Psychology and Aging
2011, Vol. 26, No. 4, 905–918

0882-7974/11/$12.00 DOI: 10.1037/a0023832

Age-Related Deficits in Low-Level Inhibitory Motor Control

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Inhibitory control functions in old age were investigated with the "masked prime" paradigm in which participants executed speeded manual choice responses to simple visual targets. These were preceded—either immediately or at some earlier time—by a backward-masked prime. Young adults produced positive compatibility effects (PCEs)—faster and more accurate responses for matching than for nonmatching prime-target pairs—when prime and target immediately followed each other, and the reverse effect (negative compatibility effect, NCE) for targets that followed the prime after a short interval. Older adults produced similar PCEs to young adults, indicating intact low-level motor activation, but failed to produce normal NCEs even with longer delays (Experiment 1), increased opportunity for prime processing (Experiment 2), and prolonged learning (Experiment 3). However, a fine-grained analysis of each individual's time course of masked priming effects revealed NCEs in the majority of older adults, of the same magnitude as those of young adults. These were significantly delayed (even more than expected on the basis of general slowing), indicating a disproportionate impairment of low-level inhibitory motor control in old age.

Keywords: aging, inhibition, motor control, negative compatibility effect, masked priming

Supplemental materials: http://dx.doi.org/10.1037/a0023832.supp

Normal aging is generally assumed to be accompanied by a decline in inhibitory control, resulting in increased distractibility and a corresponding decline in cognitive functions (Hasher, Zacks, & May, 1999). As intuitively compelling as this notion might be, experimental results are still inconclusive (see Maylor, Schlaghecken, & Watson, 2005, for examples). One reason for this might be that "inhibition" refers to a diverse, only loosely related set of cognitive functions, which might be differentially affected by aging (e.g., Andres, Guerrini, Phillips, & Perfect, 2008; Collette, Germain, Hogge, & Van der Linden, 2009; Kramer, Humphrey, Larish, Logan, & Strayer, 1994; Nigg, 2000). However, conflicting results have been obtained even within a single experimental paradigm. The negative priming effect—an increase in reaction times when a to-be-ignored distractor becomes the new target stimulus—might serve as an example: numerous studies have found reduced negative priming in older compared to young adults (e.g., Verhaeghen & De Meersman, 1998), but a more recent meta-analysis (Gamboz, Russo, & Fox, 2002) suggests that young and older adults produce negative priming effects of equivalent size.

Similarly conflicting results have recently emerged in the masked prime paradigm, an experimental procedure aimed at investigating low-level, automatic visuomotor control processes (e.g., Eimer & Schlaghecken, 1998). In this task, participants give a speeded response to a simple visual target (e.g., a left-hand response to an arrow pointing to the left). Each target is preceded by a prime stimulus, which is associated with either the same response as the subsequent target (compatible trial), with a different response (incompatible trial), or is without response assignment (neutral trial). Primes are presented very briefly (e.g., 17 or 33 ms) and are followed by a patterned backward mask. This makes them unlikely to be perceived consciously (near-threshold or subthreshold presentation), as evidenced by participants' informal verbal reports and by their inability to identify primes with more than chance accuracy (Eimer & Schlaghecken, 1998, 2002; Schlaghecken & Eimer, 1997). Yet these primes can be shown to trigger their corresponding motor activation, thereby influencing responses to the subsequently presented, clearly visible targets. Relative to neutral trials, responses are faster and more accurate on compatible and slower and less accurate on incompatible trials (positive compatibility effect; PCE) when prime and target are presented in immediate succession. However, with a short delay between prime and target (approximately 100–200 ms), the reverse is true (negative compatibility effect; NCE). This has been taken as evidence that an active inhibition process—either triggered by the sudden lack of prime information (e.g., Schlaghecken & Eimer, 2002, 2006; Schlaghecken, Rowley, Sembi, Simmons, & Whitcomb, 2007) or by the sudden appearance of the mask (e.g., Jaśkowski, 2008; Boy, Clarke, & Sumner, 2008)—suppresses the initially activated motor response.

Because the stimulus triggering the initial response activation is presented below the level of conscious perception, the presence of such an inhibitory effect is surprising: Traditionally, it has been assumed that nonconscious processes comprised only automatic

This article was published Online First May 23, 2011.

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This research was supported by a grant to EAM and FS from the Economic and Social Research Council of Great Britain (RES-000-22-2687).

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activation and passive decay, and that active inhibition required conscious awareness of the stimulus that gave rise to the to-be-inhibited response (for a brief overview, see Eimer & Schlaghecken, 2003). More recently, however, numerous exceptions to this "rule" have been reported in various experimental paradigms (e.g., Aron et al., 2003; Bermeitinger, Frings, & Wentura, 2008; Eimer & Schlaghecken, 1998; Hughes, Velmans, & De Fockert, 2009; Schlaghecken & Eimer, 2006; van Gaal, Ridderinkhof, van den Wildenberg, & Lamme, 2009), providing converging evidence for the existence of nonconsciously triggered (automatic or low-level) inhibitory control processes.

In the case of NCEs in the masked prime task, the inhibition occurs at the level of motor response representations (e.g., Schlaghecken, Klapp, & Maylor, 2009). In most tasks, response inhibition is regulated by the dorso-lateral prefrontal cortex (dlPFC) and the anterior cingulate cortex (ACC; e.g., Faw, 2003; Mansouri, Tanaka, & Buckley, 2009). However, hemodynamic (Aron et al., 2003) and patient studies (Sumner et al., 2007) suggest that in the masked prime task, it relies on subcortical and supplementary motor cortical areas, confirming the notion that low-level inhibition is different from frontally mediated (high-level) control triggered by consciously perceived stimuli. Additional evidence comes from the finding that children (seven- and 12-year-olds) produce NCEs indistinguishable from those of young adults, even though they show considerably less overt response control, corresponding to their as yet immature frontal lobes (Schlaghecken & Sisman, 2006).

With older adults, however, the picture is less clear. In line with the assumption that unlike high-level executive control, low-level inhibition is unaffected by normal aging (Andres et al., 2008; Collette et al., 2009), Sumner et al. (2007) reported robust NCEs in a group of older adults. In marked contrast, Schlaghecken and Maylor (2005) and Seiss and Praamstra (2004) found that older adults fail to produce NCEs. The aim of the present study, therefore, was to establish under which conditions—if any—NCEs can be observed in normal older adults. Experiment 1 investigated reduced processing speed, Experiment 2 perceptual limitations, and Experiment 3 the need for prolonged learning.

Experiment 1–Speed of Information Processing

Information processing speed is reduced with increasing age (e.g., Salthouse, 1996, 2004). Although the neural correlates of processing speed are as yet unknown, it is thought that a deterioration of functional networks might contribute to this effect (e.g., Andrews-Hanna et al., 2007; Damoiseaux et al., 2008). Thus, conceivably, a masked prime might take longer in older adults to activate its corresponding motor response, and inhibition of this response tendency might build up more slowly. In Schlaghecken and Maylor (2005), we tested this prediction by presenting masked primes and targets with mask-target SOAs of 0 ms (typically resulting in PCEs in young adults), 150 ms (resulting in NCEs), 300 ms and 450 ms. If prime-induced motor activation is delayed in older adults, they should produce smaller PCEs at the 0-ms SOA than young adults. Furthermore, if aging delays the onset and/or build-up of low-level inhibition, older adults should produce NCEs only at the 300- or 450-ms SOA. Interestingly, neither of these results was observed. PCEs at the 0-ms SOA were numerically larger in older than in young participants, suggesting that masked primes trigger a motor activation in older adults just as quickly and effectively as in young adults. However, older participants failed to produce statistically reliable NCEs at any of the longer SOAs. This pattern of results was taken as evidence of an age-related loss of low-level inhibitory motor control.

On the other hand, the sample size in that study was relatively small (eight participants per age group), and some of the older participants did produce NCEs at one or other of the longer (>150 ms) SOAs, though no systematic pattern governing these occasional NCEs could be identified. The present experiment, therefore, aimed to replicate the earlier results with a larger sample of young and older participants, and to extend them by investigating whether individual differences in fluid intelligence, crystallized intelligence, processing speed, or visual acuity contributed to the effects.

Method

Participants. Twenty young (17–31 years) and 22 older (65–83 years) participants completed the experiment (see Table 1).

Table 1
Background Details for Participants in Experiments 1–3, and Supplementary Experiments S1 and S2

Experiment	Group	N[1]	M;F[2]	Age[3]	FIQ[4]	CIQ[5]	Speed[6]	Visual acuity[7]
1	Young	19/20	10;9	22.3 (3.5)	102.2 (19.3)	18.8 (4.2)	74.1 (13.2)	6.58 (0.90)
	Older	22/22	11;11	71.0 (4.8)	79.6 (19.7)	25.3 (4.0)	49.7 (10.8)	5.32 (0.65)
2	Young[†]	18/20	4;14	19.1 (2.6)	—	—	—	5.61 (0.50)
	Older[*]	19/19	10;9	71.0 (5.0)	82.4 (19.6)	25.3 (4.3)	50.6 (10.3)	5.21 (0.63)
3	Older	10/10	5;5	67.0 (3.5)	76.4 (20.0)	23.0 (3.0)	53.9 (10.1)	4.80 (1.55)
S1[**]	Older	10/10	5;5	68.2 (5.4)	64.1 (18.0)	23.1 (3.9)	43.6 (8.3)	5.20 (1.32)
S2[**]	Older	8/10	4;4	75.5 (5.4)	79.3 (14.9)	23.9 (1.2)	47.4 (11.3)	5.38 (1.41)

[†] Cognitive scores were not collected from young participants in Experiment 2. [*] The older participants in Experiment 2 were the older participants in Experiment 1, minus three who did not wish to return. [**] Experiments reported in the Supplementary Material.
[1] Number of participants whose data were included in the final analyses/Number of people tested. [2] Numbers of males and females. [3] Mean age (and standard deviation). [4] Mean fluid intelligence score (and standard deviation) based on the total of Parts 1 and 2 of the AH4 test (Heim, 1968); maximum score = 130. [5] Mean crystallized intelligence score (and standard deviation) based on the multiple choice section of the Mill Hill vocabulary test (Raven, Raven, & Court, 1988); maximum score = 33. [6] Mean information processing speed (and standard deviation) based on the Digit Symbol Substitution test (Wechsler, 1981). [7] Mean visual acuity (and standard deviation) as measured by the number of lines read correctly from the Near Vision Test Card (Schneider, 2002) viewed at a distance of 16 inches whilst wearing corrective glasses, with scores ranging from 1 (16/160 – lowest acuity) to 9 (16/16 – highest acuity).

