# Confirmation of age-related alterations in inhibitory control using a modified minimally delayed oculomotor response (MDOR) task

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Considerable effort has been made to measure and understand effect of ageing on inhibitory control using a range of behavioural tasks. In the minimally delayed oculomotor response (MDOR) task, participants are presented with a simple visual target step with variable target display duration (TDD), and instructed to saccade to the target not when it appears (a prosaccade response), but when it disappears (i.e. on target offset). Using this task, we recently found higher error rates and longer latencies for correct responses in older compared to younger participants. Here we have used a modified MDOR task, in which participants were presented with static placeholders identifying potential target positions (increasing spatial information), and three TDDs rather than two (reducing temporal predictability). We found that the yield of analysable trials was generally higher with this modified task and in 28 older (mean±SD age: 65±7y) and 25 younger (26±7y) participants the total overall error rate was again higher in the older group (30±18% vs 16±11%). An analysis of the temporal distribution of responses demonstrated a pronounced peak in error production around 150ms (young) or 200ms (old) after target onset. When we recalculated the error rate focusing on these errors, it was again significantly higher in the older group. The latency of correct responses (to offsets) was significantly increased in the older group, although much of this increase was accounted for by expected age-related visuomotor slowing. However, both latency and distribution data suggested that while older participants could generate increased levels of inhibition, they could not maintain these levels as efficiently as the younger participants. In 24 participants (15 old, 9 young) who completed both versions of the MDOR task, neither latency nor error rates differed significantly between versions. These results confirm an inhibitory control deficit in healthy older participants, and suggest that the dynamics of inhibitory control are also affected by ageing. The modified MDOR task yields more data

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## Abstract

Considerable effort has been made to measure and understand effect of ageing on inhibitory control using a range of behavioural tasks. In the minimally delayed oculomotor response (MDOR) task, participants are presented with a simple visual target step with variable target display duration (TDD), and instructed to saccade to the target not when it appears (a prosaccade response), but when it disappears (i.e. on target offset). Using this task, we recently found higher error rates and longer latencies for correct responses in older compared to younger participants. Here we have used a modified MDOR task, in which participants were presented with static placeholders identifying potential target positions (increasing spatial information), and three TDDs rather than two (reducing temporal predictability). We found that the yield of analysable trials was generally higher with this modified task and in 28 older (mean±SD age: 65±7y) and 25 younger (26±7y) participants the total overall error rate was again higher in the older group (30±18% vs 16±11%). An analysis of the temporal distribution of responses demonstrated a pronounced peak in error production around 150ms (young) or 200ms (old) after target onset. When we recalculated the error rate focusing on these errors, it was again significantly higher in the older group. The latency of correct responses (to offsets) was significantly increased in the older group, although much of this increase was accounted for by expected age-related visuomotor slowing. However, both latency and distribution data suggested that while older participants could generate increased levels of inhibition, they could not maintain these levels as efficiently as the younger participants. In 24 participants (15 old, 9 young) who completed both versions of the MDOR task, neither latency nor error rates differed significantly between versions. These results confirm an inhibitory control deficit in healthy older participants, and suggest that the dynamics of inhibitory control are also affected by ageing. The modified MDOR task yields more data while not altering basic performance parameters.

## Introduction

Stopping ourselves from executing motor actions or thinking thoughts is at least as important as being able to execute those actions or generate those thoughts. Without some means of inhibitory control, both behaviour and cognition become disordered. For this reason, the phenomenon of inhibitory control has been an important subject of investigation (Bari & Robbins 2013). Given that actions, unlike thoughts, lend themselves to third party observation and measurement, various tasks have been developed to study behavioural inhibitory control with the conviction that these provide insights into underlying cognitive processes (Castiglione et al. 2019; Logan & Cowan 1984; Logan et al. 1997). A number of oculomotor tasks have been used to study inhibitory control, including ocular no go (Crawford et al. 2005), countermanding (Hanes & Carpenter 1999) and antisaccade (AS) tasks (Alichniewicz et al. 2013; Hallett 1978). To this list we recently added the minimally delayed oculomotor response (MDOR) task (Knox & Pasunuru 2020; Knox et al. 2018; Wolohan & Knox 2014).

The MDOR task is a saccade task in which the stimulus is a target step with randomised direction and timing. Participants execute a saccade to a target to the left or right of fixation, not when it appears but when it is extinguished. Target display duration (TDD) is varied so that the onset of the target does not reliably predict the time of the offset. Performance is measured using the error rate (the proportion of trials in which participants execute a target directed saccade prior to target offset) and the latency of correct responses. While the MDOR task shares features with both AS and memory-guided saccade tasks (MGS; see Hutton, 2008, for a review), it differs from both in important ways. As in the AS task, the error rate provides information about inhibitory control performance. However, in the AS task there is a competition between processes which in each trial leads to either an error prosaccade or a correct antisaccade (Munoz & Everling 2004). Because the AS error rate is the product of this race between two processes competing for behavioural expression, an error prosaccade is not necessarily the result of a failure of inhibition: the error rate, while related to the effectiveness of inhibitory control, cannot provide an unambiguous measurement of that control (Reuter et al. 2007; Reuter et al. 2005). In the MDOR task there is no such competition. Further, in the

82    MDOR task by identifying responses to target *onsets*, which by definition must be inhibition

83    failures, an error rate specifically and closely related to the effectiveness of inhibition can be

84    calculated.

85

86    We assume that there is a working memory load in the MDOR task; at a minimum there is an

87    instruction to be remembered. But any memory load is reduced compared to both AS and MGS

88    tasks, because it is not necessary to remember a target location; the target is present

89    throughout the period of central fixation prior to target offset. Visuospatial attention is

90    involved in all types of saccade tasks (Kowler et al. 1995) with a close link between the

91    allocation of attention and saccade programming (Deubel 2008). In the AS task attentional

92    resources are divided or distributed between alternative target locations (Klapetek et al. 2016).

93    In most versions of the MGS task, the disappearance of a central fixation target provides the go

94    signal for a saccade directed to the remembered target position, therefore involving two

95    distinct locations and implying some division of attentional resources.  In the MDOR task, it is

96    the offset at the target location that provides both the go signal and indicates the position to

97    which the saccade should be directed. Taken together, these considerations suggest that the

98    MDOR task provides a means of investigating behavioural inhibition relatively uncontaminated

99    by those attentional and memory processes that, along with inhibitory control, are key

100   components of executive function (Friedman & Miyake 2017; Miyake et al. 2000).

101

102   We recently used the MDOR task to investigate the effect of normal healthy ageing on

103   behavioural inhibitory control (Knox and Pasunuru 2020). Comparing a group of older

104   participants with previously collected data from younger participants, we found that error rates

105   were higher and the latency of correct responses longer in the older group. Further

106   investigation of the latency differences suggested that most of the increase in latency was

107   accounted for by general age-related slowing in the visuomotor system (Salthouse 1996;

108   Verhaeghen 2011).

109

110 However, in the task as used in that study, the stimulus display was sparse, with only simple

111 fixation and saccade targets presented. At the end of each trial, after the eccentric target was

112 extinguished (providing both the go signal and the target location for the saccade), the stimulus

113 monitor was completely blank. This led to a number of potential uncertainties on the part of

114 participants. There was no visual feedback as to the spatial accuracy of their saccade, no

115 marker for how long they should fixate the eccentric position and no formal marker of the end

116 of the trial. This in turn could lead to early blinks and breaks of fixation, and the loss of data. We

117 also only used two TDDs (200ms and 1000ms) limiting the inferences that could be made with

118 regard to the timing of errors and differences in timing and rates between errors and correct

119 responses. And we were comparing the data collected from older participants with historical

120 data collected in earlier experiments.

121

122 We have addressed these and other issues in the current experiment by the addition of static

123 placeholders (at the potential positions of the saccade targets) and the use of three rather than

124 two TDDs. The placeholders reduce the spatial uncertainty in the task; participants have

125 increased information about *where* to saccade to. The appearance and disappearance of the

126 placeholders also provides a clear indication of the start and end of trials, delineating the

127 intertrial period. The introduction of a third, intermediate TDD (600ms), reduces the temporal

128 predictability of the task, while also proving additional data about the temporal distribution of

129 responses.

130

131 In this new experiment, both old and young participants (returning and naïve) have been

132 contemporaneously tested using the modified MDOR task, in order to further investigate task

133 performance and ageing effects reported previously. Retesting previous participants with the

134 new version of the MDOR task (as well as recruiting and testing naïve participants) allowed an

135 investigation of general laboratory or task familiarity effects, as well as an explicit comparison

136 of the cumulative effects of the changes made in the new task, all issues that relate to

137 performance stability.

138

## Materials & Methods

### Ethics and Participants

Healthy, adult participants, with normal or corrected to normal vision were recruited, under ethical approval from the University of Liverpool Research Ethics committee (Reference number: 2933) and the study was conducted in accord with the ethical standards laid down in the 1964 Declaration of Helsinki.  Interested potential participants were provided with a study information sheet. Having read this, and after the experiment was explained to them and they had an opportunity to ask questions, written consent was obtained. Older participants were offered £10 to compensate them for their time and for the expense of travelling in to the University for testing. Younger participants, who were recruited from the University, were offered £5.

### Apparatus and Stimuli

We used the same apparatus as in previous MDOR experiments (Knox & Pasunuru 2020; Knox et al. 2018). Briefly, stimuli were presented on a 21" monitor (1024×768 spatial resolution, 100 Hz temporal resolution) driven by a VSG2/5 card (Cambridge Research Systems, Rochester, UK), positioned on the fronto-parallel plane 57 cm from the participant's eye.  Horizontal eye position of the left eye was recorded using a Skalar Iris IR Eye Tracker, with the eye tracker output digitised at 1kHz with 16-bit precision using a CED Power 1401 (Cambridge Electronic Design, Cambridge, UK) interface. Oculomotor data were stored for off-line, trial-by-trial analysis using custom software.

Fig 1 illustrates the modified MDOR task used in the present study. As noted in the Introduction, there are two key differences between this version of the task and the version used previously (see Fig 1A in Knox and Pasunuru, 2020, for comparison): the inclusion of two target placeholders to the left and right of fixation (within which the saccade target appeared), and the use of three, not two, target display durations (TDD). Each trial began with the appearance of a central fixation target (a 0.2° black square on a light background) and the two target placeholders. The place holders were themselves 0.8° square boxes centred 5° from the fixation target. The fixation target was presented for a randomised period of 0.5s to 1.5s.  A

167 synchronous MDOR task was used in which, when the fixation target was extinguished, the

168 saccade target appeared at the centre of one of the placeholders (5° from the fixation target)

169 and was displayed for 200ms, 600ms or 1000ms. The offset of the target was the go signal for

170 the saccade.

