Journal of Experimental Psychology:
Learning, Memory, and Cognition
2008, Vol. 34, No. 2, 257–281

0278-7393/08/$12.00 DOI: 10.1037/0278-7393.34.2.257

Prime Diagnosticity in Short-Term Repetition Priming: Is Primed Evidence Discounted, Even When It Reliably Indicates the Correct Answer?

Christoph T. Weidemann
University of Pennsylvania

David E. Huber
University of California, San Diego

Richard M. Shiffrin
Indiana University Bloomington

The authors conducted 4 repetition priming experiments that manipulated prime duration and prime diagnosticity in a visual forced-choice perceptual identification task. The strength and direction of prime diagnosticity produced marked effects on identification accuracy, but those effects were resistant to subsequent changes of diagnosticity. Participants learned to associate different diagnosticities with primes of different durations but not with primes presented in different colors. Regardless of prime diagnosticity, preference for a primed alternative covaried negatively with prime duration, suggesting that even for diagnostic primes, evidence discounting remains an important factor. A computational model, with the assumption that adaptation to the statistics of the experiment modulates the level of evidence discounting, accounted for these results.

Keywords: prime diagnosticity, perceptual identification, repetition priming, short-term priming, prime-validity effect

Short-term priming is often used as a tool for illuminating the structural properties of language and semantics by comparing situations in which primes facilitate performance in a task with situations in which primes cause no facilitation or even detriments in response time and/or accuracy. In such studies, the focus is less on the mechanisms of priming and perception than on inferences about semantic structure and modularity. In our research, rather than asking "what primes what?", we ask "how does priming work?" Our research is motivated by a view of priming that does not see the primes as separate perceptual events but as an integral part of the target perception itself. The general idea is to see the visual identification system as attempting to make the best inference on the basis of information that is often imprecise in both time and space.

Christoph T. Weidemann, Department of Psychology, University of Pennsylvania; David E. Huber, Department of Psychology, University of California, San Diego; Richard M. Shiffrin, Department of Psychological and Brain Sciences, Indiana University Bloomington.

Christoph T. Weidemann's World Wide Web page is at http://cogsci.info. This work was supported by a postdoctoral fellowship to Christoph T. Weidemann from the German Academic Exchange Service (DAAD) as well as by National Institute of Mental Health Grants 12717 (to Richard M. Shiffrin) and 63993 (to Richard M. Shiffrin and David E. Huber). This article is based on parts of a dissertation by Christoph T. Weidemann submitted to the faculty of the University Graduate School at Indiana University Bloomington in partial fulfillment of the requirements for the degree Doctor of Philosophy in the Departments of Psychological and Brain Sciences and Cognitive Science. We acknowledge helpful discussions with Kelly Addis, Jason Gold, Robert Goldstone, Shane Mueller, Adam Sanborn, and Peter Todd that benefited this article.

Correspondence concerning this article should be addressed to Christoph T. Weidemann, University of Pennsylvania, Department of Psychology, 3401 Walnut Street, Room 302c, Philadelphia, PA 19104. E-mail: ctw@cogsci.info

Previous Studies

Our previous studies used a forced-choice procedure for testing perceptual identification (e.g., Weidemann, Huber, & Shiffrin, 2005): The primes are followed by a briefly flashed target, followed by presentation of both the target and a foil for a choice response. This paradigm allowed us to prime neither choice, the target, or the foil (see Figure 1—even though we only presented one prime, it was presented twice as indicated in the figure to avoid complete overlap with the target in the target primed condition). Huber, Shiffrin, Lyle, and Ruys (2001) showed that repetition and associative priming with words arises largely from preference effects—in other words, a bias to choose whatever had been primed. Furthermore, the magnitude and direction of these preference effects proved readily changeable as a function of prime saliency (e.g., Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005). These findings place an important cautionary note on priming studies, suggesting that small changes in how primes are presented, or instructions that may draw more or less attention to primes, can produce large changes in the magnitude and direction of priming. The current studies build upon this earlier work by examining the issue of *prime diagnosticity*, defined as the relative proportion of trials on which the prime can be used to infer the correct answer.

Many priming experiments have used very brief and/or degraded prime presentations in an effort to reduce strategic use of prime diagnosticity. If participants are readily aware of the identity of prime words and also notice that a large proportion of trials involve targets related to primes, they may use this information to

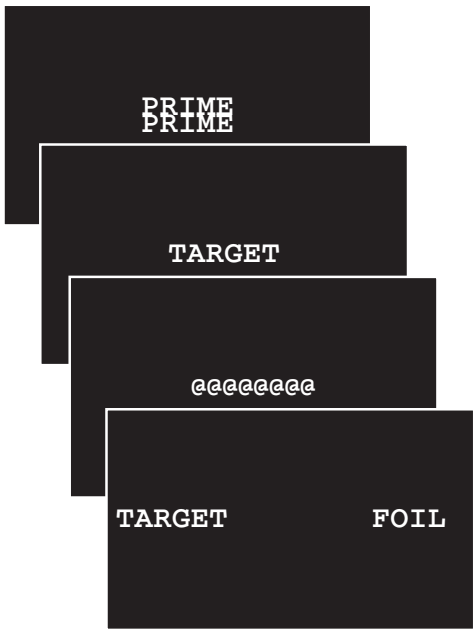

Figure 1. Illustration of the two alternatives forced-choice (2-AFC) perceptual identification task with short-term priming. The first screen (upper left corner) shows the prime presentation. The prime is presented twice in the middle of the screen to preserve symmetry and to avoid complete overlap with the target in the target primed condition. For repetition priming, the prime can be identical to the target, the foil, or neither choice word in the 2-AFC (bottom right corner). Prime duration is variable: The current studies use 50 ms for brief primes and 1,000 ms for long primes. The target is presented very briefly (for an individually adjusted time, see the *Method* section for Experiment 1) and immediately masked with a row of "@" signs. A fixation point before the onset of the prime and accuracy feedback after the response to the 2-AFC are not shown in the figure.

strategically guide their responses. Because most researchers who use priming to study language are not interested in such decision strategies, they attempt to keep prime awareness low (e.g., Bodner & Masson, 2001; Bodner, Masson, & Richard, 2006). Taking a different approach to strategy reduction, previous studies used a forced-choice procedure that contained just as many trials in which the prime indicated the incorrect choice as trials in which the prime indicated the correct choice (e.g., Huber, Shiffrin, Lyle, & Quach, 2002; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Ratcliff & McKoon, 2001; Weidemann et al., 2005). In these studies the participants were told explicitly that the primes would be nondiagnostic, and they were warned that strategic responding on the basis of prime relations to choices would prove ineffective. These experiments showed that brief (but, at least in some cases, clearly visible) primes produced a preference to choose whatever had been primed. This was revealed by higher accuracy when the target was primed and lower accuracy when the foil was primed. However, longer duration primes, or primes that were responded to, produced the opposite preference pattern, with lower accuracy when the target was primed but higher accuracy when the foil was primed (e.g., Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005).

A Model of Short-Term Priming

Huber et al. (2001) explained these findings with two distinct processes: (a) spatial and temporal confusions that cause some prime features to become mixed into the target percept (termed *source confusion*) and (b) discounting of evidence from percept features known to have been present in the prime(s) (and hence the prime[s] could have been the basis of the match). Source confusion introduces prime features into the target percept and evidence discounting works by reducing the impact of those features that are deemed likely to have been introduced by the prime. This may seem somewhat circular; if the nature of the priming is known, why would it induce source confusion? However, it is important to realize that the perceptual system may know the potential sources without knowing which specific sources are responsible for which specific pieces of information (e.g., if the prime was identified as the word "Treat," and at the time of the target the letter "T" was identified, it may be unclear whether this was a holdover from the prime or whether it was a new percept due to the target). The optimal degree of evidence discounting assigned to such features is determined by the probability of source confusion, although this model assumes that the system does not know this true probability of spatio-temporal confusions of prime and target features. Instead, the system may often misestimate the amount of source confusion (and hence the degree of required discounting). In particular, if the estimate is a little too low when primes are brief, and a little too high when primes are salient, then the complex pattern of results is predicted quite accurately, particularly including the switch from positive to negative priming as prime durations increase.

This account not only explained conditions in which only one choice alternative was primed but also conditions in which both choices or neither choice were primed. Huber et al. (2001) implemented this theory as a Bayesian model called *responding optimally with unknown sources of evidence* (ROUSE), and Ratcliff and McKoon (2001) proposed a multinomial model that relies on the same mechanisms of source confusion and discounting to account for the variable direction of priming observed in the data. An important difference between the multinomial model and ROUSE is that the former implements source confusion and discounting as explicit probabilities applied to whole words, whereas the latter uses an implicit feature-based representation on which these mechanisms operate. Both theories were able to account for a complex set of data, but the more fine-grained representation in ROUSE was crucial for generating several counterintuitive predictions that were confirmed empirically (see also Huber, Shiffrin, Lyle, & Quach, 2002; Huber, Shiffrin, Quach, & Lyle, 2002). After presenting the new experiments, we provide a more detailed overview of ROUSE.

Automatic and Controlled Processes

The pattern of results in the short-term priming tasks reviewed above is quite complex with large changes in identification performance as a function of prime saliency. Given the nondiagnosticity of the primes in these studies (i.e., the primes were equally likely to indicate the correct or incorrect choice regardless of prime saliency) and the instructions and feedback that reinforced this fact, we take these results to indicate automatic priming effects in the absence of controlled processes (e.g., Posner & Snyder, 1975a,

1975b; Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977). With nondiagnostic primes, a preference for or against the primed alternative can never increase performance: As performance for target primed trials increases, performance for foil primed trials decreases and vice versa (additionally, increased response variability may actually hurt performance; Huber et al., 2001). Nevertheless, previous results show consistent effects of increasing prime salience, which produces decreasing preference for primed stimuli over a wide range of saliency manipulations (e.g., Huber, in press; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005). This might be considered suggestive of a conscious decision strategy to discount. However, explanations based on decision strategies quickly became untenable given the complex priming patterns that emerged when using multiple prime presentations (Weidemann et al., 2005), which would require multiple simultaneous strategies as applied to different conditions on different trials with different combinations of primes. In contrast, such complex data patterns were readily captured with a computational model that employed a much smaller set of parameters than observed levels of preference.

Despite the success of source confusion and evidence discounting in explaining these priming data, it remains unclear whether this account is applicable to more traditional priming paradigms in which primes are diagnostic. Instead, it may be that automatic discounting is a mechanism that is unique to situations in which primes are nondiagnostic. To investigate this issue, we systematically manipulated the diagnosticity of the primes in the forced-choice perceptual identification paradigm reviewed above. When primes are diagnostic, they reliably indicate the correct answer, and accuracy above 50% in the two alternatives forced-choice (2-AFC) test (i.e., chance level when no information is available) is possible by making use of that diagnosticity even if no information is gained from the presentation of the target.

Diagnostic primes (especially if they are clearly visible) are likely candidates for eliciting strategic responding. Indeed, effects of prime diagnosticity are often simply assumed to index strategic responding (e.g., Hutchison, Neely, & Johnson, 2001; Pecher, Zeelenberg, & Raaijmakers, 2002). However, even when direct evidence for strategic responding is found in some conditions (e.g., Hutchison, 2007; Pecher et al., 2002), it is problematic to assume that effects of prime diagnosticity necessarily index controlled processes. In this article, we investigate the nature of prime diagnosticity effects in the forced-choice perceptual identification task and examine several variables that may help distinguish automatic from controlled processes. In all experiments, we manipulated prime type (i.e., target, foil, or neither primed trials), prime saliency (i.e., prime duration), and direction of prime diagnosticity (i.e., primes could be more likely to indicate the correct or the incorrect choice). Additionally, we investigated effects of changes in prime diagnosticity (Experiments 1 and 2), strength of prime diagnosticity (Experiment 2), quality of target information (Experiment 2), and different cues for prime diagnosticity (Experiments 3 and 4). The pattern of results across these manipulations can help determine to what extent diagnostic primes may lead participants to shift from trying to identify the target to strategic responding on the basis of the prime. To foreshadow our conclusions, the results consistently point toward automatic processes as the source for prime diagnosticity effects, thus challenging the view that prime diagnosticity effects are sufficient for indexing controlled processes. Following the presentation of the experiments, we introduce a formal model that accounts for prime diagnosticity effects by assuming that the trial statistics modulate (implicit) evidence discounting during target identification.

Experiment 1: Prime Diagnosticity and Prime Duration

In Experiment 1, we varied prime diagnosticity across different blocks of trials as a within-subjects manipulation. Diagnosticity was crossed with prime duration. The two prime durations, mixed within block, were 50 ms (a duration near those typically used to reduce prime awareness) and 1,000 ms (a duration that allows clear perception and consideration of the prime). Prime diagnosticity was positive (a prime was 3 times more likely to indicate the correct choice than the incorrect choice), negative (a prime was 3 times more likely to indicate the incorrect choice than the correct choice), or neutral (primes were equally likely to indicate the correct or incorrect choice). Participants were not told that prime diagnosticity would vary between blocks of trials. Because prime diagnosticity was blocked, the design broke the experiment into thirds. In other words, participants experienced one prime diagnosticity for the first third of the experiment, another for the second third, and the last for the final third of the experiment. These thirds are henceforth referred to as *triads* because each third was composed of three separate blocks of trials (see the *Method* section below). The fact that the three blocks within a triad all used the same level of diagnosticity allowed an analysis of learning in response to a change in prime diagnosticity.

Method

Participants. Eighty-four undergraduate students at Indiana University Bloomington participated in exchange for introductory psychology course credit. Fourteen participants each were assigned to the six prime-target diagnosticity orders.

Materials and equipment. We used two pools of 1,075 five-letter and 1,249 six-letter words with a written-language frequency of at least 4 per million as defined by Kucera and Francis (1967). We presented all words in uppercase using the fixed-width "Courier New Bold" 17-point font. The pattern mask for the target consisted of a row of six "@" signs. This mask was presented in the "Arial Narrow Bold" 13-point font, which ensured a dense and complete coverage of the target.

All stimuli were displayed on 17-in. (43.18 cm) PC CRT monitors with a vertical refresh rate of 120 Hz and a screen resolution of 800 × 600 pixels. We synchronized the display to the vertical refresh using the ExpLib programming library (A. L. Cohen & Sautner, 2001). This provided display increments of 8.33 ms. The stimuli were presented as white font against a black background. Each participant sat in an enclosed booth with dim lighting. The distance of the monitor, the presentation positions, and the font size were chosen such that the target and the primes encompassed less than 3° of visual angle. Responses for the 2-AFC test were collected through a standard computer keyboard. Participants were asked to press the "Z" key or the slash key to choose the left or right alternative, respectively.

Procedure. Every trial began with a central fixation point followed by the prime presentation. Each prime presentation consisted of the same prime word presented simultaneously in two

locations: symmetrically above and below fixation with the bottom of the top prime just touching the top of the bottom prime (as shown in Figure 1).[1] The prime was presented for either 50 ms or 1,000 ms and then immediately followed by the target presentation in the center of the computer screen. The target was replaced by an "@" sign mask after an individually adjusted duration (see below), but the combined duration of the target and the mask was fixed at 500 ms to keep the duration between prime offset and mask offset constant. Each trial ended with the presentation of two simultaneous choice words in new and distinct screen locations, one of which had to be selected as the target (i.e., a 2-AFC test). After making a correct choice, participants were shown a green check mark and the text "You identified the word correctly!", and after an incorrect choice they were shown a red "X" with the text "You did NOT identify the word correctly!" After each block, participants were given summary statistics of their accuracy and response times (this was the only time response time feedback was given because the instructions emphasized accuracy) and encouraged to take a short break. This experiment (as well as the other experiments presented here) focused on repetition priming, and thus all primes were either identical to one choice word in the 2-AFC or unrelated to both. There were three prime types mixed within block and crossed with prime duration: *neither primed* (i.e., the prime was different from both choice words), *target primed* (i.e., the prime was identical to the target), and *foil primed* (i.e., the prime was identical to the incorrect alternative of the 2-AFC).

