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# The Role of the Locus Coeruleus in Mediating the Attentional Blink: A Neurocomputational Theory

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The attentional blink refers to the transient impairment in perceiving the 2nd of 2 targets presented in close temporal proximity. In this article, the authors propose a neurobiological mechanism for this effect. The authors extend a recently developed computational model of the potentiating influence of the locus coeruleus–norepinephrine system on information processing and hypothesize that a refractoriness in the function of this system may account for the attentional blink. The model accurately simulates the time course of the attentional blink, including Lag 1 sparing. The theory also offers an account of the close relationship of the attentional blink to the electrophysiological P3 component. The authors report results from two behavioral experiments that support a critical prediction of their theory regarding the time course of Lag 1 sparing. Finally, the relationship between the authors' neurocomputational theory and existing cognitive theories of the attentional blink is discussed.

*Keywords:* attentional blink, locus coeruleus, attention, noradrenergic, P3

Recent research has suggested that the neuromodulatory brainstem nucleus locus coeruleus (LC) is critical for the regulation of cognitive performance (Aston-Jones, Rajkowski, & Cohen, 1999, 2000; Robbins, 1997). The LC exhibits a strong phasic increase in activity during the processing of motivationally relevant stimuli, leading to the release of the neuromodulatory neurotransmitter norepinephrine (NE) in widespread cortical projection areas (Aston-Jones, Foote, & Bloom, 1984). This LC-mediated noradrenergic innervation increases the responsiveness of efferent target neurons, which is thought to facilitate processing in response to a stimulus (Waterhouse & Woodward, 1980; for a review, see Ber-

ridge & Waterhouse, 2003). In contrast, local NE release within the LC has an inhibitory effect (Aghajanian, Cedarbaum, & Wang, 1977; Egan, Henderson, North, & Williams, 1983; Washburn & Moises, 1989; Williams, Henderson, & North, 1985), leading to a brief period of quiescence following the phasic response during which LC–NE-mediated facilitation of information processing is largely unavailable. The duration of this refractory-like period (cf. Usher, Cohen, Servan-Schreiber, Rajkowski, & Aston-Jones, 1999) coincides with the temporal profile of a cognitive phenomenon known as the *attentional blink*, a temporary deficit in processing of a target stimulus following successful processing of a previous target (Raymond, Shapiro, & Arnell, 1992).

In this article, we propose that the attentional blink may be caused by the specific dynamics of the LC–NE neuromodulatory system. To illustrate the explanatory power of this hypothesis in a formal fashion, we conducted simulations of performance in the attentional blink paradigm, using an existing computational model of LC function (Gilzenrat, Holmes, Rajkowski, Aston-Jones, & Cohen, 2002). With minimal modification to this model, we show that it captures several critical features of the attentional blink phenomenon. In addition, we present empirical data from two behavioral experiments that support a critical prediction of our hypothesis. The implications of the LC–NE hypothesis for understanding other empirical phenomena, such as the relationship between the attentional blink and the electrophysiological P3 component, are discussed. Finally, we point out that our efforts should not be regarded as a challenge to existing cognitive theories of the attentional blink—instead, our account may suggest a neural basis for the functional mechanisms outlined in these theories. However, our account does highlight the important influence that neuro-

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modulatory systems may have on cognitive function—something that has largely been ignored by many cognitive neuroscientific theories. We begin by introducing the attentional blink phenomenon, followed by a brief overview of the structural and functional characteristics of the LC–NE system.

### Attentional Blink

When presented with a rapid serial visual presentation (RSVP) stream containing two target stimuli (T1 and T2) and multiple distractors, presented for about 100 ms each, subjects are typically impaired at the detection or identification of T2 for a few hundred milliseconds following correct detection or identification of T1. This deficit, known as the attentional blink, is most severe around 200–300 ms (or 2–3 items) following T1, after which performance gradually recovers (e.g., Broadbent & Broadbent, 1987; Chun & Potter, 1995; J. Duncan, Ward, & Shapiro, 1994; Raymond et al., 1992). It is interesting to note that if T2 follows T1 without intervening distractors (at Lag 1, or one frame after T1), performance on T2 is often (partially) spared (Raymond et al., 1992). This phenomenon has been labeled *Lag 1 sparing*. These basic characteristics of the attentional blink are illustrated in Figure 1A, which shows behavioral performance in an attentional blink study reported by Chun and Potter (1995, Experiment 1).

Although they differ in the specific mechanisms, cognitive theories of the attentional blink have generally held that there is a capacity-limited stage in stimulus processing and that competition between different stimuli for limited attentional resources underlies the attentional blink deficit (for a review, see Shapiro, Arnell, & Raymond, 1997). For example, Shapiro and colleagues have suggested that competition between stimuli occurs in retrieval from visual short-term memory (e.g., Shapiro, Raymond, & Arnell, 1994). Several factors, including the order of entry into this temporary storage buffer, determine the probability of an item being retrieved. The attentional blink occurs when there is interference in retrieval of the correct item from visual short-term memory. Alternatively, it has been proposed that stimuli compete for entry to a limited-capacity processing stage that is necessary for the stimuli to reach awareness or to elicit a response (Chun & Potter, 1995). According to this account, the attentional blink occurs when this stage is still occupied by T1 when T2 is presented. More recent proposals have suggested a hybrid model (Maki, Frigen, & Paulson, 1997). As we outline below, the temporal dynamics of the LC–NE system exhibit the properties of such limited-capacity attentional resources.

### LC–NE System

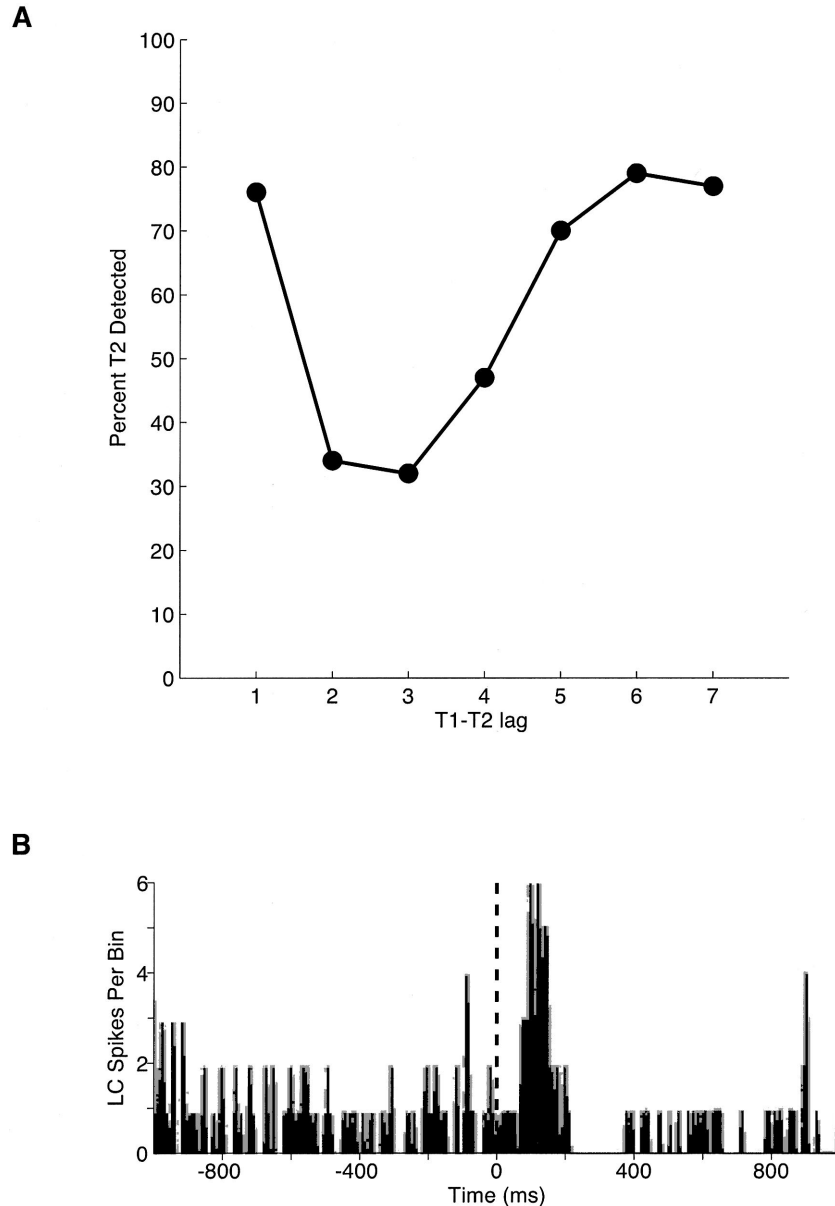
The brainstem nucleus LC is situated in the dorsal pontine tegmentum and is estimated to contain half of all noradrenergic neurons in the central nervous system. The LC projects widely to all levels of the neuraxis and is the sole source of NE-releasing fibers projecting to the forebrain (Berridge & Waterhouse, 2003), showing particularly strong innervation of areas associated with attentional processing (Morrison & Foote, 1986). Although to date it has not been possible to investigate the activation dynamics of

the LC–NE system in humans, cell recordings in nonhuman primates have yielded a wealth of information regarding these dynamics. These primate studies have implicated tonic activity of the LC–NE system in numerous aspects of cognitive and behavioral regulation, including general arousal level (e.g., Foote, Aston-Jones, & Bloom, 1980), affective state (e.g., Aston-Jones, Rajkowski, Kubiak, Valentino, & Shipley, 1996), and the sleep–wake cycle (e.g., Aston-Jones & Bloom, 1981a; Hobson, McCarley, & Wyzinski, 1975).