171 **Procedures**

172 At the beginning of each testing session, the task was explained to the participant by stepping

173 through it, before a number of trials were run at actual speed. Participants were then carefully

174 positioned by adjusting table height, chin rest and cheek pads, and the eye tracker was applied

175 and adjusted as necessary. Participants were instructed to maintain fixation centrally, until the

176 target in one of the placeholders was extinguished. Then they were to saccade to the centre of

177 the empty placeholder previously occupied by the target, and to maintain fixation there until

178 both placeholders were extinguished (after 1s), leaving the monitor blank. It remained blank

179 until the beginning of the next trial. Participants were asked to refrain from blinking except

180 during the intertrial interval when the monitor was blank. Practice trials were run; these data

181 were not retained. Each participant then completed two runs of 150 MDOR trials (in the original

182 version of the task the run length was 120 trials) with a break in between them. The quality of

183 performance was carefully monitored to ensure that it was maintained, with verbal feedback

184 given as necessary.

185 In order to calibrate eye tracker output, at the end of each MDOR run, a 32-trial calibration

186 procedure was performed (again the same procedure as used previously; Knox & Pasunuru

187 2020; Knox et al. 2018). Calibration trials were simple prosaccade trials in which, after a

188 randomised fixation period (0.5-1.5s), the fixation target was extinguished and a saccade target

189 was presented to the left or right at an eccentricity of 5° or 10° (randomised and with equal

190 frequency) for 1s. Participants were instructed to fixate the central point and saccade to the

191 target as soon as it appeared, fixating it until it was extinguished, at which point they could

192 blink and return to the centre, ready for the next trial. All older participants also completed the

193 Addenbrookes Cognitive Examination (ACE) III questionnaire (Hsieh et al. 2013).

194 **Analysis**

195   Oculomotor data were analysed, as previously (Knox & Pasunuru 2020; Knox et al. 2018), using

196   an interactive program which displayed the eye position data and the time at which the "go"

197   signal (target offset in MDOR trials) occurred. The calibration data were used to transform the

198   data from arbitrary system units into units of degrees of eye rotation. Trials with blinks or

199   unstable fixation prior to target appearance were removed from the analysis. For each valid

200   MDOR trial, the latency (the time from target offset to saccade onset) and amplitude of the

201   primary target-directed saccade were measured.

202   Data were collated in MS Excel. Any target directed saccade with an amplitude greater than 1°

203   that occurred from 80ms after target onset to 80ms after target offset was counted as an error.

204   Error responses were removed and collated separately from correct trials, and the error rate

205   calculated. Any target directed saccade occurring from 80ms to 1000ms after target offset was

206   counted as a correct response. For each participant, median correct saccade latency was

207   calculated along with the error rate for each TDD. We also calculated the overall total error rate

208   collapsing across TDDs and subsequently derived other error rates, as described in the Results

209   section. Across participant groups, latency and error rate were summarised using the mean. In

210   order to obtain an estimate of prosaccade latency, we calculated median saccade latency from

211   each participant's calibration runs, collapsed across direction, eccentricity and run.

212   Statistical analysis was conducted with SPSS v25. Repeated measures ANOVA was used to

213   compare groups and conditions with $\eta_p^2$ reported for effect size (details in Results). Cohen's $d_s$

214   (Lakens 2013) is also reported where relevant.

215

216   **Results**

217   **Participant characteristics**

218   A total of 53 participants were recruited and tested in this study. Of 28 older participants (17

219   male; mean±SD age: 65±7y; the "old" group), 16 were naïve to testing and 12 had taken part in

220   our previous study on ageing. The mean ACE III total score for the older group of 28 participants

221   was 95 (range 89-100). The cut-off for suspicion of neurological disease is a score of 88 (Noone

222  2015) and the mean score for our group was very similar to that reported in (Hsieh et al. 2013)

223  for a similarly aged healthy control group. Of 25 younger participants (mean age: 26±7y; the

224  "young" group), 21 were naïve to testing and 4 had participated in a previous experiment. A

225  total of 15 075 trials were available for analysis (7828 from the old group, 7247 from the young

226  group). The mean trial yield per participant was 93±8% and 97±4% for the old and young groups

227  respectively (t=1.98, df=51, p=0.05).

228

229  **Analysis of saccade latency**

230  The latency of correct MDOR responses across conditions followed the same pattern reported

231  previously (Fig 2). Note that one participant in the old group exhibited extremely high error

232  rates for two of the TDDs leaving fewer than 5 correct trials from which to calculate latency. We

233  therefore removed this participant's data from the latency analysis (leaving n=27).  In both

234  participant groups, raw latency (Fig 2A) was longest in those trials with the shortest TDD

235  (200ms mean±SD; old: 458±67ms, young 370±67ms) and was clearly modulated by TDD. For

236  both groups there was a monotonic relationship between TDD and latency. For the longest TDD

237  (1000ms) latency was 290±48ms and 257±34ms in the old and young groups respectively.  It

238  was consistently longer in the older group by 88ms, 47ms and 33ms for TDDs of 200, 600ms

239  and 1000ms respectively. These latency data were tested with repeated measures ANOVA,

240  treating TDD as within and group as between subjects factors. Both TDD ($F_{2,49}$=160, p<0.001,

241  $\eta_p^2$=0.87) and group ($F_{1,50}$=18, p<0.001, $\eta_p^2$=0.26) returned statistically significant results, with a

242  statistically significant interaction between factors ($F_{2,49}$=6.4, p=0.003, $\eta_p^2$=0.21). The difference

243  between groups for each TDD was statistically significant (t-test with Holm-Sidak correction for

244  multiple comparisons; p<0.01 for all pairs) with effect sizes (Cohen's $d_s$ ) of 1.17, 0.94 and 0.78

245  for TDDs of 200, 600ms and 1000ms respectively.

246

247  When we compared the latency of reflexive prosaccades in old and young groups (using data

248  from the calibration task), latency in the old group (209±44ms) was significantly longer

249  compared to the young group (181±41ms; t=2.4, df=50, p=0.02; $d_s$=0.66). Given that this

250  difference represents the general age-related slowing expected for the visuomotor system, we

251 investigated whether once this was taken into account, MDOR latency still differed between

252 groups. We calculated "corrected" MDOR latency values for each participant by subtracting

253 their median calibration latency from the MDOR latency, and then recalculated MDOR group

254 means (Fig 2B). This had the effect of reducing the latency difference between groups to 60ms,

255 18ms and 4ms for TDDs of 200ms, 600ms and 1000ms respectively. When ANOVA was run on

256 these data, the effect of TDD was unchanged ($F_{2,49}$=160, p<0.001, $\eta_p^2$=0.87), the difference

257 between groups no longer reached statistical significance ($F_{1,50}$=3.2, p=0.081, $\eta_p^2$=0.06) but

258 there remained a statistically significant interaction between the factors ($F_{2,49}$=6.4, p=0.003,

259 $\eta_p^2$=0.21). Only the difference at 200ms was statistically significant (t=2.96, df=50, adjusted

260 p=0.01) and $D_s$ was now 0.83, 0.31 and 0.10 for TDDs of 200, 600ms and 1000ms respectively.

261

262  In order to quantify and investigate the modulation of latency by TDD, taking all of the data

263 into account, we calculated the least-squares linear regression of latency on TDD for each

264 participant. For both groups this captured the relationship reasonably well, given that the mean

265 $r^2$ for these functions was 0.89±0.12 and 0.86±0.12 for old and young groups respectively. Using

266 the regression slopes to represent the overall modulation in latency, we found that the mean

267 slope for the old group was -0.21±0.08 and was -0.14±0.07 for the young group; this difference

268 between slopes was statistically significant (t=3.5, df=50, p<0.001, $d_s$=0.99). To provide

269 comparative data with values reported previously (Knox & Pasunuru 2020) we also calculated

270 the difference in latency between the 200ms and 1000ms TDD for each participant; the group

271 means for this difference were 168±61ms and 113±52ms for old and young groups respectively

272 (t=3.5, df=50, p=0.001, $d_s$ =0.97).

273

274 **Analysis of errors**

275 The overall error rate, for all errors calculated across TDD conditions, was considerably higher

276 in the old compared with the young group (30±18% vs 16±11%; t=3.3, df=44, p=0.002; $d_s$= 0.93).

277 When calculated for each TDD, the total error rate ($ER_t$) was higher in the older group and there

278 was a modulation with TDD (it increased with TDD; Fig 3). When analysed with a similar ANOVA

279 design as used above for latency, TTD returned a statistically significant result ($F_{2,50}$=94,

280    p<0.001, $\eta_p^2$=0.79) as did group ($F_{1,51}$=10, p=0.002, $\eta_p^2$=0.17), with a statistically significant

281    interaction between the factors ($F_{2,50}$=7.2, p=0.002, $\eta_p^2$=0.22). The group difference in $ER_t$ for

282    each TDD was statistically significant (adjusted p values 200ms:p=0.03, 600ms and

283    1000ms:p<0.01), and $d_s$ for each TDD was 200ms:0.67, 600ms:0.99 and 1000ms:0.81.

284

285    The $ER_t$ captured all the errors as defined in the Methods. We investigated the pattern of errors

286    further by constructing average distributions of all responses for the two participant groups (Fig

287    4). In each of the six distributions in Fig 4 there are two clear peaks, identified by the arrows.

288    The larger of these is composed of the correct responses, which occur after the target offset

289    (the vertical dashed line at 0ms in each plot). The bins prior to +80ms contain error responses,

290    and errors (all of which contribute to the $ER_t$) occur throughout the period between target

291    onset (at -200ms, -600ms and -1000ms) and target offset. Errors are not distributed uniformly

292    throughout this period, and the second clear feature in these distributions is the early peak just

293    after target onset. The timing of this peak is consistent with these responses being primarily

294    uninhibited responses to the target onset.