Young participants were mostly students at the University of Warwick who took part either for course credit or for payment of £6. Older participants were members of a volunteer panel who had been recruited through local newspapers and advertisements to join the Warwick Age Study and were paid £10 to cover their travel expenses. Data from one young participant were subsequently excluded from the analyses due to high error rate (>15%). All but four young and three older participants were right-handed.

Background measures. Fluid intelligence was assessed by the AH4 (Heim, 1968), a timed problem-solving test employing verbal and spatial problems (Table 1 shows the combined scores). Crystallized intelligence was assessed by the multiple choice section of the Mill Hill vocabulary test (Raven, Raven, & Court, 1988), in which participants have to select the best synonym for a target word from a set of six alternatives. Speed of information processing was assessed by the Digit Symbol Substitution test from the Wechsler Adult Intelligence Scale–Revised (Wechsler, 1981). Visual acuity was assessed (with glasses if worn) at the beginning of the experiment using the Near Vision Test Card (Schneider, 2002). All participants had normal or corrected-to-normal vision according to self-report. Age group differences for fluid intelligence, crystallized intelligence, speed and visual acuity were all highly significant, $t(39) = 3.58$, -4.92, 6.37, and 5.20, respectively, all ps $< .005$, revealing the typical pattern reported in the aging literature (e.g., Baltes & Lindenberger, 1997; Salthouse, 1991; Schneider & Pichora-Fuller, 2000) of higher fluid intelligence, speed and visual acuity, but lower crystallized intelligence, in young adults than in older adults.

Stimuli and apparatus. Left- and right-pointing double arrows (« and ») served as primes and targets, subtending a visual angle of 2.0° × 0.8° at a viewing distance of approximately 1 m. Masks were constructed from a virtual 8 × 6 grid (2.3° × 1.4°), randomly filled with overlapping horizontal, vertical and oblique lines of different lengths (0.1° to 1.0°; width 0.2°). A new mask was constructed on each trial to avoid perceptual learning of the mask and correspondingly increased prime identification (see Schlaghecken, Blagrove, & Maylor, 2008). Stimuli were presented in black on a white background on a 17" computer screen. A fixation cross (0.1° × 0.1° visual angle), primes and masks appeared in the center of the screen, whereas targets appeared randomly and with equal probability 1.4° above or below the center (i.e., beyond the area occupied by the mask—see Figure 1). Participants were seated in a comfortable chair in a dimly lit, sound attenuated chamber, with response buttons mounted on adjustable armrests under their left and right index fingers.

Procedure. In experimental sessions lasting up to one hour, participants first carried out a masked prime task, followed by a prime identification task. Immediately after the experiment, background cognitive measures were collected from young participants (this information was already available for older participants from an earlier testing session).

In the masked prime task (Figure 1, upper panel), trials started with the fixation cross presented for 250 ms, followed by a blank screen for 650 ms. A prime was then presented for 33 ms, replaced immediately by a 100-ms mask. Either simultaneously with the mask, or after a further blank screen lasting 50, 200, or 350 ms (resulting in mask-target stimulus onset asynchronies (SOAs) of 0, 150, 300, or 450 ms, respectively), a target was presented for 100 ms. Participants were instructed to maintain central eye fixation throughout, and to respond as quickly and accurately as possible to the direction of the target arrows (i.e., a left-hand key-press to arrows pointing to the left, and a right-hand key-press to arrows pointing to the right). The intertrial interval between target offset and the next fixation cross was 1800 ms. On compatible trials, prime and target arrows pointed in the same direction, on incompatible trials, they pointed in opposite directions.

Trials were presented in blocks of 72 trials each. Within each block, left- and right-pointing primes and targets, and compatible and incompatible trials, were presented randomly and with equal probability. In contrast, mask-target SOA was blocked, and eight blocks (two of each SOA condition) were presented in a random order. At the end of each block, there was a short rest period of at least 20 s, after which participants could initiate the next block. They were invited to take a slightly longer rest break halfway through the experiment.

The prime identification task (Figure 1, lower panel) consisted of three 50-trial blocks employing a 2-up-1-down staircase procedure. Primes and masks were presented as before, with equal numbers of left- and right-pointing primes, the mask following the prime immediately, and a 1900-ms intertrial interval. No targets were presented. Participants had to indicate the direction of the prime by pressing the corresponding key (they were instructed to "simply guess" if they felt unsure about the prime's direction). Prime duration (ranging from 17 to 167 ms in 10 17-ms steps) varied as a function of the participant's response on the previous trial. Each block began with the 167-ms duration. Following a correct answer, presentation duration on the subsequent trial was reduced by 17 ms (one screen refresh cycle, until the lower limit of 17 ms was reached, at which point correct responses were followed by a 17-ms prime). After an incorrect answer, presentation duration on the next trial was increased by 33 ms (two refresh cycles, until the upper limit of 167 ms was reached). After each block, there was a short rest period of at least 20 s. Participants were informed in detail about the staircase procedure. They were told to respond spontaneously, but that speed was not of the essence. A brief "warm-up" was given prior to the experimental blocks to familiarize participants with the task.

Data analyses. For the masked prime task, repeated measures analyses of variance (ANOVAs) were performed on mean correct response times (RTs) and error rates, combined across left- and right-hand responses, with the between-subjects factor age group (two levels: young vs. older), and the within-subject factors SOA (four levels: 0, 150, 300, and 450 ms), and compatibility (two levels: compatible vs. incompatible). In all analyses, Greenhouse-Geisser correction to the degrees of freedom was applied where appropriate, and corrected p values are reported. Follow-up analyses were conducted in the form of t tests.

For the prime identification task, because a 10-level staircase was employed and blocks always began at the top level, the first 10 trials of each block were discarded. The mean prime duration on the remaining trials—reflecting the duration at which participants were just more likely to give a correct than an incorrect response—was taken to represent identification threshold. Using a one-sample t test, this duration value was compared against the 33-ms prime duration employed during the masked prime task. Furthermore, we calculated the correlation between identification threshold and priming effects in the different SOA conditions.

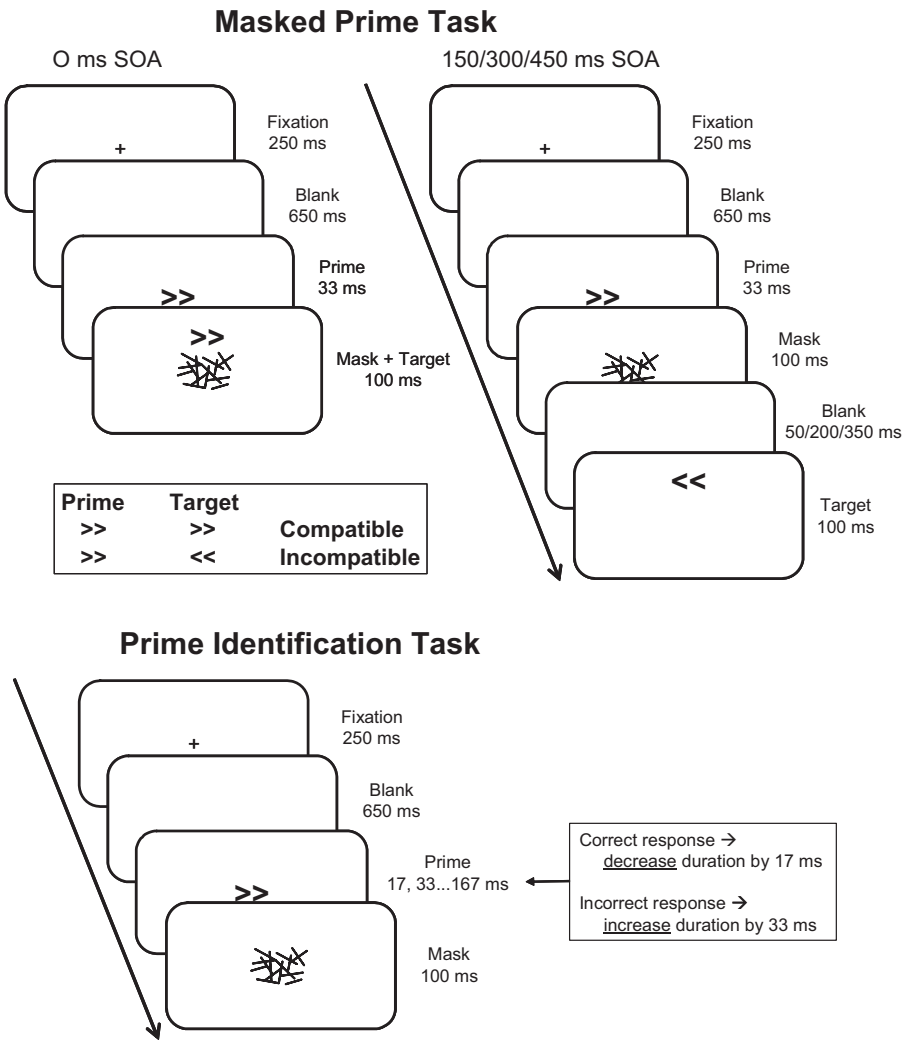

Figure 1. Schematic illustration of trials in the masked prime task (upper panel) and prime identification task (lower panel) in Experiment 1.

Results and Discussion

Prime identification performance. Mean prime durations achieved during the staircase procedure were 39.3 ms (SD = 8.4) and 42.6 ms (SD = 8.4) for young and older adults, respectively, which did not differ significantly from each other, $t(39) = -1.25$, $p > .2$, but significantly exceeded 33 ms, $t(18) = 3.26$, $p < .005$, and $t(21) = 5.34$, $p < .001$, respectively. Masked primes in the present set-up thus can be regarded as equally below threshold and subjectively subliminal for young and older adults.

Masked prime task. Results of the overall ANOVAs are listed in Table 2. As can be seen from Figure 2, young adults were around 100 ms faster (upper panel)—but no less accurate (lower panel)—than older adults, and RTs and errors generally decreased with increasing SOA. For RTs, as expected, PCEs at the 0-ms SOA were observed in both young and older participants, $t(18) = 7.87$, $p < .001$, and $t(21) = 10.44$, $p < .001$, respectively. Older participants did not show any further priming effects, all ts < 1.65, all ps $> .1$. In contrast, young participants showed substantial NCEs at the 150-ms SOA, $t(18) = -5.80$, $p < .001$, no effect at SOA-300, $t(18) < 1$, and (perhaps surprisingly) an NCE at SOA-450, $t(18) = -2.40$, $p < .03$. Comparing compatibility effects between age groups at each SOA revealed a significant difference only for SOA-150, $t(39) = -3.27$, $p < .005$.[1]

[1] To investigate whether the different pattern of priming effects in young and older adults was caused by the overall difference in RTs between groups, we selectively removed the fastest young and the slowest older responders. The age difference was successfully reduced to a nonsignificant 10 ms, $F < 1$, by comparing the slowest 6 young with the fastest 15 older participants. The main findings were essentially unaltered, with a highly significant 3-way Age × SOA × Compatibility interaction, $F(2.2, 41.5) = 4.97$, $MSE = 219.90$, $p < .01$. Compatibility effects differed between age groups at both SOA-0 (32 vs. 54 ms for young and older adults, respectively), $t(19) = -2.48$, $p < .03$, and SOA-150 (−30 vs. 2 ms), $t(19) = -2.79$, $p < .02$.

