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Journal
Proceedings of the Annual Meeting of the Cognitive Science Society, 31(31)

ISSN
1069-7977

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Publication Date
2009

Peer reviewed

A Biologically-Plausible Cognitive Model (BPCM) of Positive and Negative Congruency Effects in Masked Priming

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Abstract

Studies have shown a positive priming effect with a short time between the prime and the target. The prime increases the performance on the target if they are congruent and decreases the performance when they are incongruent. Paradoxically, a negative priming effect has been found with a long time between the prime and the target. A major hypothesis argues that the prime initiates a motor self-inhibitory process that causes these effects. This hypothesis has been criticized and the model based on this hypothesis does not fit human data. A model was developed that fits the human data. It depends on attentional neuro-modulation not motor self-inhibition.

Keywords: Masked priming; Negative congruency effect; modelling; attention; conflict.

Introduction

In masked priming tasks, a brief masked stimulus (the prime) can affect the processing of the stimulus that follows (the target). A prime, a mask, and a target are presented sequentially and the task is to make a decision on the target. The result is usually a Positive Congruency Effect (PCE), also known as the positive compatibility effect. In PCE, the prime increases the performance on the target if they are congruent and decreases the performance if they are incongruent (Dehaene et al., 1998; Schlaghecken & Eimer, 2000). Conversely, a negative priming effect has been found, called the Negative Congruency Effect (NCE). This effect is also known as the negative compatibility effect, where paradoxically the prime increases the performance on the target if they are incongruent and decreases the performance if they are congruent (e.g., Schlaghecken & Eimer, 2000, 2002; Eimer & Schlaghecken, 1998; Jaśkowski & Ślósarek, 2006). The PCE has been shown with a short mask-target Stimulus Onset Asynchrony (SOA), while the NCE has been shown mainly with a longer mask-target SOA (e.g., 100 ms). To explain these results, some researchers (Schlaghecken & Eimer, 2000; Bowman et al., 2006), based on Event Related Potential (ERP) measurements and computational modelling, argue that when SOA is short, response selection can already take place during the initial response activation phase; this is reflected as an early increase in ERP for the congruent compared to incongruent trials, and this should result in the congruency effects in the form of a PCE. When SOA is longer, responses have to be selected during the subsequent inhibitory phase. This is reflected as a late decrease in ERP for congruent compared to incongruent trials, and this should be demonstrated as a negative effect (i.e., NCE). In these studies, the reduction in ERP activity has been attributed to a motor self-inhibition, causing the NCE effect. The mask causes this inhibition to be reversed, by removing the sensory evidence for the corresponding response and initiating its suppression.

The previous model of PCE and NCE (Bowman et al., 2006) depends on motor self-inhibition and does not show a decline in NCE and overall Reaction Times (RTs) through time and shows a huge PCE eventually at very long SOAs. However, human data shows that NCE decreases and disappears or turns into a very small PCE at very long SOAs. The current model does show these effects and does not depend on motor self-inhibition, but it works through attentional modulation driven by conflict. The same model that shows the PCE with strong prime and target and NCE with weak prime and target, without any changes in the parameters, shows a PCE at short mask-target SOA and an NCE at long mask-target SOA. It also shows the effect of other factors such as degradation (Schlaghecken & Eimer, 2002), mask density (Eimer & Schlaghecken, 2002), and prime duration (Eimer & Schlaghecken, 2002). The model is based on previous models that have been used to simulate different tasks such as target detection and simple decisions in monkeys and humans (Gilzenrat et al., 2002; Usher & Davelaar, 2002; Nieuwenhuis, et al., 2005).

The Current Model

Modeling methods. The processing elements in the model are a few neurons with self-excitation, lateral inhibition, and accumulative activation that have a strong computational power in simulating basic neural and cognitive processes (e.g., Usher & Davelaar, 2002; Gilzenrat et al., 2002). It has been demonstrated that these types of reduced models can resemble the neural computation of a large group of neurons (e.g., Wong & Wang, 2006).

