A Cognitive Model of Positive and Negative Congruency Effects in Unmasked Priming: The Role of Attentional Limit and Conflict

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Abstract

Positive priming effect has been found with a short interval between the prime and the target, while negative priming effect (i.e., a congruent prime causes longer RTs) has been found with a long time between the prime and the target. Negative priming effect has been shown mainly using masked priming but some recent studies have shown it without masks (i.e., in unmasked or conscious conditions). We employed our previous model of masked priming for the unmasked condition here, only by removing mask presentation. The model successfully simulated the negative priming effect in unmasked condition found in previous experimental studies.

Keywords: Negative congruency effect; Negative compatibility effect; modeling; attention; consciousness.

Introduction

Studies on priming have long shown reliable positive effects of the congruent prime on target processing. An early study, in the age of using tachistoscopes, was one conducted by Marcel (1983) on word and color naming. The effect of masked priming showed that masked stimuli are indeed processed to the level of response. Later studies on unmasked and masked conditions showed similar results both for masked priming (e.g., Neumann & Klotz, 1994; Dehaene et al., 1998; Eimer & Schlaghecken, 2002) and masked and unmasked priming differences (e.g., Cheesman & Merikle, 1986; Dehaene, Artiges, et al., 2003; Schlaghecken & Eimer, 2002).

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 speeds up the performance on the target if they are congruent and slows down the performance if they are incongruent (e.g., Neumann & Klotz, 1994; Dehaene et al., 1998; Eimer & Schlaghecken, 2002; Jaśkowski & Ślósarek, 2007). 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., Schleghecken & Eimer, 2000, 2002, 2006; Eimer, 1999; Eimer & Schlaghecken, 1998; 2001, 2002; Lleras & Enns, 2004, 2006; Verleger et al., 2004; Jaśkowski & Ślósarek, 2006). The PCE has been shown with a short mask-target Stimulus Onset Asynchrony (SOA), while the NCE has been shown with a longer mask-target SOA (see below).

The PCE has been found usually with verbal and shape stimuli and a short mask (e.g., 71 ms, as in Dehaene et al., 1998) and no or a small interval between stimuli. In contrast, the NCE has been shown mainly with arrow stimuli and a longer mask (e.g., 100 ms). Recently, it has been replicated with other stimuli, for example shapes (Jaśkowski & Ślósarek, 2006) and faces (Bennett, Lleras, Orient, & Enns, 2007). This effect has been found by using a long mask (about 100 ms) and a long mask-target SOA (>80 ms) or a long (> 30 ms) prime-mask Inter Stimulus Interval (ISI) or mask-target ISI (e.g., Eimer & Schleghecken, 1998, 2002; Jaśkowski & Ślósarek, 2007). These manipulations all increase the prime-target SOA.

In Eimer and Schlaghecken's (2002) aforementioned experiments on the role of prime duration and mask density, participants who were better at detecting the prime showed a later change from positive to negative, and conversely those who were not good in reporting the prime showed an earlier change from positive to negative, showing that there is a close relationship between prime reportability and the direction of priming. Schlaghecken and Eimer (2000) and Eimer and Schlaghecken (2002, see also 2003) found that when there is no mask or the mask is peripheral (i.e., it does not make the prime unreportable), the result is PCE, unlike the situation with masked priming. Using their motor self-inhibition hypothesis, they argued that the inhibition is initiated (as an automatic or evolved process) when visual input disappears, otherwise is blocked by visual input. Therefore, they claimed that an NCE, being a result of this self-inhibition, occurs only in the masked condition because prime input is stopped by the mask. They added that with the reportable prime, motor self-inhibition is prevented by the prime, so a PCE occurs. However, recently Lleras and Enns (2006), by comparing different studies, showed that prime visibility has no linear relationship with NCE, meaning that NCE is not necessarily caused by prime invisibility (see below).