Table 2
Results of Repeated-Measures ANOVAs for RTs (Ms) and Error Rates (%) in Experiment 1

	RTs				Errors			
Factor	Df	*F*	*MSE*	*p*<	Df	*F*	*MSE*	*p*<
Age	1, 39	40.03	20015.88	.001	1, 39	<1	17.85	n.s.
SOA	2.5, 95.9	34.81	693.50	.001	2.6, 103.2	5.72	4.24	.005
Compatibility	1, 39	17.83	205.10	.001	1, 39	2.06	4.87	n.s.
Age × SOA	2.5, 95.9	<1	693.50	n.s.	2.6, 103.2	<1	4.24	n.s.
Age × Compatibility	1, 39	4.16	205.10	.05	1, 39	3.00	4.87	n.s.
SOA × Compatibility	2.9, 111.5	82.50	154.03	.001	2.8, 107.7	15.72	6.18	.001
Age × SOA × Compatibility	2.9, 111.5	5.02	154.03	.005	2.8, 107.7	1.24	6.18	n.s.

Although for errors the 3-way interaction was not significant, errors closely mirrored RTs (see Figure 2), with significant PCEs at SOA-0 for both young adults, $t(18) = 3.22$, $p < .01$, and older adults, $t(21) = 4.72$, $p < .001$, and a significant NCE for young adults only at SOA-150, $t(18) = -3.40$, $p < .005$. Compatibility effects differed significantly between age groups for SOA-150 only, $t(39) = -2.02$, $p < .05$.

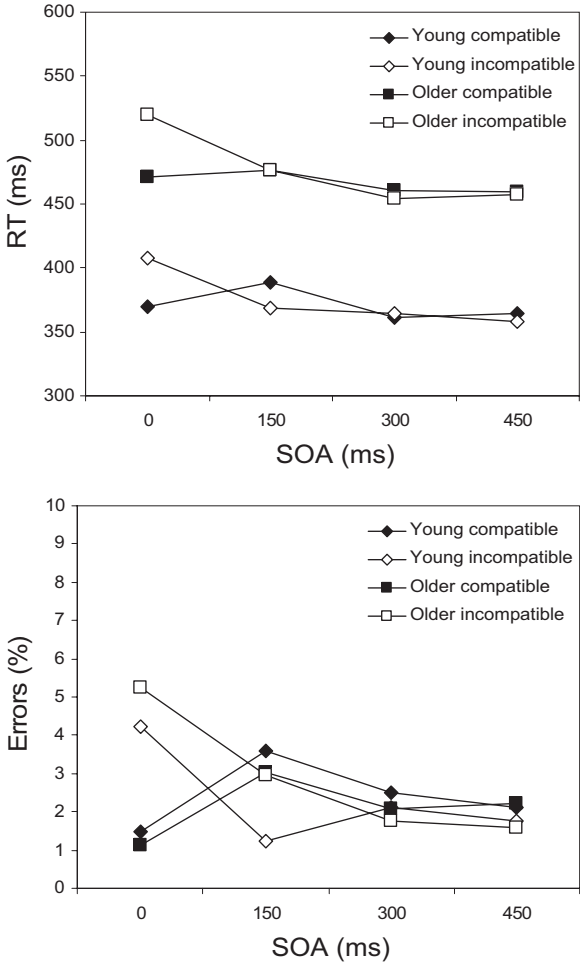

Figure 2. Mean correct response times (RTs) in ms (upper panel) and percentage error rates (lower panel) for young and older adults' compatible and incompatible trials as a function of mask-target stimulus onset asynchrony (SOA) in the masked prime task of Experiment 1.

Neither for RTs nor for error rates did priming effects at any of the four SOAs correlate significantly with prime identification threshold for either young or older adults, although for RTs, there were weak trends (both *p*s < .09) in young adults for compatibility effects at 0- and 150-ms SOAs to be more positive for those with lower prime identification thresholds.

In sum, the results replicated those obtained by Schlaghecken and Maylor (2005), up to and including numerically larger PCEs for older than for young adults, and an oscillatory trend in young adults' compatibility effects (to compare: 38, −20, 2, and −6 ms at 0, 150, 300 and 450 ms SOAs in the present study, and 39, −25, 8 and −5 ms, in Schlaghecken & Maylor), a phenomenon that has been studied in detail by Sumner and Brandwood (2008). Finally, although there were numerical NCEs for RTs at 300- and 450-ms SOAs for older adults in both the present experiment and in Schlaghecken and Maylor's study, they were small in magnitude and failed to reach significance. Thus overall, the data seem to confirm the hypothesis that masked primes trigger an activation-followed-by-inhibition process in young adults, but activation-only in older adults.

There is, however, an alternative possibility. The analyses reported above—in fact, the experimental logic itself—rest on the assumption that processes triggered by the masked prime, processes triggered by the target, and processes related to response execution all follow a fixed, stable time course. While this might be true for young participants, it is well established that for older participants, responses are more variable from trial to trial (see Hultsch, Strauss, Hunter, & MacDonald, 2008, for a review). This could affect the chance to observe certain processes or effects within an experimenter-defined time window, particularly as it is the interval between masked prime and response that determines the direction of priming effects (PCE vs. NCE), not the interval between masked prime and target as such: a response executed shortly after the prime is likely to be affected mostly by the initial activation of the prime-related response, whereas a response executed at a later point is likely to be affected mostly by the subsequent inhibition of this initial response tendency (see, e.g., Eimer, 1999). If older participants' responses are less time-locked to the target than young participants' responses, then these variations might obscure any inhibitory effect present in their data. To investigate this

possibility, we reanalyzed the data with respect to prime-response interval.

Prime-locked time course analysis. First, mean RTs on correct compatible and incompatible trials were calculated for each participant and each experimental block individually. Second, any error responses within a given block were replaced with the corresponding mean RT for that condition within this block. Third, all RTs were recalculated with respect to prime offset (mask onset) by adding the corresponding mask-target SOA. Fourth, for each participant and each compatibility condition separately (but across SOA blocks) RTs were rank-ordered. Importantly, this could—and usually did—lead to a reshuffling of RTs across SOA conditions, as relative to the prime, slow responses from a short SOA condition might be executed later than fast responses from the next longer SOA condition. Fifth, these RT series were divided into 16 latency bins of 18 trials each. Finally, priming effects were computed by subtracting compatible from incompatible mean RTs for each latency bin, and their significance determined by comparing—for each participant individually—compatible and incompatible RTs in each latency bin with an unpaired *t* test and applying a Bonferroni-corrected significance criterion.

Figure 3 presents the priming effects in each of the 16 bins for one representative young and one representative older participant. Six values were extracted from these curves: (a) the latency bin with the largest initial PCE (PCE peak latency) and (b) the magnitude of this PCE peak (PCE peak amplitude in ms); (c) the latency bin where the first significant negative effect occurred (NCE onset latency) and (d) the magnitude of the NCE onset (NCE onset amplitude in ms); (e) the latency bin with the largest significant negative effect (NCE peak latency), and (f) the magnitude of this NCE peak (NCE peak amplitude in ms).[2]

All participants—both young and older—produced an NCE in at least one latency bin, though not all of these were significant. Thus data from four older and one young participant did not enter the analysis, leaving 18 participants in each age group.[3]

The PCE peak occurred later in older than in young adults (Figure 4, top panel), $t(34) = 3.37$, $p = .002$, but PCE peak amplitudes did not differ significantly between age groups, $t(34) = 1.65$, $p > .1$ (Figure 4, bottom panel). Surprisingly, the NCE showed the same pattern: both onset latency and peak latency were substantially longer in older than in young adults, both ts > 2.56, both ps $< .02$, whereas onset and peak amplitudes did not differ between age groups, both ts < 1, both ps $> .4$. Recall that latency bins are specific to each participant's RT distribution, such that an individual's median RT—and consequently, the group's mean of medians—corresponds to a latency bin value of 8.5. Thus for young adults, NCE onset and peak both occurred in the first half of the response distribution (i.e., with latencies shorter than young participants' median RT), whereas for older adults, both occurred in the second half (i.e., latencies longer than older participants' median RT). In other words, NCEs were not merely delayed in older relative to young participants, but were disproportionally delayed beyond the overall age difference in average RTs. This can also be seen in Figure 4 by comparing age differences in prime-locked RTs between means (91 ms), NCE onset (178 ms) and NCE peak (217 ms).

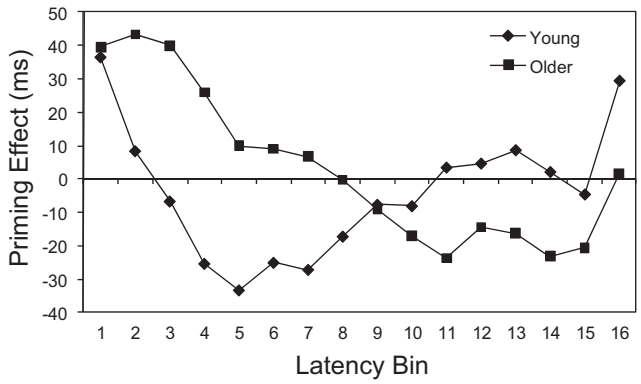

Figure 3. RT priming effects (incompatible−compatible) in each of 16 RT latency bins from the prime-locked time course analysis of Experiment 1. Data from two representative participants (one young, one older).

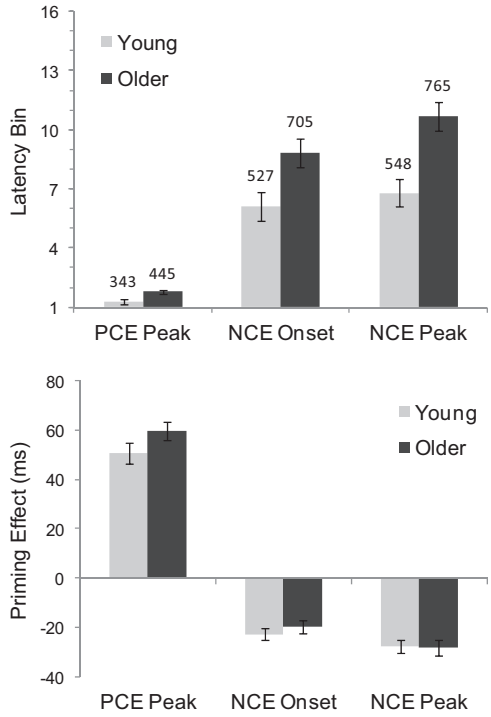

Figure 4. Mean parameters (±1 *SE*) from the prime-locked time course analysis of Experiment 1 for 18 young and 18 older adults. Upper panel: timing in terms of latency bins, with corresponding prime-locked RTs indicated in ms. Note that for each age group, the median RT in terms of latency bins is 8.5, with corresponding mean prime-locked RTs of 600 and 691 ms for young and older groups, respectively. Lower panel: RT priming effects (incompatible−compatible) in ms.

