) (**Fig 6D**).

353   Relative to target onset, ANOVA on last pre-T1 MS latency revealed main effects of hazard rate

354   ($F_{(2,30)}=144.793$, $p<0.0001$, $\eta_G^2=0.449$) and attention ($F_{(2,30)}=12.026$, $p=0.0001$, $\eta_G^2=0.103$),

16

355   but no interaction (F(4,60)=1.508, p=0.231). Pairwise t-tests showed that the time window

356   between the last pre-T1 MS and the T1 onset was the shortest for Early than Expected

357   (p<0.0001, d=1.022), which in turn was shorter than for Late (p<0.0001, d=1.052). Similarly, it

358   was shorter when attending to T1 than T2 (p=0.050, d=0.147), which in turn was shorter than for

359   Neutral (p<0.001, d=0.423) trials. These findings are in the same direction as the MS timing

360   locked to the precue.

361   A 2-way ANOVA on the median first post-T1 MS timing showed a main effect of precue

362   (F(2,30)=0.034, p=0.034, η_G^2=0.006), but not of hazard (F(2,30)<1, p>0.1; **Fig 6E**). Post-T1 MS

363   was sooner when T1 was precued than in Neutral trials (p=0.018, d=0.201; **Fig 6F**).

364   **Temporal Precision**

365   We performed 2-way (4 precision x 3 precue) ANOVAs on the median timings of the last pre-T1

366   and first post-T1 MS. On both last pre-T1 and first post-T1 MS timings, there was a main effect

367   of attention (last pre-T1 **Fig 7B**: F(2,26)=10.111, p<0.001, η_G^2=0.067; first post-T1 **Fig 7D**:

368   F(2,26)=7.847, p=0.002, η_G^2=0.017), but not of precision (last pre-T1: F<1, p>0.1; first post-T1:

369   F<1, p>0.1) or their interaction (last pre-T1 **Fig 7A**: F(6,78)=1.271, p>0.1; first post-T1 **Fig 7C**:

370   F(6,78)=1.563, p>0.1). Post-hoc pairwise t-tests showed that the last pre-T1 MS was earlier in

371   T1 (p<0.001, d=0.599) and T2 (p<0.001, d=0.506) than in Neutral trials. First post-T1 MS were

372   earlier when attending to T1 than T2 (p=0.003, d=0.273) or Neutral (p=0.006, d=0.276) trials.

DISCUSSION

Different temporal structures shape temporal expectations, influencing perception, action and attention through overlapping yet distinct mechanisms, such as modulations in neural signal strength, latency and synchrony (review: Nobre & van Ede, 2017). The brain forms temporal expectations through distinct neural correlates, which are integrated into behavioral responses, based on its temporal structure (Coull et al., 2000; Tal-Perry & Yuval-Greenberg, 2020; Grabenhorst et al., 2021). Hazard rate–mounting expectations through time passage–and temporal precision–based on probabilistic distributions–differentially influence auditory neural responses (Todorovic & Auksztulewicz, 2021) and interact with temporal attention distinctly when modulating visual performance (Duyar et al., 2024). Here, explored potential dissociations between perception and action and examined whether they can be distinguished at the oculomotor level.

We focused on the temporal dynamics of the push-pull mechanisms of MS rate signature across three stages: (1) pre-stimulus anticipatory reduction, (2) post-stimulus reflexive exogenous inhibition, and (3) rebound, which enabled us to disentangle endogenous components driven by anticipatory processes from the exogenous components triggered by external stimuli.

MS rate signature is aligned with the hazard rate, but not with precision. We analyzed MS rate signatures across varying hazard rates and across different precision levels. As the stimuli were delayed and the hazard rate increased, prestimulus anticipatory reduction was prolonged, and post-stimulus reflexive exogenous inhibition and the corresponding rebound were delayed. And notably, the post-stimulus inhibition was stronger than the anticipatory reduction, resulting in lower MS rates in Early than Late trials (**Fig 2A**). In contrast, MS rate signatures were similar across temporal precision levels (**Fig 2B**).

Temporal attention modulates hazard rate and temporal precision in an opposite manner during MS rate decrease. We analyzed MS rate signature across varying hazard rates and across different precision levels as a function of attention. In the neutral condition, MS rates

399 were lower when the target was early during the reduction/inhibition, and the corresponding

400 rebound occurred earlier; however, no such effect was observed for temporal precision (**Fig 3A**

401 **and B, top panels**). When observers attended to T1, prestimulus reduction was stronger,

402 leading to an attenuated reflexive inhibition and resulting in only one cluster during the inhibitory

403 period, highlighting the difference between Early and Late trials. As a result, the distinctions

404 between the hazard rate levels that were present in Neutral were diminished when attending to

405 T1 and disappeared when attending to T2 (**Fig 3A, middle and bottom panels**). In contrast,

406 precision levels were distinct during the pre-stimulus period when observers attended to T1 (**Fig**

407 **3B, middle panel**). Specifically, microsaccades occurred less frequently in the pre-stimulus

408 period under lower (33%) than higher (86%) temporal precision. Because hazard rate depends

409 on stimulus onset, attentional interaction's emergence in the post-stimulus period is expected.

410 However, the prestimulus emergence of the precision effect is notable, especially given its late

411 effect on auditory-evoked MEG responses compared to hazard rate (Todorovic &

412 Auksztulewicz, 2019). Furthermore, our findings indicate that temporal precision information is

413 integrated into eye movement planning primarily during attentional deployment in the

414 anticipatory period.

415 **Temporal attention modulates hazard rate and temporal precision similarly after rebound.**

416 Notably, we found that post-rebound differences emerged with attention, both for hazard rate

417 (**Fig 3A, middle and bottom panels**) and for temporal precision (**Fig 3B, middle panel**). These

418 differences were in the same direction, such that a higher probability of the stimulus onset was

419 associated with a higher MS rate in the post-rebound. This similarity suggests a potential

420 common mechanism for integrating the attended probabilistic temporal information irrespective

421 of the temporal structure from which the probability is derived. Such post-stimulus processing

422 commonality among hazard rate and temporal precision was observed in gain modulation in

423 primary auditory cortex and superior temporal gyrus (Todorovic & Auksztulewicz, 2019).

424 **Perception-action parallelism is observed for the hazard rate, but not for temporal**

425 **precision.** Temporal attention facilitates pre-stimulus anticipatory MS reduction when stimulus

426   timing is predictable (Denison et al., 2019; Palmieri et al., 2023), as well as under low temporal

427   precision (Duyar & Carrasco, 2024). Here we found that attentional modulation can extend

428   beyond T1(**Fig 4A**). Remarkably, this modulation coincides with early stimuli onsets, for which

429   hazard is the lowest (**Fig 4B**), within the window with largest attentional benefits on performance

430   (Duyar et al., 2024). Together, these results suggest a parallelism between performance and

431   oculomotor behavior. We did not observe such a parallelism for temporal precision. Attentional

432   modulation occurred earlier for Certain than all uncertain trials (**Fig 4D**).

