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The Influence of Repeated Presentations and Intervening Trials on Negative Priming

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Abstract

The effects of repeating a task-irrelevant element and inserting intervening trials between the last prime and the probe trial in a negative priming study were compared with a standard prime/probe pair. An associative model based on SAC (e.g. Reder & Schunn, 1996; Schunn, Reder, Nhouyvanisvong, Richards, & Stroffolino, 1997) was able to account for both the decrease in response times across the repeated primes and the increase in response times when the task-irrelevant element became relevant.

Introduction

Until recently, theories of attention have focused on the object of attention. These theories emphasized the facilitatory "spotlight" at the center of attention, and the remaining areas of visual space were largely ignored (e.g., Broadbent, 1958; Shiffrin & Schneider, 1977). Over the past several years, however, researchers have developed evidence of an inhibitory effect of attention on objects that fall *outside* the spotlight. In particular, if some part of a visual display (either an object or a location) is to be ignored at some point in time, responses to that same item (or to one that is sufficiently similar) will be inhibited (i.e., slower or less accurate) at subsequent points in time. This inhibitory effect of ignoring an object has been termed *negative priming* (Tipper, 1985).

A typical negative priming experiment consists of pairs of visual displays. Before any stimuli are displayed, participants are told to respond to a certain aspect of each visual display. For example, participants might be told to name the object shown in the center of the display and to ignore any other objects. In the first (or *prime*) display of each negative-priming pair, the important object is the one that is to be ignored, the one that, in this example, is not centrally located. As stated above, when this object is presented in the second (or *probe*) display, in the central, to-be-attended location, participants are slower to respond (or less accurate) relative to a control condition.

Since this phenomenon was first described, two principal explanations have been proposed to account for it. The first posits that the role of attention in negative priming is one of inhibition or active suppression. One variant of this explanation is that when an object or location is ignored, the activation of its cognitive representation is suppressed (Neill, 1979). A second variant is, instead, that although the representation of the ignored information can remain active, that activation is kept from influencing one's response (Tipper & Cranston, 1985). Thus, according to these theories, negative priming occurs because this inhibition remains in force for some period of time after it is invoked. Herein, this will be referred to as the *inhibitory* account of negative priming.

The second candidate explanation is that negative priming occurs because of associations that form between instances of a stimulus and their subsequent responses as in Logan's (1988) instance theory of automaticity (Neill & Valdes, 1992; Neill, Valdes, Terry, & Gorfein, 1992). In this case, however, the responses in question are not the overt responses measured by the experimenter; they are internal, attentional responses. As part of the process of making overt responses, however, participants' are hypothesized to be making internal responses to focus attention on the stimulus to which they are to respond and consequently to ignore the irrelevant stimulus. According to the instance theory of automaticity, associations are then formed between each of the stimuli and the internal responses they evoked. On the probe display, when the previously irrelevant stimulus becomes relevant, it triggers its associated, internal ignore-response, which slows the overt naming response. This explanation will be referred to as the *attentional association* account of negative priming.

If, as is asserted in the attentional association hypothesis, negative priming is caused by associations formed between ignored stimuli and an internal ignore-response, then repeated presentations of an ignored stimulus over time should strengthen these associations. While the ignore-response is the correct internal response, performance should improve (i.e., become faster and more accurate). If, after repeatedly being presented as an irrelevant, and hence, to-be-ignored element of the display, an item becomes relevant, there should be substantial evidence of negative priming.

Moreover, since long-term associative memory decay has been shown to be well described by a power function (e.g., Anderson & Schooler, 1991),[1] which predicts considerable retention even after fairly long periods of time, these repeated, ignored items should provide evidence of negative priming

[1]The functional form of a power function is ct^{-d}, where c is the initial level of encoding, t is the amount of time since the last presentation, and d is the rate of decay. Power functions show a relatively rapid initial decline followed by a long period of only gradual decline.

at substantial delays. Also, if the set of items shown over the course of the experiment are sufficiently distinguishable, the inclusion of even a fairly large number of intervening trials between the last prime trial and the subsequent probe trial should have a minimal effect.

It should be noted, however, that these predictions depend upon a specific associative model that will be described below. While discussing these same issues, May, Kane, and Hasher (1995) made substantially different predictions, but proposed no formal model of associative memory by which their predictions could be tested.