[2] For participants who showed two distinct negative peaks, the first one was selected for this analysis.

[3] We repeated the analyses twice, once removing the "significance" criterion and including all participants, and once applying a stricter criterion (significant NCEs in at least two successive latency bins), which led to the exclusion of 7 older and 4 young participants. Neither approach revealed a qualitatively different pattern of results.

The difference in PCE peak latency between the two age groups might be due to this later onset of inhibition in older adults. In the majority of young participants, the PCE peaked in the first latency bin already and then decreased (from 47.9 to 42.4 ms), whereas in the majority of older participants, PCE amplitude increased from the first to the second latency bin (from 48.2 to 55.5 ms). The interaction between latency bin (first vs. second) and age group was significant, $F(1, 34) = 5.80$, $MSE = 127.65$, $p < .03$, consistent with the assumption that at the (relative) time where prime-triggered motor activation is already affected by inhibition in young adults, it is still building up in older adults.

These results clearly demonstrate that despite initial appearance, inhibition of a nonconsciously triggered response does occur in the majority of older adults, but with a disproportional delay and with much greater interindividual variation.[4] This finding is particularly puzzling in light of the fact that the initial prime-triggered activation seems to be unaffected by age (note that PCEs in the first latency bin were almost identical for young and older participants). If an older adult's visuomotor system is sufficiently sensitive and efficient to quickly produce substantial motor activation in response to a fleeting, not consciously perceived, masked prime, then why is this system not capable of producing correspondingly fast inhibition? The obvious answer seems to be that initial activation and subsequent inhibition are mediated by two separate systems, one largely immune to effects of aging, the other highly sensitive to it. We will return to this in the General Discussion. First, however, an alternative possibility has to be considered.

Nonconsciously triggered inhibition is thought to involve a threshold mechanism such that only sufficiently strongly activated response tendencies become inhibited, whereas weaker activations decay passively (Schlaghecken & Eimer, 2002). If the coherence of functional neural circuits deteriorates with age (e.g., Andrews-Hanna et al., 2007; Damoiseaux et al., 2008; for an overview, see Bishop, Lu, & Yankner, 2010)—effectively decreasing the signal-to-noise ratio in the system—then it seems likely that higher motor activation levels (and correspondingly more time to accumulate the necessary input) are required to reach the threshold. If this interpretation is correct, then perceptually stronger primes—resulting in correspondingly stronger and more rapidly accumulating motor activation[5]—should cause NCEs to occur earlier in older adults, such that they become evident even with the "standard" 150-ms mask-target SOA. This was investigated in the following experiment.

Experiment 2–Perceptual Limitations

As prime identification thresholds in Experiment 1 were comparable for young and older participants, one might argue that the strength of the prime's neural representation was the same in young and in older adults. However, if the integrity of functional neural circuits deteriorates with increasing age, then accumulating motor activation from perceptual input should take longer in older than in young adults at any given level of stimulus visibility. Consequently, whereas for young adults a 33-ms masked prime is sufficient for motor activation to quickly reach the inhibition threshold, older participants might need perceptually stronger primes to accumulate motor activation at a comparable rate. Experiment 2 tested this hypothesis by presenting primes with the standard 33-ms duration and with two longer durations (50 and 67 ms), with mask-target SOAs of 150 and 300 ms.

If the above hypothesis is correct, then the increased perceptual strength of long-duration primes should cause sufficiently fast and strong motor activation in older participants to elicit NCEs at either the 150- or the 300-ms SOA. For young participants, in contrast, long primes should result in PCEs rather than NCEs. In young adults with highly efficient neural circuitry, a masked prime of increased perceptual strength should very quickly activate frontal cortical areas, which feed information back to perceptual areas via reentrant links (e.g., Caminiti et al., 1999; Lamme, 2004; Lamme & Roelfsema, 2000). A prime-related motor response thus can continue to accumulate activation even once the prime is masked: a motor activation initially supported through direct sensory input can subsequently be supported through reentrant top-down signals. According to the self-inhibition model of NCE (Schlaghecken & Eimer, 2000, 2002, 2006; Schlaghecken, Bowman & Eimer, 2006), this continued support of a motor tendency will prevent the release of self-inhibition, thus resulting in PCEs.

Method

Participants. Twenty young and 19 older participants completed the experiment. Data from two young participants were subsequently excluded from the analyses due to excessive error rates (>15%). None of the young participants had taken part in Experiment 1, whereas the older participants were those from Experiment 1 who agreed to return for a second session approximately two months later. Participants included in the analyses ranged in age from 18–29 years (young) and 65–83 years (older). All but three young and two older participants were right-handed (see Table 1 for other background details).

Procedure. Experimental sessions comprised the visual acuity test, the masked prime task, and the prime identification task. These tasks were identical to those of Experiment 1 with the following exceptions: (a) prime duration was varied between blocks at 33, 50 or 67 ms, (b) only two mask-target SOAs (150 and 300 ms) were employed, and (c) intertrial interval was shortened to 1300 ms. Following practice trials, participants completed 12 72-trial blocks of masked prime trials (two blocks for each combination of prime duration and SOA), presented in a random order. Mean correct RTs and error rates were analyzed with age group as the between-subjects variable, and SOA (two levels), prime duration (three levels), and compatibility (two levels) as within-subjects variables. Furthermore, we conducted a time-course analysis analogous to the one described for Experiment 1—with eight instead of 16 latency bins, corresponding to the reduced range of mask-target SOAs—for the 33-ms prime duration condition.

[4] Note that the standard errors for NCE onset and peak latency in young adults were driven entirely by two participants with extremely late NCEs. In older adults, by contrast, they reflect a more or less even distribution of NCE latencies.

[5] For instance, Shadlen and Newsome (2001) have shown that visuomotor neurons in the parietal cortex respond both earlier and more strongly to a perceptually strong than to a perceptually weak target stimulus, suggesting a direct relationship between the perceptual strength of a stimulus and the speed with which it impacts on subsequent perceptuomotor processing stages.

Results

Prime identification performance. Young and older adults again did not differ, with mean identification-threshold prime durations of 44.4 ms (SD = 11.1) and 48.1 ms (SD = 15.2), respectively, $t(35) = -0.84$, $p > .4$. These values significantly exceeded 33 ms for both young adults, $t(17) = 4.33$, $p < .001$, and older adults, $t(18) = 4.32$, $p < .001$.

Masked prime task. The overall means for RTs and error rates are presented in Table 3, whereas Figure 5 shows the main findings more readily as mean differences between incompatible and compatible trials (i.e., compatibility effects, with PCEs above and NCEs below zero). Overall, young adults were faster (by 115 ms) but less accurate than older adults (6.4% vs. 3.1% errors). RTs decreased slightly with increasing SOA (by 15 ms), and increased slightly with prime duration (also by 15 ms). Neither of these factors affected error rates. There were no main effects of compatibility (see Table 4).

At the 150-ms SOA, young adults showed NCEs with 33- and 50-ms primes for RTs, $t(17) = -7.52$ and -2.46, both ps $< .03$, and error rates, $t(17) = -4.89$ and -3.63, both ps $< .005$. Older adults showed an 11-ms NCE at SOA-150 with 33-ms primes, $t(18) = -2.36$, $p < .05$, and a PCE with 67-ms primes for RTs, $t(18) = 5.50$, $p < .001$. At the 300-ms SOA, both age groups showed PCEs with 67-ms primes for RTs, $t(17) = 3.85$, $p < .002$, and $t(18) = 2.55$, $p < .003$, and error rates, $t(17) = 2.48$, $p < .03$, and $t(18) = 2.47$, $p < .03$.

RT compatibility effects were significantly more positive for older than for young participants at all three prime durations for SOA-150, $t(35) = 3.00$, 2.63, and 3.21, all ps $< .02$, whereas no age differences were found for SOA-300, all ts < 1. Error priming effects differed significantly between age groups only at prime durations of 33 and 50 ms at SOA-150, $t(35) = 4.29$ and 2.95, both ps $< .01$.

Again, mean prime identification duration was not systematically associated with RT or error compatibility effects for either age group (out of 24 correlations in total, only one—in young adults—reached significance).

Prime-locked time course analysis. As the above analyses clearly suggested that with longer prime durations, older participants were less, not more, likely to produce NCEs, we restricted the time course analysis to the 33-ms prime duration. To facilitate comparisons between experiments, we labeled the latency bins "5" to "12" (corresponding to the middle portion of the 16 latency bins analyzed earlier, as the 150- and 300-ms SOAs correspond to the middle portion of Experiment 1's four SOAs of 0, 150, 300, and 450 ms). Results replicated those obtained in Experiment 1 (see Figure 6). Again, all participants produced a negative effect in at least one latency bin. For six older participants, NCEs failed to reach significance, and these participants were excluded from the analysis.[6] Both NCE onset and NCE peak were delayed in older compared to young participants, $t(29) = 2.45$, $p < .05$, and $t(29) = 2.99$, $p < .02$, respectively, with older adults' peak latency being longer and young adults' peak latency being shorter than their respective average RTs (Figure 6, upper panel). NCE onset amplitude was smaller in older than in young participants, $t(29) = 2.35$, $p < .03$, but NCE peak amplitude did not differ between age groups, $t < 1$ (Figure 6, lower panel).

Discussion

In summary, an attempt to compensate for older adults' perceptual limitations by lengthening prime durations not only failed to produce NCEs in older adults at either standard (150 ms) or longer (300 ms) mask-target SOAs with longer prime durations, but resulted in older adults showing increased PCEs, parallel to the effects observed in young adults. This suggests that the delayed inhibition found for older adults in Experiment 1 is not due to weak perceptual prime representation and delayed motor activation (see Supplementary Material for further evidence).

