Semantic Priming in a Cortical Network Model

Nicolas Brunel[1] and Frédéric Lavigne[2]

Abstract

■ Contextual recall in humans relies on the semantic relationships between items stored in memory. These relationships can be probed by priming experiments. Such experiments have revealed a rich phenomenology on how reaction times depend on various factors such as strength and nature of associations, time intervals between stimulus presentations, and so forth. Experimental protocols on humans present striking similarities with pair association task experiments in monkeys. Electrophysiological recordings of cortical neurons in such tasks have found two types of task-related activity, ''retrospective'' (related to a previously shown stimulus), and ''prospective'' (related to a stimulus that the monkey expects to appear, due to learned association between both stimuli). Mathematical models of cortical networks allow theorists to understand the link between the physiology of single neurons and synapses, and network behavior giving rise to retrospective and/or prospective activity. Here, we show that this type of network model can account for a large variety of priming effects. Furthermore, the model allows us to interpret semantic priming differences between the two hemispheres as depending on a single association strength parameter. ■

INTRODUCTION

Contextual recall such as involved in language comprehension requires dynamic access to knowledge in memory. This fundamental cognitive function is based on semantic priming processes depending on learned associations between concepts in memory. The first experimental study of semantic priming effects reported shorter reaction times with related word pairs (e.g., *butter* and *bread*) than with unrelated pairs (e.g., *tree* and *bread*) (Meyer & Schvaneveldt, 1971). Priming effects were soon reported to occur during words sequence processing, when the target was presented following a related or unrelated prime (Meyer, Schvaneveldt, & Rudy, 1972). A typical semantic priming procedure requires subjects to read the prime and give a response to the target as fast and accurately as possible according to a given task, such as lexical decision, word naming, and semantic categorization. According to the paradigm of mental chronometry proposed by Donders (see Posner, 1978), variations in a behavioral response times are assumed to reflect dynamic variations in the ''activation'' of memorized concepts by a semantically related contextual prime word or stimulus (see Neely, 1991; Meyer & Schvaneveldt, 1976; Collins & Quillian, 1969). The possibility given by reaction times studies to probe the magnitude of the semantic activation of concepts in memory made semantic priming in humans an active field of research. A large amount of data reveals a rich phenomenology of semantic processes that are still to be linked to realistic properties of cortical networks in a unified way. Semantic priming effects vary notably with the timing of experimental procedures, as well as the strength and type of the prime–target relation (see McRae & Ross, 2004; Chiarello, Liu, Shears, Quan, & Kacinik, 2003; Hutchison, 2003; Lucas, 2000; Neely, 1991 for reviews).

Semantic Priming Dynamics

The temporal dynamics of priming effects are investigated in humans by manipulating the time elapsed between prime and target onsets, or stimulus onset asynchrony (SOA). Priming effects are reported to arise at very short SOAs of a few tens of milliseconds (Perea & Rosa, 2002; Rastle, Davis, Marslen-Wilson, & Tyler, 2000; Lee, Binder, Kim, Pollatsek, & Rayner, 1999; Perea & Gotor, 1997; Lukatela & Turvey, 1994; de Groot & Nas, 1991; Sereno, 1991; Beauvillain & Segui, 1983). Priming effects increase with increasing SOAs (Coney, 2002; Rastle et al., 2000) and reach maximum amplitude that sustains up to SOAs of several seconds (Hill, Strube, Roesch-Ely, & Weisbrod, 2002; Deacon, Uhm, Ritter, Hewitt, & Dynowska, 1999; Brodeur & Lupker, 1994; Balota, 1983; Fowler, Wolford, Slade, & Tassinary, 1981; Fischler, 1977). The precise time course of priming effects has also been reported to depend on the type of semantic relation and on association strength.

Types of Relationships between Prime and Target

Studies on semantic priming have investigated how priming effects depend on the type of relationships between

[1]Université René Descartes, Paris, France, [2]Université de Nice Sophia Antipolis, Nice, France

prime and target (direct vs. indirect), and on the association strength (estimated in production norms as the percentage of production of targets associated to a given prime word among several subjects; McRae, Cree, Seidenberg, & McNorgan, 2005; Cree & McRae, 2003; Nelson, McEvoy, & Schreiber, 1999; McRae, de Sa, & Seidenberg, 1997; Battig & Montague, 1969; Shapiro & Palermo, 1968).