The model (Figure 1) is a multi-layer dynamic neural model that consists of a feed-forward component for perceptuo-motor processing from the Input Layer (IL) to an intermediate layer, called Representation Layer (RL), and from there to the Cognitive Layer (CL) and Motor Layer

(ML, not shown in Figure 1). An assumption is that the cognitive processing, including the response, is modulated by attention. The Alert Attention layer (AA) simulates attentional modulation, that is supposed to be a model of Locus Coeruleus (LC) that potentiates cortical areas through norepinephrine (Aston-Jones & Cohen, 2005). The executive attention is only modelled through its effects on AA, using a Cognitive Layer (henceforth, CL) for conflict monitoring. The CL effect on AA simulates direct cortical projections to LC (Aston-Jones & Cohen, 2005). The CL and ML are affected by both prime and target. The ML is not shown in Figure 1 for the sake of simplicity, but its architecture is identical to CL, with the exception that it sends no outputs to AA, is slower, and noisier (see Table 1). Each condition in a simulation consists of 20,000 trials (200 independent blocks of 100 trials each, with congruent and incongruent trials counterbalanced randomly within each block). A single trial takes 1100 cycles. Each block starts with 500 cycles without changes in IL to let the units in other layers reach a steady state of activation. Similarly the Inter-Trial Interval (ITI) for each trial is 500 cycles, which allows the activation of units to return to baseline following the responses. The prime is presented by clamping one of the two units in the IL to 1, intended to be smaller or larger than five, or left or right in the case of arrows. The mask units in IL are set to 1 at the time of mask presentation and are otherwise set to 0. Therefore, the recognition of the stimuli is implemented with a localized representation, for example, the left unit is turned on when the stimulus is less than five in the case of numerals and symbols, or points left in the case of arrows; otherwise the right unit is turned on. Accordingly, as will be described below, in a congruent trial the two corresponding units (e.g., the left unit of the prime and target in IL) is set to 1 or 0 at the time of stimulus presentation, while in an incongruent trial, one of the two relevant units of the prime or target is set to 1 and the other to 0.

The units in each layer make connections, via excitatory weights, to their corresponding units in other layers. The activations of these units (except IL) are calculated by a sigmoid (logistic) function of the incoming information, and a small amount of random noise. The RL sends excitatory activities to ML and CL continuously but activates AA only if a unit of the prime or target reaches a designated threshold of .62. Similarly, when one of the two units in the ML reaches the same designated threshold it triggers a manual response (i.e., initiating a hand movement). When AA is activated and its activation reaches a threshold, it starts modulating information processing in RL, CL, and ML by making the activation function of their units steeper.

Modelling details. As shown in Figure 1, the IL encodes the prime, the mask, and the target, and projects to RL through excitatory connections. For the sake of simplicity, prime and target, as well as an identical mask for each (shown as a single unit in Figure 1, for the sake of simplicity) were implemented in two separate paths. All units in RL have a self-excitation connection, intended to simulate mutual excitation among a group of neurons. Connections between mutual units (for prime and target and to the mask) from IL to RL have small cross-talks (see Table 1), indicating feature overlaps or similarities among stimuli. The units also have lateral inhibition with neighboring units within the same layer.

The mask units are activated after the prime and before the target for a specific time. They have lateral inhibition with prime and target. In addition to lateral inhibition, the model simulates the similarity of the mask to the prime and target through a lateral excitation from mask to the prime and target. It plays a role using this lateral excitation and can affect ML and AA (and CL), indirectly, through its effect on both prime and target. Moreover, the prime and target units, but not the mask, have feed-forward projections into the ML, CL, and AA layers. Therefore, the mask acquires meaning through its relationship with the prime and target. Because it comes right after the prime, it can activate the prime through its excitation. So, it can act partially like the prime and increase the attentional responses to it, forcing it to stay longer, but, on the other hand, its inhibitory effect usually dominates its excitatory effect and interrupts the prime, causing it to decay faster. This interplay depends on the similarity of the mask to the prime and target (Sohrabi, 2008).