433   **Temporal attention and hazard rate operate independently during the post-stimulus**

434   **period.** We quantified both rebound temporal shift and magnitude in hazard rate. The MS rate

435   rebound is not merely a reflexive release from prior suppression; it is delayed following an

436   oddball event (Valsecchi, Betta & Turatto, 2007; Widmann, Engbert & Schroger, 2014) and with

437   task difficulty due to prolonged sensory evidence accumulation (Ezzo et al., 2025). Its duration

438   also increases as a function of reaction times (Betta & Turatto, 2006), while its magnitude

439   diminishes following an oddball event (Valsecchi & Turatto, 2009). Moreover, the magnitude of

440   the rebound is linked to activity in the frontal eye fields (Hsu, Chen, Tseng & Wang, 2021;

441   Fernández, Hanning, Carrasco, 2023). Unlike the interaction between temporal attention and

442   hazard rate on performance, we observed no evidence for an interaction between them on MS

443   rate rebound. In particular, rebound onset was delayed with hazard (**Fig 5A**), but not with

444   attention (**Fig 5B**). Rebound offset was also delayed with hazard (**Fig 5C**), but attention

445   hastened it (**Fig 5D**), so that post-stimulus MS rate reached baseline level faster with attention.

446   We also found that rebound magnitude increased with both hazard (**Fig 5E**) and attention (**Fig**

447   **5F**). Overall, these findings suggest that temporal attention and expectation operate

448   independently during the post-stimulus period. The delay in rebound onset and offset with

449   hazard rate primarily indicates a temporal shift locked to stimulus timing, whereas attention

450   hastened rebound offset, expediting return to baseline rate without affecting its onset.

451   Furthermore, the increase in rebound magnitude with both hazard and attention may reflect their

452 separate contributions to ongoing cognitive control and decision making during the post-

453 stimulus window.

454 Precise timing of MS around stimulus onset allows for quantification of oculomotor freezing and

455 the subsequent release, revealing fine-grained temporal dynamics of perceptual and cognitive

456 processes (Bonneh et al., 2015; Yablonski, Polat, Bonneh & Ben-Shachar, 2016). When the

457 stimulus timing is certain, the last MS before stimulus onset and the first MS after stimulus onset

458 occur earlier with temporal attention (Denison et al., 2019).

459 **Perception-action dissociation for the hazard rate.** Effects of temporal attention and hazard

460 rate interact on performance (Duyar et al., 2024), but we found not such an interaction on the

461 precise timing of MS. The oculomotor freezing onset—indexed by indexed by last pre-T1 MS—

462 was delayed with the hazard rate, as the stimulus was delayed (**Fig 6A**). But regardless of

463 hazard rate, attention speeded oculomotor freezing (**Fig 6B**). Correspondingly, release from

464 freezing, indexed by the first post-T1 MS was delayed with hazard rate (**Fig 6C**) and shifted

465 earlier with attention (**Fig 6D**).

466 **Upcoming MS are scheduled in advance.** To differentiate the effects of MS timing from those

467 of stimulus latency, we analyzed MS timing relative to the stimulus within each trial. This

468 analysis revealed that the release from inhibition was consistent regardless of hazard rate, with

469 the first post-stimulus MS occurring after a similar duration following stimulus onset (**Fig 6E**). In

470 comparison, attentional effects on the release from inhibition were still present relative to

471 stimulus onset, such that the release from inhibition occurred earlier in the attended trials as

472 compared to Neutral (**Fig 6F**). This provides evidence for the preparatory scheduling of MS in

473 advance (Badde et al., 2020). Hazard rate is stimulus-dependent and dynamically updated as

474 the stimulus is delayed. Consequently, the release from inhibition cannot be pre-planned with

475 respect to hazard rate. The release timing was unaffected by hazard rate, which is inherently

476 tied to immediate, transient changes. In contrast, attentional allocation occurs earlier within the

477 trial, enabling planning of the inhibition release and advance scheduling of MS. Overall, we

478   conclude that release from inhibition can be governed by endogenous anticipatory rather than

479   transient processes.

480   **Perception-action dissociation for temporal precision.** Although temporal attention benefits

481   on visual performance increase with temporal precision (Duyar et al., 2024), we found precise

482   timing of MS is independently and consistently advanced with attention. Regardless of temporal

483   precision, the oculomotor freezing onset—the last pre-T1 and first post-T1 MS were consistent

484   (**Fig 7A & 7C**), but started (**Fig 7B**) and released (**Fig 7D**) earlier with attention.

485   The temporal scale of temporal precision and hazard rate differs. Understanding how two

486   probabilistic structures are represented may provide valuable insights regarding atypical

487   conditions, such as schizophrenia, attention deficit hyperactivity disorder, autism, and Parkinson

488   (e.g., Aitkin, Santos & Kowler, 2013; González-Gadea et al., 2015; Dankner et al., 2017; Martin

489   et al., 2017; Cannon et al., 2021; Coull & Giersch, 2022; Degos, Pouget & Missal, 2022) in

490   which temporal expectations are altered. Revealing similarities and distinctions between

491   different types of temporal expectations is important for improving cognitive models, which can

492   be useful in developing diagnostic and interventional tools. In this context, MS serves as a

493   valuable noninvasive biomarker.

494   In conclusion, expectations based on hazard rate are represented within the oculomotor system,

495   with probabilistic information through time passage contributing to fixational stability. In contrast,

496   we found no evidence that temporal expectation based on precision is represented in the

497   oculomotor system, except when temporal attention is allocated. We observed action-perception

498   dissociations for both types of expectation on the precise timing of MS, alongside parallel

499   instances in which attentional modulations on MS coincided with performance benefits with

500   increasing hazard rate. Our study provides insights into the endogenous and stimulus-driven

501   components of oculomotor dynamics, demonstrating how temporal attention and distinct types

502   of expectation work together to enhance fixational stability.