Step 1 priming corresponds to a direct association between prime and target in memory (e.g., *tiger–stripes*). It is typically reported to arise at short SOAs (Lee et al., 1999; Perea & Gotor, 1997; Perea, Gotor, & Nacher, 1997; Hodgson, 1991; den Heyer, Briand, & Smith, 1985). Some studies report late effects (Rastle et al., 2000; Smith, Briand, Klein, & den Heyer, 1987) or even no effects (de Mornay Davies, 1998; Thompson-Schill, Kurtz, & Gabrieli, 1998), especially at short SOAs on weak associates (Williams, 1996; see Hutchison, 2003 for a discussion). Such variability in the onset and magnitude of Step 1 priming effects is reported as depending on association strength (Coney, 2002; Abernethy & Coney, 1993).

Step 2 priming corresponds to an indirect association through a common associate [e.g., *lion*–(tiger)–*stripes*]. Such indirect prime–target relationships typically give rise to significant priming effects (Kreher, Holcomb, & Kuperberg, 2006; Bennett & McEvoy, 1999; Kiefer, Weisbrod, Kern, Maier, & Spitzer, 1998; Livesay & Burgess, 1998; Weisbrod, Maier, Harig, Himmelsbach, & Spitzer, 1998; Sayette, Hufford, & Thorson, 1996; Spitzer, Braun, Maier, Hermle, & Maher, 1993; McKoon & Ratcliff, 1992; McNamara, 1992; Shelton & Martin, 1992; McNamara & Altarriba, 1988; Ratcliff & McKoon, 1988; Balota & Lorch, 1986; Motes, personal communication, for a meta-analysis; but see de Groot, 1983). Step 2 priming is reported as weaker than Step 1 priming (Kiefer, Ahlegian, & Spitzer, 2005; Hill et al., 2002; Chwilla, Kolk, & Mulder, 2000; McNamara, 1992; see Weisbrod et al., 1999), and stronger than Step 3 priming reported across two intermediate items [e.g., *mane*–(*lion–tiger*)–*stripes*; see Chwilla & Kolk, 2002; McNamara, 1992]. Some authors report Step 2 priming at long SOAs only (Arnott, Chenery, Copland, Murdoch, & Silburn, 2003; Hill et al., 2002; Bennett & McEvoy, 1999; Kischka et al., 1996; Spitzer et al., 1993), or more reliable at long SOAs than at short SOAs (Kiefer et al., 2005; Moritz, Woodward, Kuppers, Lausen, & Schickel, 2003; Hill et al., 2002; but see Arnott et al., 2003; Kischka et al., 1996).

Other studies have investigated how Step 2 priming effects depend on the number n of common associates to the prime and the target (Step 2_n priming; Fischler, 1977; see McRae, 2004). The degree of overlap n between prime and target associates differs from direct association strength and can involve semantic relations between category and exemplar (e.g., bird–robin), concept and feature (e.g., bird–feathery), and co-exemplars (e.g., robin–sparrow). Step 2_n priming is reported at short SOAs (Deacon et al., 1999; Thompson-Schill et al., 1998; Ober, Vinogradov, & Shenaut, 1995; Brodeur & Lupker, 1994; Smith et al., 1987; den Heyer et al., 1985; Favreau & Segalowitz, 1983; Neely, 1977), equivalent as (den Heyer et al., 1985) or stronger than (Smith et al., 1987) Step 1 priming. The magnitude of Step 2_n priming effects is reported as increasing with overlap n (Gonnerman, Seidenberg, & Andersen, 2007; Sanchez-Casas, Ferré, Garcia-Albea, & Guasch, 2006; Ober et al., 1995; Neely, Keefe, & Ross, 1989; Lorch, Balota, & Stamm, 1986; Schwanenflugel & Rey, 1986; see also McRae, Cree, Westmacott, & De Sa, 1999; Massaro, Jones, Lipscomb, & Scholz, 1978, Experiment 1; but see McRae et al., 1999, Experiment 2). Step 2_n is also reported as occurring more reliably through highly overlapping primes and target associates (Perea & Rosa, 2002; Frenck-Mestre & Bueno, 1999; de Mornay Davies, 1998; McRae & Boisvert, 1998; Hodgson, 1991; Lupker, 1984; see Hutchison, 2003 for a discussion). At long SOA, the reported Step 2_n priming effects depend less reliably on overlap (Keefe & Neely, 1990; Smith et al., 1987; Lorch et al., 1986; Favreau & Segalowitz, 1983; Lorch, 1982; but see Grose-Fifer & Deacon, 2004; Neely et al., 1989; Lorch et al., 1986; den Heyer et al., 1985; Becker, 1980; see McRae et al., 1999). It is then important to discriminate between the number n of common associates of two words and the semantic field s of a given word, defined as its number of associates, in addition to the associative strength a. This permits to define general semantic relationships involving combinations of multiple *Step* N_n^s with variable values of N, n, and s.

