

Motor Control in Old Age: Evidence of Impaired Low-Level Inhibition

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In the masked prime task, responses to supraliminal targets are influenced by previously presented subliminal primes. When targets follow primes immediately, positive compatibility effects are obtained such that performance is better when prime and target are compatible (mapped to the same response) than when they are incompatible (mapped to opposite responses). In young adults, this pattern reverses with longer interstimulus intervals (negative compatibility effect). These effects reflect an activation-followed-by-inhibition process: Primes trigger an initial activation of their corresponding motor response, which is subsequently inhibited. The present study demonstrates that healthy older adults ($M = 76$ years) show a substantial positive compatibility effect with a short prime-target interval, but they fail to produce reliable negative compatibility effects with longer intervals, indicating an age-related impairment in low-level motor control.

SUCCESSFULLY masked visual prime stimuli can affect motor responses to subsequently presented supraliminal targets (e.g., Dehaene et al., 1999; Eimer & Schlaghecken, 1998; Leuthold & Kopp, 1998). The direction of these priming effects depends on the prime-target interstimulus interval (ISI): With a 0-ms ISI, positive compatibility effects (PCEs) occur, with performance benefits on compatible trials (prime and target mapped to the same response) and costs on incompatible trials (prime and target mapped to different responses), relative to neutral trials (prime not mapped to any response). With longer ISIs (about 100 ms), PCEs turn into negative compatibility effects (NCEs), with performance benefits on incompatible trials, and costs on compatible trials (Eimer, 1999; Schlaghecken & Eimer, 2000).

Converging evidence indicates that these effects originate within the motor system rather than at perceptual or cognitive-semantic levels (Dehaene et al., 1999; Eimer, 1999; Eimer & Schlaghecken, 1998; Eimer, Schubö, & Schlaghecken, 2002; Klapp & Hinkley, 2002; Leuthold & Kopp, 1998; Schlaghecken & Eimer, 2000). Initially, the prime activates its corresponding motor response, which in turn inhibits its competitor. Unsupported by further sensory evidence (because primes are successfully masked), the initial prime-induced activation then self-inhibits, thereby disinhibiting the competing response (see Schlaghecken & Eimer, 2002). With short prime-target ISIs, target-response preparation starts during the initial activation phase, reflected in PCEs. With longer ISIs, target-response preparation starts during the subsequent self-inhibition phase, reflected in NCEs. The fact that subliminal stimuli trigger these effects (Dehaene et al.; Eimer & Schlaghecken, 1998, 2002) suggests that they are generated at low-level stages of the visuomotor process, possibly mediated by subcortical control circuits (Aron et al., 2003; Schlaghecken, Münchau, Bloem, Rothwell, & Eimer, 2003).

The masked prime paradigm is of particular interest for the study of normal human aging in view of the influential proposal by Hasher and Zacks (1988; Hasher, Zacks, & May, 1999) that age-related cognitive deficits reflect diminished inhibitory control. In support of this hypothesis, several studies have

reported an age-related decline in the ability to voluntarily inhibit a prepotent motor response (Kramer, Humphrey, Larish, Logan, & Strayer, 1994; Nieuwenhuis, Ridderinkhof, de Jong, Kok, & van der Molen, 2000). However, the question of whether low-level, nonvoluntary motor inhibition is also impaired in old age has yet to be addressed. At least in other domains, it appears that whereas aging impairs high-level inhibitory processes, low-level processes may be preserved (e.g., Hartley & Kieley, 1995; Maylor & Henson, 2000). Nevertheless, recent evidence indicates an age-related impairment of low-level motor processes (Hartley, 2001), suggesting that, in the motor domain, low-level inhibitory control might not be spared.

In the present study we addressed these issues by comparing the performance of young and older participants in the standard masked prime task with stimulus-presentation conditions adjusted to account for possible impaired visual acuity and slower responses in older participants (conditions were similar to those used successfully with patients with Huntington Disease and age-matched controls by Aron et al., 2003). We manipulated the prime-target ISI, and we expected young participants to produce PCEs with a 0-ms ISI, and NCEs with a 150-ms ISI, replicating previous results (e.g., Eimer, 1999; Schlaghecken & Eimer, 2002). We used two longer ISIs (300 ms and 450 ms) to further test for a potential slowing of low-level activation and inhibition processes in older adults. These have not yet been investigated in young participants. Consequently, no specific predictions about priming effects at these ISIs can be made (although one might reasonably assume that any effects will disappear with sufficiently long ISIs).