Acknowledgments

507

508   The authors declare no competing interest.

509

510 REFERENCES

511 Aitkin, C.D., Santos, E.M., & Kowler, E. (2013) Anticipatory Smooth Eye Movements in Autism
512   Spectrum Disorder. PLOS ONE 8(12): e83230.https://doi.org/10.1371/journal.pone.0083230

513 Akdoğan, B., Balcı, F., & van Rijn, H. (2016). Temporal Expectation Indexed by Pupillary Response.
514   Timing & Time Perception, 4(4), 354-370. https://doi.org/10.1163/22134468-00002075

515 Amit, R., Abeles, D., Carrasco, M., & Yuval-Greenberg, S. (2019). Oculomotor inhibition reflects
516   temporal expectations. Neuroimage, 184 : 279–292.

517 Badde, S., Myers, C. F., Yuval-Greenberg, S., & Carrasco, M. (2020). Oculomotor freezing reflects
518   tactile temporal expectation and aids tactile perception. *Nature Communications*, *11*(1).
519   https://doi.org/10.1038/s41467-020-17160-1

520 Betta, E., & Turatto, M. (2006). Are you ready? I can tell by looking at your microsaccades.
521   *Neuroreport*, *17*(10), 1001–1004. https://doi.org/10.1097/01.wnr.0000223392.82198.6d

522 Bonneh, Y. S., Adini, Y., & Polat, U. (2015). Contrast sensitivity revealed by microsaccades. *Journal of*
523   *Vision*, *15*(9), 11. https://doi.org/10.1167/15.9.11

524 Bueti, D., & Macaluso, E. (2010). Auditory temporal expectations modulate activity in visual cortex.
525   *NeuroImage*, *51*(3), 1168–1183. https://doi.org/10.1016/j.neuroimage.2010.03.023

526 Cannon, J., O'Brien, A. M., Bungert, L., & Sinha, P. (2021). Prediction in autism spectrum disorder: A
527   systematic review of empirical evidence. *Autism Research*, 14(4), 604–630.
528   https://doi.org/10.1002/aur.2482

529 Chen, Y., Zhou, M., Zheng, Z., & Chen, D. (2019). Time-aware smart object recommendation in social
530   internet of things. *IEEE Internet of Things Journal*, *7*(3), 2014-2027.

531 Collewijn H., & Kowler, E. (2008). The significance of microsaccades for vision and oculomotor
532   control.*Journal of Vision,* 8(14):20.1-21. doi: 10.1167/8.14.20.

533 Correa, A. & Nobre, A. C. (2008). Neural modulation by regularity and passage of time. *Journal of*
534   *Neurophysiology,* 100, 1649–1655 .

535 Coull, J. T. (2009). Neural substrates of mounting temporal expectation. *PLoS Biology*, *7*(8).
536   https://doi.org/10.1371/journal.pbio.1000166

537 Coull, J. T., Frith, C. D., Büchel, C. & Nobre, A. C. (2000). Orienting attention in time: behavioural and
538   neuroanatomical distinction between exogenous and endogenous shifts. *Neuropsychologia*, 38,
539   808–819.

540 Coull, J.T., Giersch, A. The distinction between temporal order and duration processing, and
541   implications for schizophrenia. *Nat Rev Psychol* 1, 257–271 (2022). https://doi.org/10.1038/s44159-
542   022-00038-y

543 Dankner, Y., Shalev, L., Carrasco, M. & Yuval-Greenberg, S. (2017). Prestimulus inhibition of
544   saccades in adults with and without attention-deficit/ hyperactivity disorder as an index of temporal
545   expectations. *Psychological Science*

546   Denison, R. N., Tian, K. J., Heeger, D. J., & Carrasco, M. (2024). Anticipatory and evoked visual
547   cortical dynamics of voluntary temporal attention. Nature Communications, 15(1).
548   https://doi.org/10.1038/s41467-024-53406-y

549   Denison, R. N., Yuval-Greenberg, S., & Carrasco, M. (2019). Directing voluntary temporal attention
550   increases fixational stability. *Journal of Neuroscience*, https://doi.org/10.1101/449264

551   Degos, B., Pouget, P. & Missal, M. From anticipation to impulsivity in Parkinson's disease. *npj*
552   *Parkinson's Dis.* 8, 125 (2022). https://doi.org/10.1038/s41531-022-00393-w

553   Duyar, A., & Carrasco, M. (2024). Eyes on the Past: Gaze Stability Differs between Temporal
554   Expectation and Temporal Attention. bioRxiv. https://doi.org/10.1101/2024.06.07.598015

555   Duyar, A., Denison, R. N., & Carrasco, M. (2023). Exogenous temporal attention varies with temporal
556   uncertainty. *Journal of vision*, *23*(3), 9. https://doi.org/10.1167/jov.23.3.9

557   Duyar, A., Ren, S., & Carrasco, M. (2024). When temporal attention interacts with expectation.
558   *Scientific Reports*, *14*(1). https://doi.org/10.1038/s41598-024-55399-6

559   Engbert, R., Kliegl, R. (2003). Microsaccades uncover the orientation of covert attention, Vision
560   Research, 43: 1035-45.

561   Glasser, D. M., & Tadin, D. (2014). Modularity in the motion system: Independent Oculomotor and
562   perceptual processing of brief moving stimuli. *Journal of Vision*, *14*(3), 28–28.
563   https://doi.org/10.1167/14.3.28

564   Gonzalez-Gadea, M. L., Chennu, S., Bekinschtein, T. A., Rattazzi, A., Beraudi, A., Tripicchio, P.,
565   Moyano, B., Soffita, Y., Steinberg, L., Adolfi, F., Sigman, M., Marino, J., Manes, F., & Ibanez, A.
566   (2015a). Predictive coding in autism spectrum disorder and attention deficit hyperactivity disorder.
567   *Journal of Neurophysiology*, *114*(5), 2625–2636. https://doi.org/10.1152/jn.00543.2015

568   Grabenhorst, M., Maloney, L. T., Poeppel, D. & Michalareas, G. Two sources of uncertainty
569   independently modulate temporal expectancy. *Proc. Natl. Acad. Sci.* 118(16), 1.
570   https://doi.org/10.1073/pnas.2019342118 (2021).

571   He, P., & Kowler, E. (1989). The role of location probability in the programming of saccades:
572   Implications for "center-of-gravity" tendencies. *Vision Research, 29*(9), 1165–1181.
573   https://doi.org/10.1016/0042-6989(89)90063-1

574   Kowler, E., & Collewijn, H. (2010). The eye on the needle. *Nature Neuroscience, 13(12),* 1443–1444.
575   doi:10.1038/nn1210-1443

576   Kowler, E. (2011). Eye movements: The past 25 years. *Vision Research*, *51*(13), 1457–1483.
577   https://doi.org/10.1016/j.visres.2010.12.014

578   Kowler, E. (2024). Vision and microsaccades: Time to pay attention! *Current Biology*, *34*(4), R153–
579   R154. https://doi.org/10.1016/j.cub.2024.01.032

580   Lieberman H. R., & Pentland A.P. (1982) Microcomputer-based estimation of psychophysical
581   thresholds: The Best PEST. *Behavior Research Methods & Instrumentation,* 14, 21–25.