Priming-like Experiments in Monkeys

Experimental studies on humans provide a rich phenomenology of behavioral effects, but it is hard to infer from these data the mechanisms underlying such phenomena at the neuronal or network levels. Recent electrophysiological experiments on behaving monkeys provide invaluable information on the dynamics at the neuronal level underlying priming-like effects. Pair associate tasks used to probe neuronal correlates of learning of associations between stimuli present striking similarities with human priming protocols. In such tasks, the monkey learns associations between arbitrary visual stimuli. After learning, a typical protocol consists of: first, presentation of a (prime) image A followed, after a delay period in which no information is available on the screen, by the presentation of a (target) image B. Delays involved in these protocols in monkeys are very similar to the long SOAs used in humans (about 1000 msec). To obtain a reward, the monkey is required to hold (or release) a bar if the prime and the target are associates (e.g., Erickson & Desimone, 1999; Rainer, Rao, & Miller, 1999). This is very similar to the go/no-go task used in humans. It reveals priming effects in monkeys and in humans very similar to those revealed by lexical decision

and naming tasks specific to human studies. Another type of task requires the monkey to choose the pair associate to the prime among two presented targets by touching it on the screen (e.g., Naya, Sakai, & Miyashita, 1996; Sakai & Miyashita, 1991). In both tasks, two types of selective neuronal activity are observed during the delay period in both the temporal lobe (inferior temporal and perirhinal cortices) and prefrontal cortex. Some neurons which are selective for a stimulus maintain an elevated firing rate during the delay period following presentation of that stimulus (retrospective activity), as in classical delay match-to-sample tasks (Miyashita, 1988; Miyashita & Chang, 1988; Fuster & Alexander, 1971). Such neurons are widely believed to underlie short-term or working memory of a stimulus. In addition, some neurons also show ''prospective'' activity: Their firing rate increases during the delay period, only when the monkey expects (based on the first stimulus shown) that the preferred stimulus of the corresponding neuron will appear at the end of the delay period (Naya, Yoshida, & Miyashita, 2001, 2003; Naya, Yoshida, Takeda, Fujimichi, & Miyashita, 2003; Yoshida, Naya, & Miyashita, 2003; Erickson & Desimone, 1999; Rainer et al., 1999; Tomita, Ohbayashi, Nakahara, Hasegawa, & Miyashita, 1999; Sakai & Miyashita, 1991; Miyashita, 1988; Miyashita & Chang, 1988; see Fuster, 2001). Hence, the presentation of an item to the monkey activates not only neuronal populations representing the shown stimulus but also neurons representing stimuli that are associated to it. Those populations of neurons seem to correspond to the ''activated units'' postulated by theories of priming in humans. Furthermore, some of these experiments demonstrate priming-like effects on reaction times related to neurons' spike rates (e.g., Erickson & Desimone, 1999) that can predict behavioral data such as reaction time (Roitman & Shadlen, 2002). The striking similarity between pair associate experiments in monkeys and priming experiments in humans lead naturally to the hypothesis that priming effects in humans involve prospective activity of neuronal populations encoding for associates of the prime. Priming effects in humans would involve prospective activity of neuron populations pertaining to a set of associates to the prime, among which the actually presented target, and not only a single associated target as in monkey experiments. Such simultaneous prospective activities of several neuronal populations coding for different objects in memory are reported in studies in monkeys (Wallis & Miller, 2003).