Neurophysiological studies with monkeys have shown that in addition to these fluctuations in LC tonic activity, when the animal is actively engaged in performing a task, LC neurons exhibit a rapid, phasic increase in discharge rate to motivationally salient stimuli of many modalities (Aston-Jones & Bloom, 1981b; Foote et al., 1980; Grant, Aston-Jones, & Redmond, 1988; Rasmussen, Morilak, & Jacobs, 1986; Sara & Segal, 1991), and that this response can be operantly conditioned (e.g., Aston-Jones, Rajkowski, & Kubiak, 1997; Aston-Jones, Rajkowski, Kubiak, & Alexinsky, 1994; Rajkowski, Kubiak, & Aston-Jones, 1994). For example, such *LC phasic responses* are observed for target stimuli in a simple signal detection task in which monkeys are required to respond to rare target stimuli presented at random intervals (varying between 1.1 and 2.4 s) embedded in a train of distractor stimuli. Provided that the animal is engaged in the task, these target stimuli cause a phasic increase in LC firing rate that peaks approximately 100–150 ms posttarget and approximately 200 ms prior to the response (see Figure 1B; e.g., Aston-Jones et al., 1994; Clayton, Rajkowski, Cohen, & Aston-Jones, 2004).<sup>1</sup> It is important to note that the LC does not exhibit this type of phasic response to distractor stimuli, nor is the phasic response associated with any other task-related events (reward delivery, fixation point, response movements, etc.) once training is complete. These findings suggest that the LC selectively responds to task-relevant or otherwise salient stimuli that demand effective processing and action.

A full consideration of LC function is beyond the scope of this article, and it has received extensive treatment elsewhere (Brown, Gilzenrat, & Cohen, 2004; Cohen, Aston-Jones, & Gilzenrat, 2004; Gilzenrat et al., 2002; Nieuwenhuis, Aston-Jones, & Cohen, 2005; Usher et al., 1999). For the present purposes, we focus on the hypothesis that the function of the LC phasic discharge elicited by task-relevant stimuli is to facilitate responses to such stimuli. This effect is thought to be mediated by the neuromodulatory action of NE on cortical areas, which temporarily increases the responsivity of these areas to their afferent input (see Berridge & Waterhouse, 2003). These neuromodulatory effects have been simulated in computational models as transient changes in the *gain*

<sup>1</sup> It is interesting to note that when the animal is disengaged and inattentive, the LC phasic response is absent or attenuated, whereas overall tonic firing rate is elevated. The observed correlation between attentional state and LC firing mode has led to a theory of the LC's role in modulating attentional state, which has been described elsewhere (Aston-Jones et al., 1999, 2000; Cohen et al., 2004; Gilzenrat et al., 2002; Usher et al., 1999).



*Figure 1.* A: Typical experimental results from an attentional blink study (Chun & Potter, 1995, Experiment 1). Second target (T2) detection accuracy is plotted as a function of the lag between the first target (T1) and T2. Adapted from “A Two-Stage Model for Multiple Target Detection in Rapid Serial Visual Presentation,” by M. M. Chun and M. C. Potter, 1995, *Journal of Experimental Psychology: Human Perception and Performance*, 21, Figure 2, p. 112. Copyright 1995 by the American Psychological Association. B: Peristimulus time histogram of activity from a typical monkey locus coeruleus (LC) neuron during target trials (average of 100) in a visual target detection task. Target presentation is at time 0. During periods when the monkey is engaged in the task, LC activity is characterized by a phasic increase in firing rate posttarget, followed by a brief, refractory-like decrement in firing. From “The Role of Locus Coeruleus in the Regulation of Cognitive Performance,” by M. Usher, J. D. Cohen, D. Servan-Schreiber, J. Rajkowski, and G. Aston-Jones, 1999, *Science*, 283, Figure 3a, p. 550. Copyright 1999 by the American Association for the Advancement of Science. Adapted with permission.

of cortical processing units (Servan-Schreiber, Printz, & Cohen, 1990). It is important to note that when applied in a temporally strategic manner (e.g., when driven by the identification and evaluation of task-relevant stimuli), increases in gain produce an

increase in the signal-to-noise ratio of subsequent processing and a concomitant improvement in the efficiency and reliability of behavioral responses, such as signal detection performance (Brown, Gilzenrat, & Cohen, 2004; Gilzenrat et al., 2002; Servan-

Schreiber et al., 1990; Usher et al., 1999).<sup>2</sup> As we show below, in the case of the standard attentional blink paradigm, this translates into more effective target detection. It is important to note that although the LC phasic response itself is relatively brief in duration (typically lasting 50–100 ms), the ensuing neuromodulatory effects of NE release on target cortical areas are known to be delayed with respect to, and to last longer than, the LC phasic response. Although the precise delay and duration of NE modulatory effects and the time course of their influence on information processing have not yet been precisely characterized, the values assumed by our model (delay: <100 ms; duration: 100–200 ms) are within a physiologically reasonable range.

Although NE potentiates processing in cortical areas, local NE release within the LC is thought to be autoinhibitory, because of noradrenergic action at presynaptic and dendritic  $\alpha_2$  autoreceptors (Aghajanian et al., 1977; Egan et al., 1983; Washburn & Moises, 1989; Williams et al., 1985). This autoinhibition results in a refractory-like period after an LC phasic response, during which subsequent LC phasic discharge is rarely observed (Aston-Jones et al., 1994; Usher et al., 1999). This refractoriness peaks approximately 50–100 ms following the LC phasic response, typically 200–250 ms after the eliciting stimulus, and usually lasts 200 ms or until about 400–450 ms poststimulus (see Figure 1B). Note that this refractoriness is a population-level phenomenon, presumably a result of the residual effects of NE release, and is most likely unrelated to the potassium-mediated refractoriness of individual neurons that is observed after an action potential. During this population-wide refractory period, the processing of a subsequent target stimulus is unlikely to recruit another LC phasic response. As a result, stimuli presented during this time period do not have the benefit of NE-mediated facilitation. This property of the LC–NE system forms the basis for our hypothesis about the attentional blink.

### Hypothesis

We propose that the attentional blink is mediated by the refractory period in LC activity that occurs following an LC phasic response elicited by target stimuli. Because of the momentary unavailability of noradrenergic potentiation, subsequent target stimuli that are presented during the refractory period do not receive the benefit of LC-mediated facilitation and, therefore, suffer a deficit in processing. However, when a stimulus immediately follows T1, it may still benefit from the NE release elicited by the LC phasic response to that target (Usher et al., 1999). This occurs because of the residual effects of NE release in the cortex following an LC phasic response. We propose that this effect explains Lag 1 sparing, the finding that processing of T2 is often unimpaired when T2 is presented immediately following T1.

We conducted computer simulations to demonstrate in a formally explicit manner how our hypothesis can account for the empirical phenomena of interest. Our simulations were performed using an existing computational model of LC population-level dynamics in a simple target detection task (Gilzenrat et al., 2002). The model was built to capture the essential computational features of previous, detailed biophysical models of LC dynamics (Brown, Moehlis, et al., 2004; Usher et al., 1999), and it accurately simulates the population-level activation dynamics of the monkey

LC and its influence on target detection performance (Aston-Jones et al., 1994). We predicted that without significant modifications to the model, the simulated LC would consistently fail to show a phasic response to the second of two target items presented in close temporal proximity in an RSVP stream typical of attentional blink research. We predicted that this would produce a pattern of deficit in the model's target detection performance comparable to that associated with the attentional blink observed in empirical studies.

### Simulation

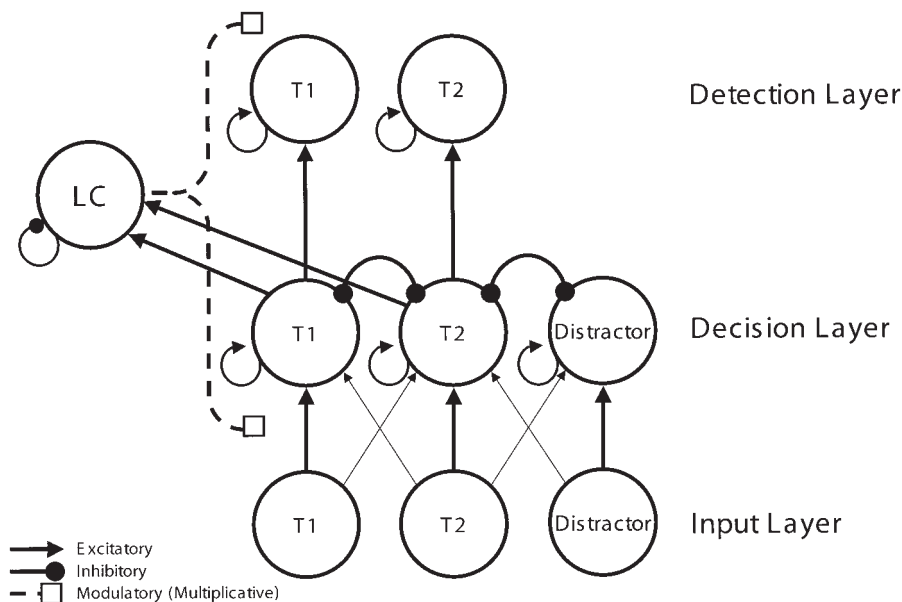
#### Method

*Model architecture.* The architecture of the model is depicted in Figure 2. The model consists of two functional components. The first component is a three-layer connectionist network designed to simulate the rudiments of stimulus processing and behavior in an RSVP-style attentional blink paradigm. The behavioral network has a very similar structure to that used in Usher et al.'s (1999) and Gilzenrat et al.'s (2002) models of target detection performance. The only change is the addition of an extra target processing pathway to be able to simulate the processing of two targets (T1 and T2) instead of one. The second component of the model is an abstracted LC that receives input from the behavioral network and, in turn, potentiates activity in the behavioral network in line with the diffuse modulatory effects of NE. The abstracted LC uses the same mathematical expressions and parameter values used to simulate LC function in Gilzenrat et al. (2002). Below, we present a brief overview of these model components and of the most essential aspects of our simulations. More detailed information is provided in the Appendix.