582   Maris, E., & Oostenveld, R. (2007). Nonparametric statistical testing of EEG-and MEG-data. *Journal of*
583   *Neuroscience Methods,* 164(1), 177–190, https://doi.org/10.1016/j.jneumeth.2007.03.024.

584 Martin, B., Franck, N., Cermolacce, M. *et al.* Fragile temporal prediction in patients with schizophrenia
585   is related to minimal self disorders. *Sci Rep* 7, 8278 (2017). https://doi.org/10.1038/s41598-017-
586   07987-y

587 Martinez-Conde, S., Macknik, S. L., Troncoso, X. G., Hubel, D. H. (2009). Microsaccades: a
588   neurophysiological analysis, *Trends in Neuroscience*, 32: 463-75.

589 Martinez-Conde, S., Otero-Millan, J., Macknik, S. (2013). The impact of microsaccades on vision:
590   towards a unified theory of saccadic function. *Nature Reviews Neuroscience* 14, 83–96.

591 Otero-Millan, J., Macknik, S. L., Serra, A., Leigh, R. J., & Martinez-Conde, S. (2011). Triggering
592   mechanisms in microsaccade and saccade generation: A novel proposal. *Annals of the New York*
593   *Academy of Sciences*, *1233*(1), 107–116. https://doi.org/10.1111/j.1749-6632.2011.06177.x

594 Palmieri, H., Fernández, A., & Carrasco, M. (2023). Microsaccades and temporal attention at different
595   locations of the visual field. *Journal of vision*, *23*(5), 6. https://doi.org/10.1167/jov.23.5.6

596 Robinson, F. R. (1995). Role of the cerebellum in movement control and adaptation. *Current Opinion*
597   *in Neurobiology*. 5: 755–762.

598 Robinson, F. R., Straube, A. & Fuchs, A. F. (1993). Role of the caudal fastigial nucleus in saccade
599   generation. II. Effects of muscimol inactivation. *Journal of Neurophysiology*. 70: 1741–1758.

600 Rohenkohl, G., Cravo, A. M., Wyart, V., & Nobre, A. C. (2012). Temporal expectation improves the
601   quality of sensory information. *Journal of Neuroscience*, *32*(24), 8424–8428.
602   https://doi.org/10.1523/jneurosci.0804-12.2012

603 Rolfs, M. (2009). Microsaccades: Small steps on a long way. *Vision Research*, 49 (20): 2415 - 2441.

604 Rolke, B. & Hofmann, P. Temporal uncertainty degrades perceptual processing. *Psychon. Bull. Rev.*
605   14, 522–526 (2007).

606 Spering, M., & Carrasco, M. (2015). Acting without seeing: Eye movements reveal visual processing
607   without awareness. *Trends in Neurosciences*, *38*(4), 247–258.
608   https://doi.org/10.1016/j.tins.2015.02.002

609 Spering, M., Pomplun, M., & Carrasco, M. (2010). Tracking Without Perceiving: A Dissociation
610   Between Eye Movements and Motion Perception. *Psychological Science*, *22*(2), 216–225.
611   https://doi.org/10.1177/0956797610394659

612 Tal-Perry, N., & Yuval-Greenberg, S. (2022). The spatiotemporal link of temporal expectations:
613   Contextual temporal expectation is independent of spatial attention. *The Journal of Neuroscience*,
614   *42*(12), 2516–2523. https://doi.org/10.1523/jneurosci.1555-21.2022

615 Tal-Perry, N., & Yuval-Greenberg, S. (2020). *Pre-Stimulus Inhibition of Eye Movements Reflects*
616   *Temporal Expectation Rather than Time Estimation*. https://doi.org/10.31234/osf.io/zwp72

617 Thomaschke, R., & Dreisbach, G. (2013). Temporal predictability facilitates action, not perception.
618   *Psychological Science,* 24(7), 1335–1340, doi:10.1177/0956797612469411

619 Todorovic, A. & Auksztulewicz, R. Dissociable neural effects of temporal expectations due to passage
620   of time and contextual probability. *Hear. Res.* 399, 107871.
621   https://doi.org/10.1016/j.heares.2019.107871 (2021).

622   Todorovic, A., & de Lange, F. P. (2012). Repetition suppression and expectation suppression are
623   dissociable in time in early auditory evoked fields. *The Journal of Neuroscience*, *32*(39), 13389–
624   13395. https://doi.org/10.1523/jneurosci.2227-12.2012

625   Todorovic, A., Schoffelen, J.-M., van Ede, F., Maris, E., & de Lange, F. P. (2015). Temporal
626   expectation and attention jointly modulate auditory oscillatory activity in the beta band. *PLOS ONE*,
627   *10*(3). https://doi.org/10.1371/journal.pone.0120288

628   Valsecchi, M., Betta, E., & Turatto, M. (2006). Visual oddballs induce prolonged microsaccadic
629   inhibition. *Experimental Brain Research*, *177*(2), 196–208. https://doi.org/10.1007/s00221-006-
630   0665-6

631   Vetter, P., Badde, S., Phelps, E. A., & Carrasco, M. (2019). Emotional faces guide the eyes in the
632   absence of awareness. *eLife*, *8*. https://doi.org/10.7554/elife.43467

633   Vishwanath, D., & Kowler, E. (2003). Localization of shapes: Eye movements and perception
634   compared. *Vision Research, 43*(15), 1637–1653. https://doi.org/10.1016/S0042-6989(03)00168-8

635   Widmann, A., Engbert, R., & Schroger, E. (2014). Microsaccadic responses indicate fast
636   categorization of sounds: A novel approach to study auditory cognition. *Journal of Neuroscience*,
637   *34*(33), 11152–11158. https://doi.org/10.1523/jneurosci.1568-14.2014

638   Yablonski, M., Polat, U., Bonneh, Y.S. & Ben-Shachar, M. (2017). Microsaccades are sensitive to word
639   structure: A novel approach to study language processing. *Sci Rep* 7, 3999.
640   https://doi.org/10.1038/s41598-017-04391-4