In this paper we test these predictions of the associative account of negative priming empirically. This is the first experiment, to our knowledge, to attempt to build up long-term associations by repeated presentations of a task-irrelevant stimulus and test negative priming with large numbers of intervening trials. We then propose a formal candidate model of the theory and determine whether the model provides an adequate account of the empirical data.

Negative Priming Experiment

In this experiment, participants were presented with 520 visual displays (one per trial), each composed of two, two-digit numbers situated one above the other. In each display, one of the numbers was presented in boldface and the other in an outline font. The participants' task was to report, as quickly and accurately as possible, the font of the number with the *smaller* magnitude. Participants made their responses by pressing one of two buttons labeled "Bold" and "Outline."

The first 40 trials, which were not distinguished from the remaining trials, were practice trials. The remaining trials were each part of one of three different kinds of trials: immediate negative prime sequences, delayed negative prime sequences, and control trials.

Immediate negative prime (INP) sequences consisted of a pair of *contiguous* trials, the prime trial and the probe trial. In these trials, a number selected to be the *critical* number was presented as the distractor (the greater of the two numbers) in the the prime trial and as the target (the lesser of the two) in the probe trial. Each participant saw 30 INP sequences randomly distributed across the 480 non-practice trials.

Delayed negative prime (DNP) sequences consisted of 17 *non-contiguous* trials. For each DNP sequence, a number was chosen as the *critical* number for that sequence. Over the course of first 16 trials in the sequence, the critical number was presented as the distractor. Although each of these 16 prime trials was separated from the others by at least one trial, the median number of intervening trials was 10. These intervening trials were not special in any way. They contained elements of INP sequences, other DNP sequences, and control trials. The last trial in the sequence, the probe trial, was separated from the last prime trial by at least four intervening trials. Again, the median number of intervening trials was 10, and these trials were, again, not special in any way. Each participant saw 20 DNP sequences. So that the DNP probe trials would not all be presented near the end of the experiment, 10 DNP sequences concluded in the first half of the non-practice trials, and 10 DNP sequences concluded in the second half.

In control trials, two numbers were presented that were never used as critical numbers in either the INP or DNP sequences. Participants saw 80 control trials throughout the non-practice trials.

The positions of the numbers and the fonts in which they were displayed was chosen randomly on all trials, so that each number in each pair was equally likely to be displayed in either font and in either position.

Each trial began with a fixation cross appearing on the screen for 500 ms. Then the fixation cross disappeared, and the stimuli were displayed until a response key was depressed. Immediately following the button press, the fixation cross reappeared to begin the next trial. Thus, the response-to-stimulus interval (RSI) is 500 ms for the INP sequences. For the DNP sequences, we did not measure the RSIs directly, but they can be estimated to be at least 6 s and have a median of about 15 s.

Results and Discussion

We excluded the data from one of the 29 participants from our analyses, because he or she appeared to be reporting the font of the greater number. The results are summarized in Figure 1.

Immediate Negative Priming Participants were not significantly less accurate in their responses to immediate negative probe trials ($M = .95$) than to control trials ($M = .96$), $t(27) < 1, p > .10$. They did, however, respond more slowly to immediate negative probe trials ($M = 1104$)[2] than to control trials ($M = 1015$), $t(27) = 4.23, p < .001$.

Delayed Negative Priming Participants' responses to delayed negative probe trials were also not significantly less accurate ($M = .95$) than their responses to control trials ($M = .96$), $t(27) = 1.06, p > .10$. Once again, however, they did respond more slowly to delayed negative probe trials ($M = 1074.61$) than to control trials ($M = 1015.18$), $t(27) = 2.38, p < .05$.

Figure 1 shows the effect of repeated priming in the delayed negative priming sequences. In that figure, and for the analyses that follow, the primes in the DNP sequence are combined into groups of two. For example, in Figure 1, the mean of participants' median response times for the first and second trials in the DNP sequence are labeled $D1$. Thus, the response times for the 16 delayed negative prime trials are represented by 8 groups of two trials each.

A repeated measures, one-way ANOVA of these eight delayed negative prime groups indicated that the repetition of distractors in the DNP sequences caused participants to respond more rapidly as indicated by the first degree polynomial

[2]Only trials in which participants responded correctly were included in these response time statistics. Also, response time means were computed as the mean of participant medians to minimize the influence of response time outliers. The same statistical results were obtained using a variety of transformations and trimming procedures.

