Aging and the Negative Priming Effect: A Meta-Analysis

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This article reports results from a meta-analysis on adult age differences in the negative priming effect (31 studies on identity negative priming and 8 on location negative priming). Both younger and older adults were found to be susceptible to the negative priming effect in identity and location tasks. Effect sizes were homogeneous for both tasks, indicating that the data are adequately described without reference to moderator variables. State trace analysis on identity tasks, in which mean latencies in negative priming conditions were regressed onto mean latencies in baseline conditions, showed (a) that in both age groups the negative priming effect is proportional rather than additive and (b) that the negative priming effect is smaller in older adults as compared with younger adults.

The term priming refers to the phenomenon that previous processing of a stimulus may have an impact on subsequent processing of the same stimulus or of related stimuli. Positive priming implies that having processed a stimulus before makes subsequent processing more fluent, resulting in shorter latency, lower error rates, or both, for a task. Negative priming refers to costs associated with previous processing of a stimulus. This phenomenon occurs when research participants are asked to provide some response to a given stimulus (the target), while at the same time ignoring an irrelevant stimulus (the distractor). If the distractor on one trial (the prime trial) becomes the target on the next trial (the test trial), performance is usually hampered. For example, one might present a research participant with two overlapping pictures of objects, one printed in red, the other in green. The participant is asked to name the object depicted in red, while ignoring the object in green. If the object depicted in red on a given trial is identical to the object depicted in green on the previous trial, naming latency is typically increased. A score for negative priming is derived by subtracting reaction time on test trials in a condition in which the target was not presented on the prime trial (a baseline condition).

The dominant explanation for the negative priming effect is that it is indicative of inhibitory attentional processes. What presumably happens is that an attentional mechanism blocks the representation of the distractor stimulus on the prime trial from access to the response systems (e.g., Hasher & Zacks, 1988; McDowd, Oesers-Kreger, & Flijon, 1995; Tipper, 1985). If that same stimulus is subsequently presented as a target on the test trial, additional processing time is required to overcome the response inhibition generated on the previous trial, resulting in longer latency for the response and sometimes in errors. This active inhibitory mechanism is extremely important for the emergence of coherent thought and behavior because it allows for selective attention by keeping activation of irrelevant stimuli low. In this sense, inhibitory mechanisms play an important role in human information processing.

The central role of inhibition in human information processing suggests that a breakdown in inhibition may have widespread effects on a large number of tasks. This observation has led some researchers to postulate that a deficit in inhibition may be the basic mechanism underlying the age differences observed in many tasks of fluid cognition (e.g., Hasher & Zacks, 1988). There is evidence that a large amount of the age-related variance among measures is indeed shared. Mean latencies of older adults, for instance, can be quite well predicted from mean latencies of younger adults, and this happens across a wide range of task complexity (for an overview, see Cerella, 1990, and Myerson & Hale, 1993). This suggests that the same age-related slowing factor is present in simple as in more complex tasks. Mediation analyses show that age differences in complex cognitive tasks such as tests for inductive reasoning, spatial performance, and recall from episodic memory are largely mediated through rather simple measures of perceptual speed and through working memory measures (for a meta-analysis, see Verhaeghen & Saltzhouse, 1997). These results imply that cognitive aging may be a more or less general process, largely caused by deficits that are situated early in the cognitive system. Hasher and Zacks (1988) have argued that a deficit in the inhibition mechanism may be a good candidate mechanism for a general cause of cognitive aging. According to their theory, a breakdown...
inhibitory processes would allow irrelevant elements to enter into working memory, thus effectively decreasing working memory efficiency, capacity, or both, and slowing down all processing.

It should be noted here, however, that problems have arisen over this view of inhibition as a cause for general age deficits. First, the concept of a global age-related deficit in inhibition has recently been challenged in two comprehensive literature reviews (Burke, 1997; McDowd, 1997). Burke has argued that, contrary to what the inhibition account of aging predicts, there are remarkable age constancies in the literature on aging and language and that evidence of an inhibitory deficit in conditions that do produce age deficits is “controversial at best” (Burke, 1997, p. 254). McDowd notes inconsistencies and improvements in the inhibition theory, such as difficulties with “identifying and reliable measures of inhibitory processes;” and “inadequately specified theory from which to generate predictions” (McDowd, 1997, p. 372). Second, correlations between different aspects of inhibition appear to be weak, and not all measures show a breakdown with advancing adult age (Kramer, Humphrey, Laish, Logan, & Strayer, 1994). Whereas stopping an overt response and adopting new rules in a categorization task were indeed age-related tasks, negative priming effects, response compatibility effects, spatial prepotent effects, and self-reported cognitive failures were found to be age invariant. Third, a meta-analysis of age differences in the Stroop interference effect (Vethaeghen & De Meersman, 1998), which presumably reflects inhibitory processing, showed that no adult age difference was present in the Stroop effect once general slowing had been taken into account. A meta-analysis of adult age difference in the negative priming effect—probably the favorite paradigm for measuring inhibition (e.g., Neill, Valdez, & Terry, 1995; Tipper, 1985)—may be helpful in the current debate.