However, the fact that older adults produced a small but significant NCE (at least for RTs) with 33-ms primes at SOA-150 was unexpected. Recall that these were participants who had previously taken part in Experiment 1. Comparing their NCEs for the same condition across experiments showed an NCE increase from -3 to -11 ms that approached significance, $t(18) = 1.92$, $p < .08$, suggesting the possibility of a learning effect. Correspondingly, the prime-locked time course analysis revealed a larger NCE peak amplitude in this experiment compared to Experiment 1. However, as this amplitude was again of the same magnitude as that produced by young participants, it might reflect a nonspecific effect of the present experimental design rather than a learning effect: perhaps having to adjust to only two different SOAs and/or being more "tuned" to the primes because of visible primes in other blocks is beneficial for larger NCEs. More promising in terms of a specific learning effect is the observation that in the present experiment, the NCE onset and peak latencies of older adults were somewhat earlier with respect to their average RTs than in Experiment 1 (compare Figures 4 and 6). Perhaps increased experience with the task can speed up inhibition in older adults sufficiently to cause NCEs at the standard 150-ms SOA, a possibility we explore in the next experiment.

Experiment 3–Prolonged Learning

NCEs are usually observed within the first few trials in young adults, with the notable exception of experiments conducted by Klapp and colleagues (e.g., Klapp & Hinkley, 2002), where NCEs were observed only on the second day of testing. In their experiments, targets were presented for only 16 ms, making target identification difficult even for young adults. Conceivably, 100-ms target presentation in our studies is equivalently difficult for older adults, which could account for older adults' NCE beginning to emerge in the 150-ms SOA condition in Experiment 2. To investigate this issue, the present experiment employed a perceptual learning design, where a new group of older adults performed the masked prime task in three testing sessions over the course of around two weeks. If learning improves inhibition—possibly by shortening inhibition latency as suggested by the prime-locked time course analysis of Experiment 2—then we expect to see an NCE develop over the course of three sessions in the 150-ms condition.

[6] As with Experiment 1, conducting the analysis with all participants did not change the overall pattern of results.

Table 3
Mean Correct Response Times (RTs) in Ms and Percentage Error Rates for Young and Older Adults' Compatible and Incompatible Trials for Mask-Target Stimulus Onset Asynchronies (SOAs) of 150 and 300 Ms and Prime Durations of 33, 50, and 67 Ms in Experiment 2

		RT (ms)				Errors (%)			
		Young		Older		Young		Older	
SOA (ms)	Prime duration (ms)	Comp	Incomp	Comp	Incomp	Comp	Incomp	Comp	Incomp
150	33	380	349	486	475	11.8	3.4	2.4	1.8
	50	370	358	484	490	9.3	4.4	2.6	1.8
	67	369	373	491	522	6.9	8.6	2.2	4.4
300	33	359	358	465	463	4.8	5.8	4.2	4.2
	50	352	356	457	463	4.5	5.9	2.6	3.7
	67	356	381	467	489	4.3	7.2	1.6	6.1

Method

Participants. Ten new volunteers aged 64–76 years participated in the experiment; all but one were right-handed (see Table 1 for other details).

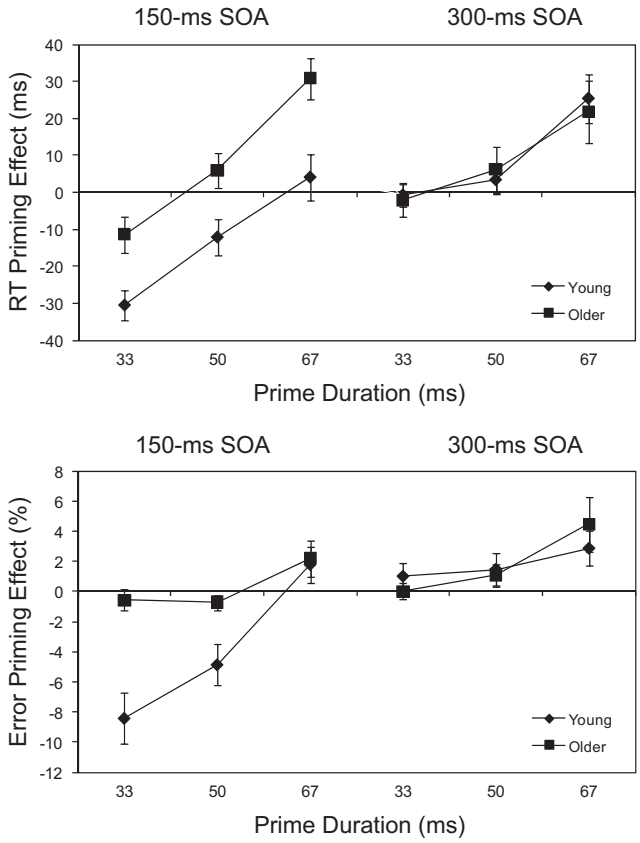

Figure 5. Mean priming effects (incompatible−compatible) ± 1 *SE* for correct response times (RTs) in ms (upper panel) and percentage error rates (lower panel) for young and older adults as a function of prime duration for mask-target stimulus onset asynchronies (SOAs) of 150 and 300 ms in Experiment 2.

Procedure. The stimuli, apparatus and procedure were identical to those in Experiment 2 with the following exceptions: (a) SOAs were 0 and 150 ms, (b) only the 33-ms prime duration was used, (c) there was no prime identification task, and (d) the experiment was repeated three times on separate days within a 7–17 day timeframe, with 1–12 days ($M = 6.7$, $SD = 2.6$) between sessions. Visual acuity was assessed at the beginning of Session 1.

Data analysis. Repeated-measures ANOVAs were conducted on correct RTs and error rates for the factors session (first, second, and third), SOA (0 and 150 ms), and compatibility. Prime-locked time course analysis was conducted for eight latency bins (Bins 1–8). We did not expect NCEs to occur in the first session, and, therefore, would not be able to extract parameters for significant NCE bins. Instead, we extracted for each participant the amplitude and the latency of the most positive and the least positive (potentially negative) priming effect in each session.

Results and Discussion

Figure 7 shows the masked prime data for RTs and errors across the three experimental sessions. Performance improved over successive sessions, as both RTs and errors decreased overall from Sessions 1–3, a trend that was nonsignificant for RTs, $F(1.9, 17.5) = 1.78$, $MSE = 1420.02$, $p > .1$, but significant for errors, $F(1.3, 12.1) = 5.36$, $MSE = 3.91$, $p < .05$. For errors, the learning effect was more evident in the SOA-0 condition, $F(1.4, 12.4) = 5.79$, $MSE = 2.24$, $p < .05$. Importantly, however, the contrast between the presence of PCEs at 0-ms SOA and the absence of any priming effects at 150-ms SOA [RT: $F(1, 9) = 28.69$, $MSE = 747.42$, $p < .001$; errors: $F(1, 9) = 7.59$, $MSE = 11.08$, $p < .03$] did not change across sessions [RT: $F < 1$; errors: $F(1.2, 10.8) = 4.15$, $MSE = 2.85$, $p > .05$]. Specifically, there was no evidence of an NCE emerging in the 150-ms SOA condition in Sessions 2 or 3—instead, in Session 2, both RTs and error rates even showed a numerical trend for a PCE.

Prime-locked time course analysis. Prolonged experience did not change the latencies of the most and least positive peaks (most positive: Bins 2.2, 1.8, and 1.8 in Sessions 1, 2, and 3, respectively; least positive: Bins 7.6, 7.5, and 7.8), nor did it change their amplitudes (most positive: 34.7, 38.6, and 33.7 ms in Sessions 1, 2, and 3, respectively; least positive: 2.0, 4.9, and 1.1 ms), all Fs < 1.73, all ps $> .2$.

Table 4
Results of Repeated-Measures ANOVAs for RTs (Ms) and Error Rates (%) in Experiment 2

	RTs				Errors			
Factor	Df	*F*	*MSE*	*p*<	Df	*F*	*MSE*	*p*<
Age	1, 35	79.74	18671.06	.001	1, 35	6.45	182.81	.02
SOA	1, 35	39.72	631.28	.001	1, 35	<1	60.28	n.s.
Duration	1.8, 62.3	17.45	670.59	.001	1.2, 41.9	<1	45.45	n.s.
Compatibility	1, 35	1.96	664.00	n.s.	1, 35	<1	11.28	n.s.
Age × SOA	1, 35	13.96	631.28	.002	1, 35	4.71	60.28	.05
Age × Compatibility	1, 35	4.38	664.00	.05	1, 35	10.85	11.28	.005
SOA × Compatibility	1, 35	9.69	356.92	.005	1, 35	21.85	16.30	.001
Duration × Compatibility	1.7, 59.9	40.80	270.44	.001	1.4, 48.5	15.78	21.15	.001
Age × SOA × Compatibility	1, 35	9.40	356.92	.005	1, 35	6.97	16.30	.02
SOA × Duration × Compatibility	1.7, 60.1	5.61	102.12	.01	1.9, 64.9	3.59	8.19	.05
Age × SOA × Duration × Compatibility	1.7, 60.1	1.53	102.12	n.s.	1.9, 64.9	7.67	8.19	.002

In sum, Experiment 3 failed to provide any evidence that prolonged experience with the masked prime task speeds up inhibition in older participants to a noticeable degree.

Individual Differences in the NCE

Although older adults' mean NCE at SOA-150 was around zero, some participants showed (almost) normal NCEs in this condition, whereas others showed PCEs. Moreover, individuals tended to show similar effects in different sessions (i.e., older adults' SOA-150 priming effects correlated significantly in Experiments 1 and 2, as well as in the three sessions of Experiment 3, all rs $> .57$, all ps $< .03$).

To investigate whether there are individual characteristics that reliably differentiate those older adults who show NCEs from those who show PCEs, RT priming effects for older adults with 33-ms masked primes and 150-ms SOAs were pooled across Experiments 1 (same participants as Experiment 2) and 3 (Session 1 only), together with data from a set of highly similar masked prime experiments (some of which are included in the Supplementary Material), resulting in a total of 80 people, 36 showing NCEs and 44 showing PCEs at the 150-ms mask-target SOA (M priming effect = 0.4 ms). However, independent samples t tests showed that these two groups did not differ significantly in terms of any of the following: age, gender, handedness, fluid intelligence (available for 52 participants only), crystallized intelligence, information processing speed, visual acuity, prime identification performance (where available), priming at 0-ms SOA (where available), overall RT at SOA-150, and overall error rate at SOA-150. In addition, both a binary logistic regression (predicting whether participants' priming was negative or positive) and a linear regression (predicting participants' actual compatibility effects), with the eight predictor variables for which there was a full dataset, revealed no significant effects. Correlations between the individual-differences variables and parameters from the prime-locked time course analysis of Experiment 1 (18 older adults) confirmed this picture: the only significant relationship was an earlier NCE onset for those with poorer prime identification. Thus, although clearly not random, older adults' priming effects were not associated with any of the measures collected on our sample.