The first 60 trials of the experiment were used to adjust the time of the target presentation such that accuracy was approximately 75%. For these calibration trials, stimuli were sampled (without replacement) from the five-letter word pool, and only the neither primed condition was used (both prime durations were used on an equal number of trials).

Following the calibration trials, there were nine blocks of 60 experimental trials during which the target duration remained fixed at the value obtained from the calibration trials ($M = 50$ ms, $SE = 2$). There were three levels of prime diagnosticity: *neutral* (i.e., 20 neither, target, and foil primed trials each per block), *positive* (i.e., 36 target primed trials and 12 neither and foil primed trials each per block), and *negative* (i.e., 36 foil primed trials and 12 neither and target primed trials each per block), with equal numbers of short and long prime durations (within each block trials were arranged pseudorandomly). Stimuli for this test phase were only sampled (without replacement) from the six-letter word pool. Prime diagnosticity was held constant for three blocks and changed after Blocks 3 and 6 (as mentioned above, we refer to these groups of three blocks with constant prime diagnosticity as *triads* below). Participants were exposed to all diagnosticities in one of the six possible orders. Participants were not explicitly informed about the diagnosticity manipulations.

Results and Discussion

Throughout the reporting of the results, it is important to keep in mind that the procedure consisted of nine blocks—but that these were broken into sets of three consecutive blocks (i.e., triads) during which the diagnosticity remained unchanged. The primary interest was performance at a given level of diagnosticity when that level occurred in the first triad and when that level followed some other level of diagnosticity. We therefore aggregated orders of triads that shared the current as well as the previous level of prime diagnosticity. To provide a concrete example, we aggregated data for the first triad (i.e., an absence of a previous prime diagnosticity) from participants who started with a positive prime diagnosticity regardless of whether they experienced neutral or negative prime diagnosticity in the second triad. Likewise, data from a triad with neutral prime diagnosticity that directly followed a triad with negative prime diagnosticity were aggregated regardless of whether the neutral prime diagnosticity occurred in the second or the third triad of the experiment.

Effects before changes in prime diagnosticity. Figure 2 shows the data for the first triad for those participants, who were first exposed to a neutral prime diagnosticity. In other words, this figure shows the data for the neutral prime diagnosticity before any change in prime diagnosticity occurred. In this article, we present the results in figures that have panels with six bars giving the probability of correct choices. Accuracy is plotted on the horizontal axis relative to the .5 chance level of the 2-AFC test. A preference for the target (symbolized by the small vertical boxes on the right side of the figure) corresponds to high accuracy, and likewise a preference for the foil (symbolized by the small vertical boxes on the left side of the figure) reduces accuracy. The fillings of the vertical boxes on either side are a graphical portrayal of the priming condition demonstrating which choice was primed (black fillings) and for how long (height of the fillings). The upper three bars give the results for short duration primes, and the lower three give the results for long duration primes. Each group of three bars, from top to bottom, gives results for neither primed, target primed, and foil primed trials, respectively.

It is obvious from Figure 2 that prime saliency had a large effect on identification performance: Comparing the upper three bars with the lower three bars reveals that brief primes produced a preference for the primed alternative, whereas long primes produced a preference against the primed alternative (all relative to the respective neither primed baseline). This is a replication of the usual pattern, discussed above, that led to the development of the ROUSE model (Huber et al., 2001).

Figure 3 shows the data for the first triad when that triad consisted of a positive (left panel) or negative (right panel) prime diagnosticity. To assess the effects of prime diagnosticity, prime type, and prime duration before any change in diagnosticity, we calculated a $3 \times 3 \times 2$ repeated measures analysis of variance (ANOVA) with these factors using only data from the first triad (i.e., the data presented in Figures 2 and 3). The details for this ANOVA are given in Table A1 in Appendix A. In summary, the main effects of prime type and prime duration were significant. In general, accuracy tended to be highest for neither primed trials and for long primes, but as is clear from Figures 2 and 3, prime type and prime duration also strongly interacted. We discussed this

[1] With repetition priming, one concern is always to make the prime presentation distinct from the target presentation (otherwise a target primed trial would just look like a long target presentation—obviously this is not a concern in priming studies that use semantic primes). We found the current display useful in achieving this goal without drawing attention away from the center of the display. We have no reason to believe that our results are limited to situations with the particular prime arrangement used in the current studies.

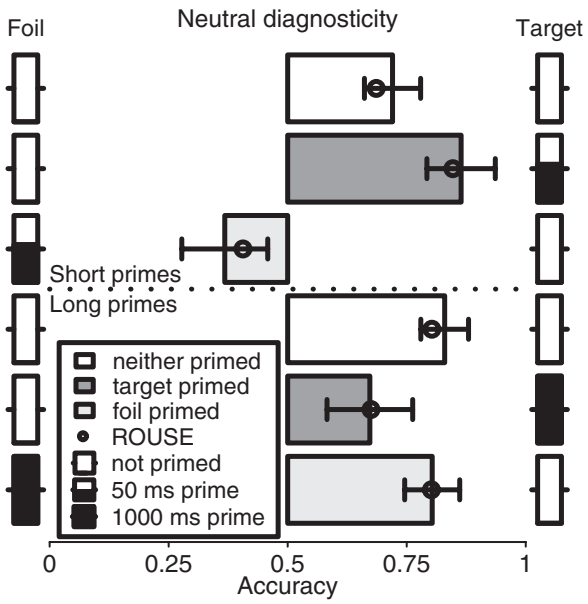

Figure 2. Accuracy for neutral prime diagnosticity at the beginning of the test phase of Experiment 1 (i.e., before any change in diagnosticity). Accuracy is plotted relative to the 0.5 chance level of the two alternatives forced-choice (2-AFC). The top three bars show accuracy for brief (i.e., 50-ms) primes, and the bottom three bars depict performance for long (i.e., 1,000-ms) primes. The boxes at the left and right edges of the figure indicate which (if any) choice word—the foil (left) or target (right)—has been primed and for how long: A completely filled box indicates that the corresponding choice word was primed for 1,000 ms, a partially filled box represents a prime duration of 50 ms, and an empty box shows that the corresponding choice word was not primed (the fill level corresponds to the logarithmically transformed prime duration to make the representations distinctive). The error bars indicate the 95% confidence intervals. The responding optimally with unknown sources of evidence (ROUSE) fits (circles) are discussed later in a special section after the experiments have been presented.

particularly striking interaction for the neutral prime diagnosticity (see Figure 2) above, and in general the preference for the primed alternative covaried negatively with prime duration. This pattern is consistent with the underdiscounting of evidence for brief (low salience) primes and the overdiscounting of evidence for long (high salience) primes. These findings and this interpretation replicate and are consistent with previous findings (e.g., Huber, Shiffrin, Lyle, & Quach, 2002; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005).

The only other significant effect was the interaction between prime diagnosticity and prime type. In Figure 3 this interaction is clearly visible by comparing the two panels with the data for the neutral prime diagnosticity shown in Figure 2: There was an increased tendency to choose the primed alternative when the diagnosticity favored such action (i.e., positive prime diagnosticity) and a decreased tendency to choose the primed alternative when the diagnosticity favored the opposite action (i.e., negative prime diagnosticity). Critically though, this diagnosticity effect did not alter the usual Prime Type × Prime Duration interaction.

Changing prime diagnosticity. Figures 2 and 3 presented the results for the first triad, prior to any changes in diagnosticity. Next, we consider combinations of current and previous diagnosticity to assess the extent to which performance adapted to the diagnosticity of the current triad. This results in the six combinations of the current diagnosticity (three possibilities) and previous diagnosticity (two possibilities considering that diagnosticities did not repeat across triads) appearing in Figure 4.

To asses the effects of changes in prime diagnosticity, we calculated three separate 3 (previous diagnosticity) × 3 (prime type) × 2 (prime duration) repeated measures ANOVAs—one for each level of the current diagnosticity. The rational for three levels of previous diagnosticity was to include the two actual previous levels, as shown in Figure 4, but also to include the lack of a previous diagnosticity, corresponding to the first triad data, to provide a baseline comparison. In other words, the data shown in Figure 2 and the middle panels of Figure 4 form the basis for the ANOVA for neutral prime diagnosticity, and the data from the left and right panels of Figure 3 combined with the top and bottom panels of Figure 4, respectively, form the basis for the ANOVAs for positive and negative prime diagnosticities. The details for these ANOVAs are given in Table A2 in Appendix A. Before considering changes to diagnosticity from these ANOVAs, we summarize the current diagnosticity results in brief: The main effects of prime type and duration were significant for all current diagnosticities, as was their interaction, replicating results from the previous analysis as well as earlier findings (e.g., Huber, Shiffrin, Lyle, & Quach, 2002; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005).

The interaction between previous diagnosticity and prime type was significant for the positive and negative current diagnosticities but only marginally significant for the neutral current diagnosticity. Finally, the three-way interaction between previous diagnosticity, prime type, and priming duration was also significant for the neutral and two negative current diagnosticities, but the sizes of these effects were small ($\eta_p^2 = .07$ and .09, respectively;[2] see Table A2 in Appendix A for details). These effects reflect the general attenuation of the current diagnosticity after a change in diagnosticity, which is evident when comparing the left and right panels of Figure 3 with the top and bottom panels of Figure 4, respectively. Indeed, there was little effect of the current diagnosticity after a change in diagnosticity, as can be seen from the relatively similar patterns of results in the different panels of Figure 4.

In separate ANOVAs that were analogous to the previous analysis but only included second triad data (i.e., only data from Figure 4), the effects involving the previous level of diagnosticity disappeared (except for a marginal interaction of previous diagnosticity with prime type and with prime duration for the neutral current diagnosticity condition—see Table A3 in Appendix A for details on this analysis). This finding suggests that the effect of previous diagnosticity can be almost entirely attributed to the difference between no previous diagnosticity (i.e., first triad data) versus situations that did involve a previous diagnosticity. However, a comparison of the left and right panels in Figure 4 reveals that the small differences were consistent with a (strongly attenuated)

[2] $\eta_p^2 = SS_{\text{effect}}/(SS_{\text{effect}} + SS_{\text{residual}})$—where *SS* stands for the sum of squared error in the ANOVA (cf. Table A2 in Appendix A)—is a measure of effect size in the sample and tends to overestimate the effect size in the population (cf. J. Cohen, 1988; Maxwell, Camp, & Arvey, 1981; Olejnik & Algina, 2000).

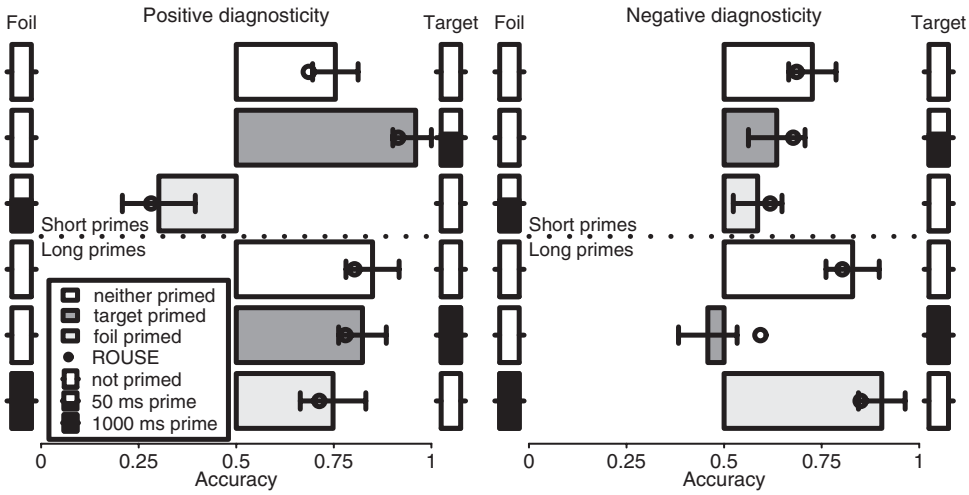

Figure 3. Accuracy for positive (left panel) and negative (right panel) prime diagnosticity at the beginning of the test phase of Experiment 1 (i.e., before any change in diagnosticity). ROUSE = responding optimally with unknown sources of evidence. See caption for Figure 2 for details.

influence of the previous diagnosticity (e.g., when comparing the middle panels of Figure 4, one can discern a slightly stronger preference for the primed alternative in the neutral prime diagnosticity conditions when the previous diagnosticity was positive compared with when it was negative).

Thus, the overall conclusions were (a) large current diagnosticity effects at the start of the experiment, (b) diminished current diagnosticity effects for subsequent diagnosticities, (c) small effects of previous diagnosticities, and (d) regardless of diagnosticity manipulations, the expected Prime Duration × Prime Type interaction remained. These results are sensible if prime diagnosticity rapidly adapts to the current situation yet remains somewhat sensitive to the previous situation, and, furthermore, if prime diagnosticity is roughly an additive factor in combination with the usual effect of prime duration. As explained in the modeling section reported after the experiments, this pattern is sensible if both prime diagnosticity and prime duration combine to set the level of discounting. Next, we consider adaptation to prime diagnosticity on a slightly finer time scale.

Learning of prime diagnosticity. Visual inspection of the individual block data revealed that identification performance adapted quickly to the prime diagnosticity. Because participants were not informed in advance of the prime diagnosticity, this adaptation must be due to learning. Figure 5 shows the data for the first and last 60 trials of the first triad (i.e., Blocks 1 and 3 of the first triad, which are shown in the left and right panels of Figure 5, respectively, and which occurred before a change in prime diagnosticity). It is evident from the similarity between these blocks that (almost) all learning of prime diagnosticity occurred within the first 60 trials.

To test whether any learning occurred after the first block in each triad, we performed additional ANOVAs (not described in detail here) that were analogous to the ones presented earlier but that excluded data from Blocks 1, 4, and 7 (i.e., the first blocks in each triad of blocks with common prime diagnosticity). In these ANOVAs, we added the two-level factor of block within the triad to compare data from the second block in each triad with those from the last blocks. In none of these ANOVAs did we find any significant effect involving block.[3] This suggests that no appreciable learning took place after the first block in each triad.

This fast adaptation to the response pattern for a particular condition is especially remarkable given the relatively long lasting attenuation effect that produced strong differences between the prime diagnosticity effect at the beginning of the experiment and that after a change in diagnosticity. It is difficult to imagine a model that could capture both effects with a single process, and unfortunately our data do not allow us to investigate these effects on a trial-by-trial basis. The model we present later assumes an estimation of "diagnostic evidence" (effectively the subjective strength of prime diagnosticity) from the respective numbers of target and foil primed trials, which is compatible with such fast learning of prime diagnosticity (i.e., this estimation presumably is based on the relative proportion of target and foil primed trials that quickly becomes less variable as trials accumulate). In this manner we assumed that the fast learning in response to current diagnosticity was essentially instantaneous. Therefore, the only aspect of learning that we specified in the model was the long range attenuation effect, which was simply captured through a single parameter for the mixing between the previous diagnosticity and the current diagnosticity (we provide details on the model in a separate section after the experiments have been presented).