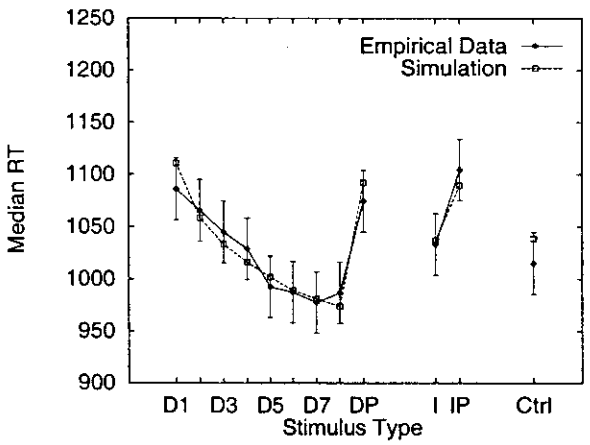

Figure 1: The best fit of the associative model to the empirical data. The stimulus types *Dn* and *I* denote delayed and immediate negative *prime* trials, respectively, the stimulus types *DP* and *IP* denote delayed and immediate negative *probe* trials, and stimulus type *Ctrl* denotes the control trials. Error bars indicate 95% confidence intervals around the mean (Loftus & Masson, 1994).

contrast $F(1, 27) = 40.65, p < .001$. No other polynomial contrast was significant.

In summary, the participants exhibited significant negative priming in both the INP and DNP conditions as predicted. Further, repetition of the distractor in the DNP sequences caused improved performance.

An Associative Model of Negative Priming

The model we propose to account for these data is an instantiation of the SAC model of memory (e.g. Reder & Schunn, 1996; Schunn et al., 1997),[3] which may be thought of as a generic semantic network model of memory most closely resembling the declarative memory structure in ACT (Anderson, 1983). SAC represents memories both as nodes with short- and long-term strengths and as connections between these nodes with short- and long-term strengths.

Although we are using an associative model to account for these data, we do not wish to argue that all instances of negative priming are due to associative processes (Reder & Weber, 1998), nor do we wish to argue that the negative priming in this experiment is due solely to associative processes. Rather, our goal is to show that an associative model that incorporates principles that have successfully accounted for a substantial number of memory phenomena can account for the data from this experiment. Thus, we are providing converging evidence that associative processes can play an important role in negative priming.

At the beginning of each simulation of the experiment, nodes representing all the two-digit numbers that are to be shown are presumed to exist and to be have identical, long-term (or base-level) activations, which we set to 1.0.

A node's base-level activation, B_N, may increase or decrease according to the power function

$$B_N = c_N \sum_i t_i^{-d_N}, \tag{1}$$

where c_N and d_N are the growth and decay constants for nodes and t_i is the time since the ith presentation. This function describes both power-law learning of practiced memories and power-law decay of memories over time (Anderson & Schooler, 1991).

In addition to its long-term, base-level activation, each node has a short-term activation, a_N, that decays exponentially. This decay may be expressed as a proportional decrease in short-term activation at each time step

$$\Delta a_N = -\rho_N a_N, \tag{2}$$

where $0 \leq \rho_N \leq 1$ is the short-term decay constant for nodes. The current activation, A, of each node is merely the sum of the short-term and base-level activations

$$A = B_N + a_N. \tag{3}$$

Activation spreads between nodes via links. Although each number node might be expected to have many links, for the purposes of this simulation, we used just two links per number node: one to an attend response and the other to an ignore response. Because other aspects of the experimental task were randomized, links to aspects of the tasks such as font or position would not have made any difference in the molar predictions of the model. Links to nodes representing information external to the task, likewise, would not influence the molar predictions inasmuch as the numbers were randomly assigned to each condition for each participant.

The link between a number node and a response node is strengthened when that response is evoked by the presentation of the number. The link will decay as time passes after the co-activation of the two nodes. Like node activation, link strength is composed of two parts, a long-term, base-level strength and a short-term strength. The long-term component for the link connecting node s to node r, $B_{L(s,r)}$, changes according to the power function

$$B_{L(s,r)} = c_L \sum_i t_i^{-d_L}, \tag{4}$$

where c_L and d_L are the growth and decay constants for links and t_i is the time since the ith co-occurrence.

