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An artificial neural network model of orienting attention toward threatening somatosensory stimuli

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Abstract

An artificial neural network model was designed to test the threat detection hypothesis developed in our experimental studies, where threat detector activity in the somatosensory association areas is monitored by the medial prefrontal cortex, which signals the lateral prefrontal cortex to redirect attention to the threat. As in our experimental studies, simulated threat-evoked activations of all three brain areas were larger when the somatosensory target stimulus was unattended than attended, and the increase in behavioral reaction times when the target stimulus was unattended was smaller for threatening than nonthreatening stimuli. The model also generated a number of novel predictions, for example, the effect of threat on reaction time only occurs when the target stimulus is unattended, and the P3a indexes prefrontal cortex activity involved in redirecting attention toward response processes on that trial and sensory processes on subsequent trials.

Descriptors: Artificial neural network, Orienting, Attention, Threat, P3a, Medial prefrontal cortex

Threatening somatosensory stimuli, many of which are painful, are well known to capture attention (Eccleston & Crombez, 1999). This ability is important in eliminating damaging stimuli and recuperating from injury. In some cases, such as patients with intractable pain that does not have any apparent signs of peripheral pathology, there is no stimulus to escape from nor does the pain aid in recovery (Gatchel, 2004; Turk & Okifuji, 2002; Wall, 1994). Yet pain's ability to capture attention can make it very difficult for these patients to perform activities of daily living and can lead to cognitive states, such as hypervigilance and catastrophization, that maintain the pain and/or make it worse (Crombez, Eccleston, Van den Broeck, Goubert, & Van Houdenhove, 2004; Eccleston & Crombez, 1999; Turk & Okifuji, 2002). These cognitive states can also lead to the anxiety disorders that are often comorbid with chronic pain (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Fox, Russo, Bowles, & Dutton, 2001; Gatchel, 2004). Hence, understanding the neural mechanisms by which threatening somatosensory stimuli capture attention might provide insights into the development and treatment of intractable pain and the anxiety disorders that often accompany it.

Our electrophysiological studies have identified two components of the somatosensory evoked potential that appear to play a role in detecting and orienting attention toward a strong, potentially threatening electrical stimulus applied to the sural nerve. These components are negative potentials recorded from over the contralateral temporal scalp between 100 and 180 ms poststimulus (CTN100-180) and from over the frontocentral scalp between 130 and 200 ms (FCN130-200). Our dipole source localization (Dowman & Darcey, 1994, Dowman & Schell, 1999) and intracranial recording studies (Dowman, Darcey, Barkan, Thadani, & Roberts, 2007) suggest that the CTN100-180 is generated in the somatosensory association areas located in the parietal operculum and insula, and that the FCN130-200 is generated in the medial prefrontal cortex, including the anterior cingulate cortex located near the ventral anterior commissure line and the overlying presupplementary motor area.

The CTN100-180 and FCN130-200 have unusual attention properties: Their amplitudes are larger when the sural nerve-evoking stimulus is presented outside of the focus of attention (unattended) than when attention is focused on it (Dowman, 2001, 2004a, 2004b, 2007a, 2007b). The increase in the amplitude of these components in the unattend condition is seen when the evoking stimulus is task relevant (e.g., a symbolic cue given at the beginning of the trial directs the subject's attention away from the sural nerve target stimulus; Dowman, 2001, 2007a) or task irrelevant (e.g., the subjects are engaged in an arithmetic task and are instructed to ignore the sural nerve-evoking stimulus; Dowman, 2004a). These data suggest that the CTN100-180 and FCN130-200 generators are involved in an automatic, stimulus-driven process that detects and reorients attention toward the sural nerve electrical stimulus when it is presented outside the focus of attention. The latencies of these components are also consistent with this hypothesis, given that electrophysiological studies suggest that event-related potential components whose amplitudes are related to voluntary changes in attention initiated

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by the evoking stimulus occur after 300 ms (see van Velzen & Eimer, 2003).

The unusual attention effects exhibited by the CTN100-180 and FCN130-200 contrast sharply with those seen with midlatency negative potentials evoked by auditory, visual (the auditory and visual evoked potential N1 peaks), weak electrical, tactile (the somatosensory evoked potential N140 peak), and painful laser (the laser evoked potential N1 and N2 peaks) stimuli, whose amplitudes are smaller when the evoking stimulus is unattended (for reviews, see Eimer & Driver, 2001; Hillyard & Anllo-Vento, 1998; Lorenz & Garcia-Larrea, 2003; Mangun, 1995; Näätänen, 1982, 1992; Näätänen & Picton, 1987). The different effects of attention on these midlatency negative potentials versus the sural nerve-evoked CTN100-180 and FCN130-200 are likely due to the sural nerve electrical stimulus having properties that make it ideal for eliciting an involuntary orienting response (see Eccleston & Crombez, 1999; Friedman Cycowicz, & Gaeta, 2001; Näätänen, 1992; Yantis & Jonides, 1990): It is strong, has an abrupt onset, and produces an unfamiliar (novel) paraesthesia/prickling pain sensation. These properties contrast with the lower subjective intensity of the weak electrical, tactile, auditory, visual, and laser pain stimuli (at least at the levels used in most laser-evoked potential studies of attention; see Dowman, 2004d), and with the familiar sensations produced by laser, tactile, auditory, and visual stimuli. Hence, the sural nerve electrical stimulus appears much better suited for studying the neural processes involved in involuntary orienting than these other stimuli. (For a more detailed comparison between the sural nerve-evoked midlatency negative components and those evoked by these other stimuli, see Dowman, 2004a, 2004d, 2007b).

Recently we proposed that the CTN100-180 component is composed of a combination of sensory and threat detector activity, given its amplitude increases in the unattend condition when the sural nerve stimulus is strong and threatening (Dowman, 2007a) but not when it is weak and nonthreatening (Dowman, 2007b). We suggested that in the unattend condition the increase in threat detector activity outweighed the decrease in sensory activity that is usually reported in electrophysiological studies of attention (see Dowman, 2007a, 2007b). The overlap between attention- and sensory-related activities has also been reported for the N1 peak of the auditory evoked potential, where attention-related activity in the auditory association cortices involved in detecting stimuli with abrupt onsets (transient detectors) and stimuli that deviate from the immediate stimulus stream (mismatch negativity) overlap with activity that is more closely associated with the stimulus properties (Näätänen, 1990, 1992; Näätänen, Jacobsen, & Winkler, 2005; Näätänen & Picton, 1987). Keep in mind, however, that the attentional processes indexed by the CTN100-180 appear to be different from those involved in transient and mismatch detection.

The increase in the CTN100-180 component amplitude when the sural nerve-evoking stimulus is unattended is only seen when the subject's attention is directed to another stimulus modality and when the evoking stimulus is strong (Dowman, 2007a). No attention effects are seen when spatial attention alone is involved (Dowman, 2004b) or when the sural nerve-evoking stimulus is weak and nonthreatening (Dowman, 2007b). The increase in the FCN130-200 component when the sural nerve-evoking stimulus is unattended, on the other hand, is much less specific. It occurs with changes in stimulus modality (Dowman, 2001, 2007a, 2007b), spatial location (Dowman, 2004b), and with weak and strong stimulus levels (Dowman, 2001, 2004b, 2007a, 2007b). This nonspecific attention effect is consistent with Botvinick, Braver, Barch, Carter, and Cohen's (2001) hypothesis that the medial prefrontal cortex is involved in monitoring any situation that requires a change in attention, and signals the lateral prefrontal cortex that such a change should be executed. Although Botvinick et al. focused on changes in attentional control that result from response conflict, they suggest that the medial prefrontal cortex may also be involved in other situations that demand a change in attentional control (see Weissman, Gopolakrishnan, Hazlett, & Woldorff, 2005,for an example). Clearly, the presence of a threatening somatosensory stimulus outside the focus of attention would qualify as such a situation.