Experiment 2: Prime Diagnosticity Strength and No-Target Conditions

Experiment 1 showed that the prime diagnosticity had a clear effect on identification performance but that discounting as a

[3] For the negative prime diagnosticity, we found a marginally significant interaction between prime type, prime duration, and block: $F(2, 162) = 3.93$, $MSE = 0.02$, $p = .05$ (Huynh–Feldt $\varepsilon = .53$ corrected; Huynh & Feldt, 1976), but the effect was very small ($\eta_p^2 = .05$).

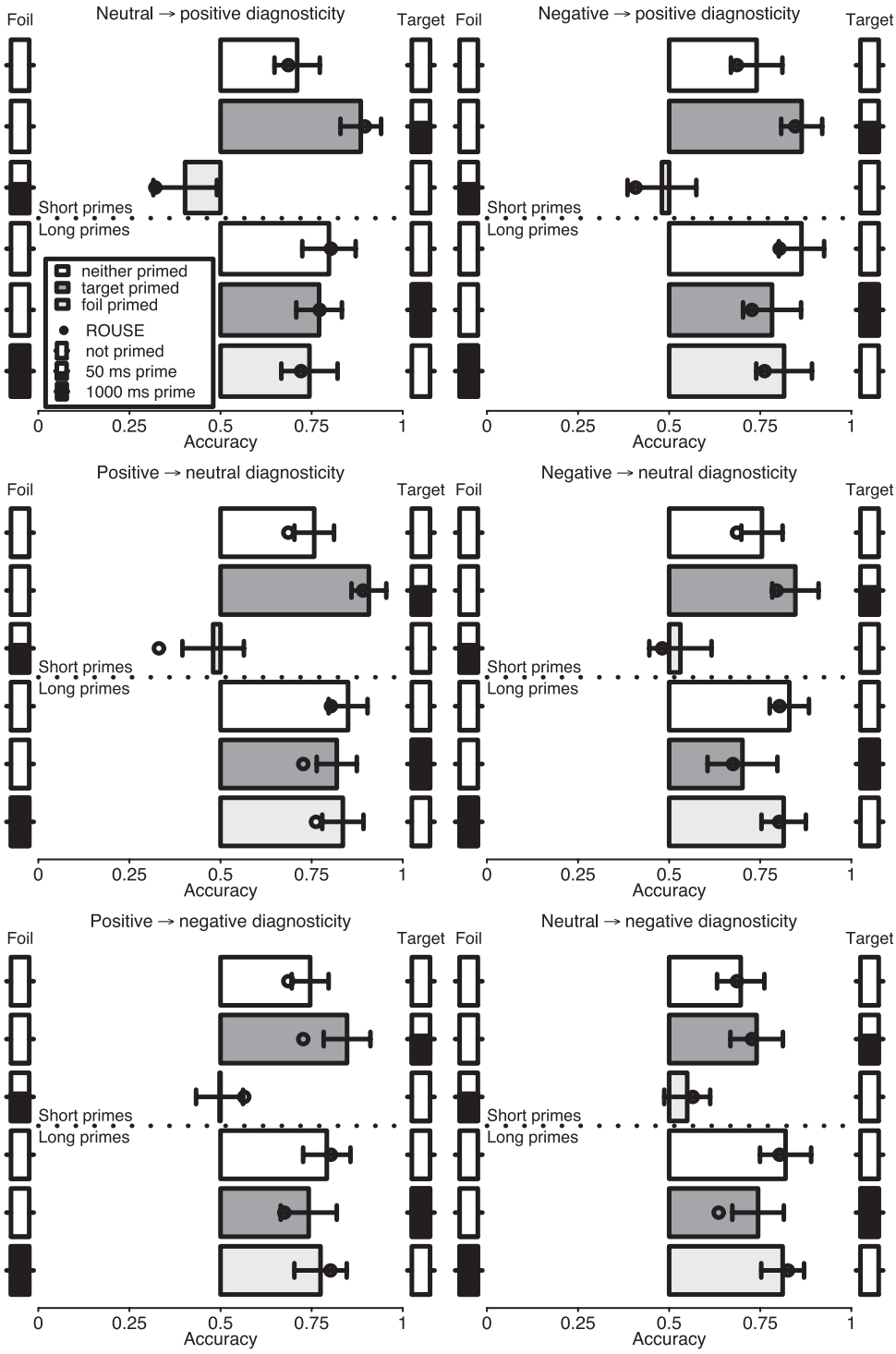

Figure 4. Accuracy for positive (top panels), neutral (middle panels), and negative (bottom panels) prime diagnosticity after a change in prime diagnosticity (as indicated at the top of each panel) in Experiment 1. ROUSE = responding optimally with unknown sources of evidence. See caption for Figure 2 for details.

function of prime duration remained a critical mechanism. If people are sensitive not only to the direction but also to the magnitude of prime diagnosticity, target identification should be influenced more for stronger diagnosticity manipulations. To investigate this issue, we used two diagnosticity strengths in this experiment, one that was weaker than that in Experiment 1 (with primes twice as likely to indicate the correct than the incorrect choice or vice versa) and another that was stronger than that in

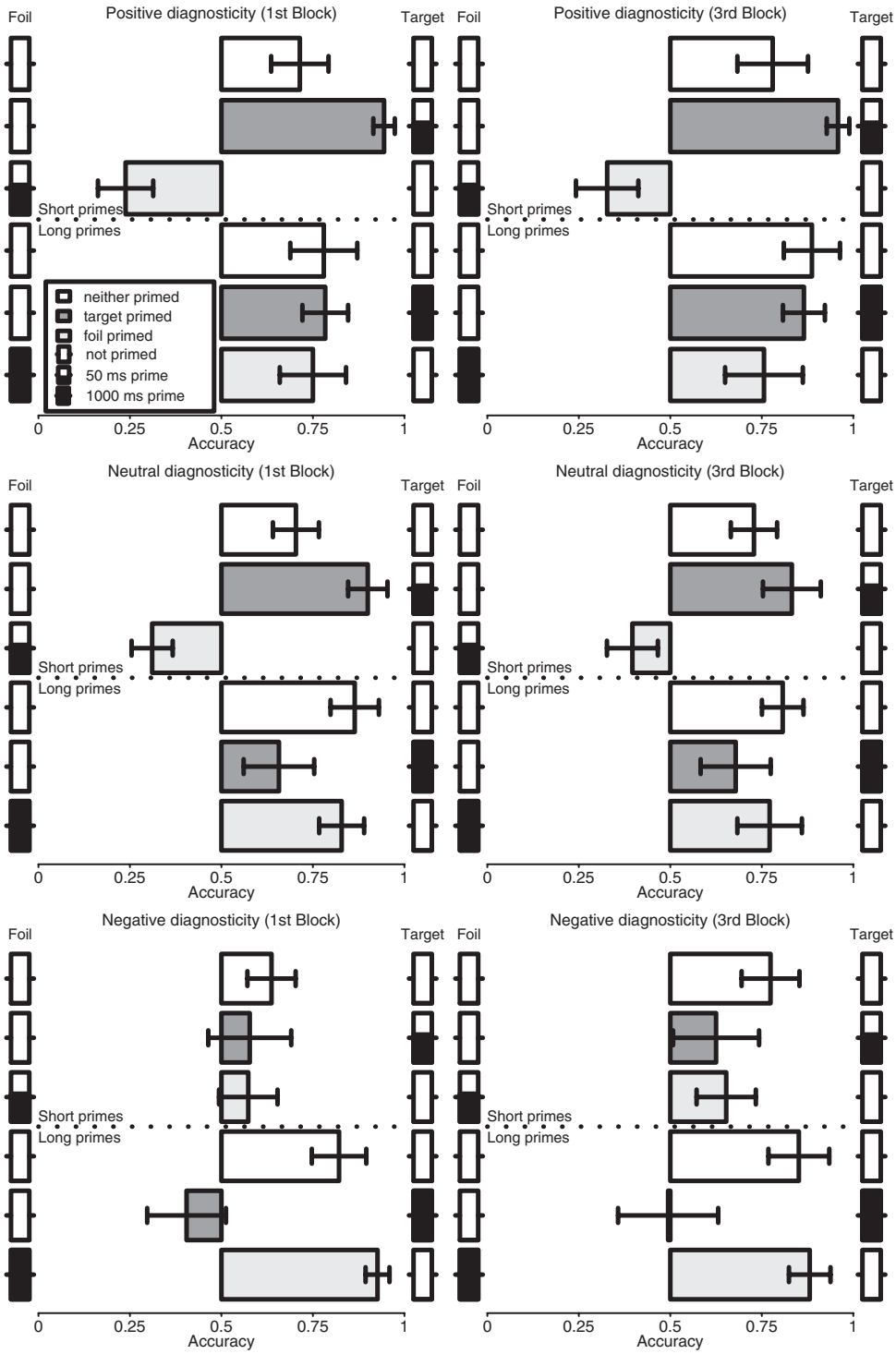

Figure 5. Accuracy for positive (top panels), neutral (middle panels), and negative (bottom panels) prime diagnosticity during the first block (left panels) and third block (right panels) of the first triad (i.e., before a change in diagnosticity) in Experiment 1. See caption for Figure 2 for details.

Experiment 1 (with primes 4 times as likely to indicate the correct than the incorrect choice or vice versa).

If no target information is available on a given trial, the strategy that maximizes performance is to always choose or always not choose the primed word depending on whether the prime diagnosticity is positive or negative. This strategy should apply regardless of the strength of the diagnosticity manipulation (provided that the prime diagnosticity is detectable). To directly assess strategic

prime-based responding (should such a strategy exist) for the different directions and magnitudes of prime diagnosticity, we omitted the target presentation on a small proportion of trials (because of the brief and masked nature of the target presentation, paired with arbitrary error feedback that reinforced participants' assumption that there was indeed a correct answer, the fact that a target was missing on some trials was not apparent). The omission of targets is particularly diagnostic because of an nonintuitive prediction the ROUSE model makes for these trials: For neutral prime diagnosticity, ROUSE predicts a preference for the primed alternative regardless of prime saliency (Huber, Shiffrin, Lyle, & Quach, 2002). This prediction, which is integral to the ROUSE model, is based on the fact that the relative preference against the primed alternative for salient primes can only be produced to the extent that evidence for prime compatible features that actually stems from the target presentation or noise is discounted (effectively "throwing the baby out with the bathwater"). Therefore, this manipulation provides an opportunity to specifically test whether prime diagnosticity effects are realized through modulations in evidence discounting. If this is the case, we predict that in cases when no target was presented, participants should be at least as likely to chose the primed alternative as they are to choose the unprimed alternative, and this ordering of conditions should occur regardless of prime diagnosticity or salience. In a separate modeling section below (after all experiments have been presented), we provide a more detailed explanation of this prediction of the ROUSE model (see also Huber, Shiffrin, Lyle, & Quach, 2002). Another purpose of this experiment was to further investigate the attenuation of prime diagnosticity effects after changes in diagnosticity that we observed in Experiment 1. To that end, we included one change in prime diagnosticity from positive to negative diagnosticity or vice versa.

Method

Participants. One hundred and twelve undergraduate students at Indiana University Bloomington participated in exchange for introductory psychology course credit. Of these, 28 participants each were assigned to the four prime diagnosticity conditions.

Materials and equipment. The materials and equipment were identical to those used in Experiment 1.

Procedure. The procedure was identical to that of Experiment 1 with the following exceptions: The mean target duration was 48 ms ($SE = 1$). The test phase consisted of four blocks of 138 trials each. Thirty trials in each block did not contain a target (a blank screen was shown in place of the target in these trials). Because of the brief target duration and the presentations of the prime and mask, the fact that a target was missing on some trials was not obvious. When a target was missing, the prime was always repeated as one of the alternatives in the 2-AFC test, and feedback was given according to the diagnosticity condition (e.g., in the strong positive condition, 80% of the trials without a target would trigger positive feedback when the primed alternative was selected in the 2-AFC test—see below).

There were four possible prime diagnosticities: strong positive, weak positive, weak negative, and strong negative. Each participant experienced one of these diagnosticities for the first two test blocks and then was shown the same-strength diagnosticity of opposite polarity for the rest of the experiment (i.e., each participant only saw either the two weak or the two strong prime diagnosticities, and they switched from positive to negative or vice versa after the second block of the test phase).

For the strong positive diagnosticity, the target was primed in 72 trials, the foil was primed in 18 trials, and in another 18 trials neither choice word was primed (the remaining 30 trials in each block did not contain a target, but the prime was repeated in the 2-AFC test). The number of target primed and foil primed trials were reversed for the strong negative diagnosticity. For the weak positive diagnosticity, the target was primed in 60 trials, the foil was primed in 30 trials, and in 18 trials neither choice word was primed. Again, 30 trials in each block did not contain a target, but the prime was repeated in the 2-AFC, and for the weak negative diagnosticity, the number of target primed and foil primed trials was reversed.

Results and Discussion

The results for the weak (upper panels) and strong (lower panels) prime diagnosticities during the first two blocks, before any change in diagnosticity, are presented in Figure 6. The results during the last two blocks, after a change in prime diagnosticity, are shown in Figure 7. The representation of the results in these figures is identical to that used for Experiment 1 with one exception: The proportion of prime compatible choices for trials in which no target was presented (accuracy is not defined in this case) is plotted alongside the data for target primed trials as a vertical dotted line.

Trials in which a target was present. Figures 6 and 7 show that the results of Experiment 2 generally replicate those of Experiment 1. In addition, greater strength of diagnosticity produced larger effects. A 2 (diagnosticity strength) $\times$ 2 (direction of the first diagnosticity) $\times$ 2 (direction of the current diagnosticity) $\times$ 3 (prime type) $\times$ 2 (prime duration) ANOVA was applied to the conditions that included a target word, the results of which are presented in detail in Table B1 in Appendix B. As is evident from comparing the left and right panels in Figures 6 and 7, the direction of the current diagnosticity influenced participants choices as in Experiment 1, with positive diagnosticities leading to more prime compatible choices and negative diagnosticities leading to fewer prime compatible choices. This main effect interacted with several other factors. The interactions of the previous diagnosticity with the current diagnosticity can be seen by comparing corresponding panels in Figures 6 and 7: Similar to Experiment 1, current diagnosticity effects were attenuated after a change in diagnosticity.