The goal of the present research then was to examine the claim that inhibition is indeed defective by conducting a meta-analysis of existing studies on aging and the negative priming effect. Data on identity and location negative priming were examined separately. Identity priming refers to negative priming in tasks in which the response is related to the identity of a stimulus (e.g., naming a pictured object or reading a letter aloud); location priming refers to negative priming in tasks in which the response is related to the location of a stimulus (e.g., pressing a key on the keyboard corresponding to the location of a particular stimulus). The reason for analyzing these two types of responses separately is that there is accumulating evidence that identity and location negative priming may reflect different mechanisms associated with different pathways in the brain (Connelly & Hasher, 1993; May, Kanc, & Hasher, 1995; McDowd, 1994). More precisely, it has been claimed that identity negative priming should be vulnerable to the effects of aging, whereas location negative priming may stay intact (May et al., 1995).

If later adulthood is indeed characterized by a deficit in (identity) inhibitory atacausal mechanisms, older adults should be less hampered when responding to a target that was a distractor on the previous trial than younger adults. Consequently, one should expect the negative priming effect to be smaller in older adults as compared to younger adults. If there were a complete breakdown of inhibitory mechanisms in later adulthood, one might expect older adults not to be slowed at all in negative priming conditions. A partial breakdown of inhibition would manifest itself by less slowing in negative priming conditions in older adults as compared to younger adults. It is interesting to note that in practice reviewers seem to have concentrated on the presence or absence of negative priming effects in older adults, rather than on the size of age differences in the negative priming effect (e.g., May et al., 1995; McDowd et al., 1995). Also, reviewers often focus on identifying conditions in which older adults are particularly sensitive (or not) to the negative priming effect, thus pointing at moderator variables for the negative priming effect in later adulthood. Some of the moderator variables mentioned in the literature include the preparatory interval (McDowd & Filion, 1995), pacing of the task (McDowd et al., 1995), response-stimulus interval (May et al., 1995), exposure duration (Kane, Hasher, Stoltzfus, Zacks, & Connely, 1994), and degree of overlap of stimuli (McDowd, 1994). The presence or absence of effects of such moderator variables is then taken as evidence for or against theories about specific effects of aging on priming or as evidence for or against specific mechanisms of negative priming.

There is an inherent danger in such an endeavor, however, because the inferred presence or absence of a negative priming effect in a particular study or a particular condition of a study is dependent on the result of a significance test, which is a function of both the size of the negative priming effect and sample size. If the effect is small and sample size insufficient, problems with statistical power may arise that can lead to Type II errors. A meta-analysis, which looks at the size of the effect regardless of its significance in primary studies, might then provide valuable information over and above the information typically reported in narrative reviews.

It should be noted that there is an additional problem with the interpretation of the results found in most aging studies concerning the negative priming effect, and this has to do with the way the effect is traditionally measured. Usually, a priming score is calculated by subtracting reaction time in the negative priming condition from reaction time in the baseline condition. If this difference score is reliably smaller in older than in younger adults (or if the Age × Condition interaction in an analysis of variance [ANOVA] is significant), researchers conclude that the inhibition effect is smaller in older adults. Interestingly, in the light of general slowing theories, this method may well be unnecessarily conservative. General slowing theories state that latencies of older adults can be described as a linear function of latencies of younger adults, with a slope larger than unity. This slope reflects the older over younger slowing ratio in central computational processes (Cerella, 1990). Such theories have been found to fit the available meta-analytic data very well (Cerella, 1990; Myerson, Hale, Wagensstaf, Poon, & Smith, 1990). A corollary of this theory is that whenever absolute age differences across two conditions are compared (such as the age difference in a baseline condition versus the age difference in a negative priming condition), the absolute age difference can be expected to be larger in the condition yielding the longest average latency (in the present case, the negative priming condition). Consequently, the traditional null hypothesis in interaction analysis in ANOVA, namely that there is an expected zero change in the age difference between baseline and negative priming
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conditions, is unduly conservative. In fact, if the age difference is truly constant across baseline and negative priming conditions, this would constitute positive evidence for an inhibition deficit in older adults. Power problems aside, finding no interaction in an Age \( \times \) Condition analysis should clearly not be taken at face value as evidence for age constancy in negative priming. The present meta-analysis overcomes this problem by explicitly examining age differences in negative priming while taking general slowing effects into account.