*The behavioral network.* The behavioral network consists of three layers: input, decision, and detection (response). There are feedforward excitatory connections between layers (simulating information flow) and mutual inhibitory connections between units in the decision layer (simulating competition among alternative representations of the presented stimulus; Cohen, Romero, Servan-Schreiber, & Farah, 1994; Desimone & Duncan, 1995; McClelland, 1993). Furthermore, the activity of these units is subject to small random variations (noise), simulating the impact of extraneous, uncorrelated afferent activity on processing. The first layer comprises three input units, corresponding to T1, T2, and distractor stimuli. For simplicity, each stimulus type (T1, T2, and distractor) is processed in a single dedicated pathway (as opposed to, e.g., each particular distractor item being represented separately). Stimulus presentation is simulated by activating the appropriate input unit. This unit activates its corresponding decision unit and, to a lesser extent, the other decision units, simulating feature similarity across stimulus categories. Activity then flows from the T1 and T2 decision units to their corresponding T1 and T2 detection units. These units represent the *output*, or response, of the model. Note that in the previous modeling work of Usher et al. (1999) and Gilzenrat et al. (2002), the output units were intended to represent motor responses, whereas in the current model, these units flag the detection of targets in the RSVP stream. If a detection unit crosses a predetermined absolute activity threshold, the

<sup>2</sup> LC-induced noradrenergic modulation is nonspecific with respect to stimulus content or location. Rather, our model proposes that LC phasic responses are selective with respect to time, serving as a temporal filter. This property of the LC–NE system stands in contrast with, and complements, attention systems specified in the classical attentional literature that typically display content- or location-specific selectivity. Our model suggests that the interaction between such classic attentional systems and the LC provides a mechanism for limiting the attentional enhancement of processing to the time at which task-relevant stimuli occur.





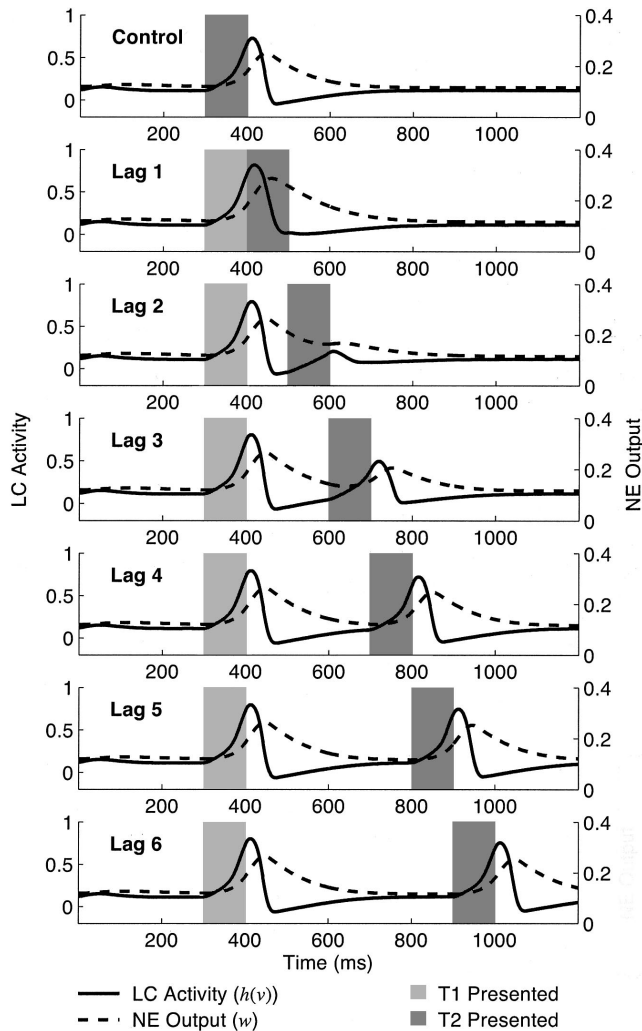
*Figure 2.* Architecture of the computational model. Circles indicate connectionist units. Activity flows across the connections indicated by the solid lines in the direction of the arrowheads. Triangular arrows indicate connections with positive (excitatory) weight; round arrows indicate connections with negative (inhibitory) weight. Line weight indicates relative connection strength. Broken lines with square ends encompass those units (all decision and detection units) innervated by the simulated neuromodulatory nucleus locus coeruleus (LC). The noradrenergic output of the simulated LC multiplicatively scales the net input to each of the units in the decision layer and the detection layer (i.e., regulates gain). For clarity of illustration, we did not indicate the cross talk connections and inhibitory connections between the first target (T1) and distractor pathways (see the Appendix for details). T2 = second target.

corresponding target has been detected by the model. Thus, target detection is a binary variable, consistent with subjective report ratings suggesting that perception during the attentional blink is all-or-none (Dehaene, Sergent, & Changeux, 2003). Because, in RSVP paradigms, subjects are not asked to report the detection of distractors, no distractor detection unit is implemented in the model. Our model also does not include mechanisms responsible for the encoding of detected targets into short-term memory (for later reporting), although target-elicited LC responses may enhance the functioning of these processes as well.

*The abstracted LC.* The abstracted LC component of the model simulates population-level dynamics of the LC–NE system observed in neurophysiological studies. It consists of a system of differential equations governing the behavior of two variables: one variable representing the state of the LC (which, in turn, is passed through an activation function returning a value that represents LC firing rate at the population level), the other variable representing its noradrenergic output (both locally and in the cortical [behavioral] network). The typical behavior of this system is illustrated in Figure 3 (top panel). When the system is in its equilibrium state, the LC-state variable is highly excitable, so when it receives sufficient afferent input, LC activation shows a large, phasic excursion, simulating the rapid posttarget increase in LC firing rate. The LC-state variable is driven directly by the activity of target units in the decision layer of the behavioral network, and it is parameterized so that when sufficient afferent activity is integrated in a relatively short time frame (typically when one of the decision units wins the competition among them), the LC crosses an internal threshold and emits a phasic response. This behavior captures evidence that LC neurons are weakly electronically coupled (Ishimatsu & Williams, 1996), so a strong, temporally coherent afferent signal can recruit a population-wide response.

In contrast to the LC-state variable, the noradrenergic-output variable changes at a much slower rate. Its activity is driven by the LC-state variable, simulating the effect of LC activity on NE release. It is important to note that noradrenergic output has an inactivating effect on the LC-state variable, capturing the effects of local autoinhibition. As a consequence, it shuts down the stimulus-driven rise in LC activity, giving the LC response its characteristic phasic form. However, as a result of the slower time constant of NE effects, the LC system remains autoinhibited for a period beyond the extinction of the phasic response. During this period, the system is particularly unexcitable (*refractory*), and new afferent inputs arriving at this time will be far less likely to recruit another LC phasic response.

Although the simulated NE release has autoinhibitory effects locally, it potentiates processing in the behavioral network. Computationally, the simulated NE release multiplicatively scales the afferent signals to the network units—that is, it transiently changes their gain. NE release affects the gain of all units in the behavioral network equally, simulating the diffuse and widespread effects of noradrenergic innervation. Note that, consistent with empirical findings, the abstracted LC is driven only by presentation of motivationally relevant stimuli (in this case, T1 and T2). Specifically, as noted above, the LC-state variable is driven by the activity of target units in the decision layer such that a phasic response is generated whenever one of these crosses its threshold (and the LC is not currently refractory). Thus, although NE release is diffuse, it is driven by the processing of target stimuli and, thus, functions as a temporal filter that facilitates responses to target stimuli but not to distractors. Such selective facilitation is assumed to be critical for accurate target detection performance under conditions such as those of the attentional blink paradigm, in which the brief presentation of stimuli in an RSVP stream of distractors



**Figure 3.** Activation dynamics of the abstracted locus coeruleus (LC) in the single-target control condition (top) and in the standard attentional blink condition for Lags 1–6. Note that LC activity and norepinephrine (NE) output are scaled on separate axes. Time = 0 ms indicates the onset of the simulated trials; 100 ms corresponds with two units of model time. Plotted data are averages across all simulated trials and, hence, include T2 correct detections and misses.

means that targets are at risk of being missed (i.e., may fail to be encoded into short-term memory for later retrieval). Accordingly, to capture this LC-mediated facilitation of responses to targets, we parameterized the behavioral network so that in the absence of an LC phasic response, the model did not reliably detect a target. Because of this reliance of target detection on the LC phasic response, and the refractoriness of this response, the LC phasic response can be viewed as a bottleneck, or *limited-capacity resource*, in our model.

**Stimulus presentation.** The model was presented with an RSVP stream typical of attentional blink studies. Each stimulus was presented to the model for a duration of two units of model time (approximately the equivalent of 100 ms; see the Appendix). On each trial, a series of 12 stimuli was presented to the model. To simulate a “midstream” arrival of T1, the first 3 stimuli were always distractors, and the 4th stimulus was always T1. Of the remaining 8 stimuli, 7 were distractors, and 1 was T2. The position of T2 ranged from

immediately following T1 (i.e., at Lag 1) to 6 stimuli following T1 (i.e., at Lag 6). We simulated 1,000 trials for each of the six T1–T2 lags. T1 and T2 detection accuracy (correct detection or miss) was recorded on each trial. In addition, we simulated a control condition in which the model was required to detect a single target only. This simulation was the same as described above, except that on each trial, T1 was replaced with a distractor stimulus. This was meant to simulate a standard control condition in attentional blink research, which involves presenting both T1 and T2 but instructing subjects to ignore T1 and to respond only to T2.<sup>3</sup>

## Results

**LC dynamics.** Figure 3 illustrates the activation dynamics of the abstracted LC during presentation of the RSVP stream as a function of the lag between T1 and T2. Irrespective of lag length, presentation of T1 leads to a sharp increase in LC activity, in turn causing augmented release of NE. As the value of NE rises, the LC is suppressed due to autoinhibition, causing LC activity to drop to subbaseline levels. In the absence of further stimulation (top panel), levels of NE track LC activation, and the two variables eventually settle to baseline. Note that the time course and shape of the simulated LC phasic response show a good correspondence with empirically observed monkey LC phasic responses (Figure 1B). In contrast to the LC response to T1, the response to T2 presentation critically depends on T1–T2 lag. If T2 is presented while simulated LC activity is suppressed below baseline due to persistent local autoinhibitory NE effects (i.e., at Lags 2–3, and, to a lesser extent, at Lag 4), T2 is unlikely to elicit a second phasic response; the LC–NE system is in a relatively unperturbable state during that period.<sup>4</sup> However, if T2 is presented after the system has recovered from its refractory state (i.e., at Lag 5 or later), the LC exhibits a robust second phasic response.