The interaction of the current diagnosticity with the prime type shows that performance for the three prime types is differentially affected by diagnosticity: As can be seen in the figures, performance in target primed trials tends to be higher for positive than for negative diagnosticities, whereas the opposite is true for performance in foil primed trials. A similar interaction exists between the previous diagnosticity and prime type. This interaction reflects the fact that accuracy is generally higher for target primed trials when the previous diagnosticity was positive, whereas the opposite is true for foil primed trials. This lasting influence of the first diagnosticity helps explain the strong attenuation of current diagnosticity effects after a change in diagnosticity; because the change was always from positive to negative or from negative to positive,

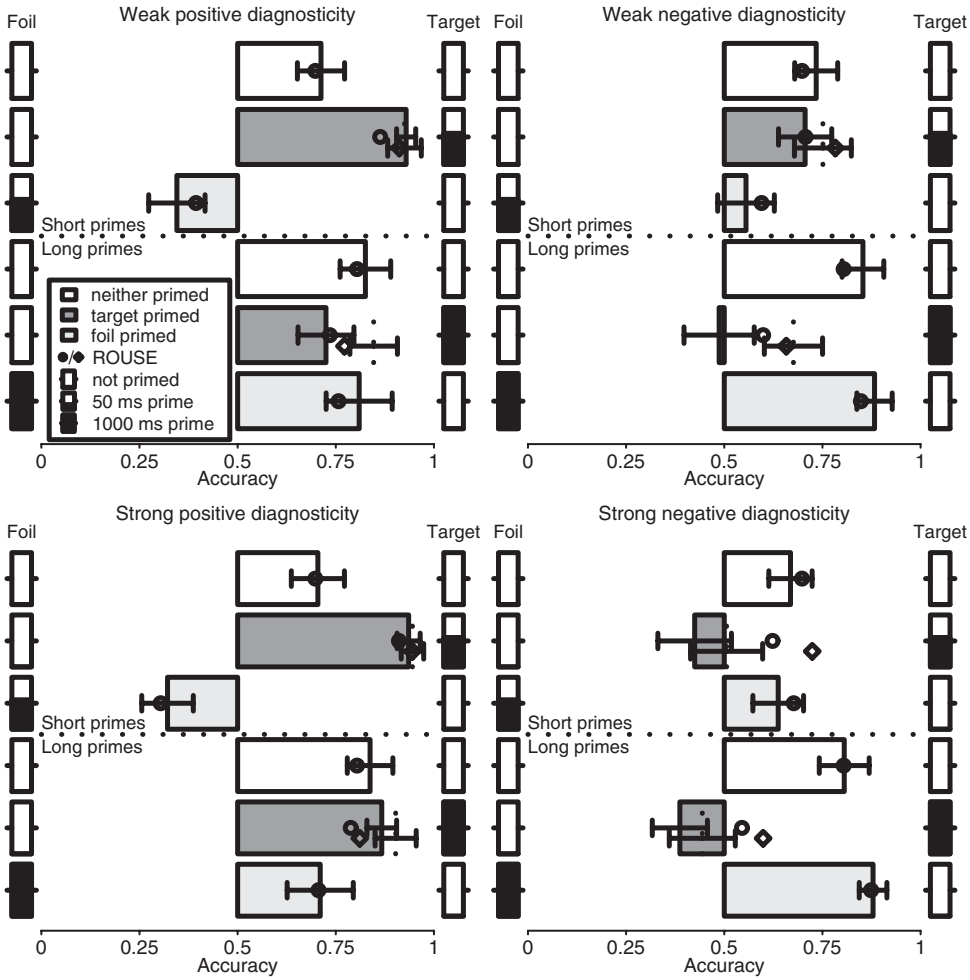

Figure 6. Accuracy for positive (left panels) and negative (right panels) prime diagnosticity at the beginning of the test phase (i.e., before any change in diagnosticity) of Experiment 2. The top two panels show accuracy for the weak diagnosticities, and the bottom two panels show accuracy for the strong diagnosticities. The vertical dotted lines plotted with the target primed conditions indicate the proportion of prime-compatible choices when no target was flashed (because of the missing target, accuracy is not defined for these conditions). The error bars indicate the 95% confidence intervals (the error bars for the conditions in which no target was flashed are shifted slightly to avoid clutter). The open circles and diamonds are responding optimally with unknown sources of evidence (ROUSE) fits for conditions with and without a flashed target, respectively, and are discussed later in a special section after all experiments have been presented. See caption for Figure 2 for other details.

the cumulative effect of the previous diagnosticity and the current diagnosticity was necessarily in the direction of neutral diagnosticity.

The interaction of the current diagnosticity with prime type is further modulated by the strength of the diagnosticity as evidenced by the significant three-way interaction between these factors (comparing the upper and lower panels of Figures 6 and 7 shows that the effects are larger for stronger diagnosticities). The significant main effects of prime type and prime duration and their interaction replicates previous findings and can be clearly seen in the figures, but the effect of the three-way interaction with strength is too small to account for much of the variance in the data ($\eta_p^2 = .05$; cf. Footnote 2).[4]

Aside from effects of diagnosticity, the findings replicate earlier research and support previous conclusions: Large differences were seen for short and long primes, suggesting that people underdiscounted evidence for brief (low salience) primes and overdiscounted evidence for long (high salience) primes. Diagnosticity clearly modulated these effects because there was a larger tendency to choose the primed alternative when there was a positive

[4] Similarly, the interaction between current diagnosticity and prime duration, and the three-way interactions of these factors with the first diagnosticity and with prime type, were also significant, but these effect were comparatively small ($\eta_p^2 = .09$ for the two-way interaction and .08 for both three-way interactions, compared with .57 for the interaction of diagnosticity with prime type and .22 for the interaction of these two factors with strength). The five-way interaction was also statistically significant, but that effect was even smaller ($\eta_p^2 = .05$).

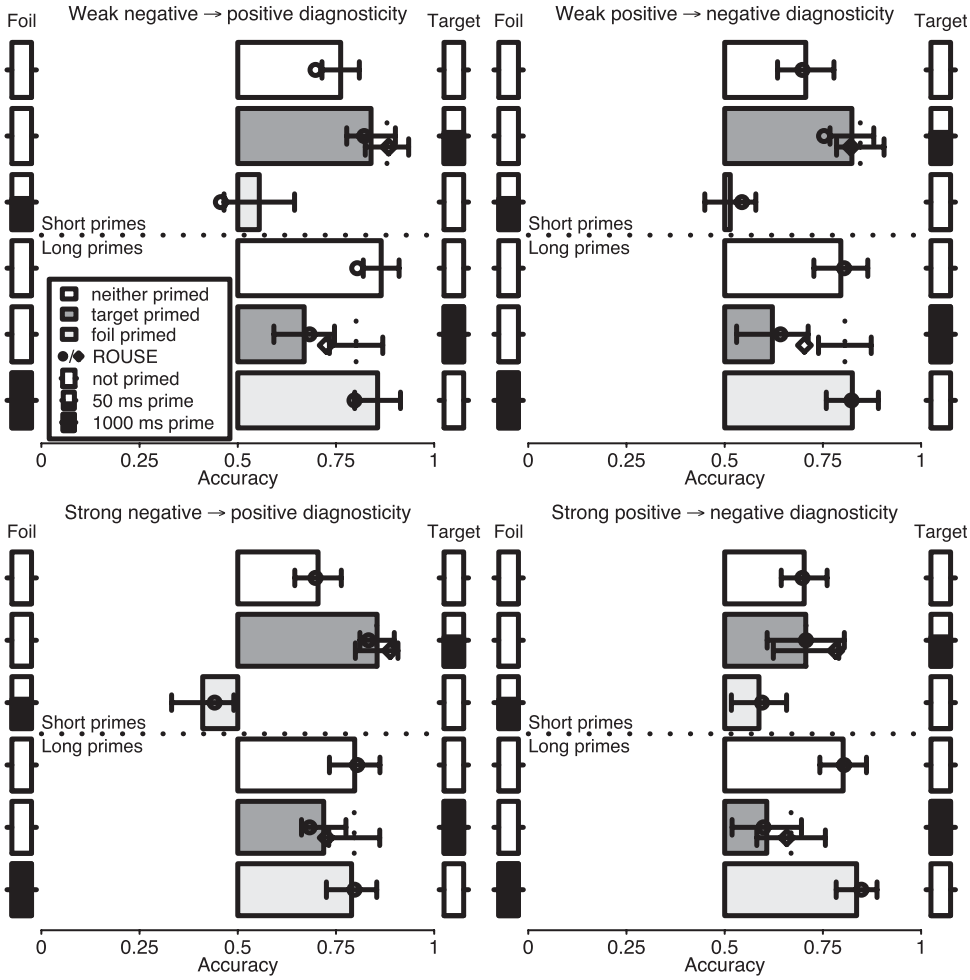

Figure 7. Accuracy for positive (left panels) and negative (right panels) prime diagnosticity after diagnosticity has changed (from negative to positive or vice versa) in Experiment 2. The top two panels show accuracy for the weak diagnosticities, and the bottom two panels show accuracy for the strong diagnosticities. ROUSE = responding optimally with unknown sources of evidence. See caption for Figure 6 for details.

prime diagnosticity and less of a tendency to choose the primed alternative when there was a negative diagnosticity. These effects were larger when primes increased in diagnosticity.

In summary, we replicated the prime diagnosticity effects and change of diagnosticity effect from Experiment 1 and once again demonstrated that increasing evidence discounting with increasing prime duration is a general finding regardless of diagnosticity manipulations. Furthermore, we found that the strength of the prime diagnosticity is important, as would be expected if diagnosticity affects implicit evidence evaluation rather than inducing a simple strategic response policy. Next we turn to the conditions in which no target was presented, which provide even stronger evidence against the use of such a policy.

Comparing trials with and without a target presentation. With our setup, target primed trials are identical to target absent trials in all respects except for the target presentation and feedback (which may be negative even if the primed alternative is chosen or positive even if it is not chosen for target absent trials). In other words, all aspects of the stimulus presentation were identical between the target primed trials and the trials without a target except that in the latter, the target presentation was replaced by a blank screen. Thus, to analyze the effect of target presentation, we compared the target primed trials with target absent trials in a 2 (diagnosticity strength) × 2 (direction of the first diagnosticity) × 2 (direction of the current prime diagnosticity) × 2 (prime duration) × 2 (target presence) ANOVA. The statistically significant effects of this ANOVA are listed in Table B2 in Appendix B.

The design included accuracy feedback in the no-target conditions that was on average in keeping with the current diagnosticity for primed trials that did contain a target (i.e., feedback in relation to the ratio of the target-primed trials vs. foil-primed trials). Thus, any strategy in relation to choosing or not choosing primed alternatives would seem equally effective in the no-target trials. Therefore, in the absence of any presented target, it seems reasonable to expect that the results of any strategic or sophisticated guessing strategies would be enhanced. If a strategic response policy explains the prime diagnosticity effects with actual targets, then the tendency to choose primed words in the no-target conditions

should clearly indicate the direction and magnitude of that policy (e.g., less than a .5 probability of choosing the primed alternative if the policy was against primed words).

By far the strongest effect was that of the direction of the current prime diagnosticity ($\eta_p^2 = .86$), reflecting the fact that for both target primed and target absent trials the proportion of prime compatible choices (for target primed trials this measure is identical to accuracy) was higher for positive diagnosticities than for negative diagnosticities (see Figures 6 and 7). This main effect of diagnosticity interacted with strength (i.e., it was stronger for stronger diagnosticities) but only marginally with prime duration. Despite these strong effects of prime diagnosticity, the tendency was never against choosing the primed word in the no-target conditions, regardless of the direction or magnitude of prime diagnosticity. In fact, in all but the strong negative prime diagnosticity conditions before a switch in diagnosticity (cf. lower right panel of Figure 6) the confidence intervals for the no-target conditions shown in Figures 6 and 7 only include accuracies above .5, indicating significant tendencies to choose the primed alternative in the absence of target information (including some situations where the diagnosticity was negative). In other words, even when the prime was 4 times as likely to indicate the incorrect choice than it was to indicate the correct choice, participants showed no tendency against choosing the primed alternative when no target was present.

To explain these results with strategic factors, one would have to assume an extremely conservative change in the decision criterion, particularly for negative prime target diagnosticities. A more parsimonious explanation is that prime diagnosticity effects are realized through modulations in evidence discounting. The fact that participants tended to show a preference for the primed alternative in the no-target conditions even for salient primes may seem surprising. However, this finding replicates earlier results and is a prediction that naturally falls out of the ROUSE model (Huber, Shiffrin, Lyle, & Quach, 2002). We discuss this aspect of the model, as well as an implementation of the idea that prime diagnosticity effects are realized through changes in evidence discounting, in detail in the modeling section after all experiments have been presented.

A second result was also quite nonintuitive: The prime was chosen more often in the no-target conditions than in the target primed conditions (i.e., chosen more often when the prime was not repeated as the target).[5] This fact is reflected in a significant main effect for target presence, which was modulated by two-way interactions with strength (the difference was larger for the weak diagnosticity), first diagnosticity (the difference was larger if the first diagnosticity was negative), and prime duration (the difference was larger for long prime presentations). The three-way interaction between strength, prime duration, and target presence was also significant, reflecting the fact that the larger difference for the long prime presentations was more pronounced at weak diagnosticities. With the exception of the significant main effects of first diagnosticity and prime duration, other statistically significant effects were comparatively small (cf. Appendix B2).

Experiment 3: Diagnosticity Cued by Prime Color

The phenomena of classical conditioning and implicit learning (e.g., Reber & Allen, 1978) show that behavior of humans and other animals can be strongly influenced by cues in the environment that have been associated with the task at hand, even if the association is not obvious. The purpose of this experiment was to investigate the degree to which prime diagnosticity effects could be extended to other cues in the environment that might have a predictive value, even when the overall prime diagnosticity was neutral. If prime diagnosticity effects stem from a response strategy, we would expect that any cue associated with a particular diagnosticity would trigger the associated strategy so long as the cue was sufficiently salient. Therefore, we presented primes in one of two different colors, and the color on any particular trial indicated whether a prime would have positive or negative diagnosticity on that trial.

Method

Participants. Forty-one undergraduate students at Indiana University Bloomington participated in exchange for introductory psychology course credit.

Materials and equipment. The materials and equipment were the same as for Experiment 1 (only the six-letter word pool was used in this experiment).

Procedure. The procedure was identical to that of Experiment 1 with the following exceptions: The mean target duration was 40 ms ($SE = 2$). In both the calibration and the test phase, words were sampled (without replacement) from the same six-letter word pool. The test phase consisted of four blocks of 92 trials each, with 12 neither primed trials and 40 trials each of target primed and foil primed trials. The primes were presented in either red or blue according to the following regimen: Half of the neither primed trials were presented in red, the other half were presented in blue. Thirty of the target primed trials in each block were presented in red, and the remaining 10 were presented in blue. These proportions were reversed for the foil primed trials. Thus, there was an equal number of red and blue primes and an equal number of target primed and foil primed trials in the experiment. However, prime color was highly diagnostic, with 30 out of 46 red primes indicating the correct choice and 30 out of 46 blue primes indicating the incorrect choice. Participants were not given any explicit information regarding the significance of prime color.

[5] Among other reasons for surprise, Huber, Shiffrin, Lyle, and Quach (2002) manipulated target duration with neutral prime diagnosticity and found that the proportion of prime-compatible choices for target primed conditions declined with target duration and was lowest for the case when no target was presented (although, as pointed out above, participants still tended to select the primed alternative more often than the unprimed alternative, even for salient primes). However, that experiment presented a mask in the no-target condition, rather than the blank screen that was used in the current experiment. We were able to account for the present results, and hence the difference from those of Huber, Shiffrin, Lyle, and Quach, by assuming that a blank screen in place of the target or a mask produces overall less (random) visual noise. We provide details of how we implemented this idea in ROUSE in a separate modeling section later in the article. Other experiments (not presented here) support this account: Weidemann (2006) found that the insertion of a blank screen is crucial for the increased number of prime compatible responses, whereas the simple substitution of the target with a noninformative consonant string leads to a lower proportion of prime compatible responses.