To investigate whether there is any differences between masked and unmasked priming, Cheesman and Merikle (1986) employed Marcel's colour priming task with modifications. They changed the ratio of congruent to incongruent trials, so that in one condition this ratio was 25:75 and in the other one it was 75:25. In the unmasked condition, they found that when the number of congruent trials was high (i.e., the 75:25 condition), the congruency effect was higher than when this number was low (i.e., the 25:75 condition). In other words, when an incongruent trial was frequently preceded by a congruent trial, the congruency effect increased, and conversely, when an incongruent trial was frequently preceded by an incongruent trial, the congruency effect decreased. This difference was not found in the masked condition. They argued that participants can use a strategy based on context only in the unmasked condition.

Jaśkowski (2007) combined Eimer and Schleghecken's paradigm and Merikle and colleagues' (Cheesman & Merikle, 1984, 1986; Merikle & Joordens, 1997) to study the difference between the masked and unmasked conditions. In a congruent to incongruent ratio of 20:80, a PCE was found in the unmasked condition with both medium (100 ms) and long (800 ms) prime-target ISI. While in the congruent to incongruent ratio of 80:20, a PCE was found in medium (100 ms) ISI but an NCE was found, interestingly enough, in long ISI condition. In another experiment, while Jaśkowski found an NCE in the masked condition with a prime-target ISI of 100 ms, he found only a non-significant NCE with a long ISI. Therefore, surprisingly, with the long ISI the NCE for the unmasked condition was larger than it was for the masked condition, ruling out the necessity of the mask and invisibility of the prime in NCE. A similar result had already been found with a Stroop task (Merikle & Joordens, 1997).

In our previous work we have modeled masked priming using a neurocomputational cognitive model (Sohrabi and West, 2009a, b; see also Sohrabi, 2008). We employed that model of masked priming for the unmasked condition here. We only removed the mask presentation to simulate the unmasked condition in human experimental studies (here, Jaśkowski, 2007).

The Model

The model is based on previous neurocomputational modeling and nurophysiological studies (e.g., Usher & Davelaar, 2002; Gilzenrat et al., 2002, see also Aston-Jones & Cohen, 2005). 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 has been described previously (Sohrabi and West, 2009a, b; see also Sohrabi, 2008 and Sohrabi and West, 2010). It is a multi-layer dynamic neural model (shown in Figure 1) that consists of a feed-forward component for perceptuo-motor processing from the Input Layer (IL) to the Representation Layer (RL) and Motor Layer (ML, not shown). 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 Task Layer (i.e., TL) for conflict monitoring. The effect of TL on AA simulates direct cortical projections to LC (Aston-Jones & Cohen, 2005). The TL and ML are affected by both prime and target. The ML's architecture is identical to TL's, 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 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 an arrow pointing left; 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 TL 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, TL, and ML by making the activation function of their units steeper (see Figure 2, as described below).

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 units, as well as an identical mask unit for each (not activated in this simulation) were implemented in two separate paths. All units in TL 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 TL 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. To simulate unmasked condition here the mask units are not activated (i.e., are not clamped) but units' baseline activities were preserved for the sake of model stability without changing the parameters.

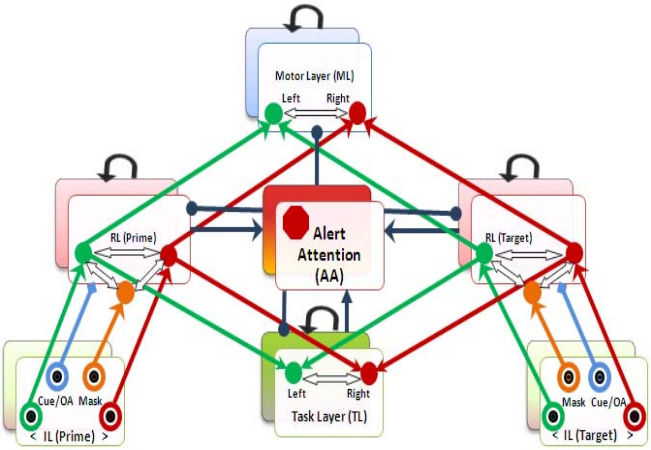

Figure 1. Architecture of the model showing hypothetical networks and connections. *Unit types:* ◉ IL ● TL and ML (not shown here) ● AA. *Attention types:* -◆ Cue/Orient Attention (OA) (not employed here) -▶ Executive (conflict driven) -● Alert. *Activation types:* ↶ Self-excitation and recurrent excitation ↔ Lateral inhibition ⟶ Feed-forward activation.