General Discussion

The present study provides evidence of a systematic age-related difference in the impact of briefly presented, backward masked primes, presented near or below the threshold of conscious awareness, on responses to subsequently presented target stimuli. In both young and older participants, primes triggered an initial activation of the corresponding motor response (as evidenced by PCEs with a 0-ms mask-target SOA of comparable magnitude). However, only in young participants was this initial activation quickly replaced by an inhibitory phase, reflected in NCEs with 150-ms mask-target SOAs. In older participants, in contrast, the 150-ms NCE was notably absent. Experiment 1, using a prime-locked time course analysis, revealed that this absence is not, as previously thought, due to a lack of inhibition, but rather due to a disproportionate delay of the inhibitory phase. For the majority of older participants, significant prime-locked NCEs were observed, which were of a similar magnitude to those produced by young participants, but were delayed even beyond the overall age-related RT increase. Experiments 2 and 3 indicated that this delay could not be overcome by increasing either the perceptual strength of the primes or the time spent on the task. This suggests that the delay does not reflect general aging effects such as impaired visual processing or slowed visuomotor learning, but represents a specific effect of aging on low-level inhibitory control. The lack of any systematic relationship between NCEs and any other age-related factors like fluid intelligence or processing speed further supports this conclusion.

As the notion of delayed inhibition stands in marked contrast to our earlier view that inhibition is absent in old age (Schlaghecken & Maylor, 2005), it seems appropriate to seek further confirmation. We first reanalyzed the Schlaghecken and Maylor dataset—obtained from eight young and eight older participants in an experimental design similar to the present Experiment 1—with the prime-locked time course method. All participants showed significant NCEs in at least one latency bin in the time course analysis. The overall pattern of results was qualitatively identical to the present Experiment 1, with significantly delayed PCE and NCE peaks in older compared with young adults, both ts > 2.33, both ps $< .04$, but similar PCE and NCE peak amplitudes, both ts < 1. Second, we conducted two further experiments (see Supplemen-

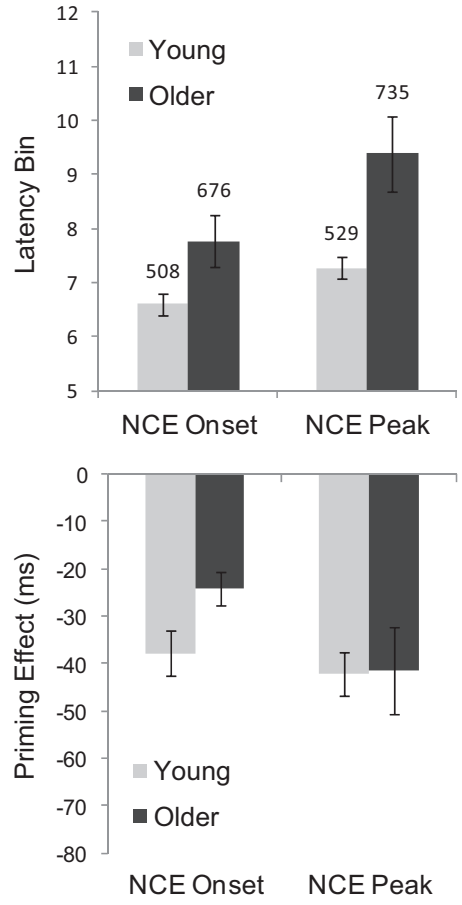

Figure 6. Mean parameters (±1 *SE*) from the prime-locked time course analysis of Experiment 2 (33-ms primes) for 18 young and 13 older adults. Upper panel: timing in terms of latency bins (note that because only mask-target SOAs of 150 and 300 ms were used, latency bins were labeled 5–12 to facilitate direct comparison with Experiment 1, which used SOAs of 0, 150, 300, and 450 ms), with corresponding prime-locked RTs indicated in ms. Median RT in terms of latency bins is 8.5, with corresponding prime-locked mean RTs of 582 and 704 ms for young and older groups, respectively. Lower panel: RT priming effects (incompatible−compatible) in ms.

tary Material) to explore the possibility that perceptual prime strength is, after all, a crucial factor, and that Experiment 2 simply failed to employ a suitable method to manipulate it. The results confirmed the failure to shorten NCE latency through manipulations of the primes' perceptual properties.

Aging and Low-Level Inhibitory Control

The pattern of intact initial activation followed by delayed inhibition in older compared to young adults is strongly reminiscent of the pattern observed for the inhibition of return (IOR) effect. IOR is the slowing of responses to visual targets at a recently attended (cued) location relative to targets at a new (uncued) location (see Klein, 2000, for a review). Like the NCE, IOR is assumed to reflect a low-level or "reflexive" inhibitory effect rather than a voluntary control process (Tipper & Kingstone, 2005), mediated by nonexecutive cortical and subcortical structures (Aron et al., 2003; Sapir, Soroker, Berger, & Henik, 1999; Shipp, 2004). Earlier studies observed equivalent IOR effects for young and older adults (e.g., Faust & Balota, 1997; Hartley & Kieley, 1995), contributing to the notion that in contrast to high-level or "cognitive" inhibition, low-level inhibition might be relatively spared in aging (see also Maylor & Henson, 2000). However, using a wider range of cue-target SOAs (from 50 to 3000 ms), Castel, Chasteen, Scialfa, and Pratt (2003) found that older adults showed larger initial facilitation and delayed subsequent inhibition relative to young adults (see also Langley, Fuentes, Vivas, & Saville, 2007).

However, it is noteworthy that in Castel et al.'s (2003) study, the initial facilitation was more than three times larger in older than in young adults. As a consequence, despite the substantial delay in IOR, the *slopes* of cueing effects (i.e., the rate of turning from facilitation to inhibition) were virtually identical for young and older participants (see Castel et al., Figure 2). One might thus argue that IOR was, in fact, unaffected by age, and that only the necessity to overcome a disproportionally large initial activation caused the apparent delay in older adults. Importantly, this argument does not apply to the present data. Although 0-ms PCEs were numerically larger in older than in young participants, this difference was nonsignificant, and was very small compared to the substantial delay in NCE peak.

To quantify this pattern, we estimated the rate of inhibition in Experiment 1 by calculating the slope of priming effects from the peak PCE (first or second latency bin, determined for each participant individually) to the peak NCE (also determined individually). The average slope of priming effects was −0.44 ($SE = 0.037$) for young and −0.32 ($SE = 0.037$) for older adults, and these differed significantly, $t(34) = 2.26$, $p < .05$.

This pattern confirms that while increased initial activation might contribute to the delayed NCE in older adults, it is unlikely to be a sufficient explanation. Rather, it suggests a genuine impairment of low-level inhibitory control in old age. It has recently been demonstrated that aging selectively delays inhibitory processes in high-level cognitive control (the voluntary suppression of

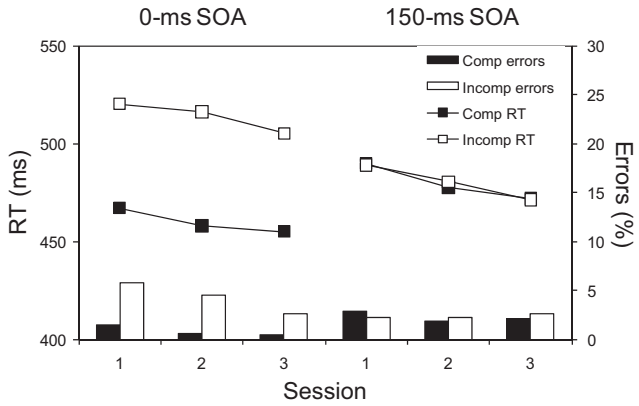

Figure 7. Mean correct response times (RTs) in ms (lines) and percentage error rates (bars) for older adults' compatible and incompatible trials across three experimental sessions for mask-target stimulus onset asynchrony (SOA) of 0 and 150 ms in the masked prime task of Experiment 3.

irrelevant information in visual working memory; Gazzaley et al., 2008; Jost, Bryck, Vogel, & Mayr, 2010). As far as we are aware, the present study demonstrates for the first time a similar phenomenon in low-level (nonvoluntary or automatic) inhibitory control.

Not enough is known to date about the precise functional organization of the brain, and the effects of aging on it, to pinpoint exactly what changes in the system would account for these effects. However, it is noteworthy that activity in prefrontal and parietal cortical areas during simple and automatic motor tasks is greater in older than in young adults (Mattay et al., 2002; Wu & Hallett, 2005), suggesting that with increasing age, motor control shifts from (highly efficient) low-level processes to (less efficient) high-level processes. At the same time, prefrontal and parietal cortex are areas particularly affected by age-related neuron loss (Good et al., 2001; Resnick, Pham, Kraut, Zonderman, & Davatzikos, 2003; Salat et al., 2004), and are consequently increasingly ill-equipped to cope with complex or rapidly changing motor task demands. In line with this, older adults show evidence for reduced intra- and intercortical inhibition, and a correspondingly reduced ability to flexibly modulate motor responses (Seidler et al., 2010). In other words, subtle motor control might be more difficult for older adults because the automatic balance of competing response alternatives provided by lateral inhibitory links is no longer fully functional. According to the self-inhibition hypothesis, the NCE reflects the activity of local opponent-process networks, comprising an excitatory and an inhibitory component, reciprocally linked to each other (Schlaghecken et al., 2006). If the inhibitory component of this circuit is selectively impaired, fast and automatic control of subthreshold motor activity would become almost impossible.[7] In order to compensate, older adults might increasingly rely on high-level (prefrontally mediated) inhibition, which would provide slower and less stimulus-triggered control. Together, these changes would manifest as an increased initial activation, and in a delay and an increase in interindividual variability in the subsequent inhibition (see also Castel et al., 2003; Gazzaley et al., 2008; Jost et al., 2010; Vallesi & Stuss, 2010).

[7] It has to be noted, though, that recent results indicate a negative relationship between NCE magnitude and GABA concentration in the supplementary motor area, that is, lower GABA concentrations were associated with larger NCEs (Boy, Evans, et al., 2010). However, the SMA might be responsible for processing the conflicting inhibited, disinhibited, and target-related motor plans (Boy, Husain et al., 2010), whereas the inhibition itself might have been generated at an earlier—possibly subcortical—processing stage (e.g., Schlaghecken, Münchau, Bloem, Rothwell, & Eimer, 2003; Schlaghecken & Sisman, 2006).