This hypothesis provides an explanation of our electrophysiological results. Namely, a threatening sural nerve-evoking stimulus presented outside the focus of attention is detected by the somatosensory association areas in the parietal operculum/insula (indexed by the CTN100-180), which is monitored by the medial prefrontal cortex (indexed by the FCN130-200). An obvious candidate component indexing the putative threat signal sent by the medial prefrontal cortex to the lateral prefrontal cortex requesting a change in attentional control is the P3a event-related potential. The P3a is thought to be an electrophysiological index of the involuntary orienting response (Friedman et al., 2001; Polich, 2003), where the P3a recorded from anterior scalp sites reflects prefrontal cortex activity redirecting attention toward the evoking stimulus (Friedman et al., 2001; Knight, 1996; Polich, 2003). The P3a is evident in our sural nerve somatosensory evoked potential at about 320–400 ms poststimulus (Dowman, 2004c), and its amplitude is larger in the unattend condition when the sural nerve-evoking stimulus is task relevant (Dowman, 2004b, 2007a). This putative threat detection process is associated with a reaction time benefit. That is, the increase in discrimination task reaction time during the unattend condition is longer for weak nonthreatening sural nerve electrical and visual stimuli (about 20%) than a strong, threatening sural nerve electrical stimulus (about 11%; Dowman, 2007a, 2007b).

In the present study we used an artificial neural network model to test the feasibility of our threat detection hypothesis. The model was based on the one developed by Botvinick et al. (2001) and Yeung, Botvinick, and Cohen (2004) to test the response conflict hypothesis. Their model has successfully simulated behavioral, hemodynamic, and electrophysiological responses related to response conflict in a number of different paradigms, including the Eriksen Flanker, Stroop, and Stem Completion tasks. More importantly, their model has explained some counterintuitive electrophysiological findings and led to novel predictions that were verified in subsequent experiments (Yeung et al., 2004). Hence this approach provides a rigorous, quantitative means of testing the feasibility of hypotheses about attentional control and can lead to novel, falsifiable predictions. Our objectives were to construct an artificial neural network model architecture that explains our behavioral and electrophysiological data on detecting and orienting attention toward threatening somatosensory stimuli and to determine whether the model could provide novel insights into the threat detection and orienting process.

Methods

The artificial neural network model was designed to simulate our endogenous cuing experiments (Dowman, 2007a, 2007b). In these experiments subjects were engaged in two tasks: a visual

discrimination task, where they made a response indicating whether a red or a yellow light-emitting diode was lit, and a somatosensory discrimination task, where they made a response indicating whether a high or low intensity sural nerve electrical stimulus was presented. A symbolic cue given at the beginning of each trial indicated whether the visual or the sural nerve electrical target stimulus was forthcoming. The target stimulus onset followed the cue offset by 1512 ms. In the validly cued condition, the cue correctly predicted the upcoming stimulus (e.g., the cue indicated that the sural nerve electrical stimulus was forthcoming and the sural nerve electrical stimulus was presented), and in the invalidly cued condition the cue incorrectly predicted the forthcoming stimulus (e.g., the cue indicated that the visual stimulus was forthcoming but the sural nerve electrical stimulus was presented). The subjects were instructed to direct their attention to the cued task, but to respond to the target stimulus regardless of whether it was validly cued or not.

We tested a number of different artificial neural network architectures. All were adapted from the basic architecture reported by Botvinick et al. (2001) and Yeung et al. (2004), where attention nodes bias activity in the sensory–response pathways mediating the tasks. The Botvinick et al. and Yeung et al. studies emphasized the role of response conflict in attentional control, where the medial prefrontal cortex monitors conflict between response nodes in the competing sensory–response pathways. Dowman, Glebus, and Shinners (2005) demonstrated that the scalp potentials time-locked to the sural nerve-evoking stimulus do not contain any components related to response conflict. Hence, the model architectures investigated here focused on threat detection, as suggested by the electrophysiological studies reviewed in the Introduction. The architecture that best accounted for our electrophysiological and behavioral data is shown in Figure 1. The connection strengths used in this model were either taken directly from or slightly modified from Yeung et al. (2004). Architectures that failed to account for our behavioral and/or electrophysiological data will be discussed below.

The architecture shown in Figure 1 contains separate sensory–response pathways for the visual and somatosensory tasks. The sensory nodes (Ss for somatosensory, Sv for visual) have feedforward excitatory connections with a middle layer (Ms for somatosensory and Mv for visual), which in turn has feedforward excitatory connections with the response layer (Rs for somatosensory, Rv for visual). The sensory nodes have bidirectional excitatory connections with sensory attention nodes (As for somatosensory and Av for visual), and the response nodes have bidirectional excitatory connections with response attention nodes (ARs for somatosensory, ARv for visual). The attention and response layers each have bidirectional inhibitory connections within their layers. The sensory attention node has a feedforward inhibitory connection with the threat detector node (Ths for somatosensory, Thv for visual). The threat detectors have feedforward excitatory connections with the medial prefrontal cortex node (mPs for somatosensory and mPv for visual), which in turn has a feedforward excitatory connection with the response attention node. In this model, the somatosensory threat detector and the orienting response it elicits (i.e., activation of the mPs and ARs nodes) are inhibited by the feedforward inhibitory connection between the sensory attention node and the threat detector when attention is focused on the somatosensory stimulus (validly cued condition).

The sensory and threat detector node activities were combined to simulate the CTN100-180 component generated in the

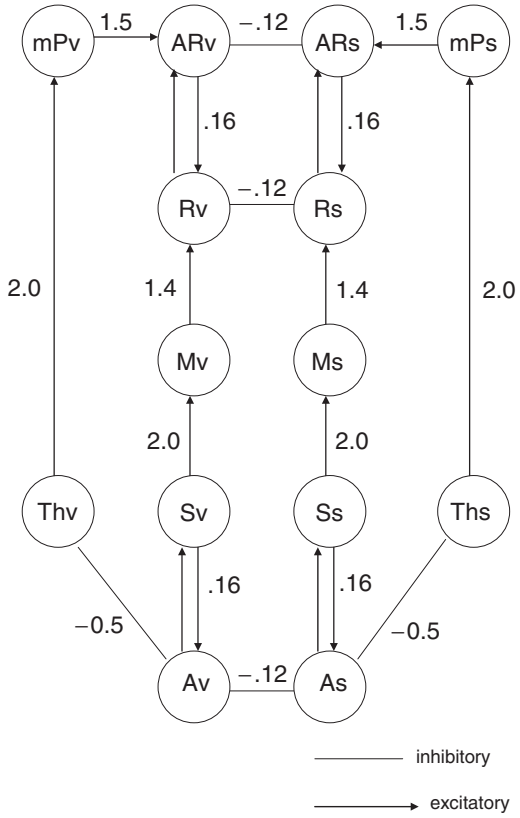

Figure 1. The artificial neural network model architecture. The numbers refer to the connection strengths between nodes. The inhibitory connections within the response and attention layers are bidirectional. Ss and Sv: sensory nodes for the somatosensory and visual pathways, respectively; Ms and Mv: middle layer for the somatosensory and visual pathways, respectively; Rs and Rv: response nodes for the somatosensory and visual pathways, respectively; As and Av: sensory attention nodes for the somatosensory and visual pathways, respectively; ARs and ARv: response attention nodes for the somatosensory and visual pathways, respectively; Ths and Thv: threat detector nodes for the somatosensory and visual pathways, respectively; mPs and mPv: medial prefrontal cortex nodes for the somatosensory and visual pathways, respectively.

somatosensory association areas of the parietal operculum and insula. The medial prefrontal cortex node simulated activity in the FCN130-200 medial prefrontal cortex generator that monitors threat detector activity in the somatosensory association areas and signals the lateral prefrontal cortex that a change in attention is required. The sensory and response attention nodes correspond to the lateral prefrontal cortex areas that provide attention-related excitatory and inhibitory biases to the sensory and motor areas (see Knight, Staines, Swick, & Chao, 1999; Miller & Cohen, 2001). These lateral prefrontal areas are presumed to contribute, at least in part, to the anterior component of the P3a event-related potential (Friedman et al., 2001). As will be explained below, the middle layer was merely added to delay the sensory-evoked activation of the response node so that it coincided with threat signals from the response attention node. As in the Botvinick et al. (2001) and Yeung et al. (2004) response conflict models, the response node reflects the combined activities of motor areas involved in response processes (e.g., basal ganglia, cerebellum, premotor cortex, primary motor cortex). These response processes do not appear to be reflected in any of the sural nerve somatosensory evoked potential components of interest

here (see Dowman et al., 2005). Rather, the response node activity is used to simulate behavioral reaction time, as was done in Botvinick et al. and Yeung et al.