Models of Priming

Theoretical modeling provides a way to bridge the behavioral and cellular levels. Psychologists have long used abstract ''connectionist'' models to account for the variety of priming effects reported in humans. According to the association-based view of priming, semantic memory is assumed to be organized as a semantic network where concepts and features are encoded in a localist way by single nodes (Anderson, 1976, 1983a, 1983b; Collins & Loftus, 1975; Collins & Quillian, 1969). In localist networks, Step 1 and Step 2_n associations of variable direct and indirect strengths would lead to priming effects through automatic spreading of activation from node to node. According to the feature-based view of priming, semantically related concepts would share common semantic features (Cree & McRae, 2003) in distributed networks (McRae & Ross, 2004; Randall, Moss, Rodd, Greer, & Tyler, 2004; Cree, McRae, & McNorgan, 1999; Becker, Moscovitch, Behrmann, & Joordens, 1997; Bullinaria, 1995; Masson, 1995; Plaut, 1995; Moss, Hare, Day, & Tyler, 1994; Sharkey & Sharkey, 1992; Masson, Besner, & Humphreys, 1991) based on attractor network architectures (Hopfield, 1982). In distributed networks, recall of a given concept in memory corresponds to convergence of the network to an attractor state, that is, a stable distributed pattern of activation or inhibition of units coding for features in a localist way. The level of overlap between prime and target features then leads to priming effects through activation/inhibition of features units, without direct associations between concepts but with direct associations between features. Another group of theorists has used more biologically realistic models of cerebral cortex to account for the monkey neurophysiological data (see, e.g., Brunel, 2004; Amit, 1995; Amit, Brunel, & Tsodyks, 1994). These models account both for retrospective activity in working memory (Amit, Bernacchia, & Yakovlev, 2003; Brunel & Wang, 2001; Haarmann & Usher, 2001; Renart, Moreno, de la Rocha, Parga, & Rolls, 2001; Wang, 2001; Amit & Brunel, 1997) and prospective activity in paired associate tasks (Lavigne, 2004; Mongillo, Amit, & Brunel, 2003; Lavigne & Denis, 2001, 2002; Brunel, 1996). However, the wide variety of semantic priming effects in human challenges cortical networks models of biophysically realistic neurons. Here, we show that elaborations of such models that have been successful in reproducing the electrophysiological data in monkeys can also reproduce many types of behavioral findings on priming experiments in humans.

METHODS

We model a local network of an area of association cortex containing cells selective to objects or concepts that show persistent activity following presentation of those objects or concepts. We study a simplified ''rate model'' in which dynamical variables represent average firing rates of populations of neurons which are selective to the same object. We assume for simplicity nonoverlapping populations of excitatory neurons coding for p distinct stimuli (Brunel & Wang, 2001; Amit & Brunel, 1997), shown schematically in Figure 1A.

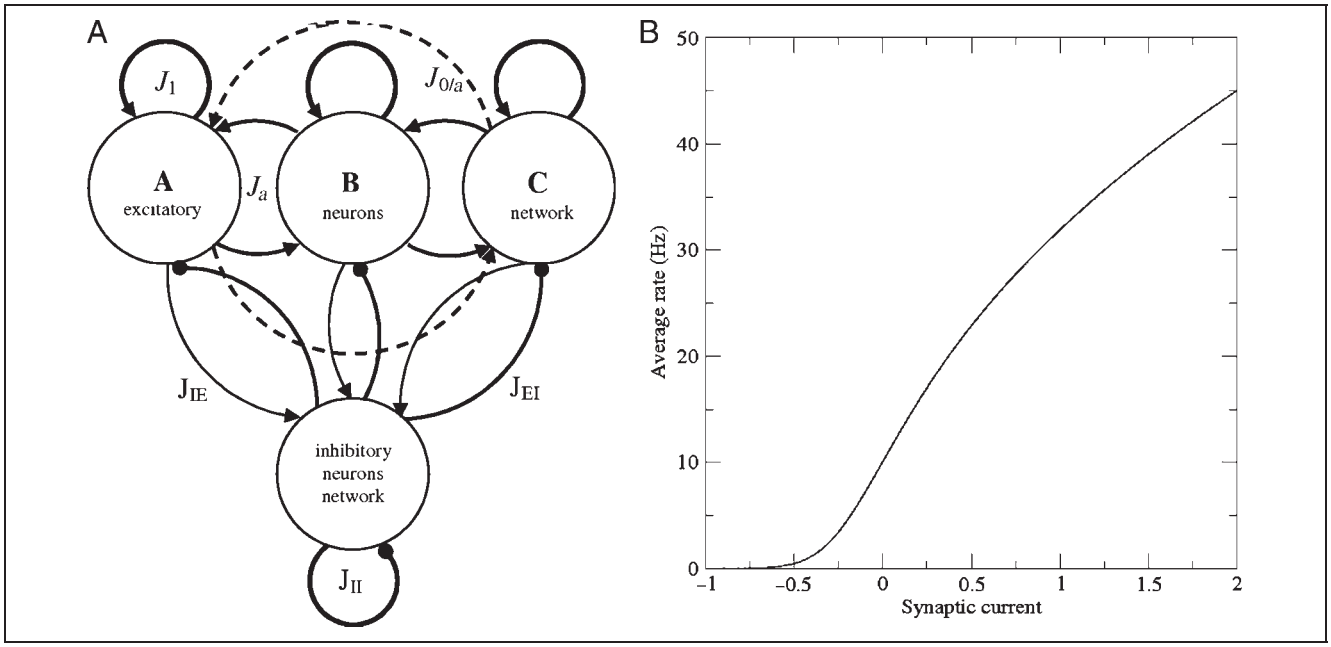

Figure 1. (A) Architecture of the excitatory–inhibitory network: Excitatory neurons are divided in p subpopulations of neurons selective for distinct stimuli. Inhibitory neurons are nonselective. Synaptic strength is indicated by line thickness; precise values of the parameters are given in Table 1. (B) Population *f–I* curve, Equation 3 specifying how the average firing rate of a population of excitatory neurons depends on the average synaptic inputs it receives.