FIGURES

A
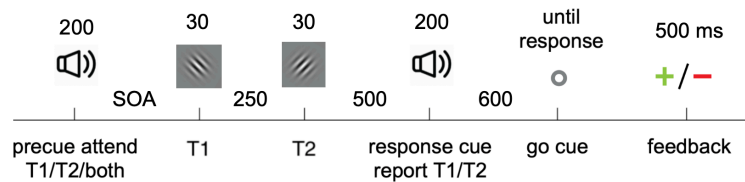
B
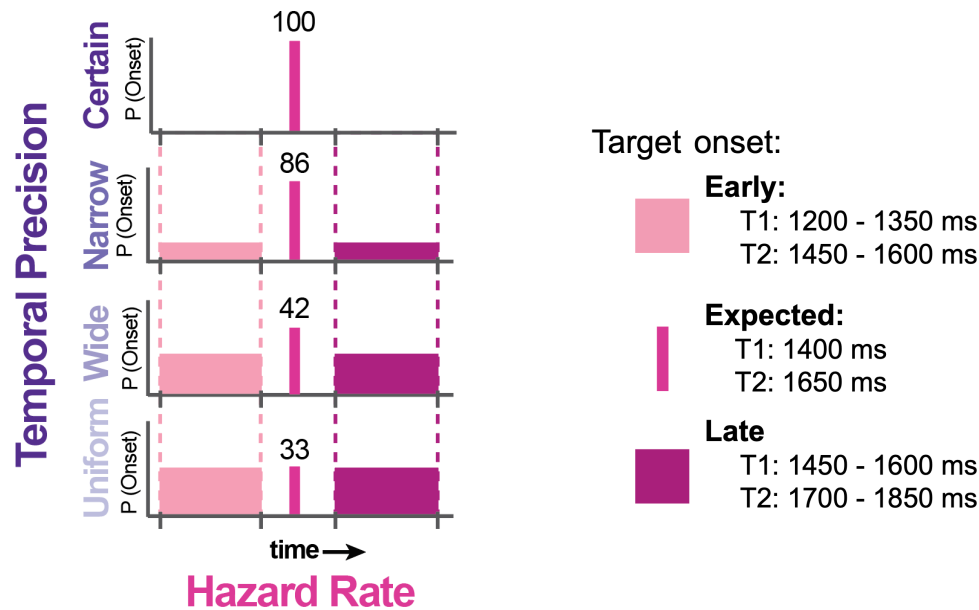
C
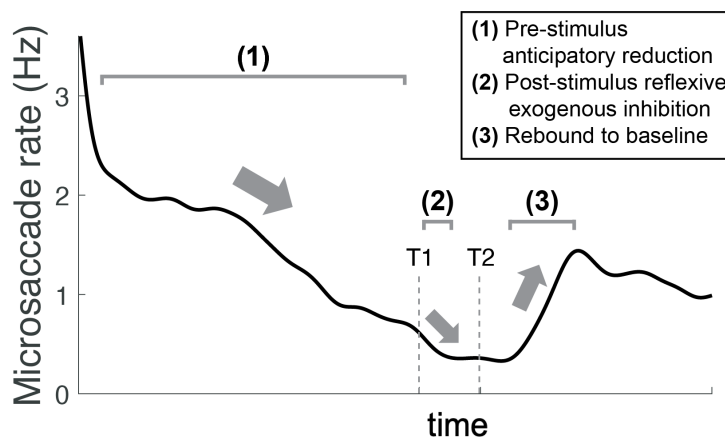
Figure 1. A. Experimental protocol (reported in Duyar, Ren & Carrasco, 2024). Attention was manipulated via the precue, and expectation was manipulated via the SOA between the precue and the targets. Observers reported the orientation (clockwise/counterclockwise relative to the reference axis) of the target indicated by the response cue at the end of the trial, with feedback provided based on accuracy. **B.** Hazard Rate was manipulated via within-trial target delay (Early, Expected, Late onsets), and Temporal Precision was manipulated via within-session onset variability (Certain, Narrow, Wide, Uniform). **C.** Typical MS rate profile.