Results

Figure 8 shows the results split by prime diagnosticity (i.e., prime color). It is evident from the figure that the results for the two diagnosticities are virtually identical, and a 3 (prime type) × 2 (prime duration) × 2 (prime diagnosticity) ANOVA confirmed that no effects involving prime diagnosticity were significant. As in previous experiments, the effects of prime type, $F(2, 80) = 13.06$, $MSE = 0.45$, Huynh–Feldt $\varepsilon = .71$; prime duration, $F(1, 40) = 50.99$, $MSE = 1.49$; and their interaction, $F(2, 80) = 86.52$, $MSE = 2.35$, Huynh–Feldt $\varepsilon = .69$ ($p < .01$ for all tests; Huynh–Feldt ε corrected where applicable; Huynh & Feldt, 1976) were all significant.

Discussion

Experiment 3 replicated the usual priming effects, showing a preference for the primed alternative for brief (underdiscounted) primes and a preference against the primed alternative for long (overdiscounted) primes. This result is not surprising, given the fact that Weidemann et al. (2005) also found priming effects when prime and target were presented in different colors. Indeed, even arguably more drastic differences between the prime and target appearances do not reduce these priming effects either: Huber et al. (2001), for example, manipulated letter case between prime and target and found similar priming effects regardless of whether prime and target matched in case. These results suggest that the features used to identify the target are higher level abstract features rather than low-level aspects pertaining to the details of the presented stimulus (see Sanborn, Malmberg, & Shiffrin, 2004, for an interpretation of this finding as a masking effect).

This experiment was identical to the beginning of Experiment 1 (i.e., before a change in prime diagnosticity) except that prime diagnosticity was indicated by prime color. The data replicate those from the neutral prime diagnosticity of that experiment (shown in Figure 2) despite the information contained in the prime color. The fact that the diagnosticity effects observed in Experiment 1 disappeared in Experiment 3 provides additional evidence against a strategic account of prime diagnosticity effects: Even though prime color was not directly relevant to the task, it was a very salient feature that could have lent itself to a strategic use of prime diagnosticity. Nevertheless, we have no evidence that participants even noticed the relationship between prime color and diagnosticity.

It is interesting to compare these results with those of Hutchison (2007), who used a naming task with semantically related primes and found a small effect of prime diagnosticity on response time for a subset of his participants (those with "high attentional control") when prime diagnosticity was cued by prime color. In his experiments, participants were explicitly informed (in one experiment before every trial) about the correspondence between prime color and prime diagnosticity, which was likely to have encouraged strategic responding. Given these instructions and the fact that prime diagnosticity was only indicated by an arbitrary cue (prime color) that has no other relevance for the task, Hutchison's conclusion that prime diagnosticity effects "from this task can be taken as a signature of conscious expectancy generation" (p. 660) seems premature. We do not dispute Hutchison's interpretation of his results in terms of expectancy generation. Instead, we are concerned by the implication that prime diagnosticity effects are in general a signature of strategic effects.

Experiments 1 and 2 demonstrated that prime diagnostic can be successfully associated with a temporal marker (i.e., the current block of trials), and the no-target conditions of Experiment 2 suggested that diagnosticity was not implemented as a general response policy for or against choosing primed alternatives. Experiment 3 provided further evidence against a strategic policy, considering that prime diagnosticity was not associated with the salient cue of prime color. However, if prime diagnosticity affects evidence discounting, then it may be possible to associate prime diagnosticity with some other cue that is known to affect how evidence is accumulated.

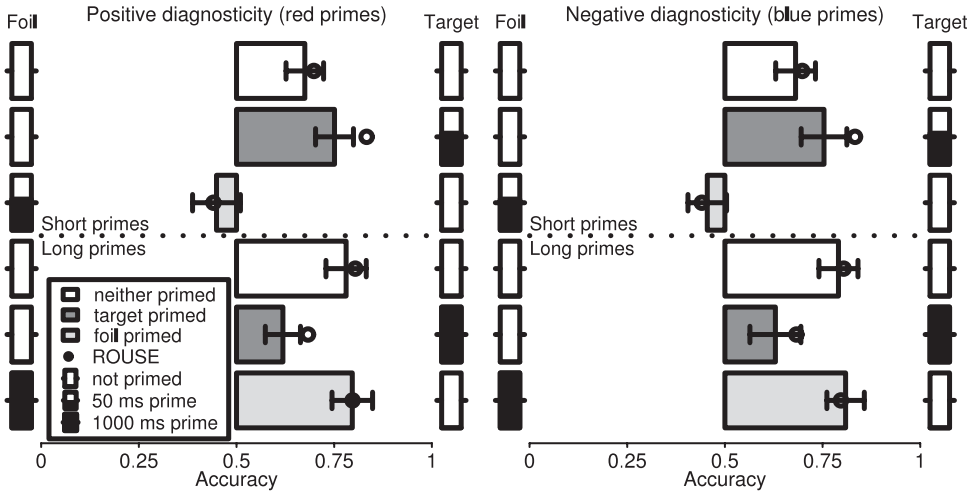

Figure 8. Accuracy for positive (left panel; red primes) and negative (right panel; blue primes) prime diagnosticity in Experiment 3. ROUSE = responding optimally with unknown sources of evidence. See caption for Figure 2 for details.

Experiment 4: Diagnosticity Cued by Prime Duration

All of our prior work with neutral diagnosticity found that prime salience is a strong cue that modifies the level of discounting, but so far we have obtained no evidence that surface cues, such as color or letter case, produce different levels of discounting (see also Huber et al., 2001). From this perspective, the failure to find a relationship between color cuing and prime diagnosticity in Experiment 3 was not surprising. On the other hand, the perceptual system might indeed be sensitive to the cuing of prime diagnosticity when that cue is provided by the duration of primes (a variable that influences evidence discounting). That possibility is explored in our last experiment.

Method

Participants. One hundred and eighteen undergraduate students at Indiana University Bloomington participated in exchange for introductory psychology course credit. Thirty participants each were assigned to the both positive and both negative diagnosticity condition, and 29 participants each experienced the positive–negative and the negative–positive conditions (see below).

Materials and equipment. The materials and equipment were the same as for Experiment 1 (only the six-letter word pool was used in this experiment).

Procedure. The procedure was identical to that of Experiment 1 with the following exceptions: The mean target duration was 44 ms ($SE = 2$). In both the calibration and the test phase, words were sampled (without replacement) from the same six-letter word pool. The test phase consisted of six blocks of 80 trials each,[6] and participants were assigned to one of four diagnosticity conditions: In the both positive condition, the prime was likely to signal the correct choice regardless of prime duration (for both short and long prime duration there were 8 neither primed, 24 target primed, and 8 foil primed trials in each block). In the both negative condition, the prime was likely to signal the incorrect choice regardless of prime duration, and the proportions of target primed and foil primed trials were reversed.

In the positive–negative and negative–positive conditions, primes were only diagnostic contingent on their duration. In the former condition, the diagnosticity was positive for short prime durations and negative for long prime durations (with the above proportions), and these diagnosticities were reversed in the latter condition. Across all trials, however, the prime diagnosticity was neutral in these two conditions.

Results and Discussion

Figure 9 shows the data for the different diagnosticity conditions. The results in the top two panels replicate those of Experiment 1 (cf. Figure 3). As is evident from the bottom two panels of Figure 9, prime diagnosticity also had a strong influence on identification performance when it was associated with prime duration, even though the overall prime diagnosticity was neutral when collapsing across prime duration. A 4 (diagnosticity condition) × 3 (prime type) × 2 (prime duration) ANOVA revealed significant main effects of prime type, $F(2, 228) = 55.05$, $MSE = 1.03$, Huynh–Feldt ε (.57) corrected $p < .01$ (Huynh & Feldt, 1976), and priming duration, $F(1, 114) = 203.72$, $MSE = 1.85$, $p < .01$. As in the previous experiments, the interaction of prime type and prime duration was also significant, $F(2, 228) = 242.25$, $MSE = 3.07$, Huynh–Feldt ε (.47) corrected $p < .01$, as was the interaction between these two factors and the diagnosticity condition, $F(6, 228) = 23.42$, $MSE = 0.30$, Huynh–Feldt ε (.47) corrected $p < .01$. Furthermore, the influence of the diagnosticity condition also manifested itself in the significant interaction with prime type, $F(6, 228) = 46.09$, $MSE = 0.86$, Huynh–Feldt ε (.57) corrected $p < .01$.

The most important findings were those from the mixed conditions in which the direction of diagnosticity was cued on a trial-by-trial basis by prime duration. In contrast to the data from Experiment 3, effects of diagnosticity cuing were clearly visible: For the lower panels of Figure 9, in which different diagnosticities were associated with different prime durations, comparisons of a condition on the left with the same condition on the right reveal sizable cuing effects. Note that these results were obtained even though the overall diagnosticity in these mixed conditions was neutral. To be more specific, trials with a particular prime duration associated with a positive prime diagnosticity produced an increased preference for the primed alternative, whereas trials with a prime duration associated with a negative prime diagnosticity showed a decreased preference for the primed alternative. Most remarkably, this occurred even for the short prime duration, which is near the threshold of prime awareness (i.e., the association between prime duration and diagnosticity exists even for primes that were not salient). Furthermore, there seems to be relatively little cross-talk between the diagnosticity of one prime duration and that associated with the other prime duration: If we compare corresponding pure conditions in the upper panels of Figure 9 with the equivalent mixed condition in the lower panels, the results look very similar. Only when comparing the lower right panel of Figure 9 with the top part of the upper right panel and the lower part of the upper left panel is a small amount of cross-talk between the two diagnosticities discernable. Thus, the system is remarkably tuned to the diagnosticity associated with a given duration of primes and does not seem to be influenced much by the overall diagnosticity of the entire block of trials. This strong contrast to the data observed in Experiment 3 lends credence to the claim that the mechanism behind prime diagnosticity is related to factors that affect evidence discounting (e.g., prime duration) but not to arbitrary factors, even if they are salient (e.g., prime color).

Modeling Prime Diagnosticity in Short-Term Repetition Priming

Experiments 1–4 yielded a complex set of data that was well accounted for by the ROUSE model (as shown by the ROUSE fits superimposed on the data in Figures 2–4 and 6–9). We now turn

[6] The number of trials per block was different from that for Experiment 3 for several reasons: Experiment 4 was run before Experiment 3, and the additional constraint in Experiment 3 to include the same number of target primed and foil primed trials for both prime durations led us to increase the number of trials per block from that used in Experiment 4 to have a similar number of observations per condition. In Experiment 3 we also decreased the total number of trials from that used in Experiment 4 because learning of prime diagnosticity was fast even when it was cued by prime duration.

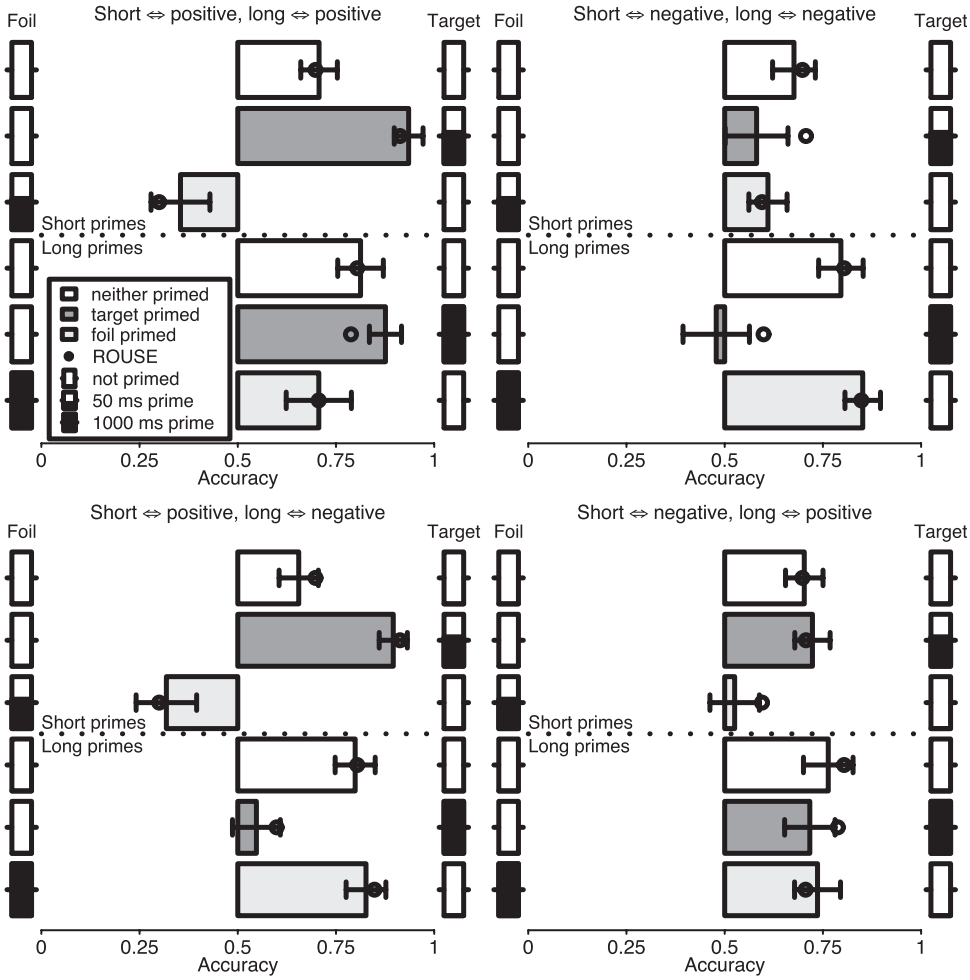

Figure 9. Accuracy for the different diagnosticity conditions of Experiment 4. The diagnosticity for the short and long primes are indicated on the top of each panel. ROUSE = responding optimally with unknown sources of evidence. See caption for Figure 2 for details.

to a description of the ROUSE model and its extension to manipulations of prime diagnosticity. The basic idea of the original model is that the primes are merged with the target percept (i.e., source confusion), but prime-compatible evidence is discounted, the degree of discounting being a function of prime saliency. If evidence discounting following brief primes is insufficient, but discounting is excessive following salient primes, ROUSE can account for the preference for a choice word that repeats a brief prime and the preference against a choice word that repeats a long prime (both relative to the neither primed baseline).

The original version of ROUSE (Huber et al., 2001) did not specify the manner in which prime diagnosticity affects performance—this was not an oversight but was merely because the previous experiments to which ROUSE was applied all used neutral diagnosticity. Of the various ways that effects of prime diagnosticity could be modeled in the ROUSE framework, several pieces of evidence led us to believe that changes in evidence discounting could effectively capture these results. Most notably, Experiments 3 and 4 showed that prime diagnosticity can be cued by prime duration, a cue that is relevant to evidence discounting, but not by prime color, which does not influence discounting (we simply modeled Experiment 3 by not allowing discounting to change with color-cued prime diagnosticity). In addition, Experiment 2 demonstrated that even when prime duration and/or prime diagnosticity induced a preference against primed words in the target present conditions, people still tended to choose the primed alternative in the target absent conditions.

Regarding changes in prime diagnosticity, Experiments 1 and 2 demonstrated that prime diagnosticity produces large effects as long as diagnosticity is constant—but much smaller effects after a change in the direction of diagnosticity (cf. Figures 4 and 7). We accounted for this effect by positing that the amount of evidence discounting is jointly influenced by the current prime diagnosticity as well as the previous one (albeit to a lesser degree).