The initial question was whether older participants would show any evidence of masked priming effects. It seems conceivable that old age compromises the subtle fine-tuning of behavioral control in general. In this case, briefly presented, subsequently masked primes should not trigger any motor processes in older adults. Alternatively, early activation and/or self-inhibition processes might be reduced or delayed, or self-inhibition might be selectively impaired, in older adults compared with young adults. In this case, older participants

Table 1. Mean Reaction Times for Compatible and Incompatible Trials and Mean Differences as a Function of Interstimulus Interval for Young and Older Adults for Session 1 and Session 2

	Young Adults			Older Adults		
	Compatible (SD)	Incompatible (SD)	Difference (95% CI)	Compatible (SD)	Incompatible (SD)	Difference (95% CI)
Session 1						
ISI = 0 ms	368 (26)	407 (23)	39** (22–56)	496 (29)	553 (36)	57** (39–74)
ISI = 150 ms	396 (33)	371 (49)	–25* (–42–8)	516 (37)	532 (35)	16 (–16–47)
Session 2						
ISI = 300 ms	353 (31)	361 (29)	8 [†] (–1–16)	499 (39)	489 (36)	–10 [†] (–20–0)
ISI = 450 ms	374 (55)	369 (53)	–5 (–13–2)	493 (37)	487 (34)	–6 (–15–3)

Notes: ISI = interstimulus interval; CI = confidence interval.

** $p < 0.01$; * $p < 0.02$; [†] $p < 0.1$.

should show priming effects, but these should differ in time course and/or polarity from those observed in young adults.

METHODS

Participants

Eight young participants (7 women and 1 man), aged 18 to 37 years ($M = 22.3$, $SD = 7.2$), were unpaid volunteers from the subject panel of the Psychology Department at Warwick University. Eight volunteers (5 women and 3 men), aged 71 to 83 years ($M = 75.6$, $SD = 3.7$), were recruited from the Department's Aging Study subject pool and were paid £5 (approximately \$7) to cover travel expenses. In both groups, all participants, but one, were right handed and had normal or corrected-to-normal vision according to self-report.

Stimuli and Apparatus

Primes and targets were left- and right-pointing double arrows (\ll and \gg), subtending a visual angle of $2.3^\circ \times 0.9^\circ$ at a viewing distance of approximately 1 m. Masks were rectangular 6×5 grids ($2.6^\circ \times 1.4^\circ$), randomly filled with overlapping lines of different lengths (0.2° to 1.0° ; width 0.2°) and orientations. A new mask was constructed on each trial. Stimuli were presented in black on a white 17-in. (43-cm) computer screen.

Procedure

Participants sat in a comfortable chair in a dimly lit, sound attenuated chamber. Response buttons, mounted on the armrests, were adjusted so that they were under the participants' left and right index fingers.

Trials started with a fixation dot ($0.3^\circ \times 0.3^\circ$ visual angle), presented for 250 ms at the center of the screen. Seven hundred and fifty ms after its offset, a prime was presented for 33 ms at fixation, replaced immediately by a 100-ms mask. Targets (presented for 100 ms) appeared randomly and with equal probability 3.3° above or below the center (just outside the area occupied by the mask). The four prime-target ISIs were blocked. In ISI-0 blocks, targets appeared simultaneously with the mask; they appeared 50 ms, 200 ms, and 350 ms after mask offset in ISI-150, ISI-300, and ISI-450 blocks, respectively. Left- and right-pointing arrows, and compatible and incompatible trials (prime and target mapped to the same or opposite response, respectively), were presented randomly and with

equal probability. The interval between target offset and the next fixation dot was 2,200 ms. Participants were instructed to maintain central eye fixation and to respond as quickly and as accurately as possible with a left or right key press to left- or right-pointing target arrows.

The experiment consisted of two sessions (Session 1, ISI-0 and ISI-150 blocks; Session 2, ISI-300 and ISI-450 blocks), at an interval of at least 1 week. There were six blocks per session (three for each ISI), containing 72 trials each. Half of the participants in each age group performed the three ISI-0 (ISI-300) blocks first; the other half performed the three ISI-150 (ISI-450) blocks first. A short practice block (24 trials) was run prior to each series of blocks. Sessions lasted approximately 30 min, including breaks.

Data Analysis

Because we predicted PCEs and NCEs for Session 1 (for young participants), whereas we made no predictions for Session 2, we analyzed data from each session separately. We did not analyze responses executed before target onset, or after onset of the next fixation dot (1.1% of all trials on average). For each experimental condition (ISI \times Compatibility), we excluded from analysis those trials in which reaction times (RTs) were more than 2 SD above or below the participant's mean RT for that condition (4.5% of the remaining trials). Error rates were extremely low—about 1.4% on average—and we did not analyze them further. We determined mean RTs on the remaining correct-response trials for each condition separately. We conducted repeated measures analyses of variance (ANOVAs) with the three variables of group (young, older), ISI (Session 1, 0 ms, 150 ms; Session 2, 300 ms, 450 ms), and compatibility (compatible, incompatible). We estimated effect sizes by using partial eta squared (η^2). Results not subsequently reported were not statistically significant ($p > .05$).