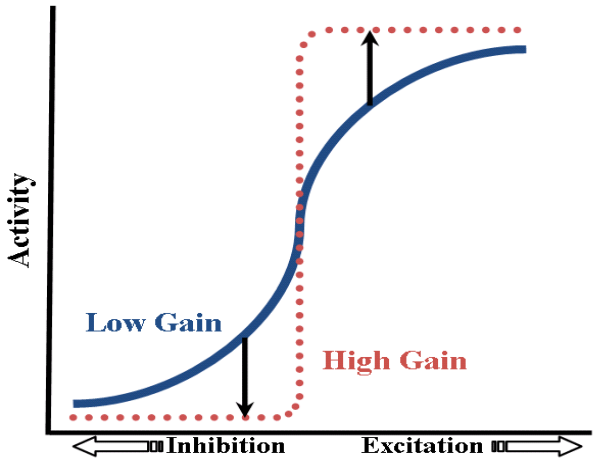

Figure 2. Effect of gain modulation on nonlinear activation function (adapted from Servan-Shreiber et al., 1990, see also Astone-Jones & Cohen, 2005).

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 TL 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 each trial or epoch, one of the prime units in the IL is turned on and the network is left active for 43 cycles, then it is turned off for 168 cycles (short prime-target SOA), 234 cycles (long prime-target SOA), or 294 cycles (very long prime-target SOA), followed by turning on the target input in IL for 200 cycles. This is similar to a trial in human data (Dehaene et al., 1998; Eimer & Schleghecken, 2002; Jaśkowski & Ślósarek, 2006; Jaśkowski, 2007).

The prime and target units in the IL are used to represent the stimulus features (here, direction). 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 here, 1 is used for the left unit if it points left, 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:

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

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

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

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:

$$f(X_i)=1/(1+\exp(-X_i g)) \quad (3)$$

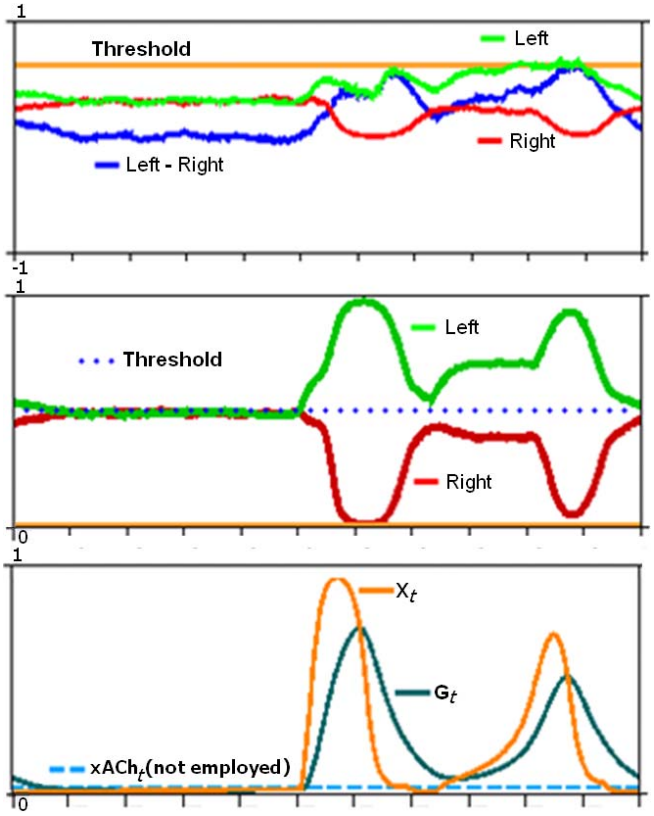

Figure 3. An unmasked congruent trial (where no conflict occurs) of 1100 cycles, including 500 cycles inter-trial interval) with 234 cycles prime-target SOA that crosses the threshold after 876 cycles (including 500 cycles inter-trial interval). From the top, ML, TL, and AA (but RL-prime, RL-target, and IL are not shown).