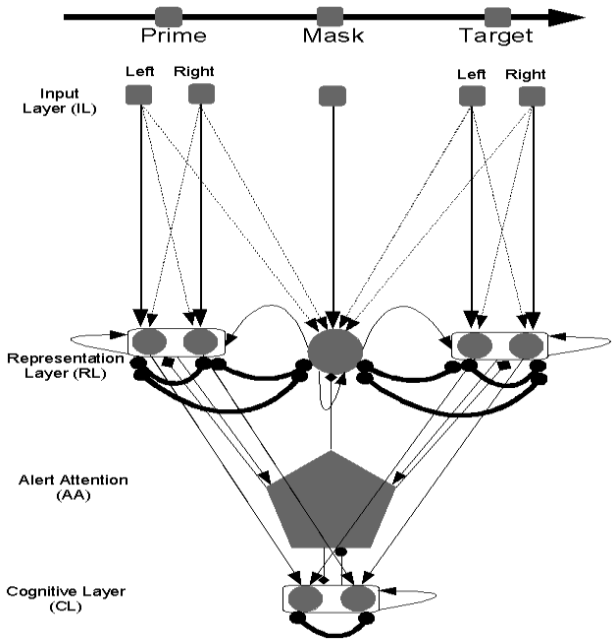

Figure 1: The architecture of the model. The IL projects to the RL. The RL excitates AA, CL, and ML (not shown). The AA modulates all other layers except IL. The CL changes the AA response mode in the event of conflict. Different connections are depicted with different arrows: –♦ modulatory; –● conflict monitoring; ↶ self-excitation and lateral excitation; ●–● Lateral inhibition; -▶ Feed-forward activation.

The units in all layers (except IL and AA) receive additive Gaussian noise (zero mean and variance σ), intended as general, irrelevant incoming activities. The activations in the model are represented using units with real valued activity levels. The units excite and inhibit each other through weighted connections. Activation propagates through the network when the IL is clamped with input patterns, leading to a final response. As will be described below, the states of units in RL, ML, and CL are adopted in a method similar to a noisy, leaky, integrator algorithm (Usher & Davelaar, 2002; Gilzenrat et al., 2002). These types of models are noisy versions of previous connectionist models.

In a typical masked trial or epoch, one of the prime units in the IL is turned on and the network is left active for 43 cycles. Then the mask units in IL are turned on for 71 cycles, followed by turning on the target input in IL for 200 cycles. This is similar to a trial in the experiment, except no forward mask is presented, for the sake of simplicity. The prime and target units in the IL are used to represent the stimulus features (i.e., direction or magnitude). However, as mentioned before, the recognition of the stimuli is not implemented in detail, but is encoded as a binary code. For example, in the case of arrows, 1 is used for the left unit if it points left, or in the case of numerical stimuli, if the number is less than five, and 0 is used for the opposite (reciprocal) unit. In the congruent condition, the RL units of the prime and target at the same side (left or right randomly) are turned on (1) or off (0) in each trial at the time of stimulus presentation. By contrast, in the incongruent condition, the two units at the opposite sides are turned on and the other two are left off, with random selection of the two possible cases.

The RL is governed by a modified version of previous models (Usher & Davelaar, 2002; Gilzenrat et al., 2002), which is calculated with discrete integrational time steps using the dynamic equation:

$$X(t+1) = \lambda_x X(t) + (1-\lambda_x)\, f\,[WX_iX_i\, X_i(t) + WX_iI_i\, I_i(t) - WX_iX_j\, X_j(t) - \theta X_i + \xi X_i] \quad (1)$$

Likewise, ML and CL are modelled in a similar way with their inputs coming from RL:

$$Y(t+1) = \lambda_y Y(t) + (1-\lambda_y)\, f\,[WY_iY_i\, Y_i(t) + WY_iX_i\, X_i(t)) WY_iY_j\, Y_j(t) - \theta Y_i + \xi Y_i] \quad (2)$$

In equations (1) and (2), X and Y denote the activity of units through time t. W is the weight of the connections between units, I is the input, and the subscripts i and j are indexes of the units. The three weight parameters in the brackets correspond to recurrent self-excitation, feed-forward excitation, and lateral inhibition, respectively. However, for the sake of simplicity in equation 1, the lateral excitation from mask units to the prime and target, $WX_iX_j\, X_j(t)$, and the cross-talk in prime and target to reciprocal units and mask units, $WX_iI_j\, I_j(t)$, are not present. The term θ is the bias, the term ξ is noise, and *f* is a sigmoid function (see equation 3). The term λ represents neural decay which is related to the discrete integrational time steps in the underlying equation (Usher & Davelaar, 2002).