295

296    Using the average distributions, we defined two further error rates. To capture the early peak,

297    we calculated the proportion of saccades contained in the five histogram bins centred on the

298    peak bin as indicated in Fig 4 (ie covering a latency range of 250ms). This was calculated for

299    each participant individually, and group means then calculated. We will refer to this as the peak

300    error rate ($ER_{pk}$). As can be seen from Fig 5A (and consistent with Fig 4), the $ER_{pk}$ was

301    consistently higher in the older group although no longer as clearly modulated by TDD. When

302    tested with ANOVA, the TDD did have a significant effect on the rate ($F_{2,50}$=43, p<0.001,

303    $\eta_p^2$=0.63) as did group ($F_{1,51}$=6.1, p<0.017, $\eta_p^2$=0.12); the TDD x Group interaction was not

304    statistically significant ($F_{1,51}$=0.2, p=0.8). For the 600ms and 1000ms TDDs, we also defined a

305    "residual" error rate ($ER_r$). As can been seen from Fig 4, errors continued to occur at a low rate

306    after the peak. While for the shortest TDD (200ms) it was difficult to distinguish between the

307    end of the initial peak and the beginning of correct responses to the target offset, this was not

308    the case for the longer TDDs. We therefore used the five bins (the same number of bins used

309    for the ER$_{pk}$ rate) from -250ms to 0ms to calculate ER$_r$ for the two longer TDDs (Fig 5B), and

310    calculated the proportion of errors contained in these bins for each participant. The difference

311    between groups was now less clear, partly because the average number of errors was relatively

312    low, particularly in the young participants. Statistically, there was now no significant effect of

313    TDD ($F_{1,51}$=2.3, p=0.133), although both the interaction ($F_{1,51}$=7.2, p=0.01, $\eta_p^2$=0.12) and the

314    group effect were significant ($F_{1,51}$=8.7, p=0.005, $\eta_p^2$=0.14).

315

316    As indicated by the arrows on Fig 4, the early peaks occurred slightly later in time in the old

317    compared to the young group. To examine the timing of these early error saccades further, we

318    used the bin ranges discussed above (five bins centred on the peak bin in each distribution, a

319    range of 250ms), calculated the median latency of saccades falling in these ranges for each

320    participant and then calculated and compared group means (Fig 6). Saccade latency was longer

321    in the old group as would be expected given the general age-related slowing in the old group

322    noted above. The latency difference was 43ms, 29ms and 30ms for the TDDs of 200ms, 600ms

323    and 1000ms respectively.  When tested with a repeated measures ANOVA similar to those

324    described previously, the difference between groups was statistically significant (Group:

325    $F_{1,47}$=51, p<0.001, $\eta_p^2$=0.52; TDD: $F_{2,46}$=94, p<0.001, $\eta_p^2$=0.80; interaction: $F_{2,46}$=0.4, p=0.68).

326    Note that there were two young and one old participant in whom no median latency was

327    available for at least one TDD; data from these participants were not included in the ANOVA.

328

329    We also investigated whether there was any relationship between each participants'

330    prosaccade latency and the latency of the error saccades comprising the early peak (Fig 7).

331    While the general context in which these prosaccades and MDOR peak error saccades were

332    executed was very different, given that in both cases these were reflexive responses to target

333    onsets, it seemed plausible that there might be a relationship.  Overall correlations (in which

334    old and young groups were combined) were calculated (note that in Fig 7 the two groups can

335    be distinguished) for each TDD separately. Correlations were generally low, although for the

336    200ms and 1000ms TDDs they reached statistical significance.  As can be seen from both Figs 6

337    and 7, latency is lowest for the longest TDD, following the same general pattern observed for

338  the correct responses. However, the peak error latency tended to be lower than the prosaccade

339  latency from the calibration task; this is particularly marked for the 1000ms TDD (Fig 7C).

340

341  In order to assess whether the two participant groups might adopt fundamentally different

342  strategies, sacrificing speed of response for more effective inhibition, we examined the data for

343  evidence of speed/accuracy trade-offs (Fig 8). Statistically there were no significant correlations

344  between either $ER_t$ and latency in either group (young Fig 8A; old Fig 8C) or $ER_{pk}$ and latency

345  (young Fig 8B; old Fig 8D). Thus there was no evidence of a difference in strategy in the two

346  groups. However, there did appear to be a systematic difference in both groups between the

347  shortest and longest TDDs. For the shortest TDD (200ms) error rate varied widely over a

348  relatively restricted latency range, whereas for the longest TDD (1000ms) latency appeared to

349  be more variable.

350

351  **Comparison of original and modified MDOR tasks**

352  We sub-divided the old group into those participants who were naïve to testing (N; n=16) and

353  those who participated in a previous experiment and returned for testing with the modified

354  MDOR task (R; n=12). These two groups were comparable in terms of age (N:65±7y; R:64±6y)

355  and ACE III score (N:95±3; R:96±3). Both latency and total error rate were indistinguishable

356  between these groups (Fig 9).

357

358  There were a total of 24 participants (15 old, 9 young) for whom data were available both from

359  the original MDOR task (MDOR-O)  and the new modified task (MDOR-M). Of the 15 old

360  participants, 12 completed MDOR-O first and MDOR-M second; only 3 completing the tasks in

361  the opposite order. In the young group 4 did MDOR-O first. One of the key differences between

362  the two versions of the task was that in MDOR-O there were only two TDDs (200ms and

363  1000ms), so these were the conditions for which comparisons could be made. As is clear from

364  Fig 10, both latency and total error rate were very similar in the two tasks whether considering

365  data from all the participants in an omnibus analysis (Latency – Fig 10A; Total Error Rate – Fig

366  10D), or separately analysing data from young (Latency – Fig 10B; Total error rate – Fig 10F) and

367  old (Latency – Fig 10C; Total error rate – Fig 10E) groups. We subjected the latency and error

368  rate data to separate ANOVA's of the same design, which treated TDD as a within and task (M

369  vs O) as between subjects factors. Unsurprisingly the task factor returned a statistically non-

370  significant result for both latency ($F_{1,46}=2.0$, $p=0.13$) and error rate ($F_{1,46}=0.12$, $p=0.74$).

371

372  **Discussion**

373  Our objectives in the current experiment were to investigate the use of a modified MDOR task

374  and further examine the effects of normal ageing that we had observed with the original task.

375  The MDOR task was originally conceived to provide a means of investigating behavioural

376  inhibitory control using  eye movements (Wolohan & Knox 2014). At that time, and

377  subsequently (Knox & Pasunuru 2020; Knox et al. 2018); see also the Introduction) we discussed

378  the advantages of the MDOR task compared to a number of alternatives. Essentially this is that

379  it provides a less contaminated measure of the ability of participants to inhibit a prepotent

380  response compared to those alternatives. Potentially this means that it might be a useful tool

381  for detecting and investigating the breakdown in inhibitory control reported to occur in a range

382  of conditions and pathologies (Kaiser et al. 2018; Manza et al. 2017; Rabi et al. 2020; Rochat et

383  al. 2013; Smith et al. 2014).

384

385  In the modified MDOR task, we increased spatial information available to participants and

386  decreased temporal predictability compared to the original version, and provided better

387  definition of trial start, end and the intertrial interval. One advantage flowing from these

388  changes appears to be an improved yield of valid trials for analysis. In our previous experiment

389  the mean yield per participant was 78% and 83% for young and old participants respectively

390  (Knox & Pasunuru 2020); here the comparable proportions were 97% and 93%. Participants

391  reported that the modified task provided them with more confidence as to how long to fixate

392  the target position towards the end of each trial; in the original version they were simply told to

393  pause at the eccentric position. They also had more confidence about when they could blink

394  because the intertrial interval was now clearly delineated. Much of our analysis focusses on

395  error trials as these provide information specifically about inhibition failure. However, errors

396   make up a minority of trials, and absolute numbers per participant can be small (particularly in

397   younger participants). So task design features that increase the information available for

398   analysis, without fundamentally altering task properties, are valuable.

399

400   The general pattern of responses observed with the modified MDOR task was identical to that

401   reported previously (Knox & Pasunuru 2020; Knox et al. 2018; Wolohan & Knox 2014). Latency

402   was related to TDD (longer latency at shorter TDDs), as was $ER_t$ (lower at shorter TDDs), and

403   latency was longer and error rates higher in older compared to younger participants. As

404   previously, most of the latency increase in the older group was due to general age-related

405   slowing in the oculomotor system, replicating our previous result (Knox & Pasunuru 2020).

406   Prosaccade latency was obtained from the task used to calibrate eye tracker output rather than

407   from a separate prosaccade task, in part to reduce the testing burden on participants. The

408   calibration task is clearly different to the MDOR task, but it provided us with an estimate of

409   prosaccade latency in a consistent manner for all participants. While a different prosaccade task

410   might have produced different absolute latencies, we would expect a similar latency difference

411   between old and young groups. The difference we did observe (28ms) was broadly consistent

412   with (although smaller than) the difference we reported previously (47ms), and consistent with

413   other reports in the literature (Eenshuistra et al. 2004; Klein et al. 2000). It provided us with a

414   means of "correcting" for general age-related slowing, a procedure recommended in the

415   literature (Verhaeghen 2011, although see also Ulrich et al. 1999). This is important because

416   general slowing accounts for some of the age-related alterations in performance observed

417   across tasks (Salthouse 1996), particularly those associated with executive function (Maldonado

418   et al. 2020). Notably after the latency "correction" a larger TDD-related modulation of latency

419   still remained in the older group, but now measured over three TDDs rather than two (compare

420   Fig 3b in Knox & Pasunuru 2020 with Fig 2B).

421

422   When the response to a target onset is successfully inhibited (so that there is no error

423   response), the result is a correct MDOR response. We have argued that the latency of these

424   correct MDOR responses reflects the level of inhibition around the time the target for the

425    saccade is extinguished (the go signal). At a TDD of 200ms, levels of inhibition at target offset

426    are high, leading to long latencies; as TDD is increased the level of inhibition (at the time the

427    correct saccade has to be initiated) falls, and so does latency. Our data suggest that older

428    participants are capable of exerting high levels of inhibition initially. As can be seen from Fig 2B,

429    at TDD=200ms the older group exhibited an additional latency increase, even once general age-

430    related slowing was accounted for; the effect size ($d_s$) for the difference between groups at

431    200ms in the corrected data was 0.83 and was statistically significant. At TDD=600ms this

432    additional latency was greatly decreased (and was not statistically significant), and it was

433    entirely absent at 1000ms.

434

435    The larger modulation in latency by TDD in the older group, a result we reported previously

436    (Knox & Pasunuru 2020), suggests that the maintenance of inhibition is more difficult for older

437    participants. The magnitude of the modulation is very similar in both studies; previously

438    comparing 200ms and 1000ms TDDs the latency difference was 164ms and 96ms in old and

439    young groups, here is was 168ms and 113ms. We might speculate that initially older

440    participants are able to generate high levels of inhibition, perhaps even higher levels than

441    young participants, as a strategy to compensate for poorer overall effectiveness of inhibition

442    (reflected in generally higher error rates); this would produce the longer latency for correct

443    responses at 200ms TDD. However, an inability to maintain inhibition as TTD increased, would

444    lead to latency dropping off, and a larger overall modulation.