666 **A**

667
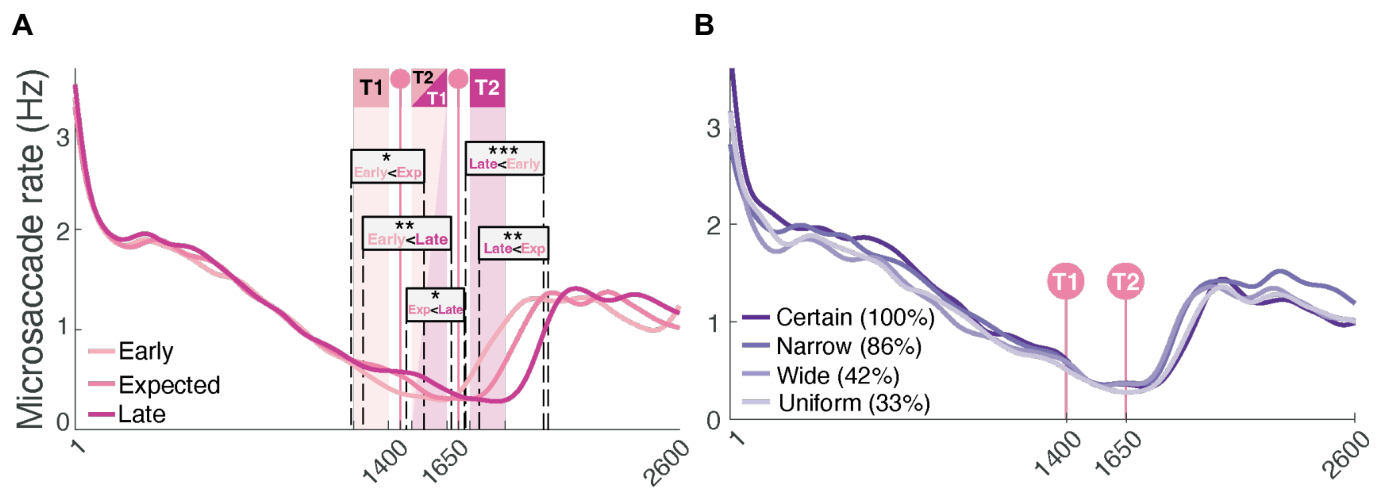
668 **Figure 2.** MS rates compared among temporal expectation conditions, regardless of attention
669 locked to precue onset. **A.** MS rate varied with hazard rate. There are three significant clusters
670 in the inhibitory period, where MS rates were higher for delayed stimuli, and two clusters during
671 the rebound, where MS rates were lower with delayed stimuli. Shaded regions indicate Early
672 and Late onsets, and the vertical lines with circles indicate the Expected moment. **B.** MS rates
673 were similar among different levels of temporal precision.*$p < 0.05$; **$p < 0.01$;*$p < 0.05$;
674 ***$p < 0.001$.
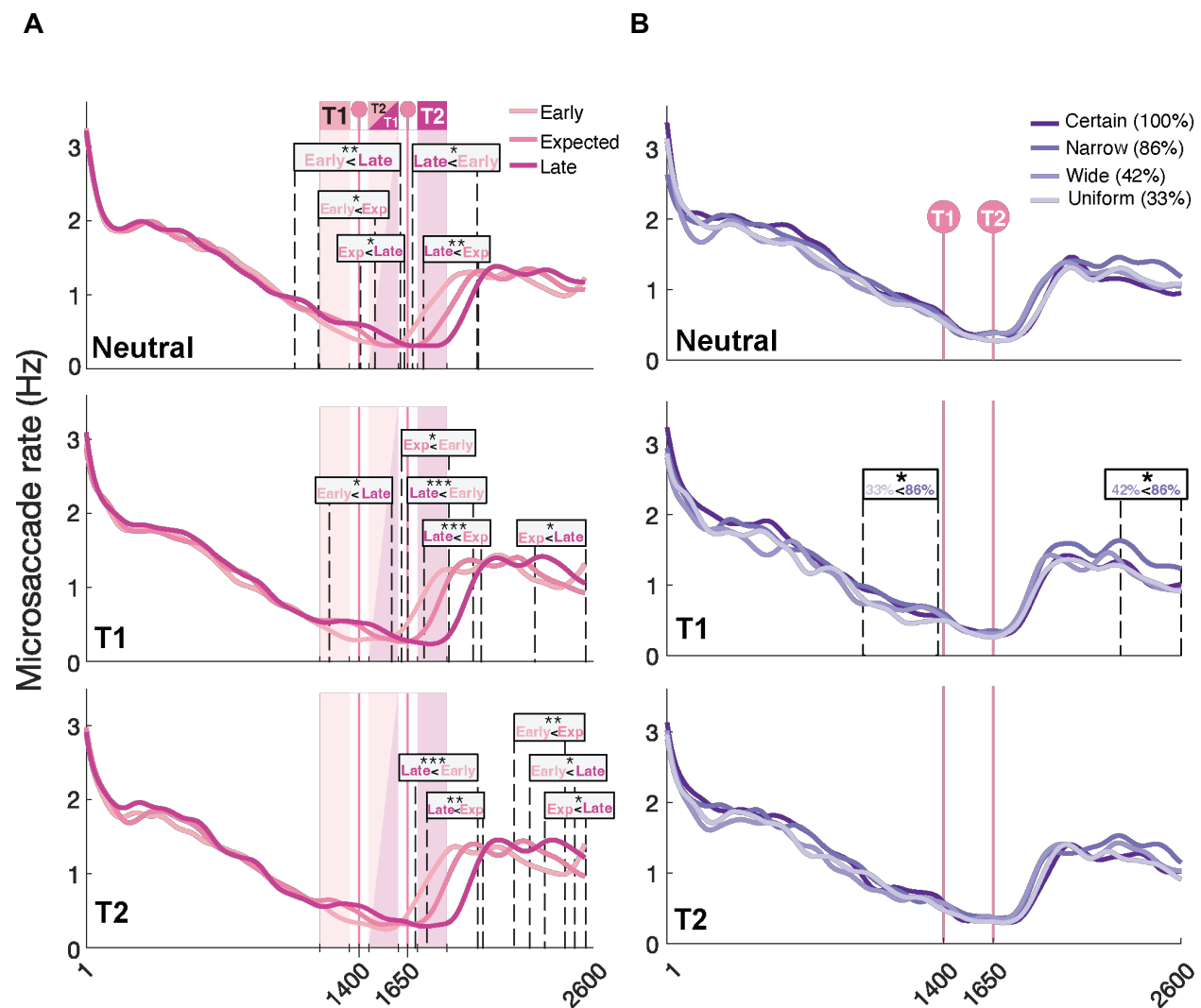
Figure 3. MS rates compared among temporal expectation conditions, separately for each attentional precue (Neutral: top panel, T1-precued: middle panel, T2-precued: bottom panel) trials). **A.** Darker colors indicate a higher hazard rate within the trial. **B.** Darker colors indicate a higher temporal precision.
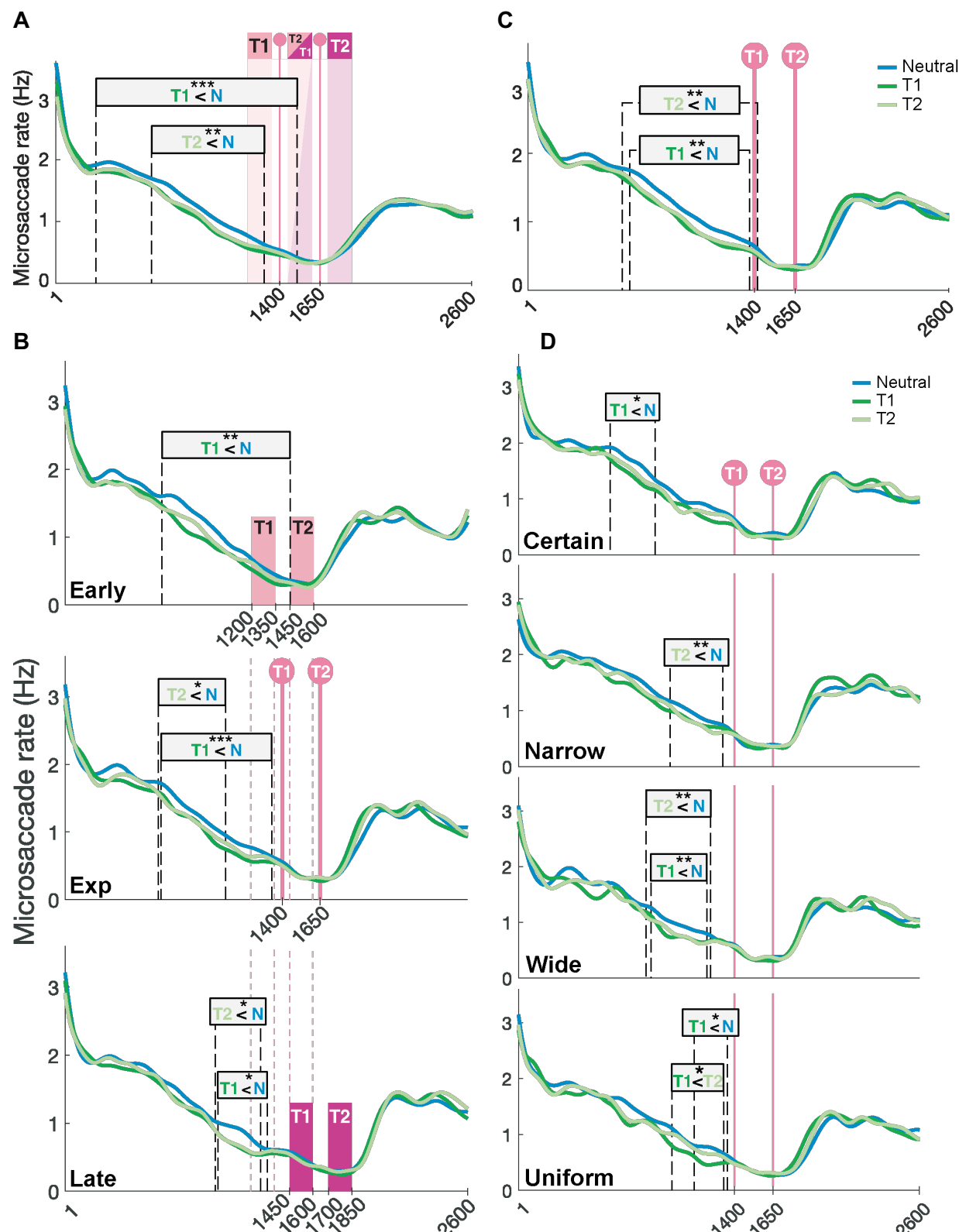
Figure 4. MS rates compared among attentional precue. Blue indicates neutral, green indicates valid attentional precue (dark green: T1-precued, light green: T2-precued). **A-B** includes trials from Wide and Uniform sessions, and **C-D** includes trials in which the stimuli occurred at the expected moment. **A.