With the above mentioned additions to the original ROUSE model, plus the assumption that there is less perceptual noise when a blank screen is presented instead of a target (Experiment 2), all four reported experiments were simultaneously fit by ROUSE with a common set of parameters. Next, we provide the specifics of the model.

ROUSE

In ROUSE, each choice word is represented by a vector of binary features. A target percept is formed and compared with these choice vectors. Thus, for each choice-word vector, a feature is activated (a match with the percept) by the prime (with probability α), the target (with probability β), or noise (with probability γ) if it is shared with the respective source. Source confusion refers to the assumption that the system only has access to the state of the activated features for the two choice words and cannot know which source activated a given feature. Source confusion produces a tendency to respond with a primed word because features that are activated by the prime cannot be distinguished from features activated by the target flash. Figure 10 illustrates this aspect of the model.

A second part of the model is a Bayesian decision process that takes into account estimated probabilities of feature activations by the different sources to determine the choice most likely to be the target. Each activated feature contributes positive evidence for the corresponding choice word, but if it is known that the feature was also present in the prime, the system lowers the amount of positive evidence contributed by that feature (because the source of activation could have been the prime instead of the target flash). This lowering of evidence in the face of alternate sources that may have caused an observation is termed *discounting* and is also known as *explaining away* in the study of Bayesian belief networks (e.g., Wellman & Henrion, 1993).

It is important to note that this discounted level of evidence still constitutes positive evidence for the corresponding choice word rather than evidence against that word. To illustrate this point, imagine that the features of a word were simply its letters and that a particular letter (e.g., "E") in one of the alternatives of the 2-AFC were activated (i.e., the system has evidence that an "E" was presented, which in turn constitutes evidence for the choice word that contains an "E"). If the prime did not contain an "E," this constitutes strong evidence in favor of the associated choice word (because it could only have been activated by the target or noise). If, however, the prime also contained an "E," this percept could stem entirely from the prime presentation, and thus the evidence for the corresponding choice word is less strong but still positive (i.e., the fact that prime also contained an "E" does not constitute evidence against the corresponding choice word). Thus, no matter how much discounting is applied, the resultant evidence still favors the choice word containing this shared feature. Because discounting can at most lower evidence compatible with the primes, it can only reverse preference to the extent that it also serves to lower (primed) target evidence. When no target is presented evidence discounting cannot overreach in this way and no preference reversal results, even if evidence discounting for the prime activated features is strong.

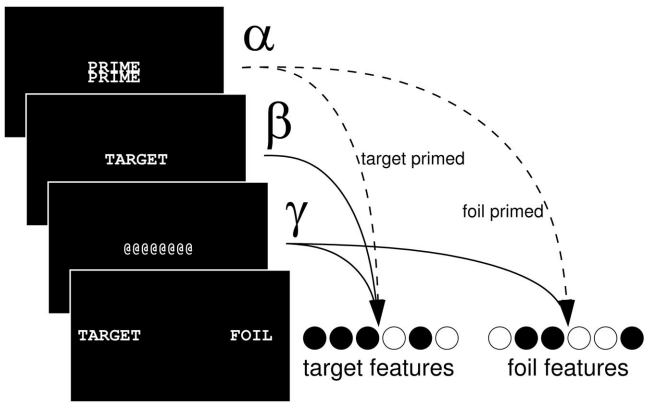

Figure 10. Source confusion in responding optimally with unknown sources of evidence (ROUSE). Binary target and foil features can be activated by the prime (with probability α), the target (with probability β), or noise (with probability γ). Feature activation by the prime presentation depends on the similarity of the prime to the choice words, which is assumed to be zero for unrelated primes and one for identical primes (this is denoted by the dashed arrows). In the actual simulations, each choice word was represented by 20 binary features. The prime is repeated in the figure solely for consistency with the prime presentations in the experiments presented here.

If the true activation probabilities were known to the system, it would be possible to discount evidence optimally so that no preference for or against a primed target would result. A central assumption of ROUSE is that the true activation probabilities are not known and have to be estimated. The relationship between the actual probability that a feature is activated by the prime (α) and its estimate (α') is crucial to account for the data. If $\alpha' < \alpha$, discounting is insufficient, and a tendency to choose the primed alternative remains. Prior fits of the model were consistent with $\alpha' < \alpha$ when accounting for brief and unattended primes (e.g., Huber et al., 2001; Weidemann et al., 2005). If, on the other hand, $\alpha' > \alpha$, discounting is excessive, and there is a tendency not to choose the primed alternative. Prior fits of the model were consistent with $\alpha' > \alpha$ when accounting for long and attended primes (e.g., Huber et al., 2001; Weidemann et al., 2005). To elaborate, excessive discounting can result in a relative preference against the primed alternative when features from the unprimed alternative are activated (because the evidence from these features is not discounted).

The odds (i.e., ratio of posterior likelihoods) for a choice of the target over a choice of the foil is given by the following Bayesian calculation (Huber et al., 2001):

$$\phi\left(\frac{\mathrm{T}}{\mathrm{F}}\right) = \frac{\prod_{i=1}^{N} \frac{p(V\{\mathrm{T}_i\}|\mathrm{T\ is\ target})}{p(V\{\mathrm{T}_i\}|\mathrm{T\ is\ foil})}}{\prod_{j=1}^{N} \frac{p(V\{\mathrm{F}_j\}|\mathrm{F\ is\ target})}{p(V\{\mathrm{F}_j\}|\mathrm{F\ is\ foil})}}, \quad (1)$$

under the assumption that each feature contributes an independent source of evidence. T and F in Equation 1 refer to the target and foil word; $V(T_i)$ and $V(F_i)$ represent binary values denoting the state of activation of the i-th feature of the target and the foil, respectively.

Each product term in the numerator and denominator of Equation 1 is the evidence provided by one of the N features of the two choices (as in previous applications of ROUSE—e.g., Huber et al., 2001; Weidemann et al., 2005—we set $N = 20$ in our simulations). The product gives the posterior likelihood that the target (numerator) and the foil (denominator) were presented during the target flash. Each feature can only contribute one of three evidence values, depending on the state of the feature activation and whether the prime could potentially have activated the feature (Huber et al.,

2001). The evidence values for all possible combinations of these factors are illustrated in Figure 11. Note that, as explained above, the evidence is always in favor of the corresponding choice word for any feature that is activated. The discounted evidence (bottom right corner of Figure 11) is smaller than the undiscounted evidence (top right corner of Figure 11) but still in favor of the corresponding choice word (i.e., the odds ratio is greater than one for $\alpha' < 1$).

For most experimental designs it is appropriate to assume equal prior probabilities for the two choice words, and in this case a normative decision process chooses the word with the greater likelihood ratio (i.e., the target would be chosen if the odds, as calculated with Equation 1, are greater than 1, and the foil would be chosen if they are less than 1—a random choice could be made in the case of equal likelihoods).

Prime diagnosticity and evidence discounting. In ROUSE, the amount of evidence discounting is determined by the estimated probability that features were activated by the prime (α'). In the following, when primes have neutral diagnosticity, we term the value of α' in a given condition α'_N. Note that to maximize performance, a completely valid prime should have $\alpha' = 0$ (no discounting), and a completely invalid prime should have $\alpha' = 1$ (complete discounting). As a way to parametrize the system's estimate of prime diagnosticity, we introduce the parameter *de* (for diagnostic evidence). We propose a simple model in which diagnostic evidence is used to calculate the proportion of change from the neutral α'_N to the 0 or 1 endpoints: For positive diagnosticity, $\alpha' = \alpha'_N - \alpha'_N \times de$, and for negative diagnosticity, $\alpha' = \alpha'_N + (1 - \alpha'_N) \times de$. If we use α'_D as a binary indicator of the endpoint values (i.e., for positive prime diagnosticities, α'_D is equal to 0; and for negative diagnosticities, it is equal to 1), these equations can be written as a weighted average between α'_N and α'_D as shown in Equation 2:

$$\alpha' = (1 - de) \times \alpha'_N + de \times \alpha'_D. \quad (2)$$

For neutral prime diagnosticity, $de = 0$, and $\alpha' = \alpha'_N$ for any value of α'_D.

Although *de* could be left as a free parameter, and allowed to vary with conditions, it turns out that a very simple estimate of *de* that is related to the actual strength of diagnosticity provides a good account of the data:

$$de = \frac{\max(t, f)}{t + f} - \frac{(t + f)/2}{t + f}, \quad (3)$$

where *t* and *f* are the number of target primed and foil primed trials, respectively. The last fraction of Equation 3 is, of course, equal to .5 regardless of the number of target primed and foil primed trials. This equation is a simple (but post hoc) way of mapping strength of diagnosticity to *de* with the constraint that $de = 0$ if $t = f$ (i.e., neutral prime diagnosticity). For other relations of *t* to *f*, *de* ranges from 0 to a maximum of .5. Equation 3 produces *de* values of .25 for the diagnostic conditions of Experiments 1 and 4, .17 for the weak diagnostic conditions of Experiment 2, and .30 for the strong diagnostic conditions of Experiment 2.

The model defined by Equations 2 and 3 assumes a piecewise linear relationship between the proportion of target primed trials and the resulting level of discounting (α') with slopes of α'_N and $1 - \alpha'_N$ for positive ($t > f$) and negative ($t < f$) diagnosticities, respectively. The midpoint in this piecewise linear relationship when $t = f$ corresponds to neutral diagnosticity and is defined by the one free parameter, α'_N. As the proportion of target primed trials increases or decreases from this neutral level, discounting decreases or increases linearly as defined above. To maximize performance, α' should approach the theoretical extremes of 0 or 1 as the proportion of target primed trials approaches 100% or 0%, respectively. However, because *de*, as defined in Equation 3, can only assume a maximum value of .5, α' approaches values that split the difference between the α'_N midpoint and the theoretical extremes of 0 and 1.

The assumptions that the relationship between discounting and the proportions of target and foil primed trials is linear and that the maximum and minimum levels of evidence discounting split the difference between neutral discounting and the theoretical extremes are admittedly somewhat post hoc. Nevertheless, this simple model produces a remarkably accurate account of the various diagnosticity manipulations and requires no additional free parameters (standard implementations of ROUSE already include α'_N as a free parameter).

The data suggest that observers adapted very quickly to the current prime diagnosticity (cf. Figure 5). Therefore, we calculated *de* on the basis of the total number of target primed and foil primed trials within the current block of trials.

Changes in prime diagnosticity. Given fast learning of prime diagnosticity in the beginning of the experiment, one might expect a rapid shift to a new level of discounting after a change in diagnosticity. Our learning analysis of the data from Experiment 1 suggests that adaptation was indeed fast, even after a change in prime diagnosticity. However, subsequent adjustments to diagnosticity failed to achieve as strong an effect as the initial adjustment to the same level of diagnosticity at the beginning of the experiment. We modeled this by supposing that the system calculates α' after a change in diagnosticity as a weighted average of the values that would have been appropriate for the current and prior diagnosticities had they both occurred at the beginning of the experiment:

$$\alpha' = (1 - prev) \times \alpha'_C + prev \times \alpha'_P, \quad (4)$$

Feature appeared in prime	State of feature activation: OFF	State of feature activation: ON
NO	$\frac{(1-\gamma')(1-\beta')}{(1-\gamma')} = (1-\beta')$ less than 1.0	$\frac{1-(1-\gamma')(1-\beta')}{1-(1-\gamma')}$ greater than1.0
YES	$\frac{(1-\gamma')(1-\alpha')(1-\beta')}{(1-\gamma')(1-\alpha')} = (1-\beta')$ less than 1.0	$\frac{1-(1-\gamma')(1-\alpha')(1-\beta')}{1-(1-\gamma')(1-\alpha')}$ greater than, but closer to, 1.0

Figure 11. Feature likelihoods in responding optimally with unknown sources of evidence (ROUSE). Each feature of the target and foil choice alternatives contributes one of three possible evidence values. An evidence value of 1 is neutral, values below 1 constitute evidence against the corresponding alternative (the smaller the value, the bigger the evidence), whereas values above 1 constitute evidence for the corresponding alternative (the bigger the value, the bigger the evidence). In these calculations, the system's estimates of the probabilities of feature activation by the prime presentation, target presentation, and visual noise (i.e., α', β', and γ', respectively) are used instead of the actual probabilities (i.e., α, β, and γ).

where α'_C is the value appropriate for the current diagnosticity (assuming no prior change), α'_P is the value for the previous prime diagnosticity (again, assuming no prior change), and *prev* is a parameter that varies between 0 and 1 to determine the relative influence of the previous diagnosticity. We kept *prev* in Equation 4 fixed across experiments even though several details (e.g., the number of trials per block) varied across experiments.[7]

Conditions without a target. Previous experiments and analyses with ROUSE (Huber, Shiffrin, Lyle, & Quach, 2002) revealed a highly nonintuitive effect that goes a long way toward explaining the conditions without target presentation: When no target is presented and noise is low, ROUSE predicts a preference for the primed alternative, even with strong evidence discounting. However, this explanation in terms of lack of discounting efficacy in the absence of a target cannot account for our finding that there were fewer prime compatible choices when the target was primed than when no target was present. Unlike these previous experiments, our Experiment 2 eliminated the target by replacing it with a briefly presented blank screen. We propose a simple assumption to account for our data: Compared with a continuous stream of visual stimuli, the insertion of a blank screen results in overall less visual noise in the system. Accordingly, we allowed a free parameter (*b* for blank) that could range between 0 and 1 to scale the probability that features were activated by noise (γ) when a blank screen replaced the target:

$$\gamma_{BL} = b \times \gamma_{TP}, \tag{5}$$

where γ_{BL} and γ_{TP} are the probabilities of random features becoming activated by noise when a blank screen is presented instead of the target or when the target is present, respectively.

Similar to the presence versus absence of a target, higher versus lower levels of noise serve to provide or take away positive evidence that is available to be reduced by discounting. Thus, a lower level of noise reduces the efficacy of discounting. Therefore, with lower perceptual noise in the target absent conditions, discounting is not as effective, and the probability of choosing the primed alternative can rise above the target primed condition. Thus, discounting efficacy takes a double hit in the no-target condition: The missing target and the lower noise both serve to reduce the evidence that can be discounted.

Details of the Fitting Procedure and Goodness of Fit

In modeling the current data, the probability that features were activated by noise was fixed to .07 for all trials in which a target was presented ($\gamma_{TP} = .07$). This is a higher level of noise than that used in previous simulations in which this probability was fixed to .02 (e.g., Huber et al., 2001; Weidemann et al., 2005). This higher level was chosen because it also enabled the modeling of the effects of trials without a target presentation in Experiment 2, where we assumed a relatively lower level of noise when the target was replaced by a blank screen (cf. Equation 5; .07 was a high enough value that it could be lowered sufficiently to produce the observed no-target effects). As in the modeling for previous experiments (e.g., Huber et al., 2001; Weidemann et al., 2005), we assumed that the probability that features were activated by the target and by noise was correctly estimated ($\beta' = \beta$, and $\gamma' = \gamma$) with one exception: In Experiment 2, γ varied with target presence, but letting γ' vary as well would imply that the system can distinguish between trials in which the target was present versus those in which a blank screen was presented instead. Therefore, we calculated γ' for Experiment 2 as follows:

$$\gamma' = \frac{tp \times \gamma_{TP} + bl \times \gamma_{BL}}{tp + bl} \tag{6}$$

where *tp* and *bl* are the number of trials in Experiment 2 in which the target was present and a blank screen was shown instead of the target, respectively—γ_{TP} and γ_{BL} are as defined in Equation 5. Equation 6 was chosen because it seemed sensible that γ' should be an appropriately weighted average of γ_{TP} and γ_{BL}—this particular assumption, however, was not crucial to produce good fits.