The activation level of each node was computed over 55 cycles. The activation levels were computed by

$$\mathbf{A} = \frac{1}{1 + e^{(4-\mathbf{N})}}, \quad (1)$$

where **A** is the vector containing the activation levels for all nodes.

The vector **N** was given by

$$\mathbf{N} = \mathbf{N} + (\mathbf{W} * \mathbf{A}) - (\mathbf{N} * \delta), \quad (2)$$

where δ is a decay constant equal to 0.1 and **W** is the matrix containing the connection weights between nodes. $\mathbf{W} * \mathbf{A}$ computes the sum of the weighted inputs to each of the nodes. The electrophysiological and behavioral responses that this model is intended to simulate were averaged over 40–120 trials. Because averaging greatly reduces trial-to-trial variability due to noise, we did not include a noise term in Equation (2).

The model simulates the voluntary allocation of attention by adding external input to the appropriate sensory attention node (e.g., for the somatosensory discrimination task, Av in the invalidly cued condition, As in the validly cued condition). Presentation of the sural nerve electrical stimulus was simulated by adding external input to the somatosensory sensory node (Ss). The external input to the somatosensory sensory node was given on cycles 6–10 (stimulus cycles). The external input to the sensory attention node was given at the first cycle and continued until the end of the stimulus cycles. Equal levels of external input were added to both response attention nodes beginning at the first cycle and continuing until the behavioral response was made, as defined by the cycle where response node activity equaled 0.2. This is comparable to the strategy used by Yeung et al. (2004) in their Eriksen Flanker task simulation, where equal amounts of external input were provided to the response nodes in both sensory–response pathways. Simulations that applied greater external input to the response attention node of the cued task than the uncued task produced essentially the same results as those described below, with the exception of larger validity effects on the reaction time and somewhat smaller threat signal responses in the response attention node. The presence of a threatening somatosensory stimulus was simulated by adding external input to the somatosensory threat detector node (Ths) during the stimulus cycles (cycles 6–10). Because the visual and somatosensory halves of the model are symmetrical, the simulations described here only involved external sensory inputs to the somatosensory sensory and threat detector nodes.

Results

The effects of cue validity on the simulated electrophysiological responses evoked by threatening and nonthreatening somatosensory stimuli are shown in Figure 2. The external inputs to the somatosensory and visual sensory attention units were 1.0 and 0.0, respectively, in the validly cued condition, and 0.0 and 1.0, respectively, in the invalidly cued condition. In our electrophysiological studies we did not have a neutral condition, where a neutral cue is presented that does not provide any information about which stimulus is forthcoming. Nonetheless, we included a neutral condition in the simulations to see if it would result in any interesting predictions for future experiments. In the neutral condition we assumed that attention is allocated equally to both the somatosensory and visual sensory attention nodes. The behavioral reaction time for the neutral condition was manipulated by varying how much external input was supplied to the sensory attention nodes: Adding 0.0 to both resulted in reaction times that were equal to the invalidly cued condition values and adding 1.0 to both resulted in reaction times equal to the validly cued condition values. For the simulations described below external inputs of 0.6 were added to both sensory attention nodes to produce neutral condition reaction times that were about midway between the validly and invalidly cued conditions. (It is not necessary to allocate exactly one half of the attention that was allocated in the validly and invalidly cued conditions in the neutral condition, because it is very unlikely that the subjects are allocating all of their attentional resources in this task.) The external inputs to the somatosensory sensory and threat detector nodes were 1.0 and 1.0, respectively, in the threat condition, and 1.0 and 0.0, respectively, in the nonthreatening condition. Both the somatosensory and visual response attention nodes received external inputs of 0.675.

The activations of the sensory and threat detector nodes shown in Figure 2 are consistent with our experimental findings. Recall from the Introduction that we proposed that the negative potential generated by the somatosensory association areas in the parietal operculum and insula at 100–180 ms poststimulus (the CTN100-180) is a combination of sensory and threat detector activities. As in our experimental studies, the amplitude of these combined activities was larger in the invalidly cued condition than the validly cued condition. This was due to the increase in the threat detector node activation outweighing the decrease in the sensory node during the invalidly cued condition. As expected, the combined sensory and threat detector activations were smaller in the invalidly cued condition than the validly cued condition in the nonthreatening condition because of the absence of threat detector activity.

We investigated how the effects of validity on the simulated somatosensory association area activation changes with stimulus intensity (Figure 3). In this simulation the external input to the somatosensory sensory and threat detector nodes was varied between 0.9 and 1.3 (external input values of less than 0.9 did not produce a behavioral response, i.e., the somatosensory response node activation never reached the 0.2 criterion). Stimulus intensity had very little impact on the validity effect in the sensory node. However, the validity effect increased with increasing stimulus intensity in the threat detector node. This resulted in the combined activation showing an increase in the validity effect with increasing stimulus intensity. This is consistent with our experimental studies, where we reported a validity effect for the CTN100-180 at strong, threatening levels of stimulation (Dowman, 2007a) but not at weak nonthreatening levels (Dowman, 2007b). The lack of a significant Validity $\times$ Stimulus Intensity interaction obtained in an experiment that involved only strong threatening levels of stimulation (Dowman, 2007a) can be explained by the limited range of stimulus intensities used in the that study.

We also tested an architecture based on the conflict hypothesis proposed by Botvinick et al. (2001) and Yeung et al. (2004). In this architecture the feedforward inhibitory connection between the sensory attention unit (As) and the threat detector (Ths) shown in Figure 1 was replaced with bidirectional inhibitory connections between the sensory and threat detector nodes.

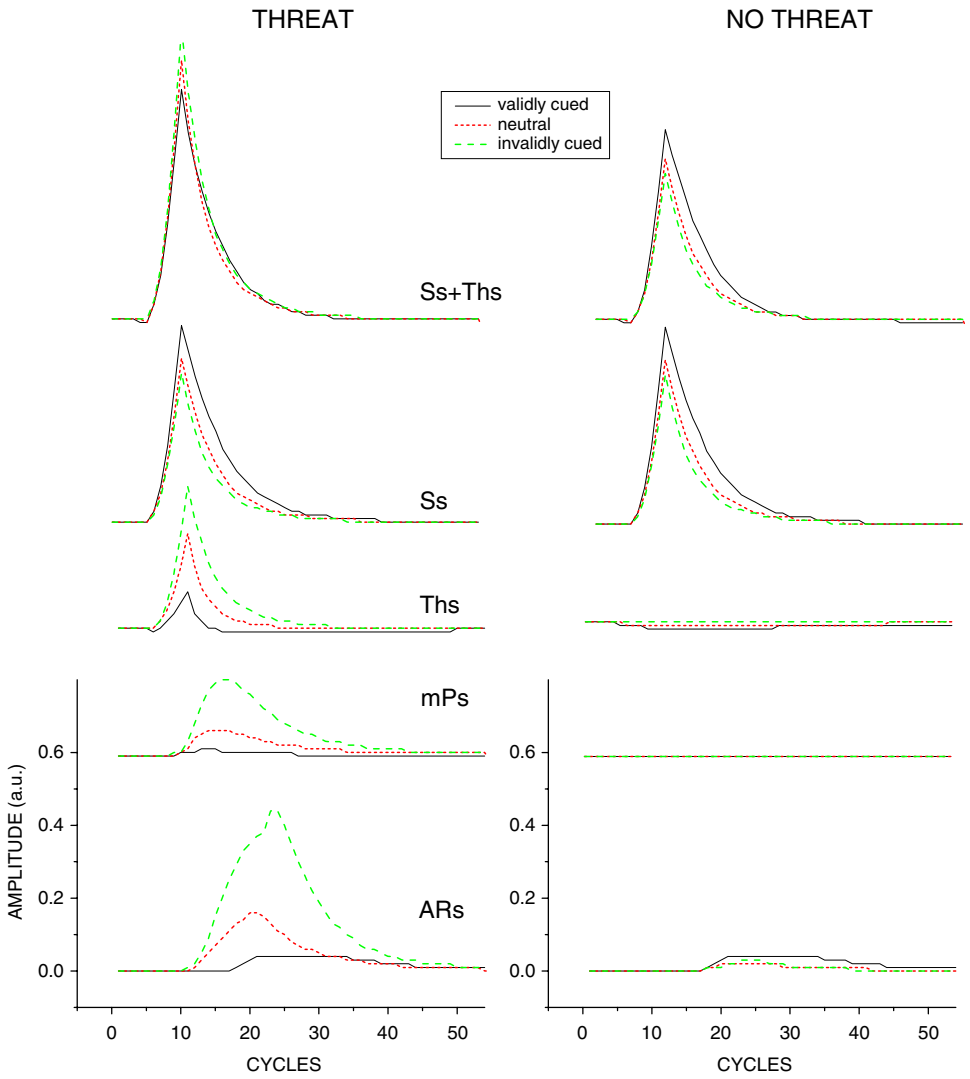

Figure 2. Activation levels of the sensory (Ss), threat detector (Ths), combined sensory and threat detector (Ss+Ths), medial prefrontal cortex (mPs), and the response attention (ARs) nodes during the invalidly cued, neutral, and validly cued simulations for the threatening (threat) and nonthreatening (no threat) somatosensory stimuli.