445

446    Independent support for the general hypothesis that the MDOR task evokes inhibition, and that

447    this is reflected in latency increases, is provided by the results of Tari et al. (2019) and Tari &

448    Heath (2019). They used the MDOR task in a task switching paradigm and found that

449    prosaccades following an MDOR task had increased latency. As with task switching costs

450    observed with mixed AS and prosaccade tasks, they interpreted this as being due to the effects

451    MDOR-induced inhibition.

452

453    While the latency of correct responses may reflect levels of inhibition, error rates reveal its

454    effectiveness; these were consistently higher in the old compared to the young group.

455    However, the distributions (Fig 4) indicate that it is important to distinguish between different

456    types of error. We have previously discussed the advantages of average, compared to pooled,

457    distributions for prosaccade (Knox & Wolohan 2014; Knox et al. 2017), antisaccades (Knox et al.

458    2012) and MDOR tasks (Knox & Pasunuru 2020; Wolohan & Knox 2014). Here we have used

459    them as a means of graphically summarising responses across groups, in a form which allows

460    the identification of significant features. For errors, the clearest feature is the peak occurring

461    just after target onset, which we used to derive the peak error rate ($ER_{pk}$). We suggest that this

462    provides a direct index of inhibitory failure as given their timing, the errors which make up this

463    peak are most likely uninhibited responses to target onsets. Note that a similar claim might be

464    made for antisaccade errors (i.e. that AS errors are uninhibited prosaccades). However, as

465    outlined in the Introduction, this interpretation is complicated by there being two processes

466    competing for behavioural expression in the AS task. An AS error therefore is not necessary the

467    result of a failure of inhibition. In the MDOR task there is no such competition.

468

469    However, if early MDOR errors were only uninhibited responses to the target onset, then there

470    is no reason to expect $ER_{pk}$ to differ with TDD. It is striking that in both participant groups $ER_{pk}$ is

471    noticeably raised in the 1000ms condition (Fig 4C,F; see also Fig 5A) compared to the two other

472    TDDs where indeed it is similar. It seems unlikely that this is a chance occurrence, particularly as

473    it was observed in the distributions in our previous study in both young and old groups (Knox &

474    Pasunuru 2020), Fig 5B,D). This might suggest some other process influencing $ER_{pk}$ at least

475    when TDD is long. However, supporting the idea that these errors are responses to target

476    onsets (and are therefore for the most part inhibitory failures), the latency difference between

477    groups was similar to that observed between the prosaccade and MDOR correct response

478    latencies (Fig 6), and there was a (modest) correlation between the peak error and prosaccade

479    latency across participants (Fig 7).

480

481    The distributions also provide further information about the dynamics of inhibition and its

482    difference between groups. Errors continued (at a low rate) throughout the period when

483    central fixation was required, up to the target offset. But for the 1000ms TDD (Fig 4C,F), the

484    error rate rose slowly as the fixation period wore on, particularly from around -400ms (600ms

485    after the target onset). With TDD=600ms (Fig 4B,E) the error rate was flat in the young group

486    and falling in the old group. The residual error rate ($ER_r$; Fig 5B), differed for 600ms TDD but not

487    1000ms TDD, consistent with the young participants being able to maintain inhibition at a

488    higher level for longer than the older group. The absolute number of errors at these timings is

489    small, so these patterns should be interpreted cautiously. But perhaps they provide evidence

490    that the increased inhibition initially evoked in the MDOR task at target onset can only be

491    maintained for a few hundred milliseconds, and that the ability to maintain inhibition declines

492    as we age.

493

494    If essentially the same processes (or their failure) accounted for both the error rates and the

495    latency of correct responses, then we would expect to see a relationship between them. A

496    participant who was able to exert high levels of inhibitory control might have a low error rate

497    (particularly a low $ER_{pk}$) and a longer latency for correct responses. There was no clear evidence

498    of this type of relationship across conditions and groups (Fig 8). What the data in Fig 8 do

499    suggest is a difference between the shortest and longest TDDs, but with a similar pattern in

500    both groups. At TDD=1000ms for both $ER_t$ and $ER_{pk}$, the error rate was highly variable between

501    participants while  latency appeared to be less variable. The opposite pattern is seen for

502    TDD=200ms. Latency is longer and error rate higher, but the relationship between them is

503    altered; latency appears to be more variable and error rate less variable. As the same general

504    pattern was seen in both old and young groups, these data provide no evidence of a different

505    control strategy between groups.

506

507    Among the older participants, we had comparable groups of naïve and returning participants.

508    The experience of having visited the lab and completed the original MDOR task did not

509    influence performance in the modified MDOR task. While there was a period of several months

510    between tests on the two different versions of the task, there was little evidence of learning or

511    exposure effects. Procedures in the lab were of course designed to produce exactly this result

512    by carefully explaining and demonstrating tasks and providing practice trials before data

513    collection began with the aim of achieving stable performance in any one session of testing.

514    Such stability in performance over time does appear to be a general feature of at least simple

515    saccade tasks. In the absence of training (exposure to large numbers of trials over a relatively

516    short period of time), this has been taken to support the concept of an oculomotor phenotype

517    (Bargary et al. 2017; Knox & Wolohan 2015). Our study was not designed to investigate the

518    stability of MDOR performance over time; longitudinal testing will be needed to demonstrate

519    that MDOR performance provides a means of measuring trait inhibitory control as opposed to

520    providing a means of measuring the inhibitory state at the time of testing.

521

522    As demonstrated in Fig 10 there was little evidence of difference in latency and total error rate

523    either overall (Fig 10A,D) or in either the young (Fig 10B,E) or old (Fig 10C,F) groups when we

524    compared the original and modified MDOR tasks in the same participants. This suggests a

525    degree of stability across versions of the MDOR task and perhaps also a lack of sensitivity of the

526    measured parameters to task context. One concern with the original MDOR task might have

527    been that with only two TDDs (200ms and 1000ms), participants might have been able to

528    predict the time of target offset, generally supressing latency. The addition a the third TDD

529    would be expected to make this less likely. If such an effect were present, then with the

530    modified task a general increase in latency might be expected . However, there is no consistent

531    evidence that this is the case. Using task design to vary performance is, of course, occasionally

532    useful depending on the type of participants being tested. In the AS task, the use of gap AS

533    tasks tends to increase error rates, usefully preventing floor effects in healthy control groups in

534    clinical studies. We have shown previously that gap and overlap effects do not influence MDOR

535    performance (Knox et al. 2018). So too the modifications made in the current study had little

536    effect on performance.

537

538    As is well recognised, inhibitory control is not a monolithic construct (Aron 2011), several

539    taxonomies have been used to describe it (Rey-Mermet et al. 2018) and many tasks have been

540    used to investigate it. Correlations between tasks tend to be poor (Friedman & Miyake 2004;

541    Rey-Mermet et al. 2018) and even tasks that might be thought of as being relatively similar (eg

542    manual go/no go and SSRT; AS and MDOR task) may actually involve different mechanisms or

543    processes (Raud et al. 2020; Wolohan & Knox 2014). The existence and nature of an age-related

544    inhibitory control deficit has been a matter of recent debate (Rey-Mermet & Gade 2017;

545    Verhaeghen 2011). However, the MDOR task does appear to offer insights into the

546    effectiveness of inhibition and perhaps also its dynamics, at least with respect to oculomotor

547    control. Using the modified MDOR task in the current study, we have confirmed that the

548    effectiveness of inhibitory control declines in healthy ageing. We suggest that the MDOR task

549    (in either original or modified versions) provides a useful means of investigating multiple

550    aspects of inhibitory control.

551

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553

554    **References**

555    Alichniewicz KK, Brunner F, Klünemann HH, and Greenlee MW. 2013. Neural correlates of saccadic
556         inhibition in healthy elderly and patients with amnestic mild cognitive impairment. *Frontiers in*
557         *Psychology* 4. 10.3389/fpsyg.2013.00467
558    Aron AR. 2011. From reactive to proactive and selective control: Developing a richer model for stopping
559         inappropriate responses. *Biological Psychiatry* 69:e55-e68.
560         http://dx.doi.org/10.1016/j.biopsych.2010.07.024
561    Bargary G, Bosten JM, Goodbourn PT, Lawrance-Owen AJ, Hogg RE, and Mollon JD. 2017. Individual
562         differences in human eye movements: An oculomotor signature? *Vision Res* 141:157-169.
563         10.1016/j.visres.2017.03.001
564    Bari A, and Robbins TW. 2013. Inhibition and impulsivity: Behavioral and neural basis of response
565         control. *Progress in Neurobiology* 108:44-79.
566         http://dx.doi.org/10.1016/j.pneurobio.2013.06.005
567    Castiglione A, Wagner J, Anderson M, and Aron AR. 2019. Preventing a thought from coming to mind
568         elicits increased right frontal beta just as stopping action does. *Cerebral Cortex* 29:2160-2172.
569         10.1093/cercor/bhz017
570    Crawford TJ, Higham S, Renvoize T, Patel J, Dale M, Suriya A, and Tetley S. 2005. Inhibitory control of
571         saccadic eye movements and cognitive impairment in Alzheimer's disease. *Biological Psychiatry*
572         57:1052-1060. http://dx.doi.org/10.1016/j.biopsych.2005.01.017