** MS rates collapsed across Hazard levels. **B.** Attentional precue effects separated by Hazard Rate (Early: top panel, Expected: middle panel, Late: bottom panel). **C.** MS rates collapsed across Precision levels. **D.** Attentional precue effects separated by Precision (Certain: top panel, Narrow: 2nd panel, Wide: 3rd panel, Uniform: bottom panel).
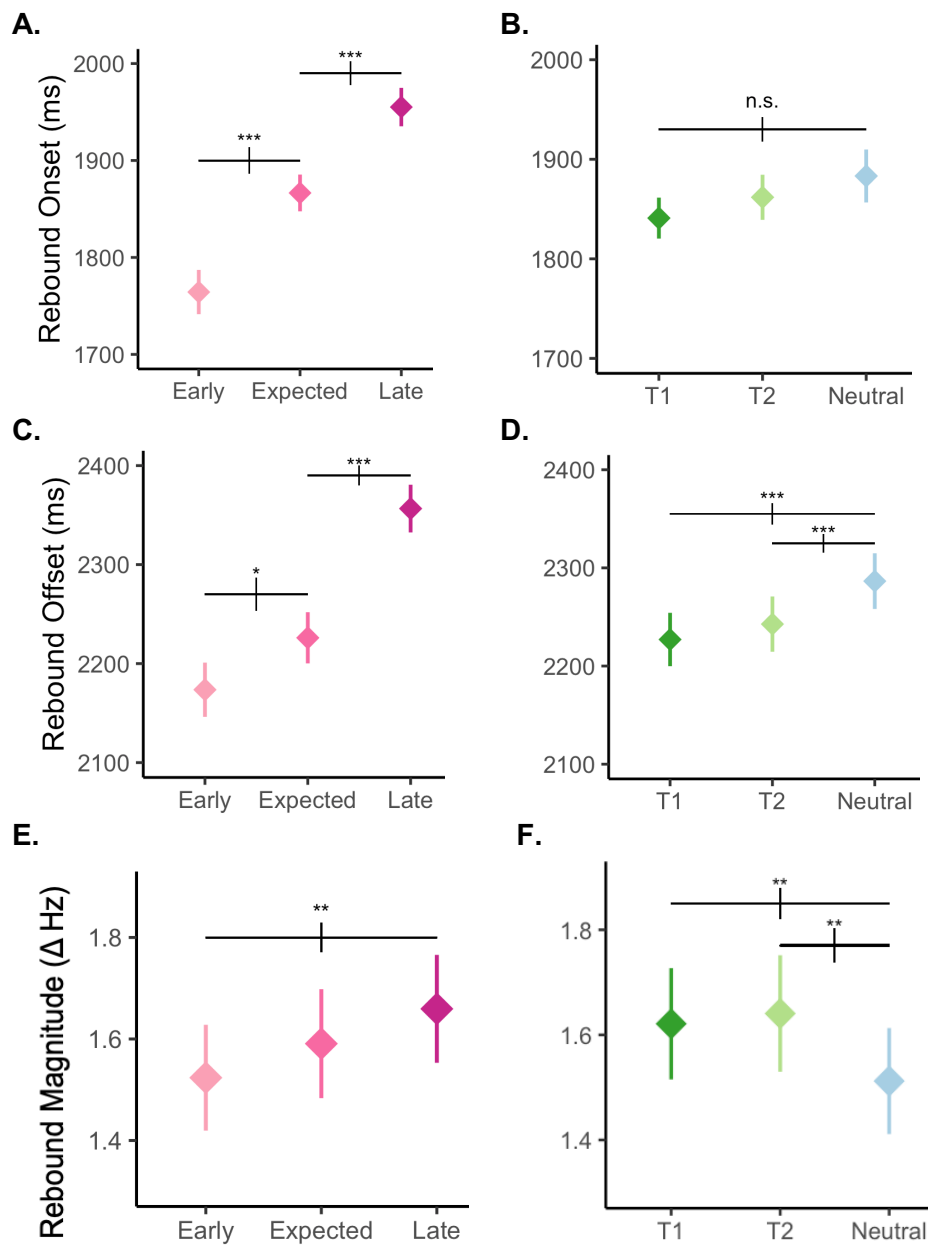
Figure 5. MS rate rebound parameters for Hazard Rate and attentional precue. Left column shows data collapsed across attention, and the right column shows data collapsed across the hazard rate. Error bars represent SEM. **A.** Main effect of Hazard on MS rebound onset. **B.** No significant effect of the attentional precue on MS rebound onset. **C.** Main effect of Hazard on MS rebound offset. **D.** Main effect attentional precue on MS rebound offset. **E.** Main effect of Hazard on MS rebound magnitude. **F.** Main effect attentional precue on MS rebound magnitude.
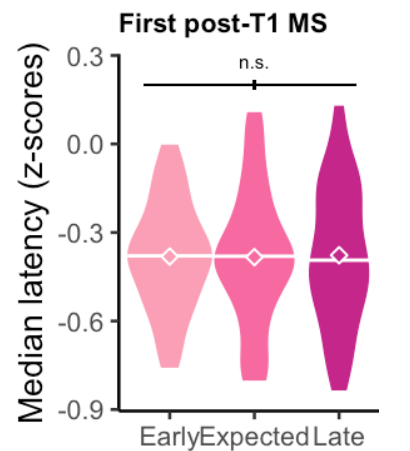
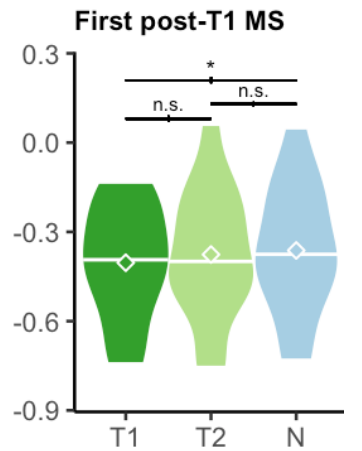**Locked to precue onset**
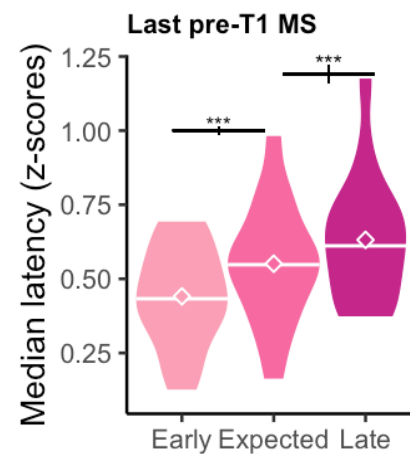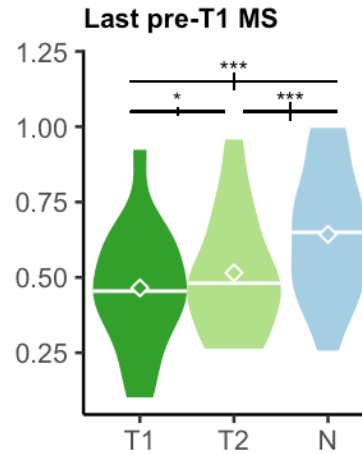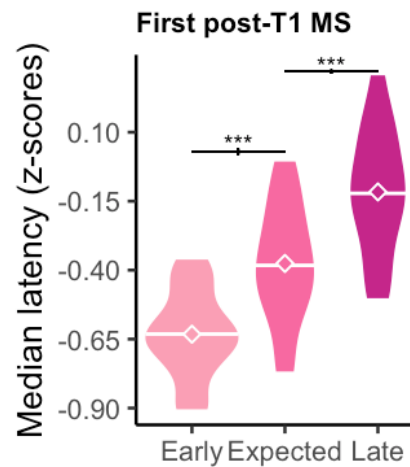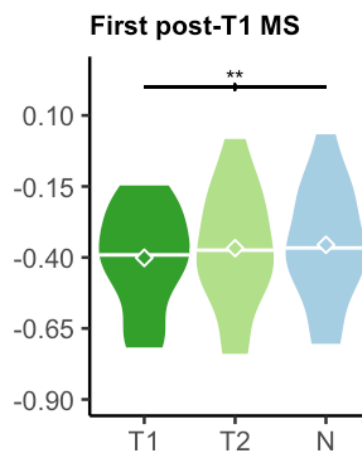
Locked to stimulus onset