Conflict was computed as the Hopfield energy between the sensory and threat detector nodes (see Botvinick et al., 2001; Yeung et al., 2004) and was added to the medial prefrontal cortex (mPs) node. We tried a number of different inhibitory connection weights between the sensory and threat detector nodes, including asymmetrical weights where the inhibition from the sensory node to the threat detector node was up to 10 times greater than that between the threat detector node and the sensory node. This architecture failed to simulate the somatosensory association area response. That is, the combined sensory and threat detector node activation was always larger in the validly cued condition than the invalidly cued condition.

The validity effects on the medial prefrontal cortex (mPs) and the response attention (ARs) nodes are also shown in Figure 2. The response attention node activation resulted from a combination of the external input simulating sustained voluntary attention toward the task, the threat signal from the medial prefrontal cortex node, and input from the response node. In experimental data the voluntary sustained attention-related activation is not time-locked to the somatosensory-evoking stimulus and does not, therefore, appear in the averages. Hence, for the simulation results, we subtracted out the response attention node activation due to voluntary sustained attention.

Under the threatening somatosensory stimulus condition the medial prefrontal cortex and response attention node activations were both larger in the invalidly cued condition than the validly cued condition. These effects are very similar to our electrophysiological studies (Dowman, 2001, 2004b, 2007a, 2007b). The medial prefrontal cortex activity at 130–200 ms poststimulus (the FCN130-200) is larger in the invalidly cued condition than in the validly cued condition. As noted in the Methods, the response attention node may correspond, at least in part, to the lateral prefrontal cortex generators of the anterior component of the P3a event-related potential. Indeed, activation of the response attention node was larger in the invalidly cued condition, as was the case for the P3a recorded in our electrophysiological studies (Dowman, 2007a).

We ran additional simulations to see if the medial prefrontal cortex (mPs) and response attention (ARs) nodes show the same stimulus intensity and task relevance effects as the sural nerve

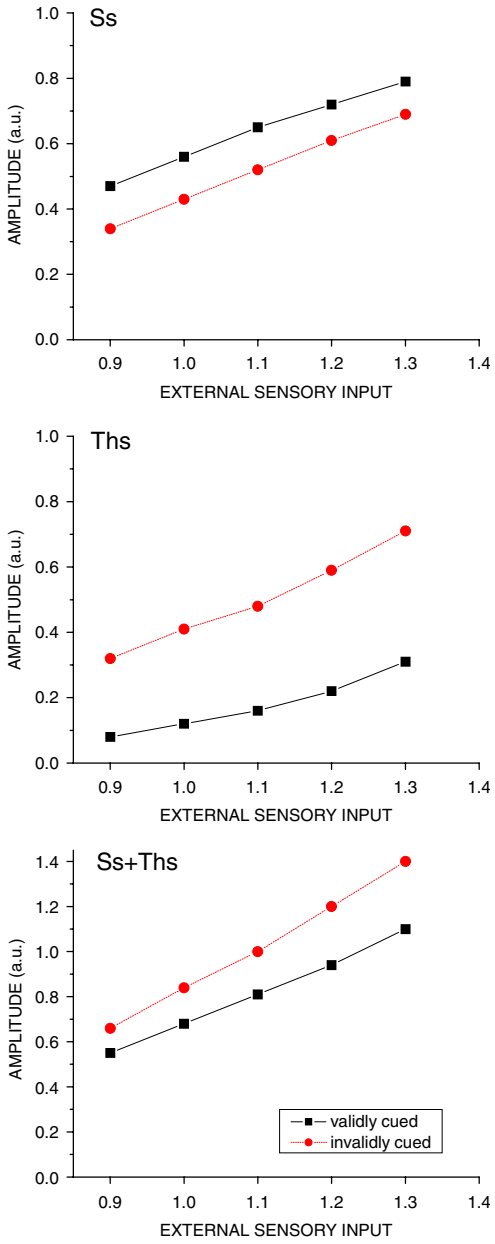

Figure 3. Effects of stimulus intensity on the somatosensory sensory (Ss), threat detector (Ths), and the combined sensory and threat detector (Ss+Ths) nodes for the validly and invalidly cued conditions during the threat condition. Increasing stimulus intensity was simulated by increasing the external input to the somatosensory sensory and threat detector nodes.

evoked FCN130-200 and P3a components (Figure 4). For stimulus intensity effects, the external inputs to the sensory and threat detector nodes were varied between 0.9 and 1.3. Task relevance was simulated by varying the external inputs to the response attention nodes between 0.0 and 0.675—the greater the external input the greater the task relevance. Nonthreatening stimuli did not activate the threat detector, the medial prefrontal cortex, or the response attention nodes (see Figure 2). Hence neither stimulus intensity nor task relevance effects were examined for the nonthreatening condition.

The medial prefrontal cortex (mPs) node exhibited the same stimulus intensity and task relevance effects as the FCN130-200 (see Dowman, 2007a): Its amplitude increased with increasing stimulus intensity and was unaffected by task relevance. The response attention node (ARs) activation elicited by the medial prefrontal cortex threat signal increased with increasing stimulus intensity and was larger when the stimulus was task relevant (Figure 4), as has been reported for the P3a event-related potential (see Dowman, 2007a; Dowman et al., 2007; Friedman et al., 2001; Polich, 2003). We tried a model architecture that replaced the feedforward excitatory connection between the medial prefrontal cortex and response attention nodes shown in Figure 1 with bidirectional excitatory connections. Although this architecture exhibited validity effects similar to those reported above, it resulted in task relevance and stimulus intensity effects that were not consistent with the electrophysiological data. Namely, the medial prefrontal cortex node showed an increase in activation with increasing task relevance and the response attention node activation did not increase with increasing stimulus intensity.

The effects of cue validity on the somatosensory response node activation and simulated reaction times are shown in Figure 5. Simulated reaction times were computed in the same manner as Yeung et al. (2004):

$$\text{Reaction time} = (c * 20) + 500, \tag{3}$$

where c is the cycle where the activity level in the response node equals 0.2, 20 is an estimate of the number of milliseconds per cycle, and 500 is a constant that accounts for response and decision processing time that is not included in the artificial neural network model. The 20 ms/cycle value was chosen to produce activations in the sensory, medial prefrontal cortex, and response attention nodes whose latencies approximated our electrophysiological data, and the 500 ms processing constant was chosen to produce validly cued reaction times that are comparable to those reported in our experimental studies.

The simulated reaction times were longer in the invalidly cued condition than the validly cued condition (Figure 5, lower panel), as was the case in our experimental studies (e.g., Dowman, 2001, 2004b, 2007a, 2007b). Likewise, the percent increase in the simulated reaction time during the invalidly cued condition (computed as ([invalidly cued − validly cued]/validly cued) * 100) was greater for nonthreatening stimuli than threatening stimuli. The percent increase in the simulated reaction times in the invalidly cued condition during the threatening (12%) and nonthreatening (20%) conditions are close to those reported in our experimental studies (11% and 20%, respectively; Dowman, 2007a, 2007b). Interestingly, the reaction time benefit afforded by threatening stimuli was seen during the invalidly cued but not during the validly cued condition. Furthermore, threat had very little effect on reaction time during the neutral condition. This contrasts with the robust effect of threat on the simulated electrophysiological data obtained during the neutral condition (Figure 2). The small reaction time threat effect seen during the neutral condition appears to be related to the magnitude of the response node activation, where the larger the activation, the smaller the threat and validity effects (Figure 5, upper and middle panels).