Figure 4 illustrates the relationship between LC dynamics and T2 detection accuracy by comparing, for one specific lag (i.e., Lag 2), LC dynamics associated with misses versus correct detections. Because of the contribution of random noise in the model, the phasic response after T1 is sometimes larger and sometimes smaller. As the figure shows, misses are associated with a relatively large LC phasic response to T1. Because of this large response, the amount of NE released is high, leading to a more pronounced refractory period. As a result, processing of T2 is

<sup>3</sup> Alternatively, this could be simulated by attenuating or eliminating the connection between the T1 decision unit and the LC (i.e., removing T1’s psychological “salience”). However, doing so would also deprive the LC of the tonic input provided by the baseline activity of the T1 decision unit, which forms half of the LC’s total baseline afferent drive. This places LC baseline state in a less excitable portion of its dynamic range and, therefore, substantially affects LC phasic responses to T2. We note that this effect reflects an undesired and unrealistic computational consequence of the simplifications made by our model that is not representative of our theory; in the brain, the LC receives tonic drive from a wealth of cortical and subcortical brain areas, and removing the tonic input from only one of these afferent sources is very unlikely to substantially influence LC baseline state.

<sup>4</sup> At Lag 1, LC activation is not below baseline, but the LC is not “reperturbable” either. Once LC state crosses its internal threshold, LC activity tracks a more or less ballistic trajectory, and the additional input of T2 activity will not significantly affect its course.

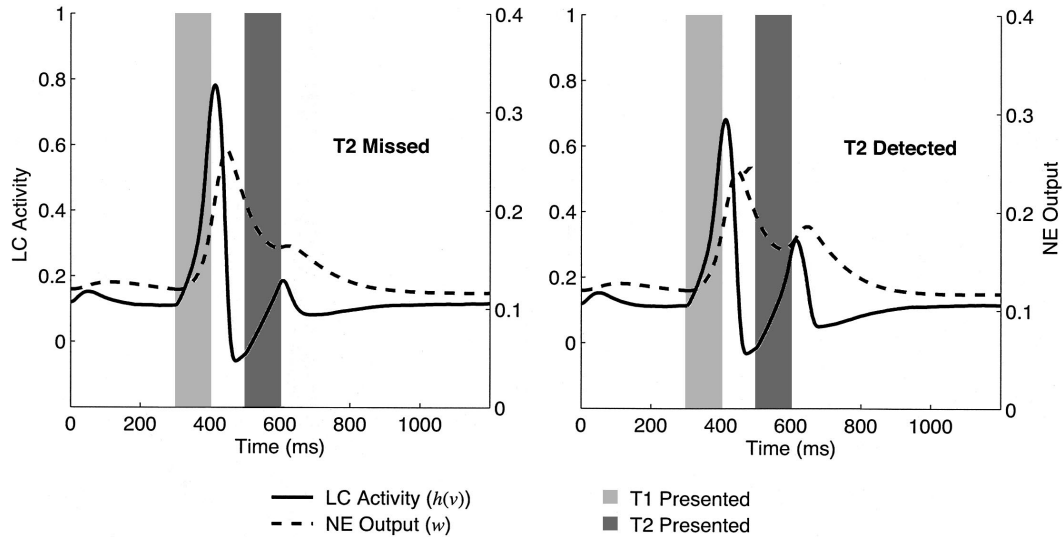


Figure 4. Average activation dynamics of the abstracted locus coeruleus (LC), plotted separately for T2 misses and correct detections. In this example, only data obtained with T1–T2 Lag 2 are shown. LC activity and norepinephrine (NE) output are scaled on separate axes. Time = 0 ms indicates the onset of the simulated trials; 100 ms corresponds with two units of model time. Note the difference between correct detections and misses in the amplitude of the LC phasic response and of the subsequent dip (i.e., refractory period).

unlikely to elicit a new LC phasic response. In the absence of LC-mediated facilitation, the detection unit for T2 is less likely to cross threshold, and therefore this target is more likely to go undetected. In contrast, correct detections of T2 are typically preceded by a smaller LC phasic response to T1 and less pronounced refractoriness, increasing the probability that T2 presentation will elicit an LC phasic response of its own and, therefore, be detected. Thus, these simulations indicate that T2 detection accuracy is heavily influenced by differences in the magnitude of the LC phasic response to T1.

*Detection accuracy for T1 and T2.* The simulated behavioral results (see Figure 5) replicate several key characteristics of attentional blink effects observed empirically. In the standard attentional blink condition, T2 detection accuracy shows a dramatic drop for Lags 2 and 3, recovering to a high level of performance by Lag 5. As outlined above, this pattern of results can be explained in terms of the degree to which the LC is suppressed at the time of T2 presentation. Indeed, in the single-target control condition, detection accuracy is at a stable asymptotic level, suggesting that T2 detection is not affected by lag alone but, rather, by the interaction between T1 and T2 processing. T1 detection accuracy, averaged across lags, was 83.4%. The model also exhibits Lag 1 sparing: Performance at the shortest lag is almost as good as the asymptotic level of performance reached for Lags 5 and further. This aspect of the simulation results can be explained in terms of the relative timing of T2 (at Lag 1) and the noradrenergic potentiation of T1 processing. As can be seen in Figure 3, the Lag 1 frame occurs well before the peak of NE release associated with T1. Thus, although T2 does not activate the LC–NE system by itself, at Lag 1, gain is still elevated sufficiently throughout the behavioral network to facilitate the processing of T2. As a consequence, performance is relatively accurate.

### Experiments

The LC–NE hypothesis of the attentional blink makes several strong predictions. For example, the presence or absence of an attentional blink on a particular trial should covary with any

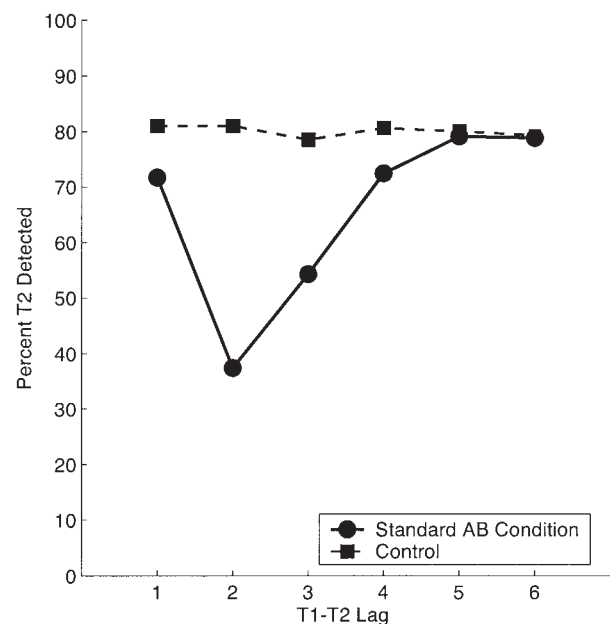


Figure 5. Simulated average T2 detection accuracy as a function of the temporal lag between the first and the second target (T1 and T2). T1 detection accuracy, averaged across lags, was 83.4% (not plotted here). AB = attentional blink.



physiological measures that index the strength of the LC phasic response to T2. Furthermore, the magnitude of the attentional blink should be affected by variables that are known to modulate the strength of the LC phasic response. As we argue in the General Discussion, considerable evidence for both of these predictions is already present in the literature. Here, we focus on a third prediction of the LC-NE hypothesis, concerning the time course of Lag 1 sparing. According to our account, the processing of items in Lag 1 benefits from the NE release elicited by the LC phasic response to T1. Note that the release and subsequent reuptake of NE is largely a time-dependent process. That is, any items that are presented during the 100–150 ms following T1 will benefit from the residual NE associated with this target. A clear prediction following from this account is that Lag 1 sparing will be preserved if an additional masking stimulus is presented between T1 and the item in the nominal Lag 1 position. It is important to note that this prediction contrasts with the predictions made by some existing models of Lag 1 sparing. For example, Raymond et al. (1992) have proposed that on the detection of T1, an attentional gate is opened that allows T1 access to a subsequent processing stage that is necessary for correct report. However, the closing of the gate is sluggish, such that the item that immediately follows T1 can also enter. If this item is T2, then T2 will be reported with greater accuracy (i.e., Lag 1 sparing). In contrast, if the item following T1 presents a source of interference, the attentional gate is immediately shut so as to exclude further interference, resulting in an attentional blink for subsequent items in the RSVP stream (i.e., Lag 2 and further). This account predicts that if a mask is inserted between T1 and the Lag 1 item, this mask will engage the protective mechanism proposed by Raymond and colleagues, leading to an attentional blink for T2s presented in the nominal Lag 1 position. Other accounts also predict that Lag 1 sparing will be observed only when T1 and T2 are of the same category and no items from other categories are presented in between (e.g., Di Lollo, Kawahara, Shahab Ghorashi, & Enns, 2005).

We conducted two attentional blink experiments to test these opposing predictions. In both experiments, subjects were required to identify two digits that were embedded in an RSVP stream of letter distractors. In Experiment 1, on half of the trials, a short-duration mask (an additional letter) was presented in between T1 and the Lag 1 item. On the other half of the trials, the mask was absent. According to the LC-NE hypothesis, the degree of Lag 1 sparing should be similar on mask-present and mask-absent trials. In contrast, according to alternative accounts, no Lag 1 sparing should be observed in the mask-present condition (Di Lollo et al., 2005; Raymond et al., 1992). Experiment 2 was similar to the first experiment, except that, in this case, on half of the trials the mask was presented simultaneously with and superimposed on T1. It is known that in and of itself, this type of superimposition mask is sufficient to produce an attentional blink (Seiffert & Di Lollo, 1997), suggesting that it leads to the immediate closing of the attentional gate, as presumed by Raymond et al. (1992). Note that with the mask in the T1 frame, the protective mechanism hypothesized by Raymond and colleagues had even more time to come into action before the arrival of the Lag 1 item. Therefore, if, in Experiment 2, we found robust Lag 1 sparing for mask-present trials, this would provide additional evidence for the LC-NE

hypothesis and against the protective mechanism account proposed by Raymond and colleagues.