A conflict-monitoring measurement was employed to take the activations of the units in the TL layer to adjust phasic and tonic response modes of AA. The activation of the TL 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 TL. 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, TL combines prime and target activations and measures conflict between its two units. When one TL unit is active and the other is inactive, conflict is low. However, when both units are active concurrently, the conflict is high. Activations in TL 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 TL reaches the designated threshold, .62, the AA is activated with a phasic or tonic mode, depending on the absence or presence of conflict in TL. The change in AA response mode usually occurs by the presentation of a target that is incongruent with the prime.

Here the AA is modeled 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):

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

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 TL 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.

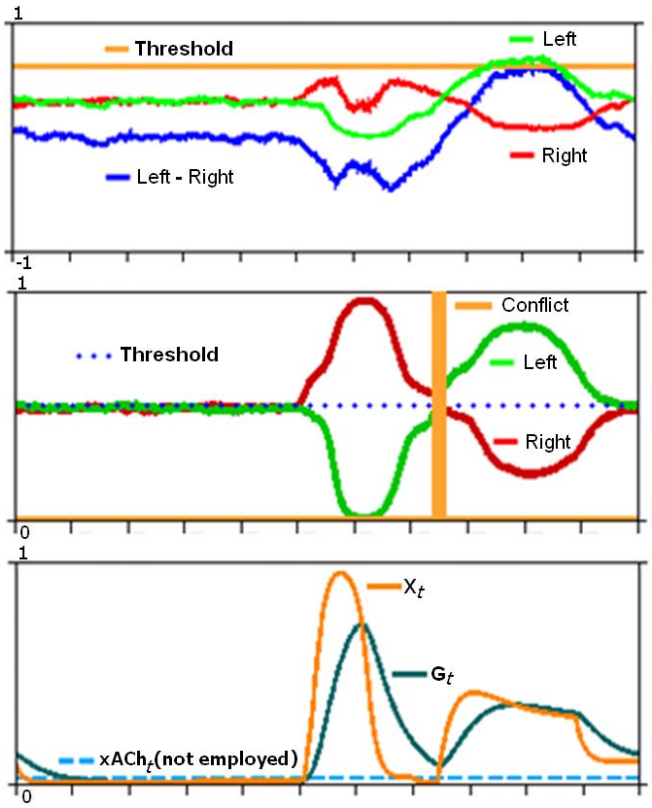

Figure 4. An unmasked incongruent trial (where no conflict occurs) of 1000 cycles, including 500 cycles inter-trial interval) with 234 cycles prime-target SOA that crosses the threshold after 876 cycles (including 500 cycles inter-trial interval). From the top, ML, TL, and AA (but RL-prime, RL-target, and IL are not shown).

Simulation Results

To create the short and long prime-target SOA conditions, a relatively short SOA (168 cycles) and two relatively long SOAs (234 and 294 cycles) were used. As shown in Figure 5, a strong PCE was found at prime-target SOA 168 cycles and a strong NCE was found at SOA 234 and 294 cycles. In the unmasked condition, in the current simulation, NCE remains high with further increases in SOA but it decreases slowly.

The simulation results in Figure 5 show a change from PCE to NCE and a drop in RTs, similar to the human data. However, the SOA in the long condition in Jaśkowski (2007) is much longer than the long conditions in the current simulation, due to a limited time course in the model, as the parameters were set for a short trial.

The activities in three layers (ML, TL, and AA) are shown for a given congruent and incongruent trial in Figures 3 and 4, respectively. There is smaller activation left in AA for the target, but it can be recovered as an effect of conflict in the incongruent condition as the phasic mode becomes tonic.