The AA modulates other layers by changing their activation from sigmoid toward binary responses. The activation function, *f*, transfers the net input, X, of a unit, and modulatory gain, g, to its activity state, implementing the firing rate of a neuron or the mean firing rate of a group of neurons:

$$f(X) = 1/(1+\exp(-Xg)) \quad (3)$$

A conflict-monitoring measurement was employed to take the activations of the units in the CL layer to adjust phasic and tonic response modes of AA. The activation of the CL units was used to measure the Hopfield energy function between units (Hopfield, 1982), as used previously (Botvinick et al., 2001). Conflict can be defined as the concurrent activation of the competing units and as the joint effect of both prime and target in CL. Hopfield energy can be calculated as

$$E = -.5\, X^t W X = -.5 \begin{bmatrix} X_1 & X_2 \end{bmatrix} \begin{bmatrix} 0 & -1 \\ -1 & 0 \end{bmatrix} \begin{bmatrix} X_1 \\ X_2 \end{bmatrix} \quad (4)$$

where E denotes energy, X denotes the activity of a unit, W is the weight of the connection between units, and the subscripts 1 and 2 are indexes of the two units.

As noted above, CL combines prime and target activations and measures conflict between its two units. When one CL unit is active and the other is inactive, conflict is low. However, when both units are active concurrently, the conflict is high. Activations in CL units are converted to 1 if they are equal to or greater than .5, and to 0 otherwise (i.e., using a threshold function). Also, E > .5 is considered as a conflict, otherwise as no conflict. When the activation of a prime or target unit in RL reaches the designated threshold, .62, the AA is activated with a phasic or tonic mode, depending on the absence or presence of conflict in CL. The change in AA response mode usually occurs by the presentation of a target that is incongruent with the prime.

Here the AA is modelled using a reduced or abstracted version of LC neurons in a Willson-Cowan type of system (e.g., Wilson & Cowan, 1972) adopted recently (Usher & Davelaar, 2002) (there are similar models and detailed implementations of this type of attention (Gilzenrat et al., 2002):

$$X(t+1) = \lambda_x X(t) + (1-\lambda_x)\, f\,[c\,(a_x X(t) - bY(t) + I_x(t) - \theta_x)],$$
$$Y(t+1) = \lambda_y Y(t) + (1-\lambda_y)\, f\,[c\,(a_y X(t) - \theta_y)],$$
$$G(t+1) = \lambda_g G(t) + (1-\lambda_g)\, X(t) \quad (5)$$

where *f* is again a sigmoid function (as in equation 3), X is the fast variable representing AA activity and Y is a slow auxiliary variable, together simulating excitatory/inhibitory neuron groups in the LC (Usher & Davelaar, 2002). The X and Y variables have decay parameters λ_x and λ_y, excitatory/inhibitory coefficients, a_x and a_y, as well as thresholds θ_x and θ_y, respectively. The G variable is the

output of the AA, which is based on X. The g (used in equation 3) is computed from G: g = G * K. The AA modulates other layers when g crosses a threshold, 1. Its activity modes can be phasic or tonic depending on the conflict state, low or high, respectively. In all conditions the CL can change the AA mode according to the conflict between prime and target (i.e., using within-trial conflict). The phasic and tonic modes of AA responses are implemented using high or low c value (3 or 1) (see equation 5). The c value is 3 at the beginning of each trial (for the prime), but it is set to 1 (for the target) if conflict occurs. The number of computer simulation cycles from the target onset until one of the ML units reached a designated threshold, .62, was considered as RT. A constant, as other sensory and motor processes, could be added to this RT, to increase the match between simulation and human data.

Simulation Results

Simulation 1: mask-target SOA. To simulate the data from previous studies i.e., a PCE and an NCE with short and long mask-target SOAs, respectively (e.g., Schlaghecken & Eimer, 2000; Jaśkowski & Ślósarek, 2006) a simulation was run with no changes in the parameters except the mask-target SOA. Seven intervals of the mask-target SOA (from 71 to 251, with 30 cycles interval) were used to show the effect of SOA on priming pattern. To maintain consistency, the duration of the mask was again 71 cycles, but a longer mask duration has a similar effect (as used in the following simulations).