573    Deubel H. 2008. The time course of presaccadic attention shifts. *Psychological Research* 72:630-640.
574         10.1007/s00426-008-0165-3
575    Eenshuistra RM, Ridderinkhof KR, and Molen MWvd. 2004. Age-related changes in antisaccade task
576         performance: Inhibitory control or working-memory engagement? *Brain and Cognition* 56:177-
577         188. https://doi.org/10.1016/j.bandc.2004.02.077
578    Friedman NP, and Miyake A. 2004. The relations among inhibition and interference control functions: A
579         latent-variable analysis. *Journal of Experimental Psychology: General* 133:101-135.
580         10.1037/0096-3445.133.1.101
581    Friedman NP, and Miyake A. 2017. Unity and diversity of executive functions: Individual differences as a
582         window on cognitive structure. *Cortex* 86:186-204.
583         https://doi.org/10.1016/j.cortex.2016.04.023
584    Hallett P. 1978. Primary and secondary saccades to goals defined by instructions. *Vision Research*
585         18:1279-1296.
586    Hanes DP, and Carpenter RHS. 1999. Countermanding saccades in humans. *Vision Research* 39:2777-
587         2791.
588    Hsieh S, Schubert S, Hoon C, Mioshi E, and Hodges JR. 2013. Validation of the Addenbrooke's Cognitive
589         Examination III in frontotemporal dementia and Alzheimer's disease. *Dementia and Geriatric
590         Cognitive Disorders* 36:242-250. 10.1159/000351671
591    Hutton S. 2008. Cognitive control of saccadic eye movements. *Brain and Cognition* 68:327–340.
592    Kaiser A, Kuhlmann BG, and Bosnjak M. 2018. A meta-analysis of inhibitory-control deficits in patients
593         diagnosed with Alzheimer's dementia. *Neuropsychology* 32:615-633. 10.1037/neu0000460
594    Klapetek A, Jonikaitis D, and Deubel H. 2016. Attention allocation before antisaccades. *J Vis* 16:11.
595         10.1167/16.1.11
596    Klein C, Fischer B, Hartnegg K, Heiss W, and M. R. 2000. Optomotor and neuropsychological
597         performance in old age. *Exp Brain Res* 135:141-154.
598    Knox PC, and Pasunuru N. 2020. Age-related alterations in inhibitory control investigated using the
599         minimally delayed oculomotor response task. *PeerJ* 8:e8401.
600         https://doi.org/10.7717/peerj.8401
601    Knox PC, Amatya N, Jiang X, and Gong Q. 2012. Performance deficits in a voluntary saccade task in
602         Chinese "express saccade makers". *PLOS One* 7:e47688. 10.1371/journal.pone.0047688
603    Knox PC, Heming De-Allie E, and Wolohan FDA. 2018. Probing oculomotor inhibition with the minimally
604         delayed oculomotor response task. *Experimental Brain Research* 236:2867-2876.
605         10.1007/s00221-018-5345-9
606    Knox PC, and Wolohan FDA. 2014. Cultural diversity and saccade similarities: Culture does not explain
607         saccade latency differences between Chinese and Caucasian participants. *PLOS One* 9:e94424.
608         10.1371/journal.pone.0094424
609    Knox PC, and Wolohan FDA. 2015. Temporal stability and the effects of training on saccade latency in
610         "express saccade makers". *PLOS One* 10:e0120437. 10.1371/journal.pone.0120437
611    Knox PC, Wolohan FDA, and Helmy MS. 2017. Express saccades in distinct populations: east, west, and
612         in-between. *Experimental Brain Research* 235:3733-3742. 10.1007/s00221-017-5094-1
613    Kowler E, Anderson E, Doshler B, and Blaser E. 1995. The role of attention in the programming of
614         saccades. *Vision Res* 35:1987-1961.
615    Lakens D. 2013. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer
616         for t-tests and ANOVAs. *Frontiers in Psychology* 4. 10.3389/fpsyg.2013.00863
617    Logan GD, and Cowan WB. 1984. On the ability to inhibit thought and action - a theory of an act of
618         control. *Psychological Review* 91:295-327. 10.1037/0033-295x.91.3.295
619    Logan GD, Schachar RJ, and Tannock R. 1997. Impulsivity and Inhibitory Control. *Psychological Science*
620         8:60-64. 10.1111/j.1467-9280.1997.tb00545.x

621 Maldonado T, Orr JM, Goen JRM, and Bernard JA. 2020. Age differences in the subcomponents of
622     executive functioning. *J Gerontol B Psychol Sci Soc Sci* 75:e31-e55. 10.1093/geronb/gbaa005
623 Manza P, Amandola M, Tatineni V, Li C-sR, and Leung H-C. 2017. Response inhibition in Parkinson's
624     disease: a meta-analysis of dopaminergic medication and disease duration effects. *npj*
625     *Parkinson's Disease* 3:23. 10.1038/s41531-017-0024-2
626 Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, and Wager TD. 2000. The unity and
627     diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent
628     variable analysis. *Cognitive Psychology* 41:49-100. http://dx.doi.org/10.1006/cogp.1999.0734
629 Munoz DP, and Everling S. 2004. Look away: the anti-saccade task and the voluntary control of eye
630     movement. *Nat Rev Neurosci* 5:218-228.
631 Noone P. 2015. Addenbrooke's Cognitive Examination-III. *Occup Med (Lond)* 65:418-420.
632     10.1093/occmed/kqv041
633 Rabi R, Vasquez BP, Alain C, Hasher L, Belleville S, and Anderson ND. 2020. Inhibitory control deficits in
634     individuals with amnestic mild cognitive impairment: a meta-analysis. *Neuropsychology Review*
635     30:97-125. 10.1007/s11065-020-09428-6
636 Raud L, Westerhausen R, Dooley N, and Huster RJ. 2020. Differences in unity: The go/no-go and stop
637     signal tasks rely on different mechanisms. *NeuroImage* 210:116582.
638     10.1016/j.neuroimage.2020.116582
639 Reuter B, Jäger M, Bottlender R, and Kathmann N. 2007. Impaired action control in schizophrenia: The
640     role of volitional saccade initiation. *Neuropsychologia* 45:1840-1848.
641     http://dx.doi.org/10.1016/j.neuropsychologia.2006.12.006
642 Reuter B, Rakusan L, and Kathmanna N. 2005. Poor antisaccade performance in schizophrenia: An
643     inhibition deficit? *Psychiatry Research* 135:1-10.
644     http://dx.doi.org/10.1016/j.psychres.2004.12.006
645 Rey-Mermet A, and Gade M. 2017. Inhibition in aging: What is preserved? What declines? A meta-
646     analysis. *Psychonomic Bulletin & Review*. 10.3758/s13423-017-1384-7
647 Rey-Mermet A, Gade M, and Oberauer K. 2018. Should we stop thinking about inhibition? Searching for
648     individual and age differences in inhibition ability. *Journal of Experimental Psychology: Learning,*
649     *Memory, and Cognition* 44:501-526. 10.1037/xlm0000450
650 Rochat L, Billieux J, Juillerat Van der Linden A-C, Annoni J-M, Zekry D, Gold G, and Van der Linden M.
651     2013. A multidimensional approach to impulsivity changes in mild Alzheimer's disease and
652     control participants: Cognitive correlates. *Cortex* 49:90-100.
653     http://dx.doi.org/10.1016/j.cortex.2011.08.004
654 Salthouse TA. 1996. The processing-speed theory of adult age differences in cognition. *Psychol Rev*
655     103:403-428. 10.1037/0033-295x.103.3.403
656 Smith JL, Mattick RP, Jamadar SD, and Iredale JM. 2014. Deficits in behavioural inhibition in substance
657     abuse and addiction: A meta-analysis. *Drug and Alcohol Dependence* 145:1-33.
658     http://dx.doi.org/10.1016/j.drugalcdep.2014.08.009
659 Tari B, Fadel MA, and Heath M. 2019. Response suppression produces a switch-cost for spatially
660     compatible saccades. *Experimental Brain Research*. 10.1007/s00221-019-05497-z
661 Tari B, and Heath M. 2019. Pro- and antisaccade task-switching: response suppression—and not vector
662     inversion—contributes to a task-set inertia. *Experimental Brain Research*. 10.1007/s00221-019-
663     05686-w
664 Ulrich R, Mattes S, and Miller J. 1999. Donders's assumption of pure insertion: an evaluation on the basis
665     of response dynamics. *Acta Psychologica* 102:43-76. https://doi.org/10.1016/S0001-
666     6918(99)00019-0
667 Verhaeghen P. 2011. Aging and executive control: Reports of a demise greatly exaggerated. *Current*
668     *Directions in Psychological Science* 20:174-180. 10.1177/0963721411408772

669    Wolohan FDA, and Knox PC. 2014. Oculomotor inhibitory control in express saccade makers.
670        *Experimental Brain Research* 232:3949–3963. 10.1007/s00221-014-4076-9