The activation level of the response node can be increased by increasing external sensory input (simulating an increase in stimulus intensity) or by increasing the connection strength between the middle and response layers (M-R) (simulating an increase in how well learned the task is). As shown in Figure 6, both the validity (computed as the percent increase in the invalidly cued condition; see above) and threat effects decrease as the

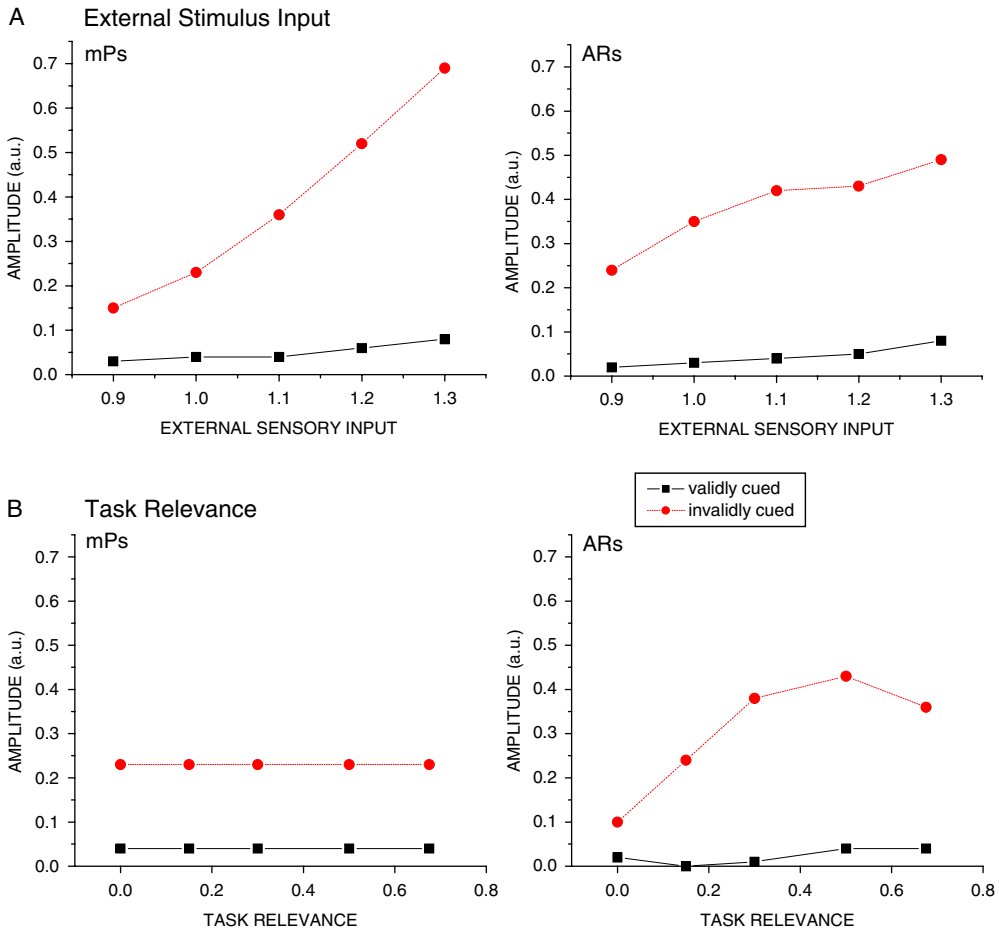

Figure 4. Effects of stimulus intensity (a) and task relevance (b) on the somatosensory medial prefrontal cortex (mPs) and response attention (ARs) nodes during the threat condition. Increasing stimulus intensity was simulated by increasing the external input to the somatosensory sensory and threat detector nodes, and increasing task relevance was simulated by increasing the external input to the response attention nodes.

strength of the M-R connection increased from 1.4 to 2.0 and as the external input to the somatosensory sensory node increased from 1.0 to 1.3.

We tested two alternative architectures that varied when and where the threat signal from the medial prefrontal cortex node was led to the attention nodes. In the one architecture the middle layer shown in Figure 1 was eliminated, and instead the sensory nodes were directly connected to the response node. In this case, the threat signal from the medial prefrontal cortex node to the response attention node occurred too late to affect the simulated behavioral reaction time. In the other architecture we eliminated the response attention node and instead led the medial prefrontal cortex threat signal directly to the sensory attention node. In this architecture the threat signal did not affect the simulated reaction time. This was the case regardless of whether the middle layer was present or if the sensory node was directly connected to the response node.

Discussion

The response properties of the combined sensory and threat detector nodes in the artificial neural network model were very similar to the CTN100-180 component it was intended to simulate (Dowman, 2007a, 2007b): Both were larger in the invalidly cued condition, and this validity effect decreased as the threat value of the evoking stimulus decreased. In the nonthreatening condition the combined sensory and threat detector activity was smaller in the invalidly cued condition than the validly cued condition owing to the absence of threat detector activation. The effect of simulated sustained attention on the sensory node activity is consistent with that reported for the N1 peak of the visual evoked potential elicited by nonthreatening visual stimuli (Dowman, 2007a, 2007b; Mangun, 1995) and with the early and mid-latency peaks of the somatosensory evoked potential elicited by weak electrical stimulation of the fingers (Desmedt & Tomberg, 1989; Garcia-Larrea, Bastuji, & Mauguiere, 1991; Garcia-Larrea, Lukaszewicz, & Mauguiere, 1995), all of which were smaller in the unattend condition. The amplitude of the CTN100-180 evoked by weak, nonthreatening sural nerve electrical stimulation, on the other hand, was the same across the validly and invalidly cued conditions (Dowman, 2007b). Sustained voluntary attention effects on sensory-related responses are more prominent at short interstimulus intervals than for long ones (see Näätänen, 1982, 1990; Näätänen & Picton, 1987). Indeed, studies showing a decrease in the amplitude of the early and mid-latency peaks of the somatosensory evoked potential had shorter interstimulus intervals (< 1.5 s; Desmedt & Tomberg, 1989; Gar-

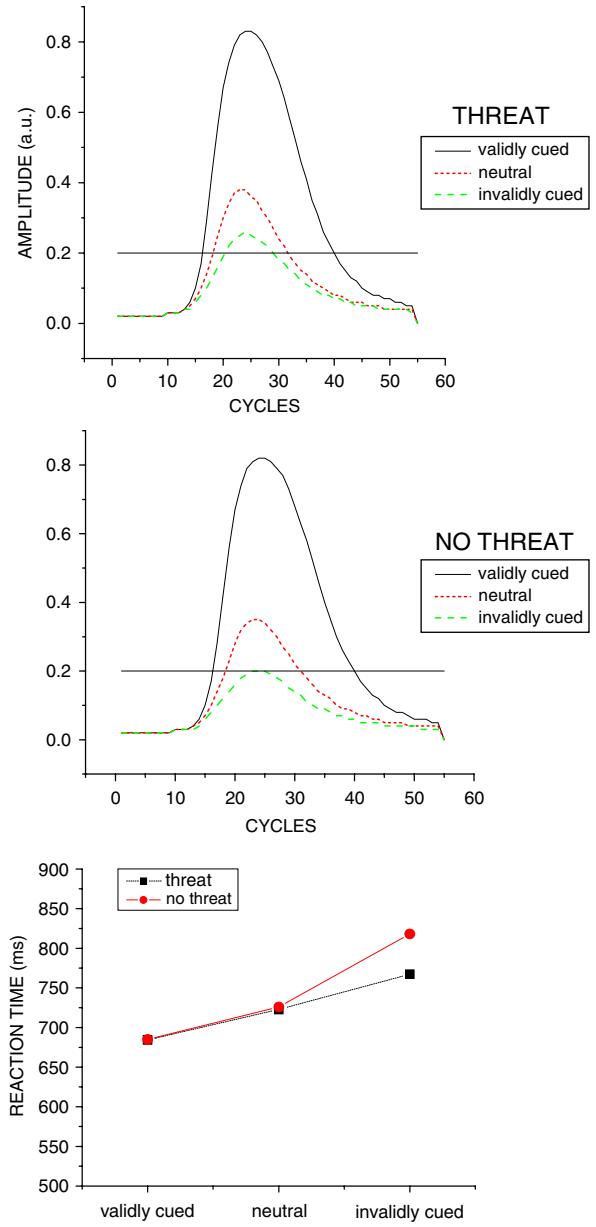

Figure 5. Top and middle panels: response node activation levels during the validly, neutral, and invalidly cued conditions for the threatening (threat) and nonthreatening (no threat) somatosensory stimuli. The horizontal line identifies the response criterion (0.2). Bottom panel: simulated reaction time during the validly cued, neutral, and invalidly cued conditions for the threatening (threat) and nonthreatening (no threat) somatosensory stimuli.