Simulation 2: stimulus degradation

A previous study (Schlaghecken & Eimer, 2002, Exp. 4) found that degradation of stimuli, by adding small random dots to all stimuli, turns NCE into PCE. Here, the degradation of stimuli was simulated by using lower input activation in IL (for both prime and target) compared to the usual 1 and 0 and increasing the noise of the prime and target in RL. Two levels of degradation were created by using .85 (opposite unit .15) and .75 (opposite unit .25), while 1 (opposite unit 0) was used to encode an intact stimulus. For a better fit between simulation and human data, the noise of the prime and target units in RL was increased from .2 to .3. The IL-RL strength for the prime and target was 2.5 and the mask-target SOA was 125 cycles. The model successfully simulated the human data as shown in Figure 3, top. With degradation, the NCE turned into PCE and RTs were increased by more degradation.

In another experiment in the same study (Schlaghecken & Eimer, 2002, Exp. 3), random dots were added to all stimuli, but the dots did not cover the target (presented above or below the target, randomly). In this case, while degradation turned the NCE into PCE, it did not increase the RTs. For simulating this experiment, a simulation was run identical to the previous one but only the prime was degraded. The result was similar to the human data. As shown in Figure 3, bottom, if the target is not degraded the RTs do not increase (because it is stronger and is processed faster).

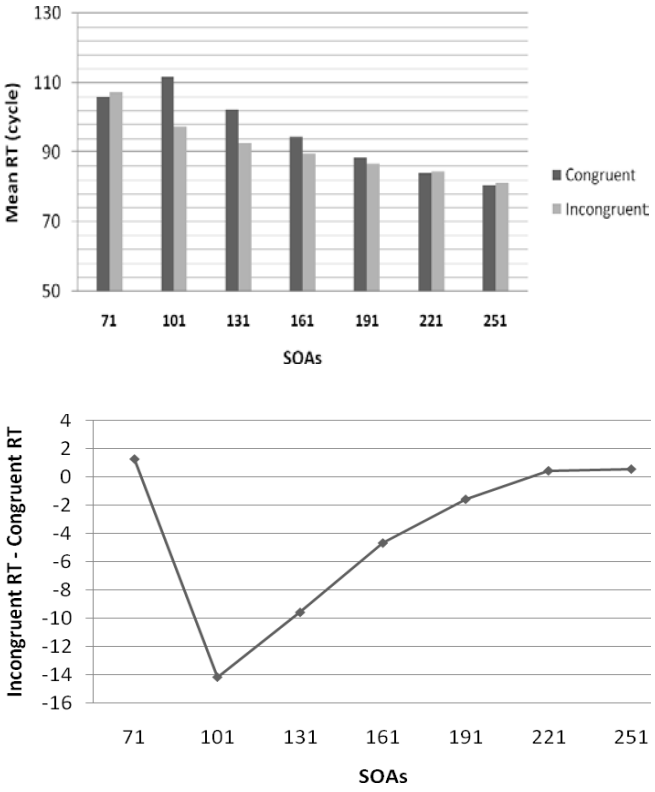

Figure 2: The result of Simulation 1, mask-target SOA. Top: Modelling results at seven levels of mask-target SOA, starting from 71 cycles. Each SOA follows 30 cycles after the previous one, with mask duration of 71 cycles. Bottom: The same result was shown by the congruency difference (Incongruent - Congruent) in the seven SOAs. This is similar to the different lags in attentional blink paradigm, showing a similar attentional basis for priming and attentional blink.

Simulation 3: mask density

It has been shown that the mask needs to be dense enough at a specific rate to cause NCE, and that decreasing the density changes NCE to PCE (e.g., Eimer & Schlaghecken, 2002), although beyond that it has no major effects. In this simulation, mask density was simulated by changing the inputs of the mask units to .55 (medium density) and .45 (low density), instead of 1 (very high density, used in simulations of usual masked conditions). The IL-RL strength for the prime and target was 2.5 and the mask-target SOA was 125 cycles. As shown in Figure 4, top, similar to human data (e.g., Eimer & Schlaghecken, 2002, Exp. 1) decreasing the mask density from 1 to .55 decreased NCE and then to .45 and 0 turned NCE to PCE (low mask density and no mask are supposed to invoke other types of processes, Sohrabi, 2007, not discussed here).