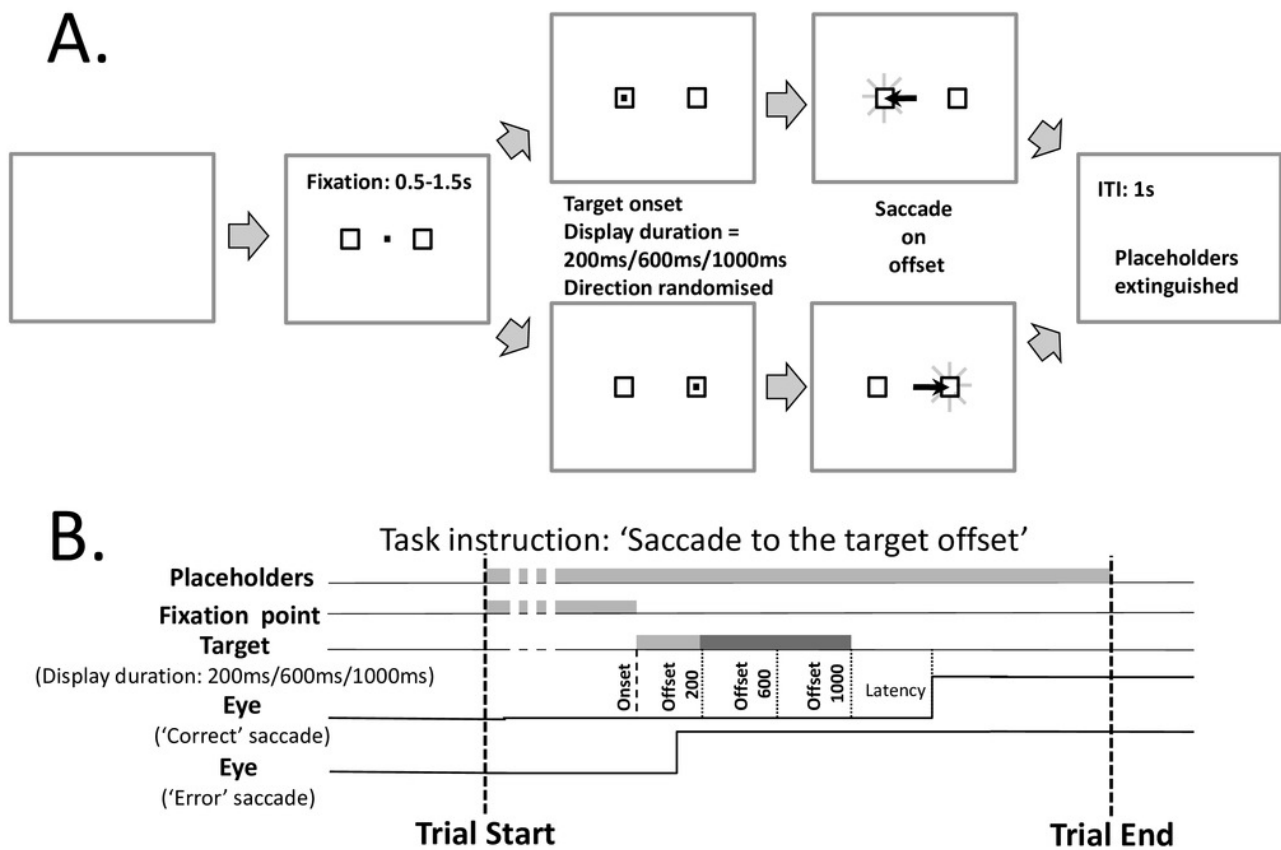
671

# Figure 1

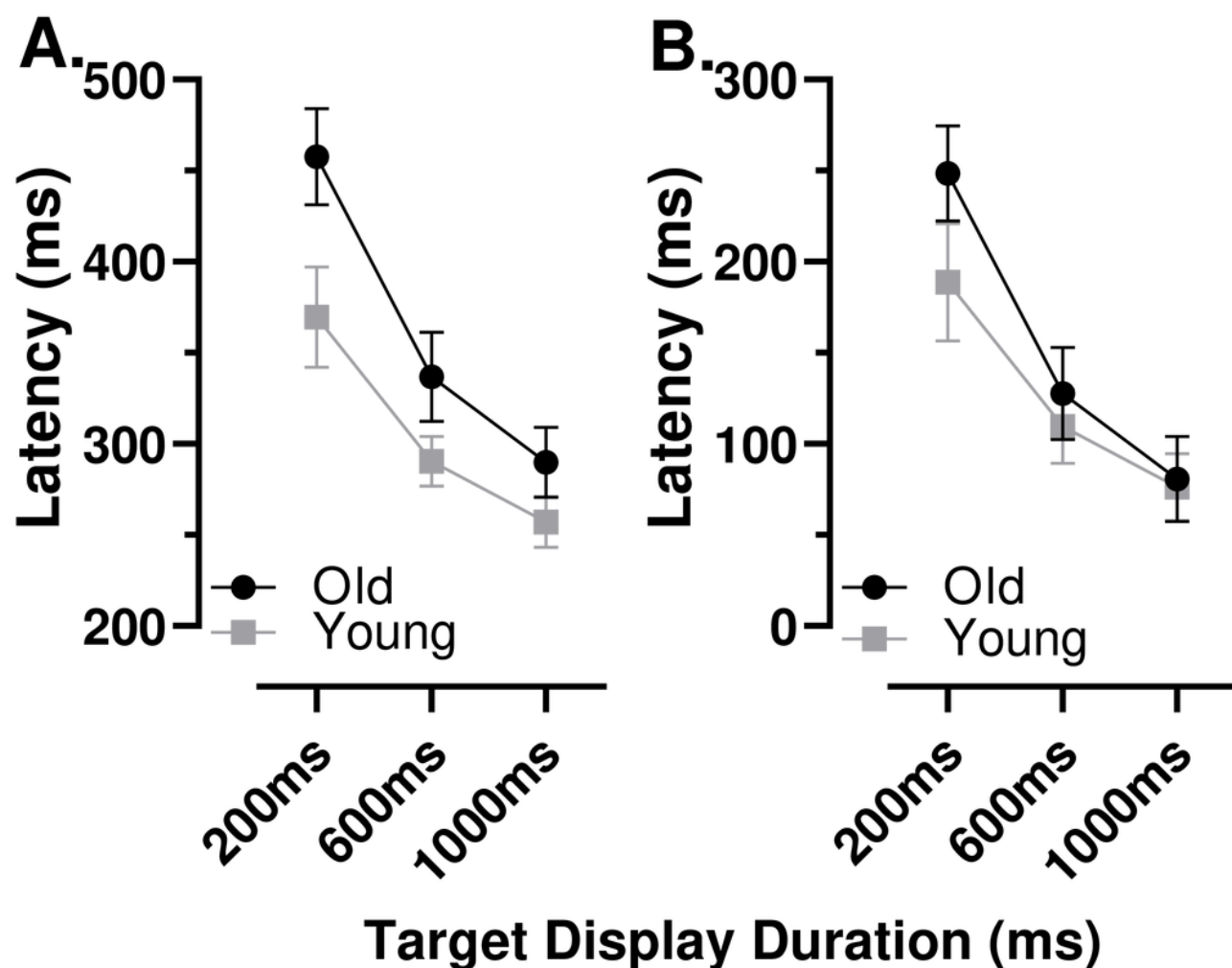
Schematic description of the modified MDOR task used in this study.

(A) Each trial began with the appearance of the central fixation target, and two 0.8° x 0.8° place holders, centred 5° to the left and right. After a random period of 0.5-1.5s the fixation target was extinguished and the target for the saccade appeared randomly at the centre of either the left or right placeholder, and was displayed for 200ms, 600ms or 1000ms (randomised). The extinction of the target was the signal for the participant to saccade to the centre of the placeholder in which the target had appeared, and maintain fixation there. After 1s the placeholders were extinguished, at which point the participant was instructed to return fixation to the centre of the display and await the beginning of the next trial. (B) The relative timing of the various events in each trial. Note that the eye trace illustrating a correct response assumes a target display duration of 1000ms.

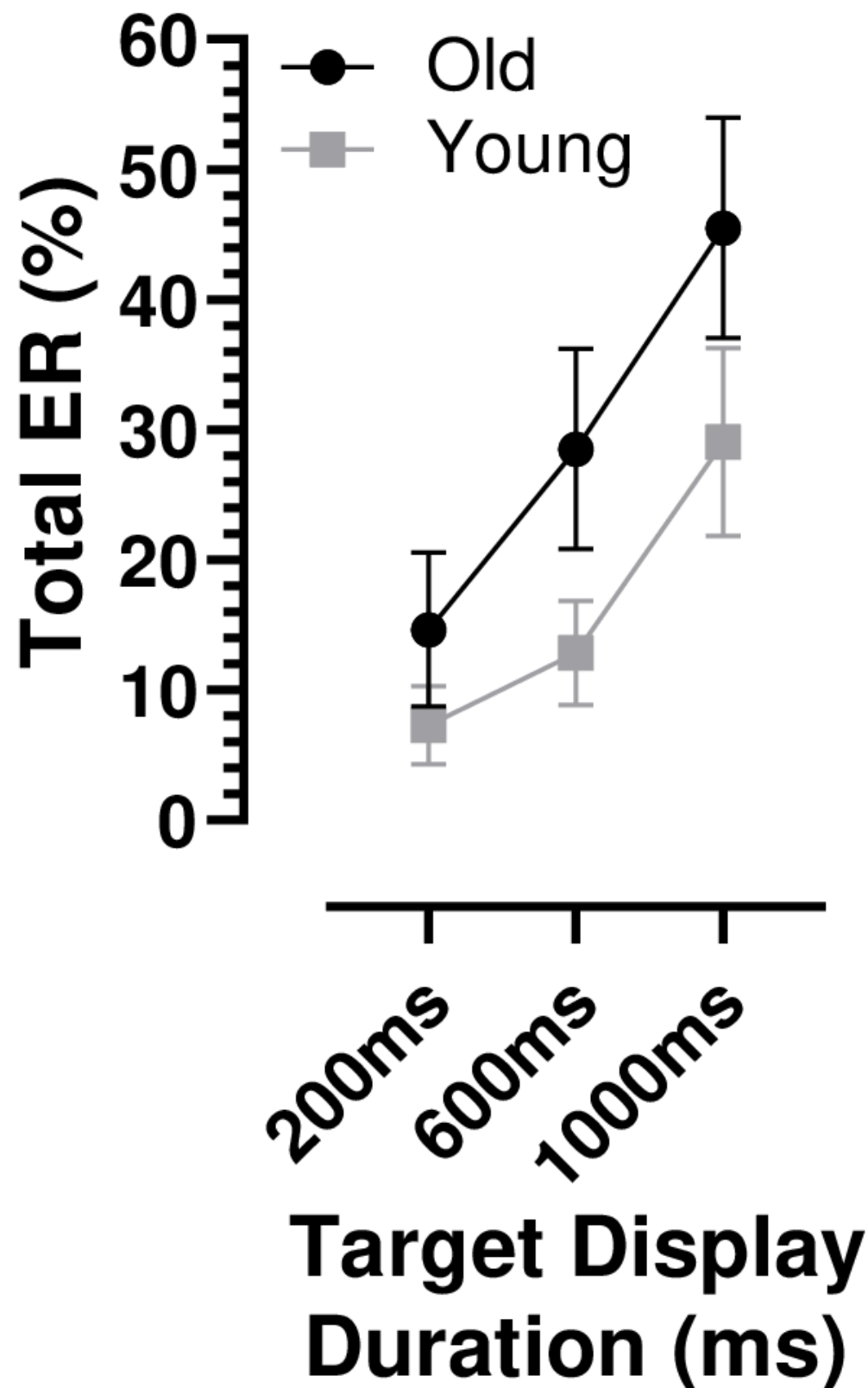
## A.

## B.

# Figure 2

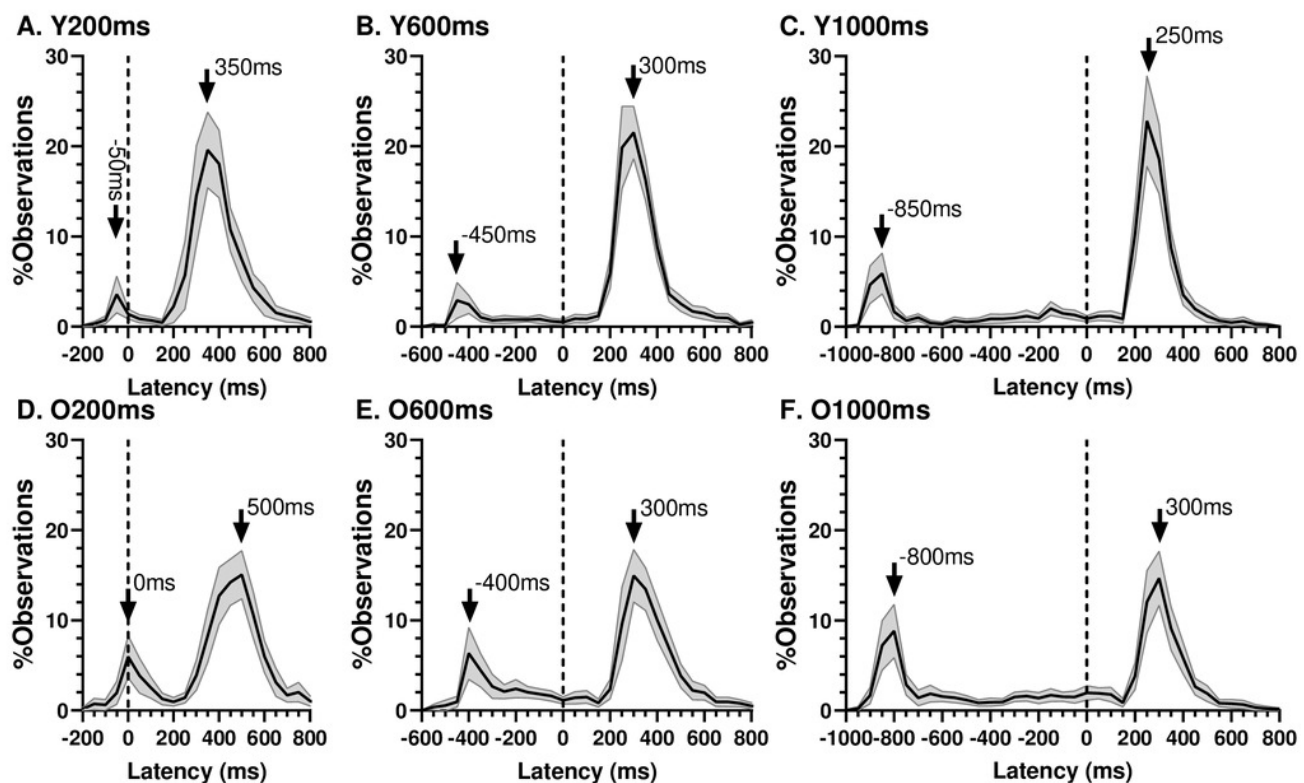
Latency of correct MDOR responses.