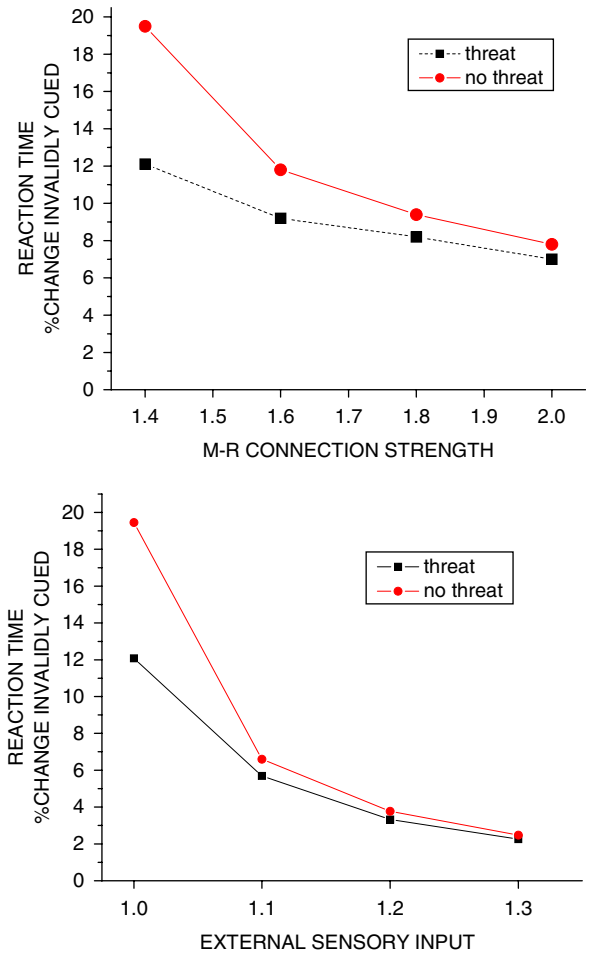

Figure 6. Effects of how well learned the task is and stimulus intensity on the reaction time validity effects for threatening (threat) and nonthreatening (no threat) somatosensory stimuli. How well learned the task is was simulated by the strength of the connection between the middle (Ms) and response (Rs) nodes (M-R) (upper panel), and increasing stimulus intensity was simulated by increasing the external input to the somatosensory sensory node (lower panel). The percent change in reaction time during the invalidly cued condition was calculated by ([invaldly cued − validly cued]/validly cued) ∗ 100.

cia-Larrea et al., 1991, 1995) than was used in our studies (4.3 s; Dowman, 2007a, 2007b). Hence the absence of a validity effect on the CTN100-180 evoked by weak nonthreatening stimuli may be due to the decay of the sustained attention effects on the sensory activity during the 4.3-s interstimulus interval. The decrease in the amplitude of the visual evoked potential N1 peak elicited by the 4.3-s interstimulus interval reported by Dowman (2007a, 2007b) implies that its sustained attention effect has a longer time course than that for the sural nerve-evoked CTN100-180. Furthermore, the fact that attention effects were seen on the threat detector but not the sensory activities at the 4.3-s interstimulus interval suggests that these also have different time courses. The time courses of these effects need to be examined in future experimental studies and, if verified, implemented in the artificial neural network model.

The medial prefrontal cortex and response attention nodes likewise exhibited properties comparable to the FCN130-200 and anterior P3a generators they were intended to simulate. Both the medial prefrontal cortex node and the FCN130-200 are positively related to stimulus intensity and are largest when the evoking stimulus was unattended, and this attention effect is seen regardless of whether the sural nerve-evoking stimulus is task relevant or not. These properties are consistent with those reported for the medial prefrontal cortex in our electrophysiological studies (Dowman, 2004a, 2004b, 2007a, 2007b; Dowman et al., 2007), and provide further support for the hypothesis that one of the roles of the medial prefrontal cortex is monitoring situations that require a change in attentional control (Botvinick et al., 2001).

Both the response attention node and the P3a are positively related to stimulus intensity and are largest when the threatening evoking stimulus initiates a change in attention, and the amplitudes of both depend on the task relevance of the evoking stimulus. As we reported in our experimental studies (Dowman, 2007a, 2007b), the threat-related activity in the response attention node was associated with a reaction time benefit, where the increase in reaction time during the invalidly cued condition was smaller for threatening somatosensory stimuli than for nonthreatening stimuli. Simulations where the threat signal from the medial prefrontal cortex node was led to the sensory attention node had no effect on reaction time. This suggests that the P3a generators are involved, at least in part, in redirecting attention toward response processing. This is consistent with a number of studies showing a close relationship between P3a amplitude and peripheral measures of the orienting response, such as skin conductance and heart rate (for review, see Friedman et al., 2001). Our results suggest that the P3a may also index changes in attention that facilitate voluntary response processes executed on that trial.

These findings do not exclude the idea that the P3a generators are involved in reorienting attention toward sensory processing. Rather, our work suggests that the reorienting of attention indexed by the P3a occurs too late to affect sensory processing for that trial. The artificial neural network model architecture shown in Figure 1 does not feed the threat signal from the medial prefrontal cortex node back to the sensory attention node, and hence the threat signal could not affect the sensory node activity level. Architectures that fed the threat signal directly to the sensory attention node did not affect the peak activation of the sensory node. That is, the sensory node peak activation was smaller in the invalidly cued than in the validly cued condition, and this effect was the same for threatening and nonthreatening stimuli (data not shown). These simulation results are consistent with the finding that the perceived magnitude of a painful stimulus is smaller during the invalidly cued than during the validly cued condition (Dowman, 2001, 2004b, 2007a; Miron, Duncan, & Bushnell, 1989). Thus, there does not appear to be any attentional facilitation of sensory processing on that trial. It is possible, however, that the reorientation of attention toward sensory processing indexed by the P3a is implemented on the next trial. Indeed, medial prefrontal cortex activity associated with response conflict has been shown to affect attentional control on subsequent trials in experimental studies and has been successfully simulated in an artificial neural network model of response conflict (Botvinick et al., 2001). Future electrophysiological and simulation studies should be aimed at determining if this is also the case for the orienting response elicited by threatening somatosensory stimuli.

In addition to successfully simulating the validity and threat effects on reaction time noted above, the artificial neural network model exhibited the decrease in reaction time with increasing stimulus intensity that has been reported in experimental studies (Bushnell, Duncan, Dubner, Jones, & Maixner, 1985; Dowman, 2001, 2004b). The model also suggests that the validity and threat effects on reaction time depend on how well learned the task is and on stimulus intensity. The threatening sural nerve electrical stimuli used in our experimental studies had a stronger intensity than the nonthreatening stimuli (Dowman, 2007a, 2007b). Hence, the smaller invalidly–validly cued reaction time difference with the threatening sural nerve electrical stimuli reported by Dowman (2007a, 2007b) could be explained by the stronger stimulus intensity and not necessarily by the stimulus' threat value. It will be necessary, therefore, to reexamine the threat effects on reaction time with stimulus intensity held constant. Likewise, future studies should examine the effect of how well learned the task is on the validity and threat effects to further validate the model.