In the present meta-analysis, then, we wanted to bring together the available data on the age effect in negative priming and to examine whether age differences are truly smaller in negative priming conditions as compared with baseline conditions, as claimed by the inhibition account of cognitive aging. This hypothesis was investigated using two methods, analogous to the methods used in our meta-analysis on aging and the Stroop effect (Verhaeghen & De Mesquita, 1998). First, traditional methods of data pooling (Hedges & Olkin, 1985) were used to calculate the negative priming effect (expressed as the mean standardized difference between the baseline and the negative priming condition) for both younger and older adults. We then examined whether this negative priming effect was smaller for older than younger adults, as predicted by inhibition theory.

Second, data were subjected to a Brinley analysis (Salthouse, 1978, 1985), that is, we examined whether a single (general slowing) or two different (inhibition theory) curves were needed to describe the relation between the performance of younger and older adults in the baseline and the negative priming conditions.

Method

Sample of Studies

Studies were collected by consulting the American Psychological Association's PsycLIT electronic database, through personal contacts, and by checking references found in the articles thus retrieved. Because negative priming effects were found to be quite small and published studies few, we decided to include unpublished studies. Therefore, abstracts from the 1994 and 1996 Cognitive Aging Conferences (Atlanta, GA) - the largest scientific meetings in the field - were searched for negative priming studies. Authors of papers or poster presentations from these meetings were contacted, and some were kind enough to provide us with their (as yet) unpublished data. The search was concluded in June 1997.

Criteria for inclusion were (a) the study examined at least one sample of younger (mean age 30 years or younger) and older adults (mean age 60 years or older); (b) negative priming data were reported, including at least a baseline condition and a negative priming condition, as described in the introduction; and (c) the data were reported in a format amenable to meta-analysis. No study was excluded for reasons other than not satisfying these criteria. Thus, to the best of our knowledge, this sample comprises the totality of the published and some of the unpublished literature on aging effects in negative priming. Studies, along with some of their characteristics, are listed in Table 1.

Data Pooling

The system for data pooling advocated by Hedges and Olkin (1985) was used for the first type of analysis. We calculated the negative priming effect for each age group (younger and older adults) for each study separately by subtracting the negative priming condition latency from the baseline condition latency and dividing this score by the pooled standard deviation, yielding the mean standardized difference, or \( g \). (see also Dunlap, Cortina, Vaslow, & Burke, 1996). \(^{1}\)

Whenever means and standard deviations were not reported in the original article or provided by the authors, the appropriate inferential statistics as advocated by Dunlap et al. (1996) were used. Correct calculation of effect sizes from inferential statistics requires an estimate of the correlation between baseline and negative priming conditions. We never found this correlation reported in the unpublished manuscripts and articles we examined. Rather than pooling all authors, we contacted the primary author of the study containing the largest sample (Pades et al., 1997 - N = 215; note that this sample size is much larger than the average sample size - 55 - and about 2.6 times as large as the size of the second-largest sample). The correlation in this study was .92. Consequently, this estimate was used in calculating effect sizes from inferential statistics. Note that it is hard to imagine that the true average would be much larger than .92. If the true correlation were smaller, the present procedure would lead to underestimates rather than overestimates of the effect size in studies for which only inferential statistics are available.

One possible effect of including potentially inaccurate effect sizes estimates is that the estimated homogeneity of the average effect size will be larger than the true homogeneity statistic (i.e., Q, if all estimates would have been exact). Note that the potential effect of misestimates due to not knowing the true correlation are equally distributed across age groups. The instances in which precise estimates were available for one age group and not the other never occurred.

A small sample correction factor was applied to the individual effect sizes in accordance with the principles outlined by Hedges and Olkin (1985, p. 81), converting the \( g \) values to \( d \). These individual effect sizes, one for each study, were then averaged using a weighting factor for sample size (Hedges & Olkin, pp. 109-117), to yield estimates of the average identity and location negative priming effect (\( d_ne \)) in younger and older adults, respectively.