When the ROUSE model was introduced, it was probabilistically simulated (Huber et al., 2001), but recently Huber (2006) developed a more efficient analytical version that was used for all modeling presented here. For the model fits, a direct search algorithm (Hooke & Jeeves, 1961) was used to minimize a chi-square error measure (Huber et al., 2001; see also Correction to Huber et al., 2001). The implementation of the fitting routine was based on publicly available C code (Johnson, 1994), which incorporated published pseudocode and improvements to the algorithm (Bell & Pike, 1966; Kaupe, 1963; Tomlin & Smith, 1969).

Table 1 shows all eight free parameters that were used in the fit to the 148 data points from Experiments 1–4. Most parameters applied to all experiments, with the exception of *prev*, which only applied to Experiments 1 and 2 (because these were the only experiments in which prime diagnosticity changed), and *b*, which only applied to Experiment 2 (because only in this experiment was the target sometimes replaced by a blank screen). We allowed β to vary (slightly) with prime duration to account for the difference in the neither primed baseline for short and long primes. Huber (2006) discussed the reliability of different parameter estimates: In terms of α and α', the most critical factor is their relative magnitude; the absolute values assigned to these parameters can usually be moved up and down in concert without greatly affecting the goodness of fit. As is evident from Figures 2–4 and 6–9, with these eight free parameters, our model could account well for an intricate set of data. The root-mean-squared deviation of the model fits from the data for all Experiments presented here (weighted by the number of observations that make up each data point) is .05.

General Discussion

We examined the role of prime diagnosticity across four different forced-choice perceptual identification experiments. Using prime diagnosticities that ranged from neutral (i.e., primes were nondiagnostic) to very strong (i.e., a prime was 4 times more likely to predict the correct choice than the incorrect choice or vice versa), we found that prime diagnosticity was an important factor for identification of the target. Nevertheless, evidence discounting remained a key element of choice behavior as revealed by the

[7] Because the data in Figure 4 were obtained by aggregating over conditions after one or two changes in prime diagnosticity, one could argue that α'_P in Equation 4 should be recursively defined to take into account all prior diagnosticities. Because the influence of prior diagnosticities falls off exponentially, considering more than the just prior diagnosticity has a negligible influence.

Table 1
Table of Fitted Parameter Values for ROUSE

Parameter	Fitted value	Applies to
α_{short}	.62	Experiments 1–4
α_{long}	.41	Experiments 1–4
α'_{short}	.43	Experiments 1–4
α'_{long}	.49	Experiments 1–4
β_{short}	.05	Experiments 1–4
β_{long}	.09	Experiments 1–4
prev	.30	Experiments 1–2
b	.77	Experiment 2

Note. The indices "long" and "short" are separate parameter values for long and short prime durations, respectively. ROUSE = responding optimally with unknown sources of evidence.

reduction or reversal of the preference for primed words when comparing short and long duration primes. These discounting effects in comparing short and long duration primes were seen in all conditions of all experiments and replicate previous findings for nondiagnostic primes (e.g., Huber, Shiffrin, Lyle, & Quach, 2002; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005).

Identification performance adapted quickly to prime diagnosticity, with an increased tendency to choose the primed alternative for positive diagnosticities (i.e., the prime predicts the correct alternative) and a decreased tendency to choose the primed alternative for negative diagnosticities (i.e., the prime predicts the incorrect alternative). These findings replicate and extend findings from a large number of previous studies that found effects of different levels of positive prime diagnosticity in short-term priming (e.g., Bodner & Masson, 2001, 2003, 2004; Bodner et al., 2006). This adaptation to prime diagnosticity revealed a primacy effect and was resistant to subsequent diagnosticity changes (Experiments 1 and 2). We discuss this finding in the context of missing prime diagnosticity effects (e.g., Pecher et al., 2002) below.

Conditions that failed to present a valid target word produced choice behavior that was inconsistent with a simple strategy against primes that were negatively diagnostic. Even in conditions where accuracy for target primed trials was lower than that for foil primed trials, participants tended to choose the primed alternative when no target was present (Experiment 2). Experiments 3 and 4 demonstrated that prime diagnosticity can be associated with prime duration but not with prime color.

Accounts for Prime Diagnosticity Effects

We have specified a formal model in which we assume that features from the prime are confused with target features. Discounting of evidence known to potentially stem from the prime presentation counteracts this blending in of prime features. Within this model, we accounted for effects of prime diagnosticity in terms of differential degrees of evidence discounting. The logic is that it is most important to discount evidence from primes that tend to signal the incorrect alternative, whereas discounting evidence from primes that tend to signal the correct alternative may actually hurt performance. This explanation naturally accounts for the finding that even with strongly negative prime diagnosticity, the proportion of prime compatible choices does not tend to go much below the chance level of .5 unless the foil is primed (this is because evidence discounting only lowers evidence for primed features but does not constitute evidence against them). It also fits nicely with the fact that prime diagnosticity can be cued by prime duration, an aspect of the prime display that also affects evidence discounting but not by an arbitrary cue—such as color of the primes, which has no influence on evidence discounting (Experiments 3 and 4). In the following discussion, we place the model proposed here in the context of alternative theories that have previously been used to account for effects of prime diagnosticity.

Automatic facilitation versus controlled inhibition. Posner and Snyder (1975a, 1975b) proposed that a preference for the primed alternative can be elicited by automatic processes, whereas inhibition of stimuli incompatible with the prime occurs as a consequence of controlled processing. The automatic processes proposed by Posner and Snyder are similar to those proposed by spreading activation theories (e.g., Collins & Loftus, 1975) and share their drawbacks: Mechanisms that assume strengthening of representations of primed stimuli can easily account for a preference for prime compatible responses but fail to capture the relative reversal of this effect as prime saliency increases.

Naccache, Blandin, and Dehaene (2002) found that masked primes require a minimal level of attentional processing to have an effect. In isolation, this result may suggest that prime diagnosticity modulates how much attention a prime receives such that a highly valid prime is attended to more than a neutral prime (cf. Bodner et al., 2006). Again, this is just another way to phrase the hypothesis that the prime representation is strengthened as prime diagnosticity becomes more positive. The present data as well as those from many other studies (e.g., Huber, Shiffrin, Lyle, & Quach, 2002; Huber et al., 2001; Huber, Shiffrin, Quach, & Lyle, 2002; Weidemann et al., 2005) are incompatible with such a simple mapping of prime diagnosticity to prime salience.

However, we do agree with Posner and Snyder's (1975a) general distinction between automatic and controlled processes and agree that true inhibition is likely the result of controlled processes. Indeed, we used the fact that we failed to observe an absolute bias against the primed alternative (unless it conflicted with the target) as evidence that the underlying processes for prime diagnosticity effects are probably automatic.

The model we present here can be viewed as specifying the automatic component in the framework put forth by Posner and Snyder (1975a). In our model, the automatic effects of the prime are not limited to strengthening the primed representation but can also produce a relative preference against the primed alternative by implicitly discounting evidence that is compatible with the prime. The distinction between an absolute bias against the primed alternative, which is due to inhibitory processes, and a relative preference against the primed alternative (as compared with the neither primed baseline), which is due to evidence discounting, is an important one because we assume that only the former reliably indexes controlled processes.

Memory recruitment. Bodner et al. (2006; see also, e.g., Bodner & Masson, 2001, 2003) found prime diagnosticity effects in lexical decision, and Bodner and Masson (2004) showed similar effects in a naming task. In the lexical decision task used by Bodner et al., the proportion of trials with a word target that were repetition primed was manipulated relative to those that were primed by an unrelated word. This setup only allowed for positive prime diagnosticities because the proportion of primes that prime a nonword response was not manip-

ulated, but their results are compatible with ours. Bodner et al. have explained prime diagnosticity effects with a memory-recruitment account that assumes that an encoded prime episode is recruited during target processing. The idea is that positive prime diagnosticity (i.e., prime validity in the language of Bodner et al., 2006) increases prime recruitment and thus leads to an increased proportion of prime compatible results.

If the amount of evidence contributed by the prime is directly related to the amount of "memory recruitment," then our model can be viewed as an instantiation of this account. Note, however, that because of evidence discounting, the amount of evidence contributed by the prime decreases as prime saliency increases. Thus, we do not assume that the prime becomes more salient or is remembered better as it becomes more valid. Indeed, the opposite would be more compatible with our account: As the prime becomes more valid it is remembered less as being distinctive from the target, and thus it is less salient as a separate perceptual event.

Adaptation to the statistics of the environment (ASE). Taking a different approach to the diagnosticity inferred by the current situation, Mozer, Kinoshita, and Davis (2004) proposed a model for response adaptation based on recent experience, which they termed ASE. This model is based on the assumption that a calculated speed-accuracy trade off is made by selecting the time of a response on the basis of recent experience. As Kinoshita, Forster, and Mozer (2008; see also Kinoshita, Mozer, & Forster, 2007) have pointed out, ASE is only applicable to response time for speeded responses and predicts no prime diagnosticity effects on accuracy for unspeeded responses. Furthermore, ASE assumes that target primed (i.e., "congruent") trials are easier than foil primed (i.e., "incongruent") trials. This assumption is difficult to reconcile with our finding that in some conditions (when prime salience is high), accuracy is as high or higher for foil primed trials than for target primed trials. Even though it has been successfully applied to response time data from parity (odd–even) decision and naming tasks (Kinoshita et al., 2007, 2008; Mozer et al., 2004), ASE is therefore inherently unsuited to account for the present data.

Changes in Prime Diagnosticity

Our studies showed that diagnosticity had strong effects in the first set of blocks but then much smaller effects after diagnosticity changed direction. We have not tried to explain why this occurs, although primacy effects are commonly observed in many perceptual and cognitive tasks. Taken together, our findings have important implications for a variety of paradigms that involve perceptual source confusion. In each paradigm and particular design, the diagnosticity of the information needs to be considered, and, as shown by the discrepancy of the results of Experiments 3 and 4, the exact nature of any cue potentially signaling diagnosticity can have a profound influence on whether this diagnosticity affects identification performance. This suggests that the proportion of positively (or negatively) primed trials is a key variable in short-term priming paradigms, even if prime duration is near threshold (cf. Experiment 4). Furthermore, changes during the experiment in terms of the proportion of primed trials need to be carefully examined, as they may substantially attenuate effects of prime diagnosticity (cf. Experiments 1 and 2). For example, if the experiment begins with a low proportion of primed trials during a practice phase rather than a high proportion, this may produce a higher level of evidence discounting across the entire experiment.[8] Finally, to compare the strength of priming in different conditions, it is important to maintain the same level of diagnosticity across these conditions (unless there is good reason to believe that the difference between the conditions does not impinge on evidence discounting, such as was the case with the color cues of Experiment 3).

In their Experiments 2A and 2B, Pecher et al. (2002) used a naming task with associative short-term priming and varied the proportion of associatively related primes (10% or 90%). For brief primes (their Experiment 2A), Pecher et al. found no effect of prime diagnosticity; however, for long primes (their Experiment 2B), they found differences in the diagnosticity conditions that were clearly influenced by strategic responding. Pecher et al. argued that automatic/implicit priming effects should not be sensitive to prime diagnosticity and, thus, interpreted their results as showing that brief primes are processed automatically. We agree that the priming effect they found for brief primes is probably not strategic, but the current experiments suggest that sensitivity to prime diagnosticity is not a good criterion to distinguish between automatic and strategic responding. Why did Pecher et al. fail to find an effect of prime diagnosticity for brief primes? As mentioned above, we found that changes in prime diagnosticity can severely attenuate prime diagnosticity effects. It turns out that in their Experiments 2A and 2B, Pecher et al. used 20 practice trials with the experimental prime diagnosticity (10% or 90%) followed by 60 threshold trials with completely nondiagnostic primes (these trials were used to individually adjust the target presentation to be near the perceptual threshold), followed by 200 experimental trials, again with the experimental prime diagnosticity (10% or 90%). Thus, when the experimental trials started, the prime diagnosticity drastically changed for a second time, which may have attenuated any effect of prime diagnosticity to the point of rendering it undetectable.

Conclusions

By augmenting ROUSE with the assumption that discounting is modulated by prime diagnosticity, the model provided an excellent quantitative account of the very complex set of data from our studies. This success, combined with the qualitative behavioral results, supports the case that (a) evidence discounting plays a critical role in all sorts of priming studies, including those that use diagnostic primes; (b) evidence discounting is an automatic process affecting evidence accumulation that can interact in nonintuitive ways with the nature of the visual displays (e.g., prime and target duration); and (c) evidence discounting is itself learnable and is modulated in accord with the prime diagnosticity in the current block of trials or even in accord with the diagnosticity of an intermixed subset of trials if the diagnosticity is signaled by the duration of the prime (a cue that itself is relevant for evidence discounting).

The experiments presented here were designed to elucidate critical and basic mechanisms by which the visual system solves a very difficult problem: forming a best percept from what is often

[8] We used neither primed trials for which prime diagnosticity is not defined (primes do not repeat as choice words) in the calibration trials at the beginning of each experiment. In other priming procedures, however, not priming the target is usually analogous to our foil primed condition (e.g., in a lexical decision task, usually either the word or nonword response is primed for any prime stimulus), and thus even a practice or calibration phase has an associated prime diagnosticity.

very noisy, incomplete, and imprecise perceptual information. Knowledge about how the effect of a prime on a subsequent identification task varies not only with the display characteristics of the prime (e.g., its duration) but also with prime-target contingencies on a global (e.g., overall prime diagnosticity in the experiment) as well as on a local level (e.g., prime diagnosticity in a subset of the experimental conditions) provides important insights into the mechanisms of the visual system. This knowledge not only refines our understanding of percept formation but is also essential to the use of short-term priming as a tool for assessing representational and linguistic structure.

References

Bell, M., & Pike, M. C. (1966). Remark on algorithm 178 (E4) direct search. *Communications of the Association for Computing Machinery, 9,* 684–685.

Bodner, G. E., & Masson, M. E. J. (2001). Prime validity affects masked repetition priming: Evidence for an episodic resource account of priming. *Journal of Memory and Language, 45,* 616–647.

Bodner, G. E., & Masson, M. E. J. (2003). Beyond spreading activation: An influence of relatedness proportion on masked semantic priming. *Psychonomic Bulletin and Review, 10,* 645–652.

Bodner, G. E., & Masson, M. E. J. (2004). Beyond binary judgments: Prime validity modulates masked repetition priming in the naming task. *Memory & Cognition, 32,* 1–11.

Bodner, G. E., Masson, M. E. J., & Richard, N. T. (2006). Repetition proportion biases masked priming of lexical decisions. *Memory & Cognition, 34,* 1298–1311.

Cohen, A. L., & Sautner, M. (2001). ExpLib (Version 1.0.1 β) [Computer Programming Library for Visual C++]. Retrieved from http://people.umass.edu/alc/explib/index.htm

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Collins, A. M., & Loftus, E. F. (1975). A spreading-activation theory of semantic processing. *Psychological Review, 82,* 407–428.

Correction to Huber et al. (2001). *Psychological Review, 108,* 652.

Hooke, R., & Jeeves, T. A. (1961). "Direct search" solution of numerical and statistical problems. *Journal of the Association for Computing Machinery, 8,* 212–229.