Like the short-term activation of a node, the short-term strength of the link connecting node s to node r, $a_{L(s,r)}$, grows by being incremented by a constant amount, which we chose to set to 100, and decays exponentially. It is incremented when a response r is made to the number represented by node s. It decays in proportion to its activation at each time step according to the difference equation

$$\Delta a_{L(s,r)} = -\rho_L a_{L(s,r)}, \tag{5}$$

[3] SAC stands for *Source of Activation Confusion.*

where $0 \leq \rho_L \leq 1$ is the short-term decay constant for links. The current strength, $S_{(s,r)}$, of each link is the sum of the long- and short-term strengths

$$S_{(s,r)} = B_{L(s,r)} + a_{L(s,r)}. \quad (6)$$

Increases in the short-term activation of nodes occur by two means. First, when a number is presented on a trial, that number's short-term activation is incremented by a constant amount (which we chose to be 100), presumably, by some perceptual process outside the model. Second, each number node's total activation spreads via links to its associated response nodes. The amount of activation that spreads to each response node depends on the total activation of the sending node and on the relative strengths of each of the sending node's links. Although in this model, each response node only received activation from a single number node, in general, the change in a response node r's short-term activation, Δa_r, is computed by summing across all the nodes that send activation to it according to the equation

$$\Delta a_r = \sum_s A_s \frac{S_{(s,r)}}{\sum_i S_{(s,i)}}, \quad (7)$$

where A_s is the activation of each sending node as defined in Equation 3, $S_{(s,r)}$ is the strength of the link between node s and node r, and $\sum_i S_{(s,i)}$ is the sum of the strengths of the links from node s to any node i, both as defined in Equation 6. Because there was no effort to systematically manipulate the number of responses associated with each number node in this experiment, this normalization of link strengths in Equation 7 is not essential to account for the negative priming data we obtained. Nevertheless, it is a key element in the mechanism employed by Anderson (1993) to account for the phenomenon known as the fan effect (e.g., Anderson, 1974). Hence, it is important in other negative priming experiments that use stimuli with variable numbers of associations (Erickson & Reder, 1998).

Once activation has spread from the number nodes to the attentional-response nodes, a measure of facilitation is computed by examining the activation of the four response nodes associated with the two numbers that were presented on the current trial. Recall that the participants' task is to report the font of the lesser number. Their performance should be facilitated by the associative system, then, if it biases the attentional system to attend to the lesser number and to ignore the greater number. The associative system will produce a facilitatory bias to attend to the lesser number to the degree that the attend response associated with that number is active; conversely, it will produce an inhibitory bias to the extent that the associated ignore response is active. This may be expressed as

$$F_{\text{small}} = A_{\text{small,att.}} - A_{\text{small,ign.}}, \quad (8)$$

where F_{small} is the degree of facilitatory bias for the lesser number, and $A_{\text{small,att.}}$ and $A_{\text{small,ign.}}$ are the respective activations of the attend and ignore nodes associated with the lesser number. Likewise, the associative system will produce a facilitatory bias to ignore the greater number to the degree that the ignore response associated with that number is active and inhibited to the extent that the associated attend response is active. This may be expressed

$$F_{\text{large}} = A_{\text{large,ign.}} - A_{\text{large,att.}}, \quad (9)$$

where F_{large} is the degree of facilitatory bias for the greater number, and $A_{\text{large,ign.}}$ and $A_{\text{large,att.}}$ are the respective activations of the ignore and attend nodes associated with the greater number. Note that these "facilitatory biases," F_{size}, where *size* is either *large* or *small*, may take on both positive and negative values. When $F_{\text{size}} < 0$, it may, of course, be thought of as either a "negative facilitation" or an inhibition.

The total amount of facilitation provided by the associative system, F_{total}, is the sum of the facilitation provided by the associations with each number, $F_{\text{total}} = F_{\text{small}} + F_{\text{large}}$. To convert that to the influence of the associative system on participants' response times, F_{total} was simply multiplied by a constant, α.

At this point, however, the model only predicts deviations from some base-line response time. We assume that there exist other systems that process the sensory input, compare the two numbers, direct attention toward the lesser number, accumulate enough information to decide in which font the lesser number is displayed, and make the appropriate response. Although more detailed modeling of these systems might become necessary if we were to attempt to account for these data at a trial-by-trial or a distributional level, at the level of representation shown in Figure 1, we can make substantial simplifying assumptions.