One advantage of the Hedges and Olkin (1985) approach is that a test statistic is available for between-group comparisons. In the present study, this statistic (the between-group homogeneity statistic, \( Q_g \), which is chi-square distributed with degrees of freedom equal to the number of groups minus 1; Hedges & Olkin, 1985, pp. 154-155) was used to test whether the negative priming effect was smaller in older as compared to younger adults. Moreover, Hedges and Olkin (pp. 155-156) propose a within group estimate of homogeneity (\( Q_w \), chi-square distributed with the number of studies minus 1 as number of degrees of freedom), indicating whether the studies can be considered to be homogeneous, that is, whether the effect size can be considered a good point estimate of a single population value. If the data are not homogeneous, an analysis of moderator variables is called for.

Brinley Analysis

For the Brinley analysis, the mean latency data of older adults were regressed onto the mean latency data of younger adults. Because of the small number of studies on location priming, Brinley analysis was conducted for identity priming only. Mean latency data were expressed in seconds needed for responding to a single stimulus. Two popular general slowing models were applied to the data.

The first model applied is the multilayered slowing model advanced by Ceco (1990). In this model, it is stated that aging brings about differential slowing in peripheral processes (i.e., input and output pro-

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\(^{1}\) Exact means and standard deviations are needed for the calculation of effect sizes in a within-subject design (Dunlap et al., 1996). If these were not provided in the article, the authors of the individual studies were contacted. If the requested information could not be obtained, inferential statistics were used to calculate effect sizes.
Table 1
Sample of Studies, Along With Effect Sizes for Negative Priming in Younger and Older Adults

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>n</th>
<th>Baseline latency (ms)</th>
<th>Negative priming latency (ms)</th>
<th>Negative priming effect size (d)</th>
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<td>Younger</td>
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<td>624</td>
<td>666</td>
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<td>514</td>
<td>500</td>
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<td>Hasher, Stoltzfus, Zacks, &amp; Reppu (1991, Exp. 1)</td>
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<td>560</td>
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<td>Kane, May, Hasher, Rabal, &amp; Stoltzfus (1997)</td>
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<td>Kramer, Humphrey, Lapish, Logan, &amp; Stiner (1994)</td>
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<td>Tipper (1991)</td>
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Location negative priming

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<th>Baseline latency (ms)</th>
<th>Negative priming latency (ms)</th>
<th>Negative priming effect size (d)</th>
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Note: Exp. = experiment.

*For letter stimuli only.*  
*Because SD was not reported, exact d could not be computed; inferential statistics were used instead.*  
*Identity condition; 0 ms stimulus onset asynchrony only.*

cessors) and control processes. Cecelia demonstrated that such a model yields young-old data that can be described by a linear function:

\[ RT_{\text{older}} = a + b \cdot RT_{\text{younger}} \]

in which RT denotes reaction time and the b parameter, or the slope of the function, describes the ratio of older over younger central processing time and thus provides an index of age-related slowing in central processing.

In order to test whether different equations are needed for the two conditions in the negative priming data, an interaction analysis approach was used (Barry & Feilman, 1985; for a brief tutorial on using this technique in the context of young-old Brinley plots, see Myerson, Waggstaff, & Hale, 1994). In this method, the data are fitted to the following regression equation:

\[ RT_{\text{older}} = a + b \cdot RT_{\text{younger}} + \alpha \cdot Cond + b \cdot Cond \times RT_{\text{younger}} \]

The Cond variable is a dummy variable, taking the value 0 in the baseline condition and the value 1 in the negative priming condition. If the \( a \) parameter is significant, different intercepts, one for each condition, are needed to describe the data adequately; if the \( b \) parameter is significant, different slopes, one for each condition, are needed for adequate description of the data. All equations were fitted using the Statistical Package for the Social Science (SPSS Inc., 1996) weighted least squares algorithm, weighting for sample size. The reader may note that this theoretically guided use of Brinley analysis is quite different from the exploratory use of the technique that has recently generated much controversy (e.g., Fisk & Fisher, 1994; Perfect, 1994).

The second general slowing model used to fit the data was the information loss model by Myerson et al. (1990). This model assumes that a constant proportion of information is lost during each processing step and that the amount of information lost is larger in older than in younger adults. This model is described by the equation

\[ RT_{\text{older}} = b \times RT_{\text{younger}} \]

The \( m \) term in this equation describes the ratio of the older over younger decay rates, that is, it gives the proportional age difference in the speed with which information is lost when propagated through the cognitive system. This equation was fitted using the SPSS (1996) nonlinear regression procedure, weighting for sample size.