Figure 6. Precise timing of MS that occurred around the stimulus. Diamond icons and the horizontal bars overlaid on the violin plots represent mean and median values, respectively. **A.** Main effect of Hazard on the last pre-T1 MS timing. **B.** Main effect of attentional precue on the last pre-T1 MS timing. **A-D** includes trials locked to precue onset; **E-F** includes trials locked to stimulus onset. **C.** Main effect of Hazard. **D.** Main effect of attentional precue. **E.** Main effect of Hazard. **F.** Main effect of attentional precue.
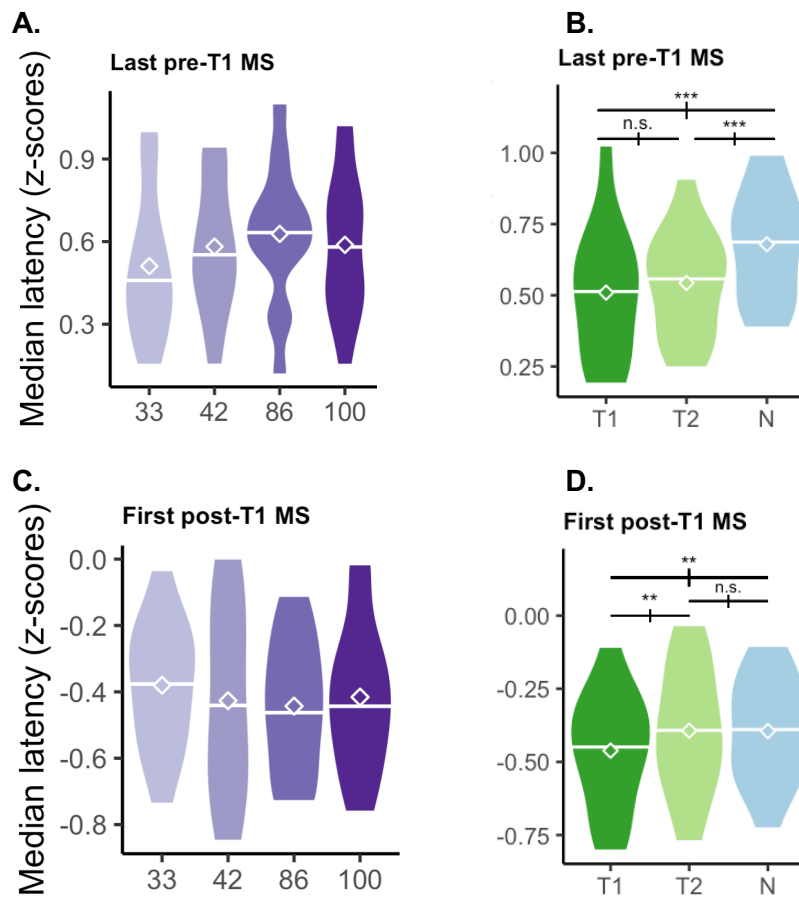
Figure 7. Precise timing of MS that occurred around the stimulus. **A.** No effect of Temporal Precision on the last pre-T1 MS timing. **B.** Main effect of attentional precue on the last pre-T1 MS timing. **C.** No effect of Temporal Precision on the first post-T1 MS timing. **D.** Main effect of attentional precue on the first post-T1 MS timing.