Simulation 4: prime duration

Prime duration has an important role in the priming effect. Stimuli with longer duration have stronger representations and also activate more attentional responses. It has been shown that increasing the prime duration increases NCE to some extent and turns it to PCE after a specific rate (Eimer & Schlaghecken, 2002). The current simulation shows the priming effects for three prime durations: 43, 48, and 53 cycles. The IL-RL strength for the prime and target was 2.5 and the mask-target SOA was 125 cycles.

As shown in Figure 4, bottom, increasing the prime duration caused larger NCE, but a further increase turned it into PCE. Interestingly, increasing the prime duration does not decrease RTs and even has an opposite effect, similar to human data (e.g., Eimer & Schlaghecken, 2002, Exp. 2) (longer duration is supposed to invoke other types of processes, Sohrabi, 2008, not discussed here).

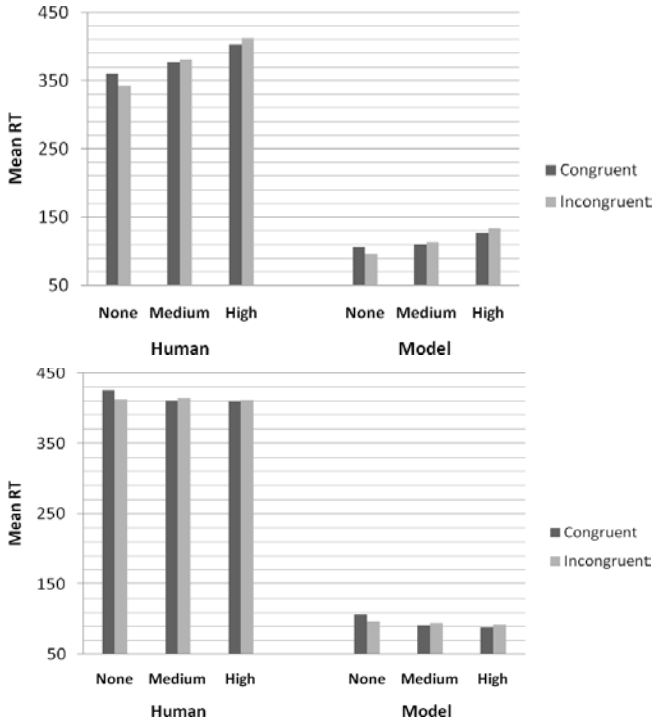

Figure 3: Results of Simulation 2, degradation effect. Top: Degrading the prime and target with three levels of prime and target inputs in IL: 1 (no degradation), .85 (medium degradation), and .75 (high degradation), as well as an increase in noise. With degraded unit activations NCE turned into PCE and RTs increased. Bottom: Degrading only the prime turned NCE to PCE but did not increase RTs.

Discussion

When the mask-target SOA was short, the target could be processed primarily with the initial activation and attentional response to the prime. When a delay was introduced between them (as with longer mask-target SOA), the second phase of attention (for the target) was not strong enough to activate the target quickly. This happened because attention showed a phasic response with a refractory period. The conflict was measured based on the incongruency in the stimuli relationship. It decreased the effect of the refractory period by putting the second phase of attention (to the target) in a tonic mode, enhancing the processing of the incongruent trials where conflict occurred. This was not the case in the congruent trials. The NCE found in previous studies (Schlaghecken & Eimer, 2000, 2002; Eimer & Schlaghecken, 1998, 2002; Jaśkowski & Ślósarek, 2006) was simulated by increasing mask-target SOA, with no other changes in the model. A PCE and an NCE were found with short and long mask-target SOA, respectively.