References

Andres, P., Guerrini, C., Phillips, L. H., & Perfect, T. J. (2008). Differential effects of aging on executive and automatic inhibition. *Developmental Neuropsychology, 33,* 101–123. doi:10.1080/87565640701884212

Andrews-Hanna, J. R., Snyder, A. Z., Vincent, J. L., Lustig, C., Head, D., Raichle, M. E., & Buckner, R. L. (2007). Disruption of large-scale brain systems in advanced aging. *Neuron, 56,* 924–935. doi:10.1016/j.neuron.2007.10.038

Aron, A., Schlaghecken, F., Fletcher, P., Bullmore, E., Eimer, M., Barker, R., . . . Robbins, T. (2003). Inhibition of subliminally primed responses is mediated by the caudate and thalamus: Evidence from fMRI and Huntington's disease. *Brain, 126,* 713–723. doi:10.1093/brain/awg067

Baltes, P. B., & Lindenberger, U. (1997). Emergence of a powerful connection between sensory and cognitive functions across the adult life span: A new window to the study of cognitive aging? *Psychology and Aging, 12,* 12–21. doi:10.1037/0882–7974.12.1.12

Bermeitinger, C., Frings, C., & Wentura, D. (2008). Reversing the N400: Event-related potentials of a negative semantic priming effect. *Neuroreport, 19,* 1479–1482. doi:10.1097/WNR.0b013e32830f4b0b

Bishop, N. A., Lu, T., & Yankner, B. A. (2010). Neural mechanisms of ageing and cognitive decline. *Nature, 464,* 529–535. doi:10.1038/nature08983

Boy, F., Clarke, K., & Sumner, P. (2008). Mask stimulus triggers inhibition in subliminal visuo-motor priming. *Experimental Brain Research, 190,* 111–116. doi:10.1007/s00221-008–1515-5

Boy, F., Evans, C. J., Edden, R. A. E., Singh, K. D., Husain, M., & Sumner, P. (2010). Individual differences in subconscious motor control predicted by GABA concentration in SMA. *Current Biology, 20,* 1779–1785. doi:10.1016/j.cub.2010.09.003

Boy, F., Husain, M., Singh, K. D., & Sumner, P. (2010). Supplementary motor area activations in unconscious inhibition of voluntary action. *Experimental Brain Research, 206,* 441–448. doi:10.1007/s00221-010–2417-x

Caminiti, R., Genovesio, A., Marconi, B., Mayer, A. B., Onorati, P., Ferraina, S., . . . Molinari, M. (1999). Early coding of reaching: Frontal and parietal association connections of parieto-occipital cortex. *European Journal of Neuroscience, 11,* 3339–3345. doi:10.1046/j.1460–9568.1999.00801.x

Castel, A. D., Chasteen, A. L., Scialfa, C. T., & Pratt, J. (2003). Adult age differences in the time course of inhibition of return. *Journal of Gerontology: Psychological Sciences, 58*B, P256–P259.

Collette, F., Germain, S., Hogge, M., & Van der Linden, M. (2009). Inhibitory control of memory in normal ageing: Dissociation between impaired intentional and preserved unintentional processes. *Memory, 17,* 104–122. doi:10.1080/09658210802574146

Damoiseaux, J. S., Beckmann, C. F., Sanz Arigita, E. J., Barkhof, F., Scheltens, Ph., Stam, C. J., . . . Rombouts, S. A. R. B. (2008). Reduced resting-state brain activity in the "default network" in normal aging. *Cerebral Cortex, 18,* 1856–1864. doi:10.1093/cercor/bhm207

Eimer, M. (1999). Facilitatory and inhibitory effects of masked prime stimuli on motor activation and behavioral performance. *Acta Psychologica, 101,* 293–313. doi:10.1016/S0001-6918(99)00009–8

Eimer, M., & Schlaghecken, F. (1998). Effects of masked stimuli on motor activation: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance, 24,* 1737–1747. doi:10.1037/0096–1523.24.6.1737

Eimer, M., & Schlaghecken, F. (2002). Links between conscious awareness and response inhibition: Evidence from masked priming. *Psychonomic Bulletin & Review, 9,* 514–520. doi:10.3758/BF03196307

Eimer, M., & Schlaghecken, F. (2003). Response facilitation and inhibition in subliminal priming. *Biological Psychology, 64,* 7–26. doi:10.1016/S0301-0511(03)00100–5

Faust, M. E., & Balota, D. A. (1997). Inhibition of return and visuospatial attention in healthy older adults and individuals with dementia of the Alzheimer type. *Neuropsychology, 11,* 13–29. doi:10.1037/0894–4105.11.1.13

Faw, B. (2003). Pre-frontal executive committee for perception, working memory, attention, long-term memory, motor control, and thinking: A tutorial review. *Consciousness & Cognition, 12,* 83–139. doi:10.1016/S1053-8100(02)00030–2

Gamboz, N., Russo, R., & Fox, E. (2002). Age differences and the identity negative priming effect: An updated meta-analysis. *Psychology and Aging, 17,* 525–531. doi:10.1037/0882–7974.17.3.525

Gazzaley, A., Clapp, W., Kelley, J., McEvoy, K., Knight, R. T., & D'Esposito, M. (2008). Age-related top-down suppression deficit in the early stages of cortical visual memory processing. *Proceedings of the*

National Academy of Sciences, USA, 105, 13122–13126. doi:10.1073/pnas.0806074105

Good, C. D., Johnsrude, I. S., Ashburner, J., Henson, R. N., Friston, K. J., & Frackowiak, R. S. (2001). A voxel-based morphometric study of ageing in 465 normal adult human brains, *Neuroimage, 14,* 21–36. doi:10.1006/nimg.2001.0786

Hartley, A. A., & Kieley, J. M. (1995). Adult age differences in the inhibition of return of visual attention. *Psychology and Aging, 10,* 670–683. doi:10.1037/0882–7974.10.4.670

Hasher, L., Zacks, R. T., & May, C. P. (1999). Inhibitory control, circadian arousal, and age. In D. Gopher & A. Koriat (Eds.), *Attention and performance XVII* (pp. 653–675). Cambridge, MA: MIT Press.

Heim, A. W. (1968). *AH4 Test.* Windsor, UK: NFER-NELSON.

Hughes, G., Velmans, M., & De Fockert, J. (2009). Unconscious priming of a no-go response. *Psychophysiology, 46,* 1258–1269. doi:10.1111/j.1469–8986.2009.00873.x

Hultsch, D. F., Strauss, E., Hunter, M. A., & MacDonald, S. W. S. (2008). Intraindividual variability, cognition, and aging. In F. I. M. Craik & T. A. Salthouse (Eds.), *The handbook of aging and cognition* (3rd ed., pp. 491–556). New York: Psychology Press.

Jaśkowski, P. (2008). The negative compatibility effect with nonmasking flankers: A case for mask-triggered inhibition hypothesis. *Consciousness and Cognition, 17,* 765–777. doi:10.1016/j.concog.2007.12.002

Jost, K., Bryck, R. L., Vogel, E. K., & Mayr, U. (2010). Are old adults just like low working memory young adults? Filtering efficiency and age differences in visual working memory. *Cerebral Cortex.* doi:10.1093/cercor/bhq185

Klapp, S. T., & Hinkley, L. B. (2002). The negative compatibility effect: Unconscious inhibition influences reaction time and response selection. *Journal of Experimental Psychology: General, 131,* 255–269. doi:10.1037/0096–3445.131.2.255

Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences, 4,* 138–147. doi:10.1016/S1364-6613(00)01452–2

Kramer, A. F., Humphrey, D. G., Larish, J. F., Logan, G. D., & Strayer, D. L. (1994). Aging and inhibition: Beyond a unitary view of inhibitory processing in attention. *Psychology and Aging, 9,* 491–512. doi:10.1037/0882–7974.9.4.491

Lamme, V. A. F. (2004). Separate neural definitions of visual consciousness and visual attention; a case for phenomenal awareness. *Neural Networks, 17,* 861–872. doi:10.1016/j.neunet.2004.02.005

Lamme, V. A. F., & Roelfsema, P. R. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neurosciences, 23,* 571–579. doi:10.1016/S0166-2236(00)01657-X

Langley, L. K., Fuentes, L. J., Vivas, A. B., & Saville, A. L. (2007). Aging and temporal patterns of inhibition of return. *Journal of Gerontology: Psychological Sciences, 62*B, P71–P77.

Mansouri, F. A., Tanaka, K., & Buckley, M. J. (2009). Conflict-induced behavioural adjustment: A clue to the executive functions of the prefrontal cortex. *Nature Reviews Neuroscience, 10,* 141–152. doi:10.1038/nrn2538

Mattay, V. S., Fera, F., Tessitore, A., Hariri, A. R., Das, S., Callicott, J. H., & Weinberger, D. R. (2002). Neurophysiological correlates of age-related changes in human motor function. *Neurology, 58,* 630–635.

Maylor, E. A., & Henson, R. N. A. (2000). Aging and the Ranschburg effect: No evidence of reduced response suppression in old age. *Psychology and Aging, 15,* 657–670. doi:10.1037/0882–7974.15.4.657

Maylor, E. A., Schlaghecken, F., & Watson, D. G. (2005). Aging and inhibitory processes in memory, attentional, and motor tasks. In R. W. Engle, G. Sedek, U. von Hecker, & D. N. McIntosh (Eds.), *Cognitive limitations in aging and psychopathology* (pp. 313–345). New York: Cambridge University Press. doi:10.1017/CBO9780511720413.013

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126,* 220–246. doi:10.1037/0033–2909.126.2.220

Raven, J. C., Raven, J., & Court, J. H. (1988). *The Mill Hill vocabulary scale.* London: H. K. Lewis Co. Ltd.

Resnick, S. M., Pham, D. L., Kraut, M. A., Zonderman, A. B., & Davatzikos, C. (2003). Longitudinal magnetic resonance imaging studies of older adults: A shrinking brain. *Journal of Neuroscience, 23,* 3295–3301.

Salat, D. H., Buckner, R. L., Snyder, A. Z., Greve, D. N., Desikan, R. S., Busa, E., . . . Fischl, B. (2004). Thinning of the cerebral cortex in aging. *Cerebral Cortex, 14,* 721–730. doi:10.1093/cercor/bhh032

Salthouse, T. A. (1991). *Theoretical perspectives on cognitive aging.* Hillsdale, NJ: Erlbaum.

Salthouse, T. A. (1996). The processing-speed theory of adult age differences in cognition. *Psychological Review, 103,* 403–428. doi:10.1037/0033-295X.103.3.403

Salthouse, T. A. (2004). What and when of cognitive aging. *Current Directions in Psychological Science, 13,* 140–144. doi:10.1111/j.0963–7214.2004.00293.x

Sapir, A., Soroker, N., Berger, A., & Henik, A. (1999). Inhibition of return in spatial attention: Direct evidence for collicular generation. *Nature Neuroscience, 2,* 1053–1054. doi:10.1038/15977

Schlaghecken, F., Blagrove, E., & Maylor, E. A. (2008). No difference between conscious and nonconscious visuomotor control: Evidence from perceptual learning in the masked prime task. *Consciousness and Cognition, 17,* 84–93. doi:10.1016/j.concog.2006.11.004

Schlaghecken, F., Bowman, H., & Eimer, M. (2006). Dissociating local and global levels of perceptuo-motor control in masked priming. *Journal of Experimental Psychology: Human Perception and Performance, 32,* 618–632. doi:10.1037/0096–1523.32.3.618

Schlaghecken, F., & Eimer, M. (1997). The influence of subliminally presented primes on response preparation. *Sprache & Kognition, 16,* 166–175.