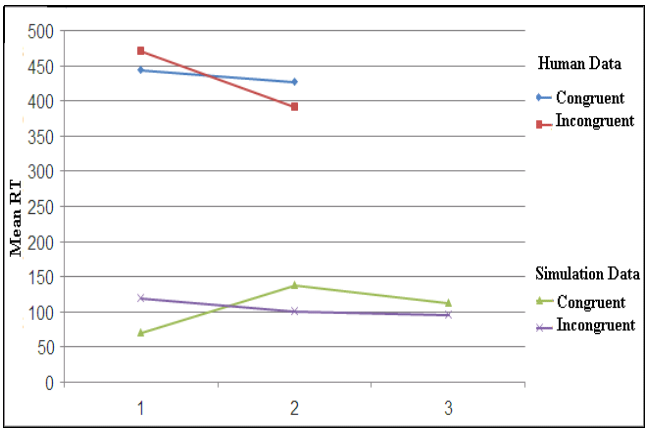

Figure 5. Unmasked priming using 168, 234, and 294 cycles prime-target SOAs, indicated by 1, 2, and 3, respectively, compared to 116.7 and 816.7 SOAs in Jaśkowski (2007), indicated by 1 and 2, respectively.

Discussion

A model that we have used for simulating masked positive and negative priming previously could simulate unmasked priming effect as well. Because there was no interruption by the mask, in the relevant unmasked prime condition, a PCE was found for short prime-target SOA. In this case, the PCE was large, consistent with the unmasked condition in Jaśkowski (2007). We assumed that a relevant or predicting prime as in Jaśkowski (2007) evokes a phasic activation in the so called alert attention to the prime but can lead to a refractory period of attentional response to the target.

An unmasked prime caused large PCE and NCE at short and long prime-target SOA, respectively. A few studies have previously shown an NCE in the unmasked condition. Here it is assumed that this effect was found in those studies because they used a medium (Koechlin et al., 1999) and long (Jaśkowski, 2007) prime-target SOA, and especially the tasks required action on (which requires attention too), or attention to, the prime, respectively. In the former, especially because of controlling physical repetition priming (and an action on the prime was required as on the target), and in the second, especially because of prime relevance (participants were told that prime highly predicts the target), the NCE was large. It could be caused by the strong refractory period created by attention to the unmasked prime. To simulate this phenomenon, in this simulation the prime was unmasked and AA mode for the prime was put in the high phasic mode ($c=3$), as with simulations of masked conditions.

At longer prime-target SOA, the relevant unmasked prime caused an NCE even larger than an equivalent masked condition (see Sohrabi and West, 2009a, b; see also Sohrabi, 2008; Sohrabi and West, 2010), consistent with Jaśkowski & Ślósarek, (2006) and Jaśkowski (2007). Interestingly, the conflict period caused by an unmasked incongruent prime (in all unmasked

conditions) was longer than that of masked incongruent prime consistent with Dehaene et al. (2003) that have shown more brain activations in unmasked incongruent compared to congruent condition.

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

WX_iI_i (IL to RL) [P & T] & WY_iX_i (RL to ML) [P & T]	3 & 1.5
WX_iI_i (IL to RL) [M] & WY_iX_i (RL to TL) [P & T]	1.5 & 1
WX_iX_i (RL) [P & T], WX_iX_i (RL) [M], WY_iY_i (TL), & WY_iY_i (ML)	1.5, 1.25, 1, & .9
WX_iX_j (RL) & WY_iY_j (ML & TL)	1 & 1
WX_iX_j (RL) [M to P & T] & WX_iI_j (IL to RL)	.75±.1 & .33
K (AA)	4.52
α & β (RL, TL, & ML) [M, P, T]	1 & 1
θ_x, θ_y (AA), θ_x (RL), θ_y (TL), & 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 (TL), 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; TL= Task Layer; ML=Motor Layer; AA=Alert Attention; P=Prime; T=Target; M=Mask.

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References

Aston-Jones, G. & Cohen, J. D. (2005). An Integrative Theory of Locus Coeruleus-Norepinephrine Function: Adaptive Gain and Optimal Performance, *Nature Review Neuroscience*, 28, 403-450.

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

Bowman, H., Schlaghecken, F., Eimer, M. (2006). A neural network model of inhibitory processes and cognitive control. *Visual Cognition,* 13, 401–480.

Cheesman, J. & Merikle, P. M. (1984). Priming with and without awareness. *Perception & Psychophysics*, 36, 387–395.