Mean (±95% CI) latency is plotted for each target display duration for old and young groups. (A) Uncorrected latency. (B) Corrected latency; each participants calibration task latency was subtracted from their MDOR latency, and group mean latency calculated.

# Figure 3

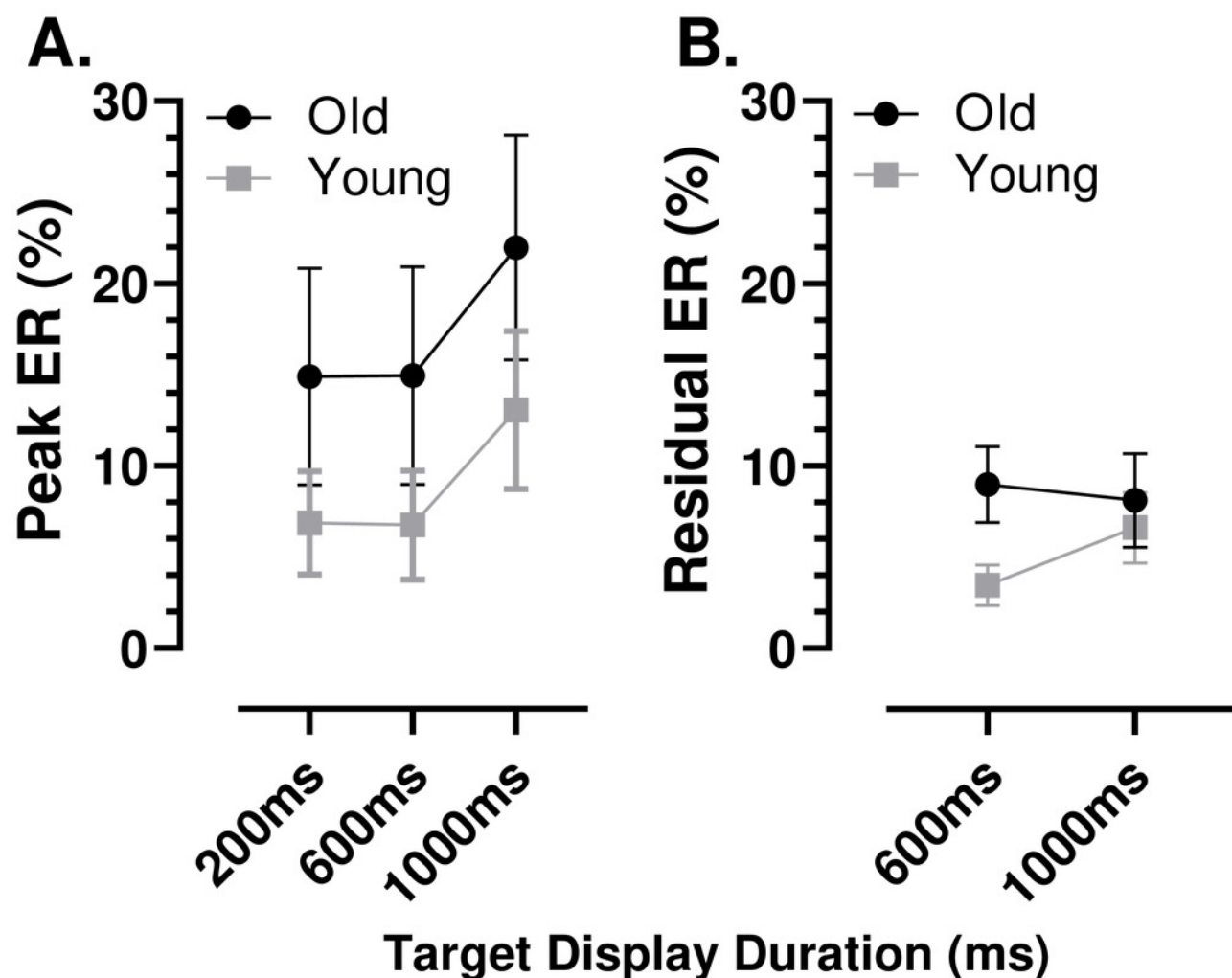
Total error rate.

Mean (±95% CI) total error rate (%) is plotted for each target display duration for old and young groups.

# Figure 4

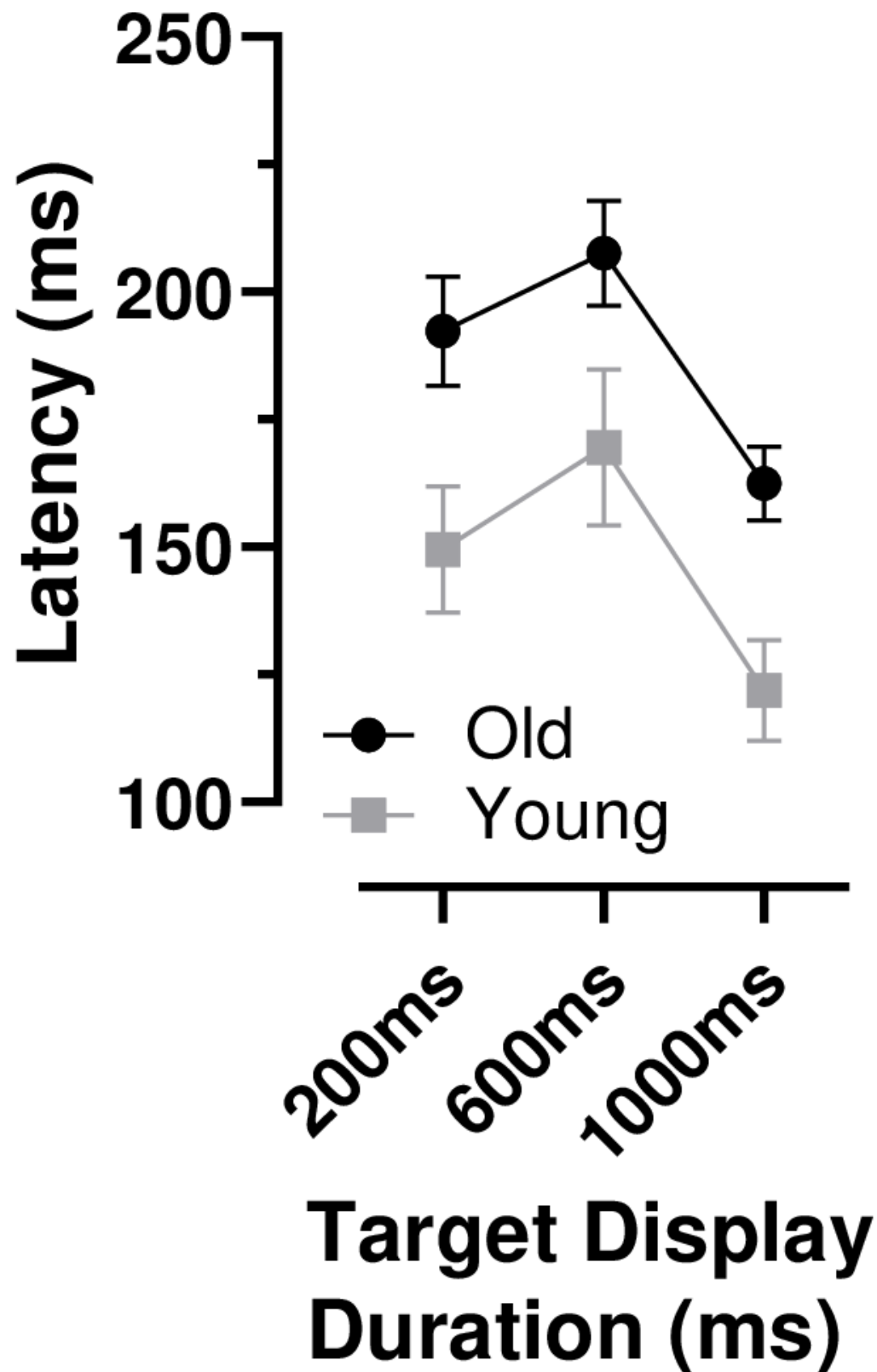
Average distribution plots for young (A,B,C) and old (D,E,F) participant groups.

Bin width=50ms. For each participant, the % frequency distribution for all responses was calculated. The mean (±95% CI) was then calculated for each bin across participants in each group. The black central line is plotted through the each mean bin value, while the grey range shows ±95% CI. The data were not smoothed. Target onset is at -200 for 200ms TDD (A,D), -600 for 600ms TDD (B,E) and -1000ms for 1000ms TDD (C,F); target offset (the go signal) is at 0ms (vertical dotted line). Arrows mark peak bins in each distribution for both errors and correct responses, and the timing of that bin is also shown.