The artificial neural network model predicts that the reaction time benefit afforded by a threatening somatosensory stimulus (i.e., reaction times faster for threatening than nonthreatening stimuli) will only occur when it is presented outside the focus of attention (invalidly cued) and not when it is attended (validly cued). To our knowledge there are no studies directly addressing this prediction using the endogenous cuing paradigm. There is, however, some support for it in visual search studies. Ohman, Flykt, and Esteves (2001, experiment 1) found that visual search times were slower for nonthreatening visual target stimuli (flowers, mushrooms) than biologically threatening targets (spiders and snakes) when they were presented outside the focus of attention, whereas reaction times for the threatening and nonthreatening stimuli were the same when they were presented within the focus of attention. Clearly, it will be important to demonstrate this effect using threatening and nonthreatening somatosensory stimuli in the endogenous cuing paradigm to provide further support for the artificial neural network model.

Interestingly, the effect of voluntary attention on reaction time benefits associated with threatening stimuli suggested by our artificial neural network model and by Ohman et al.'s (2001) visual search data is also seen with nonthreatening visual target stimuli that have abrupt onsets. Using an endogenous cuing paradigm similar to that used here, Yantis and Jonides (1990) demonstrated that the reaction time benefit afforded by visual target stimuli with abrupt onsets occurred when the target was presented outside the focus of spatial attention (invalidly cued) but not when it was presented within the focus of spatial attention (validly cued). This abrupt onset effect may be related to the transient detector described by Näätänen and his colleagues (Näätänen, 1990, 1992; Näätänen & Picton, 1987). The transient detector component of the auditory evoked potential N1 peak increases with the abruptness of the evoking stimulus onset and when the evoking stimulus is unexpected (e.g., invalidly cued; Näätänen, 1992; Näätänen & Picton, 1987). The artificial neural network model described here could account for the transient detector/abrupt onset stimulus effects by replacing the threat detector node with a transient detector node. This model would account for the reaction time benefits being evident in the invalidly cued but not the validly cued condition reported by Yantis and Jonides (1990) as well as the electrophysiological indices of transient detectors in the auditory association areas reported by Näätänen and colleagues (Näätänen, 1992; Näätänen & Picton, 1987).

A number of authors have posited that the reaction time differences between the validly cued and neutral conditions and between the invalidly cued and neutral conditions index processes involved in engaging and disengaging attention, respectively (for examples, see Fox et al., 2001; Van Damme, Crombez, Eccleston, & Goubert, 2004). Our modeling studies present an alternative explanation. In our model the neutral condition was simulated by adding equal amounts of external inputs to the visual and somatosensory sensory attention nodes. Where the neutral condition reaction times fell relative to those of the validly and invalidly cued conditions depended on the magnitude of the external inputs: Decreasing the external inputs drove the

neutral condition reaction times toward the invalidly cued times and increasing the external inputs drove them toward the validly cued times. We arbitrarily chose external inputs that put the neutral condition reaction times about midway between the validly and invalidly cued condition values. In theory, the actual values will depend on a number of factors, including the salience of the target stimuli and the task demands.[1] In our model the difference between the neutral and invalidly cued reaction times is due solely to how well the evoking stimulus engages attention (i.e., the magnitude of the threat detector and orienting responses it elicits) and does not require a disengagement process. Cohen and Huston (1994) made a similar observation with their artificial neural network model of spatial attention.

Our experimental data likewise suggest that the effects of threatening somatosensory stimulation on the reaction time validity effect reflect how well the evoking stimulus engages attention and does not require an attention disengage process. As reported by Dowman (2007a), the reaction time validity effect was longer for the nonthreatening visual target stimuli than for the strong, threatening sural nerve target stimuli. There are at least two possible interpretations of these data: (1) Reaction time to the invalidly cued visual stimulus is longer than that for the sural nerve stimulus because of the extra time required to disengage attention from the expected threatening sural nerve electrical stimulus. (2) Reaction time to the invalidly cued threatening sural nerve stimulus is shorter than that for the nonthreatening visual stimulus because the former is better at engaging attention (i.e., it elicits stronger threat detector and orienting responses). We tested these two hypotheses (Dowman, 2007b) using weak nonthreatening sural nerve electrical target stimuli and the same visual target stimuli used by Dowman (2007a). If the attention disengage hypothesis is correct, then the weak nonthreatening sural nerve and visual target stimuli should show the same reaction time validity effect as was seen for the threatening sural nerve targets by Dowman (2007a), because in all cases attention was being disengaged from a nonthreatening visual stimulus. If the attention engage hypothesis is correct, then the reaction time validity effects for the weak nonthreatening sural nerve target stimulus should be the same as that for the visual targets used by Dowman (2007a), because in all cases the targets will be eliciting a weak orienting response. The reaction time data were consistent with the attention engage hypothesis. That is, the reaction time validity effects for the visual and the weak nonthreatening sural nerve targets were the same (20% increase in the invalidly cued condition), and all were longer than that for the strong threatening sural nerve targets (11% increase in the invalidly cued condition). Our modeling and experimental data do not, of course, prove that an attention disengage process does not exist. Rather, these data suggest that the disengage process is not important in this situation. Examples of situations where the disengage process may be operating can be found in reports by Fox et al. (2001) and Van Damme et al. (2004).

[1]This implies that asymmetries in the amount of attention allocated to the threatening and nonthreatening stimuli in the invalidly cued condition could account for the shorter invalidly cued validly cued reaction time differences for threatening stimuli observed in our experimental studies. It does not, however, account for the threat effects observed in the electrophysiological responses, namely, the increase in the CTN100-180, FCN130-200, and P3a evoked by the strong threatening sural nerve stimulus during the invalidly cued condition.

Our experimental and modeling results provide support for Ohman's threat feature detectors. Ohman (2000) proposed that the ability of biologically relevant threats to preferentially capture attention is mediated in part by threat feature detectors that are activated at an early preattentive stage of perceptual processing. This hypothesis is based in part on the finding that the attentional bias toward biological threats observed in individuals suffering from anxiety disorders occurs even when the threat is perceptually masked and does not enter consciousness (Ohman, 2000). It is thought that these threat feature detectors arose from evolutionary pressure to develop a rapid detection of and response to stimuli that are detrimental to our survival and well-being (Ohman, 2000). Clearly, body sensations that have a sudden onset, are intense, and have an unusual quality, such as those elicited by the sural nerve electrical stimulus, would be expected to activate threat feature detectors. Our experimental data suggest that body threat feature detectors are located in the somatosensory association areas in the parietal operculum and/or insula, and that activity in these body threat detectors is monitored by the medial prefrontal cortex, which in turn signals the lateral prefrontal cortex to redirect attention to facilitate behavioral responses to the threat.

In sum, the artificial neural network model presented here demonstrates the feasibility of the threat detection hypothesis derived from our experimental studies. Perhaps more importantly, the model generated a number of novel, falsifiable hypotheses: (1) The effects of threat and validity on the behavioral reaction times depend on stimulus intensity and how well learned the task is. (2) The anterior P3a component generators redirect attention toward sensory and response processes related to the task, with the latter being effective on that trial and the former on the subsequent trial. (3) The reaction time benefits afforded by threatening somatosensory stimuli will be evident when the target stimulus is presented outside the focus of attention but not when it is presented within the focus of attention.

The artificial neural network model described here does, of course, have limitations. It does not, for example, model all of the perceptual, motor, and decision-making processes associated with the task. Rather, it takes a large-scale approach that simulates when these processes are engaged, their magnitudes, and how they interact. This scale appears to be compatible with that indexed by scalp event-related potentials and brain hemodynamic responses. Consequently, these electrophysiological and hemodynamic responses can be used to constrain the artificial neural network model architecture, as was done here and by Botvinick et al. (2001) and Yeung et al. (2004). Work on artificial neural network model architectures on smaller scales, that is, models of the processes, should eventually be integrated with the large-scale models to yield more comprehensive models of the brain. For example, the suppression of threat detector activity during the validly cued condition was implemented in our model by a feedforward inhibitory connection between the sensory attention node and the threat detector node. It may be case that the suppression of the threat detector activity actually involves a template-matching process, as has been suggested for the mismatch negativity (Näätänen, 1990, 1992; Näätänen et al., 2005) and simulated by Friston (2005) using empirical Bayes and generative models. Note that in the model proposed by Friston (2005), a mismatch between the template and the stimulus results in increased activation of the neural elements. Our model captures the essence of this template-matching model without specifying its details.