Significance level of all statistical tests was set at .05.
Results

Data Pooling

Effect sizes for the individual studies are reported in Table 1. In order to test for the assumption that identity and location negative priming are indeed separate constructs, we conducted a within-age-group-between-negative priming-type analysis. Because the Hedges and Olkin (1985) method calls for a comparison between independent effect sizes, the three studies that examined both identity and location negative priming in a within-subject design were discarded from the analysis, leaving us with 18 studies on identity negative priming and 5 studies on location negative priming. Effect sizes for younger adults were 0.26 (limits of the 95% confidence interval were 0.13 and 0.38) for identity negative priming and 0.61 for location negative priming (limits of the 95% confidence interval were 0.31 and 0.90). For older adults, effect sizes were 0.11 (limits of the 95% confidence interval were -0.00 and 0.22) for identity negative priming and 0.27 for location negative priming (limits of the 95% confidence interval were 0.01 and 0.54). The between-group homogeneity statistic reached statistical significance in younger adults ($Q_h = 4.60$), but not in older adults ($Q_h = 1.26$). This result shows that in younger adults, effect sizes for location priming are indeed reliably larger than for identity priming. Consequently, it seems wise to consider both types of priming as separate constructs.

The average effect in the 21 identity negative priming studies was 0.27 for the younger adults (limits of the 95% confidence interval were 0.15 and 0.39) and 0.13 for older adults (limits of the 95% confidence interval were 0.02 and 0.23). Thus, the identity negative priming effect was quite small, especially in older adults. However, the effect was significantly different from zero for both age groups. The effect size did not differ reliably across groups, as indexed by nonsignificant between-group homogeneity, $Q_h(1) = 3.11$, n.s., indicating that the negative priming effect was as large in older adults as in younger adults. Note, however, that the probability associated with the $Q_h$ statistic was quite low ($p < .10$). Both effect sizes were homogeneous, as indicated by nonsignificant within-group homogeneity statistics, $Q_w(20) = 5.96$ and 10.36, for younger and older adults, respectively. This within-group homogeneity indicates that the effect sizes can be interpreted as good point estimates of a single effect size for each age group. Thus, the identity negative priming data are adequately described without reference to moderating variables. Consequently, no moderator analysis was undertaken.

The average effect in the 8 location negative priming studies was 0.38 for the younger adults (limits of the 95% confidence interval were 0.16 and 0.61) and 0.24 for the older adults (limits of the 95% confidence interval were 0.03 and 0.45). Thus, the location negative priming effect was significantly different from zero for both age groups. The effect size did not differ reliably across groups, as indexed by nonsignificant between-group homogeneity, $Q_h(1) = 0.77$ (this statistic did not approach significance, $p > .50$), indicating that the negative priming effect was as large in older adults as in younger adults. Both effect sizes were homogeneous, as indicated by nonsignificant within-group homogeneity statistics, $Q_w(7) = 8.55$ and 1.41, for younger and older adults, respectively. This within-group homogeneity indicates that the effect sizes can be interpreted as good point estimates of a single effect size for each age group. Thus, the location negative priming data are adequately described without reference to moderating variables. Consequently, no moderator analysis was undertaken.

Brinley Analyses on Identity Tasks

A Brinley plot of the young-old data for identity negative priming is provided in the left-hand panel of Figure 1. Mean

![Figure 1](image-url)
latency (weighted for sample size) in the baseline condition was 558.2 ms for younger adults and 643.2 ms for older adults; weighted mean latency for the negative priming condition was 578.4 ms for younger adults and 656.8 ms for older adults. The mean negative priming effect was thus 20.2 ms for younger adults and 12.6 ms for older adults.

The multilayered slowing model fit these data quite well ($R^2 = .95$; 42 data points), with $a = 0.05$, and $b = 1.04$. Fitting Equation 2 to the data resulted in the following estimates for the parameters: $a_1 = 0.036$, $a_2 = -0.0022$, $b_1 = 1.05$, and $b_2 = -0.01$. In this equation, neither the $a_2$ parameter nor the $b_2$ parameter were significant, indicating that there was no reliable difference between the two age groups. This suggests that the data merely reflect a strong general slowing effect. When the information loss model was fitted to the data, with the restriction that $m$ was to be equal to or larger than 1, estimated $m$ equaled 1, so that the estimated information loss model reduced to a linear model. Consequently, it was decided to retain the multilayered model as best reflecting the pattern in the data.