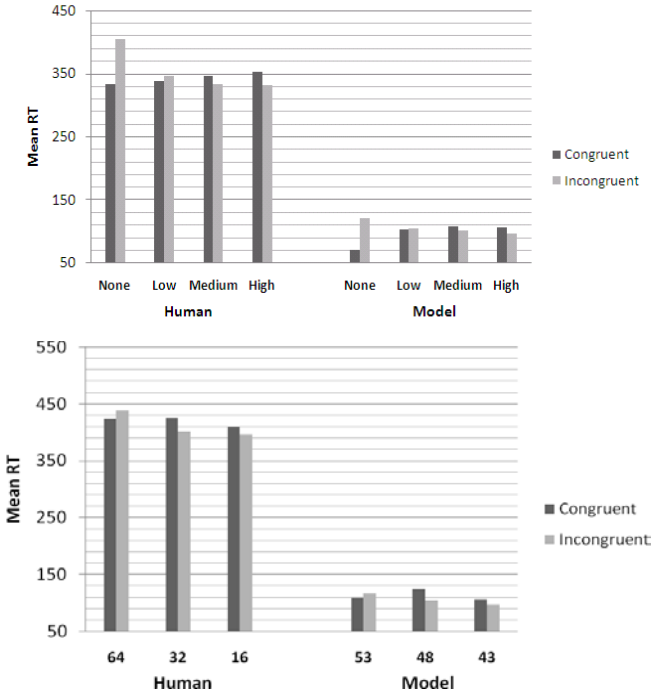

Figure 4: Results of Simulations 3 (mask density) and 4 (prime duration). Top: Four levels of mask density were employed: 1 (no mask), 2 (low density), 3 (medium density), and 4 (high density), simulated by IL mask unit inputs 0, .45, .55, and 1 compared to masks with 15, 10, 5, and 0 random lines in human data, respectively (Eimer & Schlaghecken, 2002, Exp. 1). Bottom: Simulation results for three levels of prime duration: 53 cycles (long), 48 cycles (medium), and 43 cycles (short), compared to 64, 32, and 16 ms in human data (Eimer & Schlaghecken, 2002). Increasing the prime duration increased the NCE but a further increase turned the NCE into PCE.

The model also showed the effects of other factors on priming directions such as prime duration, stimulus degradation, and mask density. For example, a prime with longer duration and less degradation has a strong representation that causes a large NCE if the target comes late (and a large PCE if it comes early). The model also

showed that decreasing the activation of input units (e.g., from binary, 1 and 0, to real normalized numbers, .9 and .1, or less, for simulating stimulus degradation) turns NCE into PCE. This supports the idea that the NCE is not caused merely by a decrease in the incoming perceptual information but by a decrease in the representation strength. The current model, in addition to being more biologically compelling, showed many dynamic effects in RT and error patterns that have not been shown previously (such as the changes in RT and the size of priming effects through time). While in the current model the NCE disappeared and became a very small PCE at very long SOAs, the previous model (Bowman et al., 2006) showed a huge PCE at very long SOAs inconsistent with human data (e.g., Jaśkowski & Ślósarek, 2006). The present model is similar to some other previous neuro-computational models, especially those employed to simulate the attentional blink (Mathis & Mozer, 1996; Nieuwenhuis, et al., 2005). In these models blink for the second target occurs at lag 2 (after 100 ms from the first target) and no blink occurs at lag 1 (if the second target appears during 100 ms after the first target), related to NCE and PCE in the current model, respectively.

Table 1. *Parameters in the model, fixed for all simulations, unless otherwise mentioned.*

WX_iI_i (IL to RL) [P & T] & WY_iX_i (RL to ML) [P & T]	2-3 & 1.5
WX_iI_i (IL to RL) [M] & WY_iX_i (RL to CL) [P & T]	1.5 & 1
WX_iX_i (RL) [P & T], WX_iX_i (RL) [M], WY_iY_i (CL), & WY_iY_i (ML)	1.5, 1.25, 1, & .9
WX_iX_j (RL) & WY_iY_j (ML & CL)	1 & 1
WX_iX_j (RL) [M to P & T] & WX_iI_j (IL to RL)	.75 & .33
K (AA)	4.52
θ_x, θ_y (AA), θ_x (RL), θ_y (CL), & θ_y (ML)	1.25, 1.5, .5, .85, & 2
b,c, a_x & a_y (AA)	4, 1-3, 2, & 3
λ_x, λ_g, & λ_y (AA)	.92, .98, & .996
λ (CL), λ (ML), & λ (RL)	.75, .925, & .95
σ (CL), σ (RL) [P & T], σ (ML) & σ (RL) [M]	.025, .2, .25, & 1.25

IL=Input Layer; RL=Representation Layer; CL= Cognitive Layer; ML=Motor Layer; AA=Alert Attention; P=Prime; T=Target; M=Mask.

Acknowledgement

This research was supported by the Carleton Cognitive Modelling Lab, Ottawa, Ontario, Canada and the University of Kurdistan, Sanandaj, Kurdistan, Iran.

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