Dehaene, S., Naccache, L., Le Clec'H, G., Koechlin, E., Mueller, M., Dehaene-Lambertz, G., van de Moortele, P. F., and Le Bihan, D. (1998). Imaging unconscious semantic priming. *Nature*, 395, 597-600.

Dehaene, S. et al. (2003). Conscious and subliminal conflicts in normal subjects and patients with schizophrenia: the role of the anterior cingulated, *Proceedings of the National Academy of Sciences of the United States of America,* 100, 13722-13727.

Eimer, M. & Schlaghecken, F. (1998). Effects of masked stimuli on motor activation: Behavioural and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance*, 24, 1737-1747.

Eimer, M. & Schlaghecken, F. (2002). Links between conscious awareness and response inhibition: evidence from masked priming. *Psychonomic Bulletin & Review,* 9, 514–520.

Gilzenrat, M. S., Holmes, B. D., Rajkowski, J., Aston-Jones, G., & Cohen, J. D. (2002). Simplified dynamics in a model of noradrenergic modulation of cognitive performance. *Neural Networks, 15,* 647–663.

Hopfield, J. J. (1982). Neural networks and physical systems with emergent collective computational abilities. *Proceedings of the National Academy of Sciences, USA, 79,* 2554-2558.

Jaśkowski, P. & Ślósarek, M. (2007). How important is a prime's gestalt for subliminal priming? *Consciousness and Cognition*, 16, 2, 485-497.

Lleras, A., Enns, J.T. (2006). How much like a target can a mask be? Geometric, spatial, and temporal similarity in priming. A reply to Schleghecken & Eimer (2006). *Journal of Experimental Psychology: General* 135, 495–500.

Marcel, A.J. (1983). Conscious and unconscious perception: experiments on visual masking and word recognition. *Cognitive Psychology* 15, 197-237.

Merikle, P. M., & Joordens, S. (1997). Parallels between perception without attention and perception without awareness. *Consciousness and Cognition*, 6, 219–236.

Neumann, O., Klotz, W. (1994). Motor responses to unreportable, masked stimuli: Where is the limit of direct motor specification. In C. Umiltà and M. Moscovitch (Eds.), *Attention and Performance XV: Conscious and non-conscious information processing* (pp. 123-150). Cambridge: MIT Press.

Nieuwenhuis, S., Gilzenrat, M. S., Holmes, B. D. & Cohen, J. D. (2005). The Role of the Locus Coeruleus in Mediating the Attentional Blink: A Neurocomputational Theory. *Journal of Experimental Psychology: General*, 134, 3, 291–307.

Schlaghecken, F., & Eimer, M. (2002). Motor activation with and without inhibition: Evidence for a threshold mechanism in motor control. *Perception & Psychophysics, 64,* 148–162.

Schlaghecken, F., Eimer, M., (2000). A central/peripheral asymmetry in subliminal priming. *Perception and Psychophysics,* 62, 1367–1382.

Servan-Schreiber, D., Printz, H., Cohen, J.D. (1990). A network model of catecholamine effects gain signal to noise ratio and behaviour. *Science* 249,892–895.

Sohrabi, A. (2008). Positive and Negative Congruency Effects in Masked and Unmasked Priming: Match of representation strength, Attention, and Consciousness. Ph.D. dissertation, Carleton University.

Sohrabi, A. and West, R. L. (2010). Cognitive Science of Primed Decision Making. *VDM-Verlag Publishing.*

Sohrabi, A. & West, R. L. (2009a). Positive and Negative Congruency Effects in Masked Priming: A Neuro-computational Model Based on Representation, Attention, and Conflict, *Brain Research, 1289, 124-132.*

Sohrabi, A. & West, R.L. (2009b). A Biologically-Plausible Cognitive Model (BPCM) of Positive and Negative Congruency Effects in Masked Priming. Proceedings of the 31st annual meeting of the Cognitive Science Society, Amsterdam, Netherlands, pp. 911-916.

Usher, M., & Davelaar, E. J. (2002). Neuromodulation of decision and response selection. *Neural Networks, 15,* 635–645.

Wilson, H., & Cowan, J. (1972). Excitatory and inhibitory interactions in localized populations of model neurons. *Biological Cybernetics*, 12, 1–24.