Given the trend towards an age difference in the identity negative priming effect size, two additional analyses were undertaken. The first was to examine whether the slope estimated from the regression analysis might not be a systematic distortion of the effect present in the individual experiments. It is possible, for instance, that the data points for the baseline condition systematically tend to lie above the regression line, whereas the data points for the negative priming condition tend to lie below the regression line (for a discussion of this and related problems with Brinley analysis, see Perfect, 1994). In that case, the regression line is not a good estimator of the average within-study effect, and one would expect that the slopes of the lines connecting the baseline data point with the interference data point in individual studies would systematically be smaller than the slope estimated from the regression analysis. When we calculated the slopes from all 21 individual studies and averaged these, weighting for sample size, the mean slope was equal to 0.70 ($SD = 1.07$). Even though this slope is not statistically different ($p = .14$) from the mean slowing effect in the data (1.05), the trend for a smaller slope is indeed present.

The second additional analysis was a state trace analysis. In this analysis, we regressed the negative priming condition latencies onto the baseline latencies. The rationale for applying a linear model to negative priming data is made explicit in the Appendix. A linear model indeed fit the data extremely well ($R^2 = .988$; 42 data points). The estimated slope was 1.07 and the estimated intercept equaled -0.38. If, however, the negative priming effect is different in younger and older adults, one would expect different slopes or intercepts for the regression lines describing the baseline-negative priming relation in the two age groups. Statistical analysis shows that the two age groups differ in the slope of the state trace, because fit was improved significantly by introducing an Age × Condition interaction term, $\Delta R^2 = .001, F(1, 39) = 4.68, p < .05$. (The reader may note that even though the change in $R^2$ is extremely small, it was significant; when evaluating the size of the increment, it should be taken into account that the fit of the simple linear model was extremely high already, thus not allowing for large increments in $R^2$.) Fit was not further improved by introducing a condition term, indicating that the intercept was identical for younger and older adults. In the final model, then, slopes were 1.10 for the younger adults and 1.08 for the older; the intercept value common to both age groups was -0.36. Both of the slopes were significantly larger than 1, and the intercept was significantly smaller than zero, indicating that the multiplicative model as outlined in the Appendix seems to fit the data better than the additive model, which would predict a slope of 1, and a positive intercept. Consequently, conditions of negative priming do not add a constant amount of time to the processing time, but rather have a multiplicative effect on the processes that are susceptible to it.

The slope values immediately return the size of the multiplicative negative priming effect (see Appendix). The fact that the slope relating negative priming to baseline performance is reliably smaller in older adults than in younger adults indicates that older adults are indeed less susceptible to the negative priming effect than younger adults. The fact that the slope is larger than unity in older adults as well as in younger adults implies that both age groups are indeed susceptible to the negative priming effect.

From the intercept, one can use Equation A5 to calculate back what amount of time is needed for executing the processes not susceptible to the negative priming effect (i.e., presumably perceptual and motor processes). These are estimated to take 358 ms in the younger adults and 475 ms in the older. Consequently, the estimated age-related slowing factor in these processes is 1.33. Entering the value for nonsusceptible processing into Equation A4 indicates that the processes susceptible to negative priming (presumably the identification process) takes 200 ms for younger adults and 168 ms for older adults in the baseline condition. Thus, in these processes, older adults are actually faster than younger adults.

The results from the second additional analysis suggest that the nonsignificance in the previous analyses on identity priming, where only a nonsignificant trend toward smaller negative priming in older adults was noted, may have been due to power problems in our meta-analysis, resulting from a combination of the rather small sample of studies, the modest size of the average negative priming effect, and scatter because of sampling differences. If sampling differences are taken out of the data by using the individual studies as their own control in a state trace analysis, a significant difference between younger and older adults does manifest itself.

**Discussion**

A number of findings from the present meta-analysis are noteworthy.

First, we found that both younger and older adults are susceptible to the negative priming effect, and this is true for both identity and location negative priming. Two lines of evidence support this conclusion. First, using data-pooling techniques, a significant negative priming effect was found in older adults both under conditions of identity and location negative priming. Second, state trace analysis on identity negative priming data showed that the slopes of the function relating negative priming condition mean latencies to mean baseline latencies were reliably larger than 1 in both younger and older adults.