### *Method: Experiment 1*

*Subjects.* Thirteen students (8 male, 5 female) from Vrije Universiteit Amsterdam, the Netherlands, ranging in age from 17 to 35 years ( $M = 22.0$ ), participated in the experiment. All subjects had normal or corrected-to-normal vision and were naive as to the purpose of the experiment. Subjects received a financial compensation of €4 (U.S.\$4.80).

*Stimuli, procedure, and design.* Stimulus generation and response recording were done using E-Prime (Psychology Software Tools, Inc., Pittsburgh, PA). Each trial started with a 1,000-ms blank period, followed by a  $0.5^\circ \times 0.5^\circ$  fixation cross, presented for 1,000 ms in the center of the display. Subsequently, the fixation point was replaced by an RSVP stream of 21 letters, each measuring approximately  $0.8^\circ \times 0.8^\circ$ . The entire stream was presented in black on a gray ( $40 \text{ cd/m}^2$ ) background. Each letter was randomly drawn without replacement from the alphabet and presented for 50 ms, followed by a 50-ms blank. *I*, *O*, *Q*, and *S* were left out because of their resemblance to digits. On each trial, two of the letters were replaced with digits, randomly drawn without replacement from the set 2–9. The first digit was presented 10–13 temporal positions from the start of the stream. The temporal distance between the first digit (T1) and the second digit (T2) was quasirandomly varied between 1, 2, 3, and 7 items, corresponding to lags of 100, 200, 300, and 700 ms. On half of the trials (and unpredictably for the subject), the blank period between T1 and the Lag 1 item was replaced by an additional 50-ms letter mask, its identity different from the other letters in the stream. The subject's task was to identify both T1 and T2. An unsped response was made at the end of each trial by typing in the digits on a standard keyboard. Trials on which T1 and T2 were identified accurately but in the wrong order were treated as correct. Following the response, a feedback display was presented for 300 ms, indicating whether T1 and T2 were correctly reported (*oo* = both correct, *ox* = T1 correct and T2 incorrect, etc.). Following the feedback, the next trial started.

The experiment started with 32 practice trials, followed by eight blocks of 32 trials each, resulting in a total of 32 trials for each combination of condition (mask present or absent) and lag. The experiment lasted approximately 30 min. Subjects were instructed to guess whenever they failed to identify a digit. All instructions were automated and presented onscreen. Apart from initial setup and final payments, there were no interactions between subjects and the experimenter, who was a lab assistant naive as to the main purpose of the experiments. T1 and T2 identification accuracy data were submitted to analyses of variance with condition (mask present or absent) and lag as repeated-measurement factors. A Greenhouse-Geisser correction was applied where appropriate.

### *Method: Experiment 2*

*Participants.* Thirteen students (8 male, 5 female) from Vrije Universiteit Amsterdam, ranging in age from 17 to 30 years ( $M = 20.8$ ), participated in the experiment. All subjects had normal or corrected-to-normal vision and were naive as to the purpose of the experiment. Subjects received a financial compensation of €4 (U.S.\$4.80).

*Stimuli, procedure, and design.* All details were the same as in Experiment 1, except as noted below. With the exception of T1, which was white ( $65 \text{ cd/m}^2$ ), the entire stream was presented in light green ( $51 \text{ cd/m}^2$ ) on a black background. All items in the stream measured approximately  $1.0^\circ \times 1.0^\circ$ . In this experiment, the additional letter mask was placed not between T1 and the Lag 1 item but in the same frame as T1 (cf. Seiffert & Di Lollo, 1997). That is, on half of the trials (and unpredictably for the subject), T1 and the additional (light green) letter were displayed simultaneously in the same location. Note that the increased intensity of T1 helped to distinguish T1 from the superimposed letter.

*Results: Experiment 1*

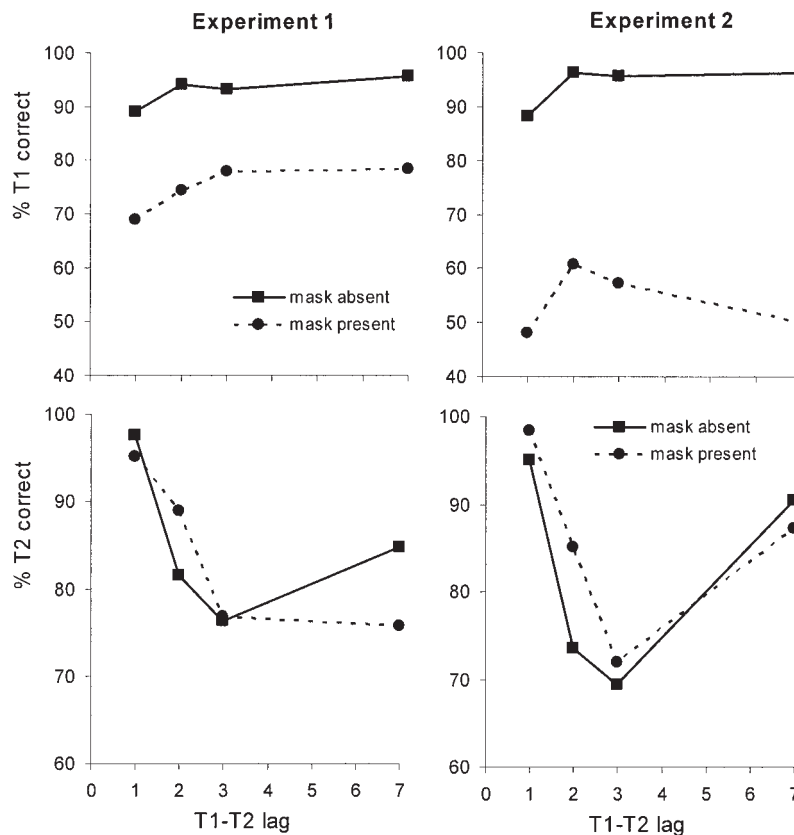
The left panels of Figure 6 show T1 accuracy and T2 accuracy as a function of T1–T2 lag and condition (mask present or absent). In both experiments, T2 accuracy was based on only those trials on which T1 was correctly identified. As expected, T1 accuracy was reduced by the presence of the additional letter mask, as indicated by a significant main effect of condition,  $F(1, 12) = 37.56$ ,  $MSE = 226.43$ ,  $p < .001$ . The main effect of lag was also significant,  $F(3, 36) = 7.98$ ,  $MSE = 44.20$ ,  $p = .001$ , reflecting impaired T1 accuracy for shorter lags. There was no Lag  $\times$  Condition interaction ( $F < 1$ ). The pattern of T2 accuracy showed substantial Lag 1 sparing, followed by an attentional blink for longer lags. This time course was reflected in a significant effect of lag,  $F(3, 36) = 14.28$ ,  $MSE = 195.33$ ,  $p < .001$ . The presence of a mask did not reliably affect T2 accuracy ( $F < 1$ ). The Lag  $\times$  Condition interaction was also nonsignificant,  $F(3, 36) = 3.51$ ,  $p > .05$ .

We computed the degree of Lag 1 sparing as the difference between T2 accuracy at Lag 1 and T2 accuracy at Lag 3, the lag at which the attentional blink was most pronounced. It is important to note that there was no reliable difference between the degree of Lag 1 sparing in the mask-present condition (18.4%) and that in

the mask-absent condition (21.3%),  $F(1, 12) = 0.44$ ,  $MSE = 124.60$ ,  $p = .52$ .

*Results: Experiment 2*

The right panels of Figure 6 show T1 accuracy and T2 accuracy as a function of T1–T2 lag and condition (mask present or absent). The results were similar to those in Experiment 1. T1 accuracy was lower in the mask-present condition than in the mask-absent condition,  $F(1, 12) = 230.78$ ,  $MSE = 181.65$ ,  $p < .001$ , and varied significantly with lag,  $F(3, 36) = 12.14$ ,  $MSE = 66.70$ ,  $p < .001$ . The Lag  $\times$  Condition interaction was nonsignificant,  $F(3, 36) = 2.87$ ,  $MSE = 57.26$ ,  $p > .05$ . T2 accuracy showed the typical dip as a function of lag,  $F(3, 36) = 20.73$ ,  $MSE = 304.91$ ,  $p < .001$ . Overall, the presence of a mask did not reliably affect T2 accuracy,  $F(1, 12) = 3.99$ ,  $MSE = 83.28$ ,  $p > .05$ . However, unlike in Experiment 1, the Lag  $\times$  Condition interaction was significant,  $F(3, 36) = 3.82$ ,  $MSE = 89.72$ ,  $p = .033$ . Most important, there was no reliable difference between the degree of Lag 1 sparing in the mask-present condition (26.6%) and that in the mask-absent condition (25.6%),  $F(1, 12) = 0.05$ ,  $MSE = 124.45$ ,  $p = .83$ .



*Figure 6.* Performance in Experiments 1 and 2 as a function of condition (mask present or absent) and the lag between the first target (T1) and the second target (T2). Top: Mean percentages of trials on which T1 was correctly identified. Bottom: Mean percentages of trials on which T2 was correctly identified, given accurate identification of T1. In Experiment 1, the mask was presented between T1 and the item presented at Lag 1. In Experiment 2, the mask was presented simultaneously with and superimposed on T1.