# Figure 5

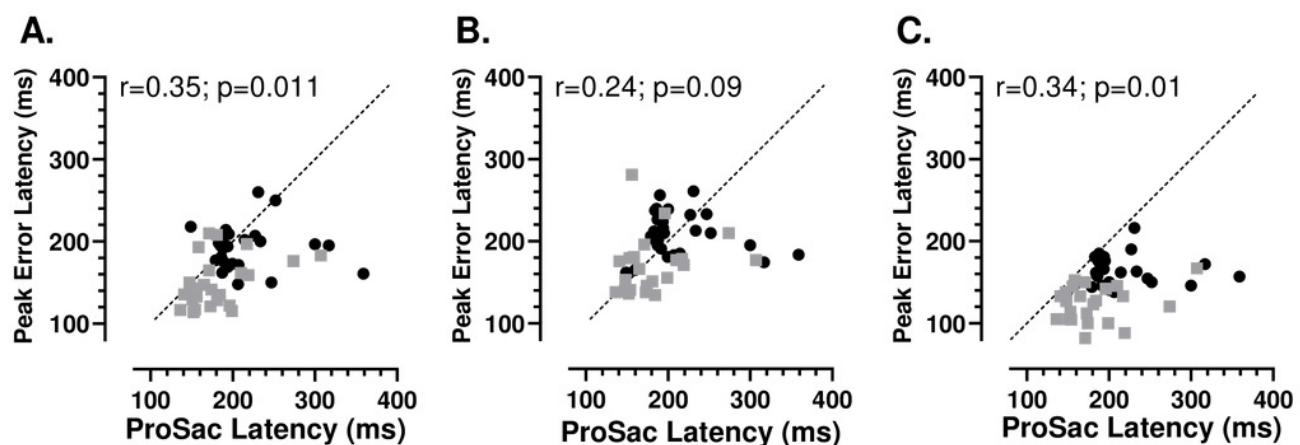
Peak and residual error rates

Mean (±95% CI ) MDOR peak error (A) and residual error rates (B) in old and young participant groups. The derivation of these error rates is described in the text.

# Figure 6

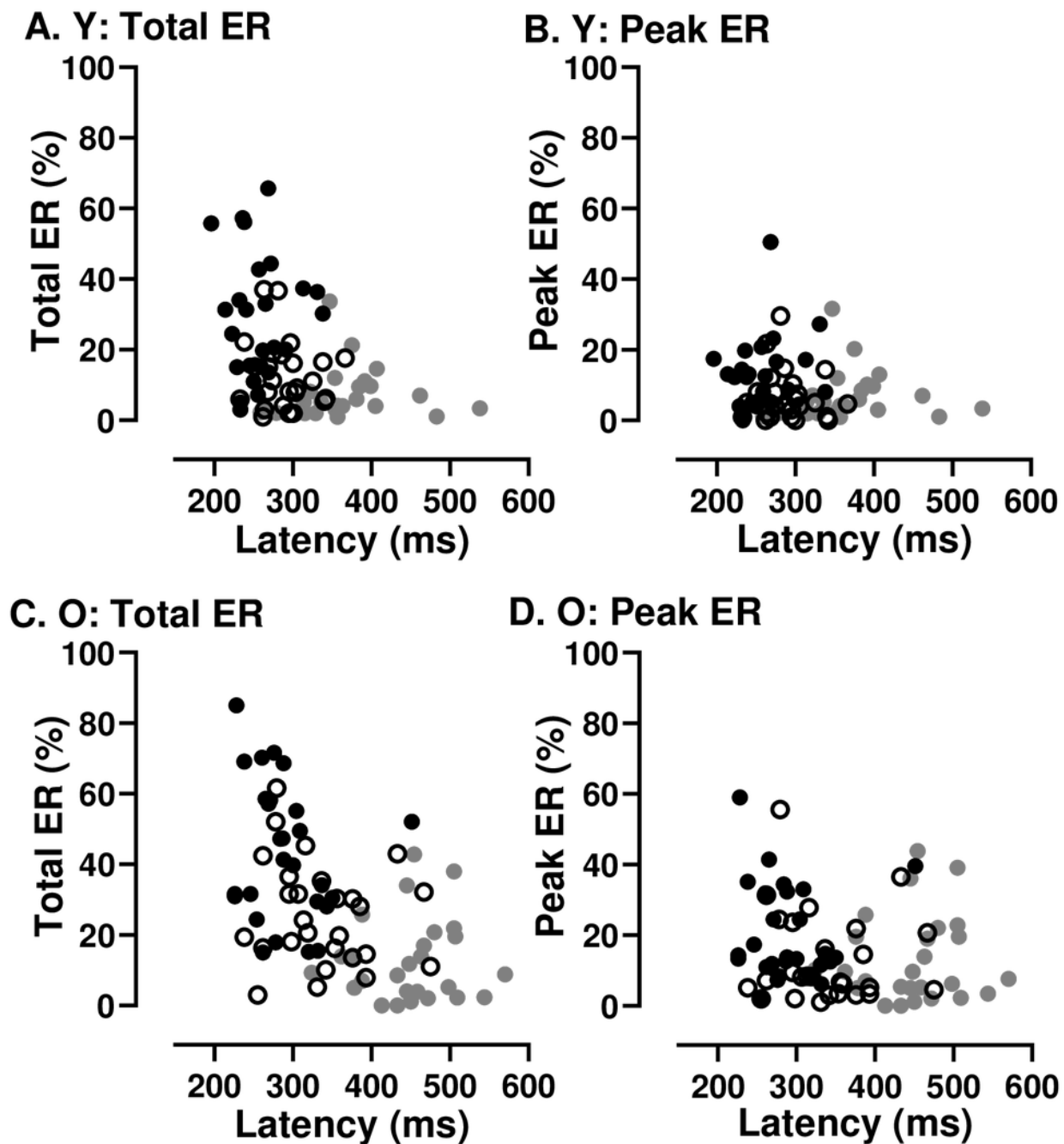
Latency of "peak" errors.

Mean (±95% CI ) latency of peak errors for old and young participant groups. Details of how these were calculated are in the text.

# Figure 7

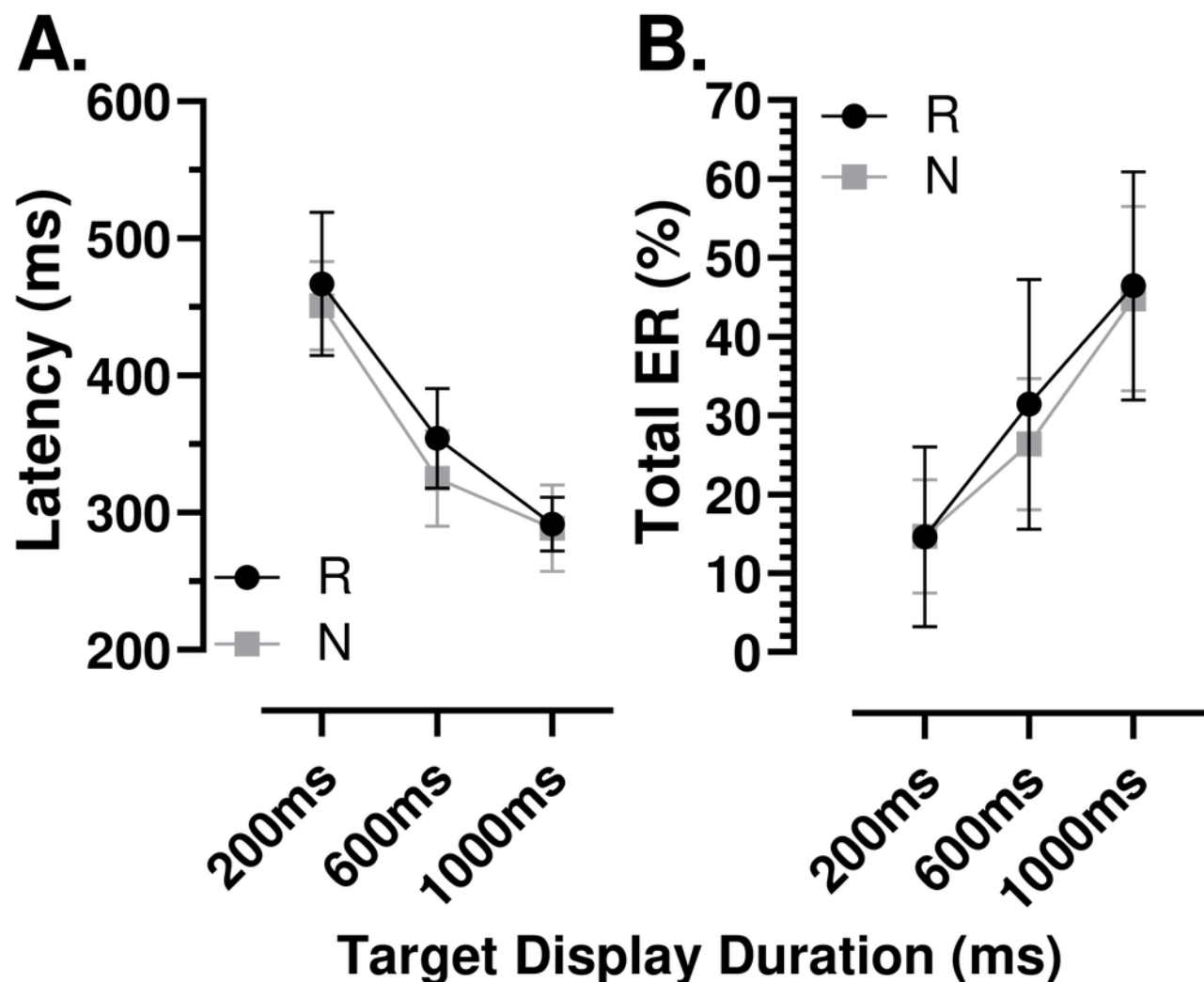
Correlation between MDOR peak error and prosaccade latencies.

Correlation between MDOR error saccade latency for saccades occurring in the peak error range and median pro-saccade latency calculated from the calibration task. (A) TDD 200ms; (B) TDD 600ms; (C) TDD 1000ms. On each plot the Pearson correlation coefficient is shown (with accompanying p value), calculated for the whole dataset (ie old and young data combined), although on each plot the groups can be distinguished (old: black circles; young: grey squares). Dashed line shows the line of equality (x=y); points falling below this line indicate that the pro-saccade latency > MDOR error latency.

# Figure 8

Relationship between error rates and latency of correct responses.

Plots of Error Rate (Total ER, $ER_t$ : A,C; Peak ER, $ER_{pk}$ : B,D) against latency for each participant in both young (A,B) and old (C,D) groups. In each plot, data from the three TDDs are distinguished: 200ms gray symbols ; 600ms unfilled symbols; 1000ms black symbols.

# Figure 9

Comparison in MDOR performance between naïve and experienced participants.

Comparison of performance in the modified MDOR task (MDOR-M) between those old participants who were naïve to testing (N) and those who had participated in a previous MDOR experiment with the original MDOR task (MDOR-O), and who had returned for testing (R). (A) Mean (±95% CI ) raw latency for correct responses. (B) Total error rate.

# Figure 10

Comparison in perfomance between the original and modified MDOR tasks.

Comparison of performance in the original MDOR task (MDOR-O) and the modified task described in the current study (MDOR-M), Mean (±95% CI ) latency (A-C) and total error rate (D-E) is shown for all participants for whom data were available (A and D; n=24) and then divided into young (B and E; n=9) and old (C and F; n=15) participants.