Second, there was evidence that the identity negative priming
effect was smaller in older than in younger adults. In two analyses, namely an analysis of the slopes relating older adults’ mean latency to younger adults’ mean latency in the two conditions, and in an analysis of the slopes of individual studies, there was a nonsignificant trend. When sampling variance was held under control by looking at slopes of the function relating mean latency of the negative priming conditions to mean latency of the baseline conditions, reliably different slopes did indeed emerge for the two age groups. For location negative priming, no evidence for a differential age deficit was found. Thus, location negative priming mechanisms seem to remain largely intact in later adulthood. However, the number of studies on location tasks is rather limited; more research may be needed before definitive conclusions can be reached.

A third and somewhat surprising finding, given the current debate in the literature (May et al., 1995; McDowd, 1994, 1997), was that there was no evidence for the influence of moderator variables on the location or identity negative priming effect, as evidenced by a lack of significant heterogeneity in the data pooling procedure. For identity negative priming, the extremely good fit of the function relating mean performance in the negative priming condition to mean performance in the baseline condition strongly suggests that mean latency in the negative priming conditions is almost exclusively (99% of the variance explained) governed by mean latency in the baseline condition.

One possible source for the confusion in the literature regarding moderator variables is the potential lack of power in individual studies. The size of the average negative priming effect is quite small, especially in older adults, and sample sizes are typically modest. This combination is a cause of concern for the interpretation of the results from single studies because the power of experiments, that is, the probability of rejecting a null hypothesis that indeed should be rejected, is precisely dependent on both sample size and the size of the effect. If we take an imaginary average study that has the average effect size and average sample size found in our analysis (viz., 0.07 0.13, and 1.27 and 3.4, for younger and older adults respectively), we find that the probability of correctly rejecting the null hypothesis (provided a one-tailed test is used) is very adequate in younger adults (97), but much less so in older adults (59; a handy tutorial on statistical power and how it can be calculated can be found in Levin, 1997). Thus, it is quite unsurprising that many researchers fail to find a significant negative priming effect in older adults. In order to obtain a power of 80, which is usually considered respectable (Cohen, 1988; Levin, 1997), the sample size for studies with older adults, given a within-subject effect of 0.23, should be 60 or more.

The lack of power due to typically inadequate sample sizes may also be the reason for the assertion (e.g., May et al., 1995) that older adults show decreased identity negative priming but spared location negative priming. The effect size for location negative priming is larger than that for identity priming, and thus fewer participants are needed in order for the effect to reach statistical significance.

A fourth finding of our meta-analysis is that the mechanism responsible for the negative priming effect is multiplicative, rather than additive, on the processes that are susceptible to the effect. This was evidenced by the result that the slope of the function relating mean latency in the negative priming condition to mean latency in the baseline condition was reliably larger than 1. As explained in the Appendix, the slope of this function can be taken as reflecting the multiplicative effect of negative priming on those processes that are susceptible to negative priming. Because this slope was found to be reliably larger than 1, we can conclude that negative priming results in an increment in time needed for processing that has to be expressed in terms of percentage increment (more specifically a 10% increment in younger adults and an 8% increment in older adults) rather than in terms of an increment expressed in milliseconds, as is usually done. Results of our analysis thus suggest an extremely simple model for identity negative priming in both younger and older adults: Latency in negative priming conditions is a simple function of latency in baseline conditions, and there are no moderator variables present. Further research is necessary to determine whether this model is fully adequate but, given the present state of research, this single mechanism explanation seems to offer a sufficient account of the identity negative priming data examined here. Occam’s razor can hardly be wielded further.

The finding of a multiplicative relation has implications for data analysis. If one simply wants to determine whether a negative priming effect is present, traditional methods for analysis (t tests, one-way ANOVA) may well serve their purpose. However, if one has a hypothesis about differential effects of negative priming under certain conditions, or about the differential effects of negative priming in different groups, results from a traditional interaction analysis in ANOVA are not to be trusted. To illustrate, assume a researcher has reasons to posit the hypothesis that showing overlapping stimuli would result in a larger negative priming effect than showing nonoverlapping stimuli. Assume that the task is to name pictures and that target stimuli are presented in a different color than the distractor stimuli. One might suppose that mentally disentangling overlapping pictures would take some extra processing time. In fact, such studies have already been conducted: For their overlapping pictures, Sullivan and Faust (1993) found a mean latency of 669 ms in younger adults; for nonoverlapping pictures, Tipper (1991) found a mean latency of 569 ms in younger adults. In the present meta-analysis, the equation relating latency in the negative priming condition to baseline condition in younger adults was found to be:

$$T_{NP} = 1.1(T_{BL} - 36\ ms).$$

If we substitute the baseline data from Sullivan and Faust (1993) and Tipper in Equation 4, we get an expected mean latency in the negative priming condition of 700 ms for overlapping pictures and 590 ms for nonoverlapping pictures. In other words, the expected negative priming effect is 31 (i.e., 700 - 669) ms for overlapping stimuli and 21 (i.e., 590 - 569) ms for nonoverlapping stimuli. It is important to note that these expected effects reflect the average trend in the data and nothing else, that is, there is absolutely no real condition-specific aspect to these expected effects. If these data had been gathered in a single experiment, and the number of participants had been large enough, one might have concluded that using overlapping stimuli has a differential impact on the negative priming effect. One way to remediate the problem of artifactual interactions could
be to include more conditions, estimate the general effect from these conditions, and then explore deviations from this pattern (for further explanation and an illustration, see Madden, Pierce, & Allen, 1992).

A fifth conclusion concerns the locus of age differences in the tasks traditionally used to measure identity negative priming effects (i.e., usually naming tasks of one sort or another). The results of the state-trace analysis suggest that when processes not sensitive to the negative priming effect (i.e., presumably perceptual and motor processes) are taken out of the analysis, no age-related slowing is present in the processes involved in the baseline condition. Rather, the data point to a slight speeding up of these processes with advancing age. Consequently, it seems that the processes associated with naming are not exempt from the negative effects of aging. This result might be linked to studies finding age equivalence in retrieval of semantic information and of semantic activation during language processing (for an overview, see Burke, 1997). In the present data set, however, perceptual and motor processes appear to be slowed in late adulthood, with a slowing factor of about 1.3.

The present findings have implications for theories about inhibition as a core mechanism for cognitive aging. On the one hand, it was found that older adults are indeed subject to the negative priming effect. This implies that there is certainly not a complete breakdown of inhibitory processes in late adulthood. On the other hand, older adults do seem somewhat less vulnerable to the identity negative priming effect than younger adults. This might imply that older adults have more difficulty inhibiting irrelevant aspects of a stimulus than younger adults. However, recent studies suggest that inhibition is not the only process responsible for the negative priming effect, but that a more general process (e.g., a motivational process) may be active as well (Kane, May, Hasher, Rahhal, & Stoltzfus, 1997; Neill & Valdes, 1992; Neill et al., 1995). Briefly, the memory account for the negative priming effect states that on the prime trial a distractor acquires information that it has to be ignored. This nonresponse or ignored task will be retrieved if the distractor becomes the stimulus to be responded to on the test trial. This may slow down processing because it might interfere with algorithmic computation of the response, or it might interfere with the retrieval of more useful episodic information. A corollary of this theory is that persons with a better memory will be more susceptible to the negative priming effect. It is a well-established finding that older adults perform less well than younger adults on both primary memory tasks (e.g., Verhaeghen, Marcon, & Guossens, 1993; Verhaeghen & Saltz-Neill, 1997). Thus, it is quite possible that the smaller negative priming effect in older adults as compared to younger adults is due to age-related problems with the memory component of the task, rather than to an age-related deficit in inhibitory processes proper. This hypothesis seems certainly worthy of further investigation.

A word of caution is in order with regard to the generality of these conclusions. Any meta-analysis is limited by the quality of the studies incorporated. It is possible that the smaller effects of negative priming in later adulthood are due to artifacts of the paradigms used. For instance, recent work by Johnson-Peterson, Rocchi, West, McConkey, and Hackney (1998) has shown that older and younger adults have different optimal times of day for their susceptibility to the negative priming effect: Older adults show the largest negative priming effect in the morning, whereas younger adults show the same effect later in the day. Inasmuch as primary researchers have not tested their participants at their optimal time of day, this may have confounded the results.

In sum, then, it was found that both younger and older adults suffer from the effects of negative priming. For identity negative priming, the effect was slightly smaller for older adults. A simple multiplicative model could explain the data very well, and no evidence for the influence of any moderating variables was found.

References

References marked with an asterisk denote studies included in the meta-analysis.

*Kane, M. J., May, C. P., Hasher, L., Rahhal, T., & Stoltzfus, E. R.


(Appendix follows)