## Discussion

We investigated whether the insertion in the RSVP stream of an additional masking stimulus, either between T1 and the Lag 1 item (Experiment 1) or superimposed on T1 (Experiment 2), would affect Lag 1 sparing. As expected, in both experiments, T1 identification accuracy was substantially affected by the presence of the mask, indicating that the mask interfered with the processing of T1. It is important to note that, irrespective of the presence or absence of the mask, there was considerable Lag 1 sparing in both experiments. Indeed, there was essentially no difference between the degree of Lag 1 sparing in the mask-present and mask-absent conditions. These results are consistent with the LC–NE hypothesis, which predicts that Lag 1 sparing is time dependent and should not be affected by presentation of additional task-irrelevant items before Lag 1. In contrast, the results are inconsistent with an alternative account, according to which the additional mask should engage a protective mechanism (denying subsequent items the access to a further processing stage) that will be in place by the time the Lag 1 item is processed. The results—at least those of Experiment 1—are also inconsistent with a recently proposed hypothesis by Di Lollo et al. (2005). According to this hypothesis, Lag 1 sparing occurs because T2, if it is from the same category as T1, can be processed with the same system configuration as T1. However, if T1 is followed by an item from another category, this will trigger an exogenous change in the system configuration, which as a consequence will no longer be tuned to process items of the target category. This leads to an attentional blink for subsequently presented targets. With regard to Experiment 1, this hypothesis predicts that an additional short-duration mask following T1 should trigger a system configuration, resulting in an attentional blink for a subsequent target in the nominal Lag 1 position. The results from Experiment 1 demonstrate that this prediction is incorrect.

## General Discussion

We have proposed a new hypothesis concerning the neural mechanisms underlying the attentional blink. According to this hypothesis, the attentional blink is mediated by the temporal dynamics of activity of the LC–NE system. Phasic bursts of activity of this system play an important role in facilitating responses to task-relevant stimuli. These phasic responses typically occur within a short latency of the eliciting stimulus, and they are followed by a period of functional refractoriness as a result of noradrenergic autoinhibition of the LC. Our hypothesis suggests that this refractoriness in LC activity is responsible for the attentional blink: The temporary unavailability of LC-mediated noradrenergic facilitation leads to a processing deficit for T2. This account is consistent with neurophysiological data and with a recent theory regarding the role of the LC–NE system in regulating attention and goal-directed action (Aston-Jones et al., 1999, 2000; Cohen et al., 2004; Usher et al., 1999). It is also consistent with previous computational modeling efforts, which have shown how an abstract computational model of LC dynamics can account for detailed aspects of target detection performance in monkeys (Gilzenrat et al., 2002; see also Usher et al., 1999). In the present study, we used this model to formally articulate our hypothesis of

the attentional blink, making only a few modifications to capture features of the attentional blink paradigm. The model accurately simulated the time course of the attentional blink, lending support to our hypothesis.

Apart from an early drop followed by a steady recovery in performance, the model also simulated some other key properties of the attentional blink. Most important, the model exhibited Lag 1 sparing, consistent with typical human behavioral data (Raymond et al., 1992; Vogel, Luck, & Shapiro, 1998). In terms of our hypothesis, this can be understood as a result of the diffuse facilitative effects of NE and their temporal dynamics. That is, the noradrenergic boost resulting from an LC phasic response to one stimulus can influence the processing of other stimuli during a critical window of time. Although further physiological research is needed to determine the exact width of this time window, the value assumed by our model is within a physiologically plausible range (Berridge & Waterhouse, 2003). Thus, in the attentional blink paradigm, if one target immediately follows another (i.e., with Lag 1), the residuum of NE release associated with T1 benefits processing of T2, allowing it to escape the disrupting effect of LC refractoriness. This account of Lag 1 sparing is also consistent with the finding of *+1 posttarget intrusion errors*—that is, naming of the to-be-reported feature of the item immediately following a target stimulus (cf. Raymond et al., 1992). An interesting question for future simulations is whether at very short lag lengths (e.g., <50 ms), T2 may benefit even more than T1 itself from the noradrenergic potentiation associated with T1 presentation (cf. Potter, Staub, & O'Connor, 2002).

Our account predicts that Lag 1 sparing should be preserved as long as T2 is processed during the critical time window during which it can benefit from residual NE associated with T1. In two experiments, we tested this prediction by presenting a short-duration mask simultaneously with or immediately following T1 but before the item in the nominal Lag 1 position. The LC–NE hypothesis suggests that under these conditions, Lag 1 sparing should be preserved, because the time interval between T1 (and the associated noradrenergic release) and Lag 1 remains constant. The experimental results confirm this prediction, indicating pronounced Lag 1 sparing regardless of whether the short-duration mask was present or absent. In contrast, the results are inconsistent with alternative accounts of Lag 1 sparing that claim that any task-irrelevant items interfering with T1 (like our short-duration mask) will trigger a process resulting in an attentional blink for subsequent items (e.g., T2 in Lag 1; see, Di Lollo et al., 2005; Raymond et al., 1992). Hence, the reported results provide evidence that specifically supports our account of Lag 1 sparing.

A potential problem for our theoretical account is the absence of Lag 1 sparing in a number of attentional blink studies (reviewed in Visser, Bischof, & Di Lollo, 1999). Why would processing of T2 benefit from a residuum of NE release in some experiments but not in others? Visser, Bischof, and Di Lollo (1999) noted that Lag 1 sparing is not found when the two targets are displayed in different spatial locations. For example, Visser, Zuvic, Bischof, and Di Lollo (1999) manipulated the location of T1 and T2 (Central or eccentric) in a  $2 \times 2$  factorial design, and observed Lag 1 sparing only when both targets were presented in the same location. It is possible that the absence of Lag 1 sparing with different target locations reflects the detrimental effects of attention still being

occupied by T1 while T2 is being presented in another location.<sup>5</sup> Indeed, it is well-known that it takes longer than 100 ms (i.e., one lag) to fully process an item at one location and move attention to a new item at a different location, and that during this “attentional dwell time,” processing of the to-be-attended item is impaired (e.g., J. Duncan et al., 1994). Under such conditions, the cost of attention being focused elsewhere may outweigh the benefit of residual NE, resulting in the absence of Lag 1 sparing.

Another result of the simulations is that the size of the simulated attentional blink exhibited a negative correlation with the size of the model’s LC phasic response to T1. This finding appears to be compatible with results from a recent magnetoencephalography (MEG) study of attentional blink performance (Shapiro, Schmitz, Martens, Hommel, & Schnitler, 2005). Shapiro et al. (2005) identified the magnetic equivalent of the electrophysiological P3 component (i.e., the *mP3*) and investigated how the *mP3* elicited by T1 and T2 (presented at Lag 2) covaried with attentional blink magnitude. They found that failure to detect T2 was associated with a larger *mP3* to the preceding T1. Similarly, they found that individuals showing a larger *mP3* to T1 exhibited increased attentional blink magnitudes for T2. These findings support a limited-capacity account of the attentional blink: Increasing the processing capacity allocated to T1 decreases the processing capacity available to accurately process T2. Our hypothesis offers an account of the neural basis of this limited-capacity mechanism. This account rests on the assumption, supported below, that the P3 (*mP3*) is an electrophysiological (magnetic) correlate of the LC phasic response and its influence on information processing in neocortex. On this assumption, variations in the size of the *mP3* to T1 reflect variations in the size of the LC phasic response, leading to variations in NE release in neocortex. Along the same lines, the enlarged attentional blink associated with large-amplitude *mP3*s reflects the more pronounced refractoriness following large LC phasic responses.

A potential challenge for the LC–NE hypothesis concerns the finding that the magnitude of the attentional blink is reduced if the frame immediately following T1 (i.e., at Lag 1) is left blank (Chun & Potter, 1995; Raymond et al., 1992). It is generally thought that this effect is mediated by backward masking of T1 by the Lag 1 item (e.g., Brehaut, Enns, & Di Lollo, 1999; Seiffert & Di Lollo, 1997). Our computational model does not incorporate specific mechanisms for simulating backward masking and, hence, does not directly address the effect of T1 masking on T2 performance. Nevertheless, there are two ways in which the LC–NE hypothesis might be able to explain this effect. First, empirical results and analysis of a detailed biophysical model of the LC suggest that the occurrence of a refractory period is dependent on the time course (i.e., strength and duration) of target-related inputs to the LC (Brown, Moehlis, et al., 2004). More specifically, for a refractory period to occur, target-related input to the LC must be punctate; inputs that are more protracted in time result in a weaker or absent refractory period. This suggests that targets that are masked, and therefore result in a more punctate neural response (Keyser & Perrett, 2002), drive the LC in a way that produces a stronger refractory period (and, hence, a more pronounced attentional blink) than do unmasked targets, which are associated with a more protracted neural response. Second, if T1 is not masked, processing of T1 will presumably be completed earlier, thereby alleviating

any bottleneck (independent from the LC) that may impede the processing of T2 (cf. Chun & Potter, 1995). The beneficial effects of this mechanism may be additive to the deleterious effects of the LC refractory period, resulting in a net attenuation of the attentional blink. Although this explanation invokes an additional mechanism outside our theoretical framework (and is, therefore, costly in terms of parsimony), it is unlikely that any single mechanism can explain the plethora of experimental factors that are known to modulate the attentional blink. Indeed, additional mechanisms may also be needed to explain some other observations that the LC–NE hypothesis in its present form leaves unaddressed. These include the effect of the nature of distractor stimuli in the RSVP stream (e.g., Chun & Potter, 1995; Isaak, Shapiro, & Martin, 1999), the strongly reduced attentional blink following T1 duration judgments (Sheppard, Duncan, Shapiro, & Hillstrom, 2002), and the role of spatial parameters in the attentional blink (e.g., Kristjánsson & Nakayama, 2002).

Our theory differs considerably from another recently proposed neurocomputational model of the attentional blink (Dehaene et al., 2003). Dehaene and colleagues postulated that a target stimulus can gain conscious access as a result of gamma-band oscillatory interactions between sensory brain areas and higher order association areas, leading to the top-down amplification of stimulus processing. For the period during which this global reverberant state is dedicated to the processing of the target stimulus, sensory processing of other incoming stimuli cannot receive similar top-down amplification, leading to an attentional blink. This hypothesis is formalized in terms of a detailed model of thalamocortical interactions at the single-neuron level. The model accurately simulates the drop of performance in reporting the second of two targets presented in close succession, and it makes interesting predictions regarding the relationship between the attentional blink and the power of gamma-band electrophysiological activity in widespread areas of the brain. At the same time, a shortcoming of the model is that it does not explain Lag 1 sparing. Furthermore, one might ask whether insight into the origin of the attentional blink necessarily requires a model at the level of single-neuron properties. An attractive feature of our model is that it captures in a relatively simple fashion the computational features of the biological mechanisms of interest that are relevant to function at the systems level. One beneficial consequence of this is that the abstracted LC is computationally tractable and can therefore be applied to simulations of LC effects in a wider array of behavioral task models (e.g., Gilzenrat et al., 2002). Of course, it is possible that oscillatory activity is a reflection of target processing that is enhanced by LC function, suggesting that exploration of the relationship between these two models may be a profitable avenue for future research.