Huber, D. E. (2006). Computer simulations of the ROUSE model: An analytic method and a generally applicable technique for producing parameter confidence intervals. *Behavioral Research Methods, Instruments, and Computers, 38,* 557–568.

Huber, D. E. (in press). Immediate priming and cognitive aftereffects. *Journal of Experimental Psychology: General.*

Huber, D. E., Shiffrin, R. M., Lyle, K. B., & Quach, R. (2002). Mechanisms of source confusion and discounting in short-term priming: 2. Effects of prime similarity and target duration. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 28,* 1120–1136.

Huber, D. E., Shiffrin, R. M., Lyle, K. B., & Ruys, K. I. (2001). Perception and preference in short-term word priming. *Psychological Review, 108,* 149–182.

Huber, D. E., Shiffrin, R. M., Quach, R., & Lyle, K. B. (2002). Mechanisms of source confusion and discounting in short-term priming: 1. Effects of prime duration and prime recognition. *Memory & Cognition, 30,* 745–757.

Hutchison, K. A. (2007). Attentional control and the relatedness proportion effect in semantic priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 33,* 645–662.

Hutchison, K. A., Neely, J. H., & Johnson, J. D. (2001). With great expectations, can two "wrongs" prime a "right"? *Journal of Experimental Psychology: Learning, Memory, and Cognition, 27,* 1451–1463.

Huynh, H., & Feldt, L. S. (1976). Estimation of the Box correction for degrees of freedom from sample data in randomized block and split-plot designs. *Journal of Educational Statistics, 1,* 69–82.

Johnson, M. G. (1994). Nonlinear optimization using the algorithm of Hooke and Jeeves [C source code]. Retrieved from http://www.netlib.org/opt/hooke.c

Kaupe, A. F., Jr. (1963). Algorithm 178: Direct search. *Communications of the Association for Computing Machinery, 6,* 313–314.

Kinoshita, S., Forster, K. I., & Mozer, M. C. (2008). Unconscious cognition isn't that smart: Modulation of masked repetition priming effect in the word naming task. *Cognition.* Advance online publication. doi: 10.1016/j.cognition.2007.11.011

Kinoshita, S., Mozer, M. C., & Forster, K. I. (2007). *Modulation of congruence effects with masked primes in the parity decision task.* Manuscript submitted for publication.

Kucera, H., & Francis, W. N. (1967). *Computational analysis of present-day American English.* Providence, RI: Brown University Press.

Maxwell, S. E., Camp, C. J., & Arvey, R. D. (1981). Measures of strength of association: A comparative examination. *Journal of Applied Psychology, 66,* 525–534.

Mozer, M. C., Kinoshita, S., & Davis, C. J. (2004). Control of response initiation: Mechanisms of adaptation to recent experience. In *Proceedings of the 26th Annual Conference of the Cognitive Science Society* (pp. 981–986). Hillsdale, NJ: Erlbaum.

Naccache, L., Blandin, E., & Dehaene, S. (2002). Unconscious masked priming depends on temporal attention. *Psychological Science, 13,* 416–424.

Olejnik, S., & Algina, J. (2000). Measures of effect size for comparative studies: Applications, interpretations, and limitations. *Contemporary Educational Psychology, 25,* 241–286.

Pecher, D., Zeelenberg, R., & Raaijmakers, J. G. W. (2002). Associative priming in a masked perceptual identification task: Evidence for automatic processes. *The Quarterly Journal of Experimental Psychology, 55A,* 1157–1173.

Posner, M. I., & Snyder, C. R. R. (1975a). Attention and cognitive control. In R. L. Solso (Ed.), *Information processing and cognition: The Loyola symposium* (pp. 55–85). Hillsdale, NJ: Erlbaum.

Posner, M. I., & Snyder, C. R. R. (1975b). Facilitation and inhibition in the processing of signals. In P. M. A. Rabbitt & S. Dornic (Eds.), *Attention and performance V* (pp. 669–682). London: Academic Press.

Ratcliff, R., & McKoon, G. (2001). A multinomial model for short-term priming in word identification. *Psychological Review, 108,* 835–846.

Reber, A., & Allen, R. (1978). Analogic and abstraction strategies in synthetic grammar learning: A functionalist interpretation. *Cognition, 6,* 189–221.

Sanborn, A. N., Malmberg, K. J., & Shiffrin, R. M. (2004). High-level effects of masking on perceptual identification. *Vision Research, 44,* 1427–1436.

Schneider, W., & Shiffrin, R. M. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review, 84,* 1–66.

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.

Tomlin, F. K., & Smith, L. B. (1969). Remark on algorithm 178 (E4): Direct search. *Communications of the Association for Computing Machinery, 12,* 637–638.

Weidemann, C. T. (2006). *Identifying brief stimuli: Perceptual, preferential, and decisional aspects.* Unpublished doctoral dissertation, Indiana University.

Weidemann, C. T., Huber, D. E., & Shiffrin, R. M. (2005). Confusion and compensation in visual perception: Effects of spatiotemporal proximity and selective attention. *Journal of Experimental Psychology: Human Perception and Performance, 31,* 40–61.

Wellman, M. P., & Henrion, M. (1993). Explaining "explaining away." *IEEE Transactions on Pattern Analysis and Machine Intelligence, 15,* 287–307.

(*Appendixes follow*)

Appendix A

Analyses of Variance for Experiment 1

Table A1
Analysis of Variance for the Conditions of Experiment 1 Before a Change in Prime Diagnosticity

Factor	*df*	*SS*	*MS*	*F*	ε	*p*
Diag.	2	0.21	0.10	1.86		.16
Residuals	81	4.54	0.06			
Prime	2	2.45	1.23	50.42		<.01*
Diag. × Prime	4	4.69	1.17	48.20	.60	<.01*
Residuals	162	3.94	0.02			
Duration	1	1.57	1.57	87.17		<.01*
Diag. × Duration	2	0.06	0.03	1.75		.18
Residuals	81	1.46	0.02			
Prime × Duration	2	6.79	3.39	199.04		<.01*
Diag. × Prime × Duration	4	0.10	0.03	1.52	.46	.23
Residuals	162	2.76	0.02			

Note. All *p* values are based on the epsilon-corrected degree of freedom when applicable. *df* = uncorrected degree(s) of freedom; *SS* = sum of squared error; *MS* = mean squared error; ε = Huynh-Feldt epsilon correction; Diag. = current diagnosticity; Prime = prime type; Duration = prime duration.
* $p < .05$.

Table A2
Analyses of Variance for All Conditions of Experiment 1

Factor	*df*	*SS*	*MS*	*F*	ε	*p*
Positive prime diagnosticity						
Prev. Diag.	2	0.13	0.06	0.70		.50
Residuals	81	7.43	0.09			
Prime	2	6.48	3.24	130.73		<.01*
Prev. Diag. × Prime	4	0.55	0.14	5.54	.69	<.01*
Residuals	162	4.02	0.02			
Duration	1	1.87	1.87	100.04		<.01*
Prev. Diag. × Duration	4	0.02	0.01	0.54		.58
Residuals	81	1.51	0.02			
Prime × Duration	2	4.97	2.48	175.37		<.01*
Prev. Diag. × Prime × Duration	4	0.12	0.03	2.12	.63	.11
Residuals	162	2.29	0.01			
Neutral prime diagnosticity						
Prev. Diag.	2	0.36	0.18	2.43		.09
Residuals	81	5.96	0.07			
Prime	2	2.78	1.39	65.37		<.01*
Prev. Diag. × Prime	4	0.22	0.06	2.63	.70	.06
Residuals	162	3.45	0.02			
Duration	1	1.34	1.34	101.50		<.01*
Prev. Diag. × Duration	2	0.06	0.03	2.45		.09
Residuals	81	1.07	0.01			
Prime × Duration	2	5.26	2.63	169.92		<.01*
Prev. Diag. × Prime × Duration	4	0.18	0.05	2.94	.64	.04*
Residuals	162	2.51	0.02			
Negative prime diagnosticity						
Prev. Diag.	2	0.18	0.09	1.20		.31
Residuals	81	6.20	0.08			
Prime	2	0.68	0.34	12.57		<.01*
Prev. Diag. × Prime	4	2.08	0.52	19.33	.67	<.01*
Residuals	162	4.35	0.03			
Duration	1	1.13	1.13	55.91		<.01*
Prev. Diag. × Duration	2	0.08	0.04	1.96		.15
Residuals	81	1.63	0.02			
Prime × Duration	2	3.01	1.50	103.12		<.01*
Prev. Diag. × Prime × Duration	4	0.22	0.06	3.80	.61	.02*
Residuals	162	2.36	0.01			

Note. All *p* values are based on the epsilon-corrected degree of freedom when applicable. *df* = uncorrected degree(s) of freedom; *SS* = sum of squared error; *MS* = mean squared error; ε = Huynh-Feldt epsilon correction; Prev. Diag. = previous diagnosticity; Prime = prime type; Duration = prime duration.
* $p < .05$.

(*Appendixes continue*)

Table A3
Analyses of Variance for Conditions of Experiment 1 After at Least One Change in Prime Diagnosticity

Factor	*df*	*SS*	*MS*	*F*	ε	*p*
Positive prime diagnosticity						
Prev. Diag.	1	0.13	0.13	1.14		.29
Residuals	54	6.08	0.11			
Prime	2	2.85	1.43	54.96		<.01*
Prev. Diag. × Prime	2	0.09	0.05	1.74	.66	.19
Residuals	108	2.80	0.03			
Duration	1	1.11	1.11	68.01		<.01*
Prev. Diag. × Duration	1	0.01	0.01	0.56		.46
Residuals	54	0.88	0.02			
Prime × Duration	2	2.67	1.33	94.00		<.01*
Prev. Diag. × Prime × Duration	2	0.01	0.00	0.27	.58	.64
Residuals	108	1.53	0.01			
Neutral prime diagnosticity						
Prev. Diag.	1	0.07	0.07	0.82		.37
Residuals	54	4.49	0.08			
Prime	2	1.55	0.78	32.53		<.01*
Prev. Diag. × Prime	2	0.16	0.08	3.45	.69	.05
Residuals	108	2.57	0.02			
Duration	1	0.77	0.77	61.87		<.01*
Prev. Diag. × Duration	1	0.05	0.05	4.09		.05
Residuals	54	0.67	0.01			
Prime × Duration	2	2.67	1.34	92.62		<.01*
Prev. Diag. × Prime × Duration	2	0.01	0.01	0.39	.61	.58
Residuals	108	1.56	0.01			
Negative prime diagnosticity						
Prev. Diag.	1	0.00	0.00	0.04		.84
Residuals	54	4.48	0.08			
Prime	2	0.87	0.43	18.67		<.01*
Prev. Diag. × Prime	2	0.13	0.07	2.87	.79	<.07
Residuals	108	2.51	0.02			
Duration	1	0.86	0.86	38.52		<.01*
Prev. Diag. × Duration	1	0.07	0.07	3.07		.09
Residuals	54	1.20	0.02			
Prime × Duration	2	1.45	0.72	59.64		<.01*
Prev. Diag. × Prime × Duration	2	0.06	0.03	2.41	.80	.11
Residuals	108	1.31	0.01			

Note. All *p* values are based on the epsilon-corrected degree of freedom when applicable. *df* = uncorrected degree(s) of freedom; *SS* = sum of squared error; *MS* = mean squared error; ε = Huynh-Feldt epsilon correction; Prev. Diag. = previous diagnosticity; Prime = prime type; Duration = prime duration.
* $p < .05$.

Appendix B

Analyses of Varience for Experiment 2

Table B1
Statistically Significant ($p < .05$) Effects for Experiment 2 (Target Present)

Factor	*df*	*SS*	*MS*	*F*	ε	*p*
Diag.	1	0.58	0.58	42.86		<.01
Start × Diag.	1	0.21	0.21	15.83		<.01
Residuals	108	1.46	0.01			
Prime	2	2.72	1.36	42.00		<.01
Start × Prime	2	2.82	1.41	43.47	.63	<.01
Residuals	216	7.00	0.03			
Duration	1	3.39	3.39	179.29		<.01
Residuals	108	2.04	0.02			
Diag. × Prime	2	6.45	3.22	144.67		<.01
Strength × Diag. × Prime	2	1.33	0.66	29.72	.56	<.01
Residuals	216	4.81	0.02			
Diag. × Duration	1	0.09	0.09	10.08		<.01
Start × Diag. × Duration	1	0.08	0.08	9.84	.94	<.01
Residuals	108	0.92	0.01			
Prime × Duration	2	12.64	6.32	222.98		<.01
Strength × Prime × Duration	2	0.33	0.17	5.84	.59	.01
Residuals	216	6.12	0.03			
Diag. × Prime × Duration	2	0.20	0.10	9.22		<.01
Strength × Start × Diag. × Prime × Duration	2	0.11	0.06	5.13	.50	.03
Residuals	216	2.37	0.01			

Note. All *p* values are based on the epsilon-corrected degree of freedom when applicable. *df* = uncorrected degree(s) of freedom; *SS* = sum of squared error; *MS* = mean squared error; ε = Huynh-Feldt epsilon correction; Diag. = direction of the current diagnosticity (positive or negative); Start = direction of the first diagnosticity (positive or negative); Prime = prime type; Duration = prime duration; Strength = strength of the prime diagnosticity.

Table B2
Statistically Significant ($p < .05$) and Marginally Significant ($p = .05$) Effects for Experiment 2 for the Comparison Between Target Primed and Target Absent Trials

Factor	*df*	*SS*	*MS*	*F*	ε	*p*
Start	1	0.84	0.84	27.13		<.01
Residuals	108	3.34	0.03			
Diag.	1	11.97	11.97	682.89		<.01
Strength × Diag.	1	0.80	0.80	45.53		<.01
Residuals	108	1.89	0.02			
Duration	1	1.30	1.30	68.34		<.01
Strength × Duration	1	0.17	0.17	8.99		<.01
Residuals	108	2.06	0.02			
Target	1	5.79	5.79	138.23		<.01
Strength × Target	1	0.66	0.66	15.67		<.01
Start × Target	1	1.43	1.43	34.17		<.01
Strength × Start × Target	1	0.17	0.17	4.04		.05
Residuals	108	4.53	0.04			
Diag. × Duration	1	0.033	0.03	4.31		.05
Strength × Start × Diag. × Duration	1	0.049	0.05	6.40	.84	.02
Residuals	108	0.828	0.01			
Strength × Diag. × Target	1	0.22	0.22	8.45		.01
Start × Diag. × Target	1	0.62	0.62	23.96	.57	<.01
Strength × Start × Diag. × Target	1	0.17	0.17	6.59		.03
Residuals	108	2.78	0.03			
Duration × Target	1	0.97	0.97	51.25		<.01
Stength × Duration × Target	1	0.19	0.19	10.04	.70	<.01
Residuals	108	2.05	0.02			

Note. All *p* values are based on the epsilon-corrected degree of freedom when applicable. *df* = uncorrected degree(s) of freedom; *SS* = sum of squared error; *MS* = mean squared error; ε = Huynh-Feldt epsilon correction; Start = direction of the first diagnosticity (positive or negative); Diag. = direction of the current diagnosticity (positive or negative); Strength = strength of the prime diagnosticity; Duration = prime duration; Target = target presence (present or absent).

Received January 26, 2007
Revision received September 19, 2007
Accepted November 5, 2007 ■