Because our associative system only predicts facilitation and inhibition around a base-line response time, we assumed that these other systems were responsible for generating the base-line response time. Moreover, because the data we were trying to fit were distributed across many trials, we allowed for some systematic variation in the base-line response times over the course of the experiment. In particular, we assumed that the base-line response times would decrease according to the power law of practice (e.g. Newell & Rosenbloom, 1981), which predicts that response times should decrease over the course of the experiment according to the (now-familiar) formula

$$T_p = T_0 N^{-\gamma}, \quad (10)$$

where T_p is the response time for a given trial, T_0 is the initial response time, N is the trial number, and γ is the rate at which performance times change.

We fit this function to the mean of participants' median response times from practice and control trials grouped into 13 blocks of 40 trials[4] allowing T_0 and γ to vary freely. The best fitting values of $T_0 = 1486.45$ and $\gamma = 0.0590$, yielded $r^2 = .4194$.

[4] Although each block ranged over 40 trials total, recall that only $80/480 = 1/6$ of the non-practice trials were control trials. Thus, the expected number of trials per participant per block was only $6.\bar{6}$.

Table 1: Parameter values used to fit SAC to the negative priming data.

Parameter	$\mathcal{M}_0$	$\mathcal{M}_1$	Eqn.
α	0.0000*	0.1795	11
c_N		7.5242*	1
d_N		0.175*	1
ρ_N		0.1442*	2
c_L		1.3235*	4
d_L		0.12*	4
ρ_L		0.1442*	5
SSE	38,884.42	2,347.05	
r^2	.1217	.8991	

Note: Values marked with a * were held constant. SSE indicates the sum squared error between the model predictions and the empirical data over 12 different conditions. r^2 indicates the squared Pearson correlation coefficient computed between model predictions and the empirical data.

By combining this fit of the power law of practice to the control trial data with the predicted facilitation provided by the associative system, the model's final response time prediction is

$$RT = 1486.45\, N^{-0.0590} + \alpha\, F_{\text{total}}, \qquad (11)$$

where RT is the predicted response time, N is the trial number, α is the associative system scaling constant, and F_{total} is the total amount of facilitation provided by the associative system.

Simulations

Rather than estimate all the parameters in the model freely, we converted ones that had appeared in the implementation of SAC by Schunn et al. (1997) to values appropriate for the time-scale in this experiment. This conversion was necessary because the average duration of each trial in their experiment was about 15.5 s whereas the average duration of each trial in our negative priming experiment was about 1.5 s. We set the new exponential decay parameter for short-term link strengths, ρ_L, to be the same as the exponential decay parameter for short-term node activation, ρ_N, for the sake of parsimony. Thus, although SAC's behavior is governed by several different parameters, *only one parameter, α, was allowed to vary in fitting the data.* As stated, the other parameters were previously determined in an entirely different paradigm. The parameter values along with the equation number where they are defined, and fit statistics are shown in Table 1.

We simulated the experiment by presenting the exact same sequence of trials seen by experimental participants to the model. This is important both because the interval between presentations of a stimulus influences the model's predictions and because the predicted responses times in each condition depended on the exact position of each trial due to the power law of practice.

To show that the proposed associative system based on SAC was important to account for the data, we fixed $\alpha = 0.0$ and ran the simulation to evaluate how well the data could be accounted for by the power law of practice alone. The fit was quite poor, only accounting for 12% of the variance. Moreover, this fit predicted values that were too slow, on average 40.3 ms too slow.

We then fit the complete model to the data by allowing α to vary freely and minimizing the sum of squared error (SSE). The predicted response times are shown together with the data in Figure 1. Considering that this fit only added a single free parameter, the improvement is remarkable. The model accounted for 90% of the variance in the data and showed clear, qualitative negative priming effects.

General Discussion

This research makes two contributions to the negative priming literature. First, to our knowledge, this is the first research that has systematically manipulated the repetition of an ignored item in a negative priming experiment and has shown negative priming of familiar stimuli after the completion of large numbers of intervening trials (up to 90). Second, to our knowledge, this is the first attempt to flesh out a specific, formal associative model of attention that accounts for RT patterns in a negative priming paradigm (cf. Houghton & Tipper, 1994; Houghton, Tipper, Weaver, & Shore, 1996).

Although the bulk of this paper has been spent describing how attentional association can account for negative priming data, we reiterate that we do not wish to claim that this is the exclusive source of negative priming effects. Much as other authors have described (Houghton et al., 1996; May et al., 1995; Tipper & Milliken, 1996), it seems likely that attentional association and inhibition both contribute to negative priming.