<sup>5</sup> Visser, Zuvic, et al. (1999) also considered this account but rejected it. They argued that it cannot explain why T1 accuracy was similar regardless of whether T1 was presented in the same or in a different location than the preceding distractor items. We note, however, that subjects were always informed where each target item would appear. As a consequence, subjects could focus their attention on the anticipated location of T1 at the beginning of each trial, thus preventing the need for an “expensive” spatial attention shift for T1. This would not be so for T2.



### *Relationship With the P3, Neuroimaging Data, and Neuropsychological Data*

Previous research has revealed a close relationship between the attentional blink and the P3—a broad, positive event-related brain potential with a modal peak latency of about 300 ms poststimulus (McArthur, Budd, & Michie, 1999; Rolke, Heil, Streb, & Henninghausen, 2001; Vogel et al., 1998). Vogel et al. (1998) studied event-related brain potential components elicited by stimuli presented during the attentional blink. Whereas components associated with early sensory processing (P1 and N1) and a later component associated with semantic analysis (N400) were unaffected by the attentional blink, the P3 was completely suppressed. Rolke et al. (2001) later qualified this finding: T2 stimuli elicited no P3 when they were unidentified but evoked a clear P3 when they were identified by the subject (for similar findings, see Dell'Acqua, Jolicœur, Pesciarelli, Job, & Palomba, 2003; Kranczioch, Debener, & Engel, 2003; Shapiro et al., 2005). Other researchers have found that the magnitude of the attentional blink varies with the difficulty and frequency of T1 and that an attentional blink can be elicited by novel distractor stimuli (e.g., Barnard, Scott, Taylor, May, & Knightley, 2004; Crebolder, Jolicœur, & McIlwaine, 2002; McArthur et al., 1999). Task difficulty, frequency, and novelty are also key determinants of P3 amplitude (see Picton, 1992).

The LC–NE hypothesis of the attentional blink offers a natural account of the link between the attentional blink and the P3. It has been proposed that scalp-recorded P3 activity reflects the NE-induced phasic enhancement of neural responsiveness in neocortex (Pineda, Foote, & Neville, 1989). In recent work, we have argued that this view is consistent with several properties of the LC (Nieuwenhuis et al., 2005). First, the relatively long conduction times of NE-releasing fibers are consistent with the P3 latency in animals and humans (Aston-Jones, Segal, & Bloom, 1980; Berridge & Waterhouse, 2003). Second, the high divergence in combination with regional specificity of the LC efferent projection system is consistent with the broad scalp distribution of the surface-recorded P3 and the distribution of P3-like potentials recorded intracranially in animals and humans (for reviews, see Frodl-Bauch, Bottlender, & Hegerl, 1999; Soltani & Knight, 2000). Furthermore, the more or less simultaneous release of NE in LC projection areas is consistent with the uniformity of P3 latency at the spatially distributed sites identified with intracranial recordings. Third, brain lesions (Ehlers & Chaplin, 1992; Pineda et al., 1989) and pharmacological manipulations (e.g., C. C. Duncan & Kaye, 1987; Joseph & Sitaram, 1989; Swick, Pineda, & Foote, 1994) that affect the LC–NE system strongly affect P3 amplitude. Fourth, LC activity, like P3 amplitude and attentional blink magnitude, is modulated by stimulus frequency (Aston-Jones et al., 1994, 1997) and novelty (Sara & Segal, 1991). Finally, the LC refractory period appears to be mirrored by a similar refractory period in P3 elicitation (Woods, Hillyard, Courchesne, & Galambos, 1980). Nieuwenhuis et al. (2005) have presented a detailed review of the literature concerning the relationship among the LC–NE system, the P3, and information processing.

Thus, according to the LC–NE hypothesis, the P3 is an electrophysiological correlate of noradrenergic potentiation of responses to motivationally relevant stimuli. This hypothesis explains the positive correlation between T2 accuracy and the amplitude of the

P3 elicited by T2 (Rolke et al., 2001; Vogel et al., 1998). In contrast, our simulations predict a negative correlation between T2 accuracy and the amplitude of the P3 elicited by T1, a prediction that is consistent with empirical findings (Martens, Johnson, Elmallah, & London, 2005; McArthur et al., 1999; Shapiro et al., 2005). Furthermore, our account explains why stimulus novelty and frequency, which modulate the size of the LC phasic response and the P3, significantly influence the attentional blink. The LC–NE hypothesis can also serve to generate detailed new predictions regarding the relationship between the attentional blink and the P3. For example, it makes the somewhat counterintuitive prediction that the P3 should be substantially reduced for T2s presented at Lag 1, even though performance at Lag 1 is relatively spared. According to our account, these targets benefit from residual NE associated with T1 but—due to refractoriness—are unlikely to elicit an LC phasic response and corresponding P3. This prediction has been confirmed in a recent study that found no clear P3 for T2s at Lag 1 irrespective of whether the target was detected (Kranczioch et al., 2003).

The LC–NE hypothesis seems also consistent with the limited available evidence from neuroimaging and patient studies that have used the attentional blink paradigm. First, studies using functional imaging methods have suggested that target processing in the attentional blink task is mediated by a widespread cortical network including parietal cortex, anterior cingulate cortex, and lateral frontal cortex (Marois, Chun, & Gore, 2000) and that activation differences in these cortical areas correlate with T2 performance (Kranczioch, Debener, Schwarzbach, Goebel, & Engel, 2005; Marois, Yi, & Chun, 2004). Converging evidence for a relationship between activity in this network and T2 performance has been reported in a recent MEG study (Gross et al., 2004). It is interesting to note that the parietal and frontal cortex are the cortical areas with the densest noradrenergic innervation (Levitt, Rakic, & Goldman-Rakic, 1984; Morrison & Foote, 1986). This may suggest that the performance-related activity differences in these brain areas reflect differential degrees of norepinephrine-induced phasic enhancement of neural responsiveness.

Second, various neuropsychological and neuroimaging studies have suggested a greater involvement of the right than of the left hemisphere during performance of the attentional blink task (e.g., Giesbrecht & Kingstone, 2004; Husain, Shapiro, Martin, & Kennard, 1997; Marois et al., 2000). Consistent with this, the limited available evidence suggests that the right hemisphere contains a higher concentration of NE than the left hemisphere (e.g., Oke, Keller, Mefford, & Adams, 1978; Robinson, 1979). Finally, Giesbrecht and Kingstone (2004) have reported neuropsychological evidence suggestive of a subcortical locus of the limited-capacity mechanism underlying the attentional blink. They tested a split-brain patient in the attentional blink task, presenting T1 and T2 in the same visual field (and thus to the same hemisphere) or in opposite visual fields (to different hemispheres). An attentional blink was found in both cases, indicating that the two hemispheres draw from the same limited-capacity resources. Because subcortical connections are preserved in split-brain patients, it is likely that these resources (e.g., NE) are distributed from a subcortical locus with bilateral connections with both cortices (e.g., the LC).



### *Relationship With Cognitive Theories of the Attentional Blink*

The neurobiological account that we have proposed should not necessarily be seen as an alternative to existing cognitive theories of the attentional blink. Rather, the biological mechanisms outlined by our account may provide a neural basis for the processes that are postulated by these cognitive theories. Despite their differences, most cognitive theories have in common that they distinguish between an early processing stage during which all stimuli are processed to some degree (and targets and distractors are preattentively differentiated) and a later processing stage during which targets and other RSVP items compete for limited attention resources (e.g., Chun & Potter, 1995; Jolicoeur, 1998; Shapiro et al., 1994). While these resources are dedicated to processing of T1, less attention is available for T2, leaving it vulnerable to decay and interference from a variety of sources. The LC–NE hypothesis corresponds well with this functional account of the attentional blink. In our model, all items are processed to a considerable degree (up through the decision layer). However, under the data-limited conditions presented by the RSVP stream, this processing is not sufficient to consistently detect targets (i.e., to drive the response units above their detection threshold). Accurate detection relies on LC-mediated noradrenergic modulation, which functions as a temporal filter by facilitating the processing of responses to target stimuli. This noradrenergic modulation is a limited-capacity attentional resource: Because of LC refractoriness, it is less available immediately following a first target, and the larger the response of the LC–NE system to the first target, the more pronounced this refractoriness.

It is important to note that we do not claim that LC-induced noradrenergic potentiation is always necessary for target detection under data-limited conditions. Some prepotent stimuli may have an inherent baseline activation (or strength of connectivity) that is so high that very little processing is needed to reach the detection threshold, and hence, noradrenergic potentiation is not needed (e.g., one's own name; Shapiro, Caldwell, & Sorensen, 1997). Furthermore, the LC–NE hypothesis allows for the possibility that brain areas other than the LC are able to pick up graded levels of activation in the decision layer. These may include brain areas involved in semantic analysis, which is consistent with findings that missed T2 items can semantically prime subsequent stimuli and elicit an N400 event-related brain potential when they are semantically incongruous with a preceding context (e.g., Vogel et al., 1998).

Cognitive theories have generally ascribed Lag 1 sparing to the sluggish closing of an attentional gate or attentional window (e.g., Chun & Potter, 1995; Raymond et al., 1992). The gate opens rapidly on presentation of T1 but closes slowly, thus permitting the next item in the stream (at Lag 1) to enter a higher processing stage. However, Shapiro, Arnell, and Raymond (1997) have noted that "such explanations are post hoc in nature and lack any suggestion of a plausible mechanism able to account for this outcome" (p. 295). An attractive feature of the LC–NE hypothesis is that it proposes a single mechanism that provides a unified account of the attentional blink and Lag 1 sparing. During the attentional blink, noradrenergic potentiation is unavailable, but items immediately following T1 may still benefit from the NE release associated with

T1. The time window of this T1-induced noradrenergic potentiation may thus present a neural correlate of the attentional gate or window invoked by cognitive theories of the attentional blink.