RESULTS

Session 1

Unsurprisingly, in Session 1 (see Table 1), young participants responded faster than older participants, $F(1, 14) = 88.62$, $MSE = 3478.45$, $p < .001$, $\eta^2 = .864$. A significant effect of compatibility, $F(1, 14) = 17.85$, $MSE = 421.14$, $p < .002$, $\eta^2 = .560$, was accompanied by a significant ISI \times Compatibility interaction, $F(1, 14) = 44.32$, $MSE = 251.82$, $p < .001$, $\eta^2 = .760$,

as compatibility effects were large (positive) in ISI-0 blocks and small in ISI-150 blocks. This overall compatibility effect was significantly more positive for older than for young adults (Group \times Compatibility interaction: $F(1, 14) = 8.04$, $MSE = 421.14$, $p < .02$, $\eta^2 = .365$).

We conducted additional ANOVAs for each ISI condition separately. In ISI-0 blocks, we observed no age difference in compatibility effects, and subsequent paired t tests confirmed that both groups produced significant PCEs: young, $t(7) = 5.46$, $p < .002$; older, $t(7) = 7.69$, $p < .001$. In contrast, compatibility effects were significantly different between age groups in ISI-150 blocks, $F(1, 14) = 7.24$, $MSE = 459.67$, $p < .02$, $\eta^2 = .341$. Young participants showed reliable NCEs, $t(7) = 3.47$, $p < .02$, whereas older participants tended to produce (nonsignificant) PCEs in this condition, $t(7) = 1.18$, $p > .27$.

Session 2

Again, as shown in Table 1, young participants were faster than older participants, $F(1, 14) = 45.36$, $MSE = 5769.18$, $p < .001$, $\eta^2 = .764$. The only other significant results were the interaction of Group \times Compatibility, $F(1, 14) = 5.85$, $MSE = 58.23$, $p < .04$, $\eta^2 = .295$, and the three-way interaction of Group \times ISI \times Compatibility, $F(1, 14) = 5.36$, $MSE = 51.61$, $p < .04$, $\eta^2 = .277$. In ISI-300 blocks, young participants produced numerical PCEs and older participants produced numerical NCEs, both approaching significance (both $t_s > 2.20$, both $p_s < .064$). We observed no reliable priming effects in the ISI-450 condition (both $t_s < 1.72$, $p_s > .13$).

Conclusions

In a masked priming task, both young and older participants produced substantial PCEs with a prime-target ISI of 0 ms, but only young participants produced NCEs with a 150-ms ISI. With a 300-ms ISI, older participants showed a tendency toward NCEs, whereas with a 450-ms ISI, neither group showed statistically reliable priming effects.

Two main conclusions can be drawn from these results. First, the fact that older participants showed substantial PCEs in response to briefly presented, subsequently masked stimuli in ISI-0 blocks confirms that the masked prime paradigm can be used successfully to assess low-level motor control processes in older adults. Second, the failure of older participants to produce NCEs with a 150-ms ISI provides strong evidence for impaired self-inhibition in old age. The absence of a reliable NCE at 300 ms for older participants further seems to suggest that this is not due to an overall delay in self-inhibition with increasing age. (Although the sample size was small, the NCE for young participants at 150 ms indicated that power was sufficient at .84 to detect a similar, but delayed, effect at 300 ms in older participants.) It is noteworthy, however, that several older participants did show NCEs at the 300-ms ISI, suggesting a delay rather than a complete breakdown of self-inhibition for at least some of the older participants.

These results confirm and extend results reported in a study by Seiss and Praamstra (2004), which provided initial evidence of impaired self-inhibition in older adults. However, these authors did not use an age-adjusted design (primes were small and presented noncentrally, and the shortest possible prime-target ISI for obtaining NCEs of 100 ms was used), so that an

interpretation of age-related NCE differences in terms of impaired visual acuity or generalized slowing in old age could not be ruled out. In contrast, in the present study we aimed to minimize the potential impact of such confounding factors. The fact that we still obtained age-related priming differences under these conditions thus provides strong evidence that these effects reflect differences within the low-level inhibitory motor control system.

The present findings therefore extend the reduced inhibition hypothesis of Hasher and Zacks (1988) of aging to low-level motor inhibition processes. Interestingly, such processes are not thought to involve the frontal cortex but are assumed to be mediated by basal ganglia–thalamic networks (Aron et al., 2003; Schlaghecken et al., 2003). It has been suggested that age-related inhibition deficits will only be observed to the extent that performance depends on the frontal lobes (e.g., Kramer et al., 1994). According to this view, low-level motor inhibition would not be expected to decline in old age. However, there is also a substantial loss of neural tissue with old age in regions connected to the frontal lobes, namely, the thalamus and the basal ganglia (Raz, 2000). Our study therefore suggests that the masked prime paradigm may provide a promising avenue for further investigations of aging and inhibition within subcortical control structures.

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