Nevertheless, by combining the results from our empirical study with a successful formal model of the data, we have provided substantial evidence that associative processes play an important role in negative priming as proposed by Neill and his colleagues (Neill, 1979; Neill & Valdes, 1992; Neill et al., 1992).

In summary, we found comparable negative priming effects for sequences of both immediate and delayed prime and probe trials. These results were compatible with an associative view of negative priming. We showed that an associative model, a variant of SAC, could account for these data. Although we do not claim that this model provides a complete account of negative priming, we suggest that it provides an important step in the process of generating formal models of this phenomenon.

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References

Anderson, J. R. (1974). Retrieval of propositional information from long-term memory. *Cognitive Psychology, 6*, 451–474.

Anderson, J. R. (1983). *The architecture of cognition*. Harvard University Press, Cambridge, MA.

Anderson, J. R. (1993). *Rules of Mind*. Erlbaum, Hillsdale, NJ.

Anderson, J. R., & Schooler, L. J. (1991). Reflections of the environment in memory. *Psychological Science, 2*, 396–408.

Broadbent, D. E. (1958). *Perception and Communication*. Pergamon Press, Elmsford, NY.

Erickson, M. A., & Reder, L. M. (1998). Inhibition and interference: A memory model account of short- and long-term negative priming. In preparation.

Houghton, G., & Tipper, S. P. (1994). A model of inhibitory mechanisms in selective attention. In Dagenbach, D., & Carr, T. H. (Eds.), *Inhibitory processes in attention, memory, and language*, pp. 53–112. Academic Press, San Diego, CA.

Houghton, G., Tipper, S. P., Weaver, B., & Shore, D. I. (1996). Inhibition and interference in selective attention: Some tests of a neural network model. *Visual Cognition, 3*, 119–164.

Loftus, G. R., & Masson, M. E. J. (1994). Using confidence intervals in within-subject designs. *Psychonomic Bulletin & Review, 1*, 476–490.

Logan, G. D. (1988). Toward an instance theory of automatization. *Psychological Review, 95*, 492–527.

May, C. P., Kane, M. J., & Hasher, L. (1995). Determinants of negative priming. *Psychological Bulletin, 118*, 35–54.

Neill, W. T. (1979). Switching attention within and between categories: Evidence for intracategory inhibition. *Memory & Cognition, 7*, 283–290.

Neill, W. T., & Valdes, L. A. (1992). Persistence of negative priming: Steady state or decay?. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18*, 565–576.

Neill, W. T., Valdes, L. A., Terry, K. M., & Gorfein, D. S. (1992). Persistence of negative priming: II. Evidence for eposodic trace retrieval. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18*, 993–1000.

Newell, A., & Rosenbloom, P. S. (1981). Mechanisms of skill acquisition and the power law of practice. In Anderson, J. R. (Ed.), *Cognitive Skills and their Acquisitions*. Erlbaum, Hillsdale, NJ.

Reder, L. M., & Schunn, C. D. (1996). Metacognition does not imply awareness: Strategy choice is governed by implicit learning and memory. In *Implicit Memory and Metacognition*. Erlbaum, Hillsdale, NJ.

Reder, L. M., & Weber, K. H. (1998). Non-contiguous habituation and expectancy effects in target localization: A new look at inhibition and facilitation in a distractor task. Under Review.

Schunn, C. D., Reder, L. M., Nhouyvanisvong, A., Richards, D. R., & Stroffolino, P. J. (1997). To calculate or not to calculate: A source activation confusion model of problem familarity's role in strategy selection. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 23*, 3–29.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review, 84*, 127–190.

Tipper, S. P. (1985). The negative priming effect: Inhibitory priming by ignored objects. *Quarterly Journal of Experimental Psychology, 37A*, 571–590.

Tipper, S. P., & Cranston, M. (1985). Selective attention and priming: Inhibitory and faciliatory effects of ignored primes. *Quarterly Journal of Experimental Psychology, 37A*, 591–611.

Tipper, S. P., & Milliken, B. (1996). Distinguishing between inhibition-based and episodic retrieval-based accounts of negative priming. In Kramer, A. F., Coles, M. G. H., & Logan, G. D. (Eds.), *Converging operations in the study of visual selective attention*, pp. 337–363. American Psychological Association, Washington, DC.