Schlaghecken, F., & Eimer, M. (2000). A central/peripheral asymmetry in subliminal priming. *Perception & Psychophysics, 62,* 1367–1382. doi:10.3758/BF03212139

Schlaghecken, F., & Eimer, M. (2002). Motor activation with and without inhibition: Evidence for a threshold mechanism in motor control. *Perception & Psychophysics, 64,* 148–162. doi:10.3758/BF03194564

Schlaghecken, F., & Eimer, M. (2006). Active masks and active inhibition: A comment on Lleras and Enns (2004) and on Verleger, Jáskowski, Aydemir, van der Lubbe, and Groen (2004). *Journal of Experimental Psychology: General, 135,* 484–494. doi:10.1037/0096–3445.135.3.484

Schlaghecken, F., Klapp, S. T., & Maylor, E. A. (2009). Either or neither, but not both: Locating the effects of masked primes. *Proceedings of the Royal Society B, 276,* 515–521. doi:10.1098/rspb.2008.0933

Schlaghecken, F., & Maylor, E. A. (2005). Motor control in old age: Evidence of impaired low-level inhibition. *Journal of Gerontology: Psychological Sciences, 60*B, P158–P161.

Schlaghecken, F., Münchau, A., Bloem, B. R., Rothwell, J., & Eimer, M. (2003). Slow frequency repetitive transcranial magnetic stimulation affects reaction times, but not priming effects, in a masked prime task. *Clinical Neurophysiology, 114,* 1272–1277. doi:10.1016/S1388-2457(03)00118–4

Schlaghecken, F., Rowley, L., Sembi, S., Simmons, R., & Whitcomb, D. (2007). The negative compatibility effect: A case for inhibition. *Advances in Cognitive Psychology, 3,* 227–240. doi:10.2478/v10053-008–0027-y

Schlaghecken, F., & Sisman, R. (2006). Low-level motor inhibition in children: Evidence from the negative compatibility effect. *Advances in Cognitive Psychology, 2,* 7–19. doi:10.2478/v10053-008–0041-0

Schneider, B. A., & Pichora-Fuller, M. K. (2000). Implications of perceptual deterioration for cognitive aging research. In F. I. M. Craik & T. A.

Salthouse (Eds.), *The handbook of aging and cognition* (2nd ed., pp. 155–219). Mahwah, NJ: Erlbaum.

Schneider, J. (2002). *Near vision test card.* Retrieved November 16, 2007, from http://www.i-see.org/block_letter_eye_chart.pdf

Seidler, R. D., Bernard, J. A., Burutolu, T. B., Fling, B. W., Gordon, M. T., Gwin, J. T., . . . Lipps, D. B. (2010). Motor control and aging: Links to age-related brain structural, functional, and biochemical effects. *Neuroscience and Biobehavioral Reviews, 34,* 721–733. doi:10.1016/j.neubiorev.2009.10.005

Seiss, E., & Praamstra, P. (2004). The basal ganglia and inhibitory mechanisms in response selection: Evidence from subliminal priming of motor responses in Parkinson's disease. *Brain, 127,* 330–339. doi:10.1093/brain/awh043

Shadlen, M. N., & Newsome, W. T. (2001). Neural basis of a perceptual decision in the parietal cortex (area LIP) of the rhesus monkey. *Journal of Neurophysiology, 86,* 1916–1936.

Shipp, S. (2004). The brain circuitry of attention. *Trends in Cognitive Sciences, 8,* 223–230. doi:10.1016/j.tics.2004.03.004

Sumner, P., & Brandwood, T. (2008). Oscillations in motor priming: Positive rebound follows the inhibitory phase in the masked prime paradigm. *Journal of Motor Behavior, 40,* 484–489. doi:10.3200/JMBR.40.6.484–490

Sumner, P., Nachev, P., Morris, P., Peters, A. M., Jackson, S. R., Kennard, C., & Husain, M. (2007). Human medial frontal cortex mediates unconscious inhibition of voluntary action. *Neuron, 54,* 697–711. doi:10.1016/j.neuron.2007.05.016

Tipper, C., & Kingstone, A. (2005). Is inhibition of return a reflexive effect? *Cognition, 97,* B55–B62. doi:10.1016/j.cognition.2005.02.003

Vallesi, A., & Stuss, D. T. (2010). Excessive sub-threshold motor preparation for non-target stimuli in normal aging. *Neuroimage, 50,* 1251–1257. doi:10.1016/j.neuroimage.2010.01.022

van Gaal, S., Ridderinkhof, K. R., van den Wildenberg, W. P. M., & Lamme, V. A. F. (2009). Dissociating consciousness from inhibitory control: Evidence for unconsciously triggered response inhibition in the stop-signal task. *Journal of Experimental Psychology: Human Perception and Performance, 35,* 1129–1139. doi:10.1037/a0013551

Verhaeghen, P., & De Meersman, L. (1998). Aging and the negative priming effect: A meta-analysis. *Psychology and Aging, 13,* 435–444. doi:10.1037/0882–7974.13.3.435

Wechsler, D. (1981). *Manual for the Wechsler Adult Intelligence Scale-Revised.* New York: Psychological Corporation.

Wu, T., & Hallett, M. (2005). The influence of normal human ageing on automatic movements. *Journal of Physiology, 562,* 605–615. doi:10.1113/jphysiol.2004.076042

Received August 4, 2010
Revision received February 18, 2011
Accepted February 23, 2011 ■

Correction to Schlaghecken, Birak, and Maylor (2011)

The article "Age-related deficits in low-level inhibitory motor control," by Friederike Schlaghecken, Kulbir S. Birak, and Elizabeth A. Maylor (*Psychology and Aging*, Vol. 26, No. 4, 905–918) reported that, whereas conventional analysis of data from the masked prime paradigm suggested intact low-level motor activation but absent low-level motor inhibition in old age, analysis of each young and older adult's time course of masked priming effects revealed evidence of inhibition (albeit much delayed) in the majority of older adults. However, we have since discovered that the method proposed for individually extracting priming effects from time course analysis may lead to some spurious effects. The ranking procedure involved ensures that each latency bin is not independent of those preceding it and hence can produce differences at several latency bins even for random sets of numbers. Moreover, a non-negligible proportion of these may be "significant" despite Bonferroni adjustment.

To gain insight into possible implications for Schlaghecken et al.'s (2011) data, we repeated the analysis with randomized data. First, we combined all the prime-locked RTs from compatible and incompatible trials from the 22 older adults in Experiment 1 into a single pool of 12,672 ($2 \times 22 \times 288$) RTs. Second, we randomly selected 576 RTs from this pool and arbitrarily assigned 288 to a "compatible" condition and the remaining 288 to an "incompatible" condition. Third, the RTs were analyzed as described on page 910 of the article, that is, they were ranked from the fastest to the slowest in each condition, split into 16 bins with 18 trials per bin, and then compatible and incompatible RTs were compared for each latency bin using unpaired *t*-tests, applying a Bonferroni correction to the critical *p*-value ($<.003125$). Fourth, this was repeated 100 times to simulate 100 older participants.

Mean "priming" effects were small in absolute magnitude (<3 ms) at all latency bins except for the 16th bin ($M = 5.6$ ms), which produced highly variable priming effects that almost never reached significance. Crucially, even though "compatible" and "incompatible" RTs were sampled from the same distribution, there were surprisingly large numbers of significant priming effects, with an average of 7.6 latency bins per simulation producing a significant positive or negative compatibility effect (PCE/NCE). Only one "participant" out of 100 produced no significant priming effect at any latency bin.

Considering all 16 latency bins, 65% of the simulated participants produced at least one significant NCE, with a mean peak magnitude of −28 ms ($SD = 12.6$). This is the same peak magnitude as observed in the 18/22 (81.8%) older adults in Schlaghecken et al.'s (2011) Experiment 1 who produced at least one significant NCE. However, in the actual data, there were highly reliable PCEs in the early latency bins for older adults and so it is perhaps more appropriate to focus only on the later latency bins (8-16) in the simulation. This resulted in 58% showing at least one significant NCE, with a mean peak magnitude of −25 ms ($SD = 11.6$). Thus it appears that spurious and substantial NCEs can be identified in a majority of simulated participants.

Schlaghecken et al. (2011) also applied a stricter criterion that required significant NCEs in at least two successive latency bins, which led to 15/22 (68.2%) older adults showing NCEs. Applying this criterion to latency bins 8-16 in the simulation resulted in NCEs in 45% of cases, with a mean peak magnitude of −28 ms ($SD = 11.6$). Note that for both criteria, approximately five more older adults produced NCEs than would be predicted on the basis of these simulations (i.e., 18 rather than 12.8 produced at least one significant NCE; 15 rather than 9.9 produced consecutive significant NCEs). Therefore it would be premature to rule out the possibility that at least some older participants produced genuine NCEs.

In view of possible spurious effects from our application of time course analysis, we therefore adopted an alternative strategy that retains our attempt to take an individual approach to identifying NCEs in older participants who may vary more than young participants in terms of the prime-target SOA at which NCEs initially appear. Thus, for each participant, correct RTs for compatible and

incompatible trials were compared using unpaired *t*-tests separately for each of the four SOAs of Schlaghecken et al.'s (2011) Experiment 1. On this basis, nine of the 22 older participants produced a significant NCE ($M = -33$ ms), with four first showing a significant NCE at the 150-ms SOA, four first showing a significant NCE at the 300-ms SOA, and one first showing a significant NCE at the 450-ms SOA. In contrast, 12 of the 19 young participants produced a significant NCE ($M = -28$ ms), with 11 first showing a significant NCE at the 150-ms SOA, and one first showing a significant NCE at the 300-ms SOA. Thus an NCE was reliably present in a sizeable minority of older participants but its appearance was somewhat delayed relative to that seen in the majority of young participants. We therefore conclude that NCEs in the masked prime paradigm, as indicative of low-level inhibitory motor control, are neither entirely absent in older adults (as suggested by the original conventional analysis) nor present to almost the same extent as in young adults (as suggested by the time course analysis).

Finally, it is important to point out that time course analysis has been employed successfully elsewhere to explore a number of phenomena such as Stroop and Simon effects (see Houghton & Grange, 2011, for examples). The method itself as used to characterize the unfolding of effects over time is not the issue here; rather, it is the search within each individual's data for latency bins showing significant effects using *t*-tests (even Bonferroni adjusted) that is questionable.

Houghton, G., & Grange, J. A. (2011). CDF-XL: Computing cumulative distribution functions of reaction time data in Excel. *Behavior Research Methods, 43,* 1023–1032. doi:10.3758/s13428-011-0119-3

DOI: 10.1037/a0028835