### *Conclusions and Directions for Future Research*

Neurophysiological data and computational modeling have suggested a crucial role for the LC–NE system in attention and goal-directed behavior. Here, we propose that the attentional blink, a phenomenon that plays a central role in the attention literature, may be explained as a result of the refractoriness displayed by the LC–NE system following salient stimuli. Using model simulations, we demonstrated that this and other properties of the LC–NE system may produce the strong nonlinearity that is typical of the performance accuracy functions obtained in attentional blink experiments. Our research highlights the value of computational modeling in understanding complex dynamical phenomena such as the attentional blink, and it produces further evidence suggesting that brainstem neuromodulatory nuclei may play a central and critical role in cognitive processing.

The current research suggests several directions for future research. One important goal for future research will be to search for and validate potential noninvasive measures of LC–NE activity in humans that can then be correlated with attentional blink performance. Our model is based primarily on the dynamics of LC firing and NE release in the monkey, so an important imperative is to confirm that the dynamics of these functions are similar in the human. As we have noted, the electrophysiological P3 potential is an important candidate for doing so, and considerable progress has been made in validating this potential as a correlate of LC-induced noradrenergic potentiation (Nieuwenhuis et al., 2005). Additional research is also needed to elaborate the possible correlational relationship between pupil diameter and LC activity that is suggested by findings from nonhuman primate studies (e.g., Aston-Jones, Ennis, Pieribone, Nickell, & Shipley, 1986). Preliminary experimental evidence suggests that phasic and tonic changes in pupil diameter closely track the time course of LC activity (Gillenrat, Cohen, Rajkowski, & Aston-Jones, 2003; Rajkowski, Kubiak, & Aston-Jones, 1993). Furthermore, the current work may motivate the development of new methods (e.g., noradrenergic ligands) that will enable the use of neuroimaging methods for imaging the LC with sufficient anatomical precision.

A second promising direction for future research will be to examine the impact on attentional blink performance of drugs that influence activity of the LC–NE system. Pharmacological studies have already established that clonidine, a noradrenergic autoreceptor agonist, affects target detection performance in humans and nonhuman primates (e.g., Coull, Middleton, Robbins, & Sahakian, 1995). The current theory may be used as a guide in formulating specific predictions about how such drugs should modulate target detection performance in the attentional blink task. Of course, the theory may also inspire new predictions for standard behavioral experiments. As an example, Olivers and Nieuwenhuis (2005) have recently found that the attentional blink is significantly ameliorated under task circumstances that induce a more distributed state of mind. This research was motivated in part by the strong dependence of the LC phasic response on tonic LC activity, an important determinant of general arousal state (see Footnote 1).

Finally, a critical objective for future research will be the development of an attentional blink paradigm that is suitable for primate research. This will allow a direct test of the link between LC activity, as measured with single-cell recordings, and attentional blink performance.

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## Appendix

### Details of Model and Simulations

#### Model Units

The behavioral network of the model consists of several connectionist units, each representing an assembly of cells dedicated to a particular computation in the information processing stream (Rumelhart & McClelland, 1986). Generally, the state  $X_i$  of each unit  $i$  is updated on a cycle-by-cycle basis by numerically integrating the following ordinary differential equation:

$$\dot{X}_i = -X_i + \sum_j w_{ij} f(X_j) + \xi_i \quad (1)$$

where  $j$  iterates over all units in the network (including  $i = j$ , allowing for recurrent connectivity), and  $w_{ij}$  is the connection strength (or weight) from unit  $j$  to unit  $i$ . The term  $-X_i$  represents decay (or “leak”), and the term  $\xi_i$  represents stochastic noise, drawn from a Gaussian distribution with 0 mean and standard deviation  $\sigma$  (fixed to 0.15 in the current simulations), independently for each unit. The function  $f$  describes the sigmoidal activation function,

$$f(X_i) = \frac{1}{1 + e^{-g(X_i - b_i)}} \quad (2)$$

where  $b_i$  is a tonic bias input to unit  $i$  (fixed to 1.75 in the current simulations), and  $g_t$  is the multiplicative gain on the unit’s input at time  $t$ . Gain is a variable that depends on the current activity of the abstracted LC and, thus, changes dynamically over the course of each simulated trial. In connectionist terms,  $X_i$  is the net input of unit  $i$ , and  $f(X_i)$  is interpreted as its activity.

#### Behavioral Network

##### Input Layer

The input layer consists of three units, one for representing each stimulus category (T1, T2, distractor). Unlike other units in the behavioral network, the activity of each input unit is fixed (“hard-clamped”) to either 0 or 1, depending on which stimulus is being presented to the model.

##### Decision Layer

The decision layer comprises three units. Activity of these units represents the degree to which the model has classified the current input stimulus as one stimulus category over the others. Each decision unit receives afferent activity from its corresponding input unit over a connection of fixed weight 1.5. Each decision unit also receives input activity from the other two input units over a connection of fixed weight 1/3. These “cross-talk” connections are intended to represent stimulus ambiguity. Furthermore, the decision units share mutual inhibitory connections (of fixed weight  $-1.0$ ), simulating competition among alternative representations of the presented stimulus. Each decision unit additionally receives a self-recurrent connection of fixed weight 2.5, simulating mutual excitatory influences within the cell assemblies represented by each connectionist unit.

##### Detection Layer

The detection layer comprises two units, one for the detection of each of the two target categories (T1 and T2). Because, in the typical attentional blink paradigm, subjects are not required to report the detection of distractors, no distractor detection unit is modeled. Each detection unit receives afferent activity from its corresponding decision unit over a connection of fixed weight 3.5. Furthermore, each detection unit has an excitatory recurrent self-connection (of fixed weight 2.0). A successful detection of a target is recorded if activity of the corresponding detection unit crosses a threshold value (0.67) in the course of a simulated trial.

#### Abstracted LC

The abstracted LC consists of the modified FitzHugh–Nagumo system detailed in Gilzenrat et al. (2002), including the same parameter values. This system is governed by the interaction of two variables,  $v$  and  $u$ , which are given by the system of ordinary differential equations

$$\tau_v \dot{v} = w_{vX} [f(X_{T1}) + f(X_{T2})] + v(a - v)(v - 1) - u \quad (3)$$

and

$$\tau_u \dot{u} = h(v) - u \quad (4)$$

We take  $v$  to represent the state (analogous to “net input” in connectionist terms) of the abstracted LC unit, and we take  $u$  to represent its noradrenergic output. The time constant of LC state ( $\tau_v$ ) is much smaller than the time constant of noradrenergic output ( $\tau_u$ ): 0.05 and 5.0, respectively. Equation 3 indicates that the change in state of the LC is determined by three terms. The first term represents external input to the nucleus, which in our model comes from the activity of the T1 and T2 decision units— $f(X_{T1})$  and  $f(X_{T2})$ , respectively—over a connection of weight  $w_{vX} = 0.3$ . The second term is a cubic function of the LC’s own state, which provides the excitable dynamics that are characteristic of a FitzHugh–Nagumo system (see Gilzenrat et al., 2002; Keener & Sneyd, 1998). The parameter  $a$  governs the excitation threshold of the LC and is set at 0.5. The third term provides the inactivating effect of the variable  $u$ , simulating the local autoinhibitory effect of NE.

Equation 4 indicates that noradrenergic output  $u$  is driven by  $h(v)$ , which we consider an activation function of LC state  $v$  described by

$$h(v) = Cv + (1 - C)d. \quad (5)$$

Here,  $C$  is a coefficient (which can range from 0 to 1) that scales the relative contribution to LC activity  $h(v)$  of its afferent inputs ( $v$ ) versus intrinsic, uncorrelated activity in the nucleus as a whole (represented by the parameter  $d$ , which is fixed at 0.5). Gilzenrat et al. (2002) have explored the relationship between the  $C$  coefficient and changes in LC firing mode. In the present simulations,  $C$  was fixed at 0.9, placing the LC in a firing mode characteristic of a state of focused, selective attention.

The simulated noradrenergic output of our system,  $u$ , has direct inhibitory effects locally. However,  $u$  also impacts processing in the behavioral network by scaling the effects of gain. In this way, changes in simulated NE output dynamically adjust the level of potentiation of LC target units (all units in the decision layer and the detection layer). The relationship between noradrenergic output  $u$  and gain  $g$  is modeled as

$$g_t = G + ku_t, \quad (6)$$

where  $G$  is a base level of gain that is independent of  $u$ , and  $k$  is a scaling constant.  $G$  and  $k$  were set at 0.5 and 1.5, respectively.

### Simulation Procedures

Simulations were conducted via numerical integration of the differential equations presented above using a simple Euler method. Each run of the simulations consisted of a series of trials, each of which was 44 units of model time in duration. Time was discretized at a granularity of 0.02 (i.e.,  $dt = 0.02$  for each integration step, for a total of 2,200 iterations per trial). All unit activities were initialized to 0 prior to the onset of a trial. During the first 20 units of model time, all network units were allowed to settle to stable levels of activation in the absence of stimulus input. The remaining time involved the actual presentation of the RSVP stream, as described in the *Method* section.

We chose to present each stimulus for 2 units of model time. In previous modeling work using identical abstracted LC parameters and similar behavioral network parameters, Gilzenrat et al. (2002) found that equating 1 unit of model time with 54.6 ms of actual time yielded the best fit of simulated data to empirically established LC temporal dynamics and target detection response latencies in monkeys. Because attentional blink paradigms typically require unspeeded responses, and no empirical data regarding LC temporal dynamics in humans are available, we could not validate this relationship in the present research. Therefore, as a convenient approximation of the previously established relationship, we defined 2 units of model time as 100 ms, yielding simulated interstimulus intervals typical of attentional blink research.

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