REFERENCES

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and non-anxious individuals: A meta-analytic study. *Psychological Bulletin*, *133*, 1–24.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624–652.

Bushnell, M. C., Duncan, G. H., Dubner, R., Jones, R. L., & Maixner, W. (1985). Attentional influences on noxious and innocuous cutaneous heat detection in humans and monkeys. *Journal of Neuroscience*, *5*, 1103–1110.

Cohen, J. D., & Huston, T. A. (1994). Progress in the use of interactive models for understanding attention and performance. In C. Umilta & M. Moscovitch (Eds.), *Attention and performance XV. Conscious and nonconscious information processing* (pp. 454–476). Cambridge, MA: MIT Press.

Crombez, G., Eccleston, C., Van den Broeck, A., Goubert, L., & Van Houdenhove, B. (2004). Hypervigilance to pain in fibromyalgia. *Clinical Journal of Pain*, *20*, 98–102.

Desmedt, J. E., & Tomberg, C. (1989). Mapping early somatosensory evoked potentials in selective attention: Critical evaluation of control conditions used for titrating by difference the cognitive P30, P40, P100 and N140. *Electroencephalography and Clinical Neurophysiology*, *74*, 321–346.

Dowman, R. (2001). Attentional set effects on human innocuous somatosensory and pain pathways. *Psychophysiology*, *38*, 451–464.

Dowman, R. (2004a). Distraction produces an increase in pain-evoked anterior cingulate activity. *Psychophysiology*, *41*, 613–624.

Dowman, R. (2004b). Electrophysiological indices of orienting attention towards pain. *Psychophysiology*, *41*, 749–761.

Dowman, R. (2004c). The pain-evoked P2 peak is not a P3a event-related potential. *Brain Topography*, *17*, 3–12.

Dowman, R. (2004d). Topographic analysis of painful laser and sural nerve electrical evoked potentials. *Brain Topography*, *16*, 169–179.

Dowman, R. (2007a). Neural mechanisms of detecting and orienting attention toward unattended threatening somatosensory targets. I. Modality effects. *Psychophysiology*, *44*, 407–419.

Dowman, R. (2007b). Neural mechanisms of detecting and orienting attention toward unattended threatening somatosensory targets. II. Intensity effects. *Psychophysiology*, *44*, 420–430.

Dowman, R., & Darcey, T. M. (1994). SEP topographies elicited by innocuous and noxious sural nerve stimulation. III. Dipole source analysis. *Electroencephalography and Clinical Neurophysiology*, *92*, 373–391.

Dowman, R., Darcey, T. M., Barkan, H., Thadani, V., & Roberts, D. (2007). Human intracranially-recorded cortical responses evoked by painful electrical stimulation of the sural nerve. *NeuroImage*, *34*, 743–763. Available at: http://dx.doi.org/10.1016/j.neuroimage.2006.09.021.

Dowman, R., Glebus, G., & Shinners, L. (2005). Effects of response conflict on pain-evoked medial prefrontal cortex activity. *Psychophysiology*, *42*, 555–558.

Dowman, R., & Schell, S. (1999). Evidence that the anterior cingulate and supplementary somatosensory cortices generate the pain-related negative difference potential. *Clinical Neurophysiology*, *110*, 2117–2126.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: A cognitive-affective model of the interruptive function of pain. *Psychological Bulletin*, *125*, 356–366.

Eimer, M., & Driver, J. (2001). Crossmodal links in endogenous and exogenous spatial attention: Evidence from event-related brain potentials. *Neuroscience and Biobehavioral Reviews*, *25*, 497–511.

Fox, E., Russo, R., Bowles, R., & Dutton, K. (2001). Do threatening stimuli draw or hold visual attention in subclinical anxiety? *Journal of Experimental Psychology: General*, *130*, 681–700.

Friedman, D., Cycowicz, Y. M., & Gaeta, H. (2001). The novelty P3: An event-related brain potential (ERP) sign of the brain's evaluation of novelty. *Neuroscience and Biobehavioral Reviews*, *25*, 355–373.

Friston, K. (2005). A theory of cortical responses. *Philosophical Transactions of the Royal Society B*, *360*, 815–836.

Garcia-Larrea, L., Bastuji, H., & Mauguiere, F. (1991). Mapping study of somatosensory evoked potentials during selective spatial attention. *Electroencephalography and Clinical Neurophysiology*, *80*, 201–214.

Garcia-Larrea, L., Lukaszewicz, A.-C., & Mauguiere, F. (1995). Somatosensory responses during selective spatial attention: The N120-to-N140 transition. *Psychophysiology*, *32*, 526–537.

Gatchel, R. J. (2004). Comorbidity of chronic pain and mental health disorders: The biopsychosocial perspective. *American Psychologist*, *59*, 795–805.

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. *Proceedings of the National Academy of Sciences, USA*, *95*, 781–787.

Knight, R. T. (1996). Contribution of human hippocampal region to novelty detection. *Nature*, *383*, 256–259.

Knight, R. T., Staines, W. S., Swick, D., & Chao, L. L. (1999). Prefrontal cortex regulates inhibition and excitation in distributed neural networks. *Acta Psychologica*, *101*, 159–178.

Lorenz, J., & Garcia-Larrea, L. (2003). Contribution of attentional and cognitive factors to laser evoked brain potentials. *Neurophysiogie Clinique*, *33*, 293–301.

Mangun, G. R. (1995). Neural mechanisms of visual selective attention. *Psychophysiology*, *32*, 4–18.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Reviews of Neuroscience*, *24*, 167–202.

Miron, D., Duncan, G. H., & Bushnell, M. C. (1989). Effects of attention on the intensity and unpleasantness of thermal pain. *Pain*, *39*, 345–352.

Näätänen, R. (1982). Processing negativity: An evoked-potential reflection of selective attention. *Psychological Bulletin*, *92*, 605–640.

Näätänen, R. (1990). The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behavioral and Brain Sciences*, *13*, 201–288.

Näätänen, R. (1992). *Attention and brain function*. Hillsdale, NJ: Erlbaum.

Näätänen, R., Jacobsen, T., & Winkler, I. (2005). Memory-based or afferent processes in mismatch negativity (MMN): A review of the evidence. *Psychophysiology*, *42*, 25–32.

Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, *24*, 375–425.

Ohman, A. (2000). Fear and anxiety: Evolutionary, cognitive, and clinical perspectives. In M. Lewis & J. M. Haviland-Jones (Eds.), (pp. 573–593) *Handbook of emotions*. New York: Guilford Press.

Ohman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology: General*, *130*, 466–478.

Polich, J. (2003). Theoretical overview of P3a and P3b. In J. Polich (Ed.), *Detection of change: Event-related potential and fMRI findings* (pp. 83–98). Boston: Kluwer Academic Press.

Turk, D. C., & Okifuji, A. (2002). Psychological factors in chronic pain: Evolution and revolution. *Journal of Consulting and Clinical Psychology*, *70*, 678–690.

Van Damme, S., Crombez, G., Eccleston, C., & Goubert, L. (2004). Impaired disengagement from threatening cues of impending pain in a cross modal cueing paradigm. *European Journal of Pain*, *8*, 227–236.

van Velzen, J., & Eimer, M. (2003). Early posterior ERP components do not reflect the control of attentional shifts toward expected peripheral events. *Psychophysiology*, *40*, 827–831.

Wall, P. D. (1994). Introduction. In P. D. Wall & R. Melzack (Eds.), *Textbook of pain* (pp. 1–7). New York: Churchill Livingstone.

Weissman, D. H., Gopolakrishnan, A., Hazlett, C. J., & Woldorff, M. G. (2005). Dorsal anterior cingulate cortex resolves conflict from distracting stimuli by boosting attention toward relevant events. *Cerebral Cortex*, *15*, 229–237.

Yantis, S., & Jonides, J. (1990). Abrupt visual onsets and selective attention: Voluntary versus automatic allocation. *Journal of Experimental Psychology: Human Perception and Performance*, *16*, 121–134.

Yeung, N., Botvinick, M. W., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*, 931–959.

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