Each population $i = 1,\ldots,p$ is described by an average firing rate whose dynamics is described by a standard Wilson–Cowan type equation (Equation 1):

$$\tau \frac{dv_i}{dt} = -v_i + \Phi\left[\frac{1}{p}\sum_{j=1}^{p} J_{ij}v_j + I_i^{ext} + I_i^{sel} - I_{inh}\right] \qquad (1)$$

where τ represents the time constant of firing rate dynamics, J_{ij} represents the total synaptic strength from population j to population i, I_i^{ext} represents the external inputs to population i, I_i^{sel} represents the selective inputs to population i, and I_{inh} represents a global inhibitory current regulating the activity of all excitatory populations, which is here for the sake of simplicity proportional to the average activity of excitatory populations (Equation 2).

$$I_{inh} = \frac{J_I}{p}\sum_{j=1}^{p} v_j \qquad (2)$$

where J_I represents the strength of inhibition. This description of inhibition corresponds to a scenario in which inhibitory neurons have linear input–output relationship and time constants that are much faster than excitatory neurons. The external input in the absence of external stimuli is chosen such that all populations in the network have some prescribed level of background activity $v_0 = 5$ Hz. The synaptic strength between two excitatory populations can take three values. Synaptic efficacy from a population to itself is $J_{ij} = J_1$. Synaptic efficacy between two populations that are selective to two unrelated items is $J_{ij} = J_0$, where $J_0 < J_1$. Finally, the synaptic efficacy between two populations that are selective to two associated items is $J_{ij} = J_a = J_0 + a(J_1 - J_0)$, where $0 < a < 1$ represents the strength of the association (Mongillo et al., 2003; Brunel, 1996). J_1, J_0, and J_I are chosen so that both a nonselective background state and selective attractors corresponding to either a single item (for $a = 0$) or groups of items (for $a > 0$) are present in the network, and J_0 is calculated as function of a to guarantee a constant level of spontaneous activity of 5 Hz independently of the value of a. Finally, Φ describes the static current-to-rate transfer function (or *f–I* curve) (Equation 3). Here, we take this function to be the transfer function obtained analytically for quadratic integrate-and-fire neurons in presence of background noise, which is expected to be qualitatively (and even quantitatively in some conditions) similar to the one of cortical excitatory neurons (Brunel & Latham, 2003). The function Φ is given by

$$\Phi(I) = \frac{1}{\sqrt{\pi}\tau_m}\left[\int_{-\infty}^{\infty} dz \exp\left[-Iz^2 - \sigma^4 z^6/48\right]\right]^{-1} \qquad (3)$$

where $\sigma = 0.5$ and τ_m is the membrane time constant. The transfer function is shown in Figure 1B.

We consider the dynamic properties of the present model according to several scenarios with different semantic relationships between $p = 100$ items stored in

memory, organized in p_g groups of p_i items ($p = p_i p_g$). In all scenarios, the synaptic matrix can have three different values, depending on the relationship between items encoded by the pre- and postsynaptic populations. The diagonal term (connections between neurons coding for the same item) is J_1. Connections between populations coding for unrelated items have strength J_0. Finally, connections between populations coding for related items have strength $J_a = J_0 + a(J_1 - J_0)$ where a measures associative strength. The matrix describing the network structure involves $p = 100$ items. For the sake of simplicity, we show below for each scenario a subset of the synaptic matrix with $p = 6$, $p_g = 2$, and $p_i = 3$, where we take $J_1 = 1$ and $J_0 = 0$.

A. Nonoverlapping Homogeneous Groups

In this scenario, items are all associated with each other within a group (Parga & Rolls, 1998; Brunel, 1996). In the case $p = 6$, $p_g = 2$, and $p_i = 3$, the synaptic matrix has the following form:

$$M = \begin{vmatrix} 1 & a & a & 0 & 0 & 0 \\ a & 1 & a & 0 & 0 & 0 \\ a & a & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & a & a \\ 0 & 0 & 0 & a & 1 & a \\ 0 & 0 & 0 & a & a & 1 \end{vmatrix} \quad (4)$$

In the simulations, we use $p = 100$, $p_g = 10$, and $p_i = 10$. Hence, for simulations each item is associated to $p_i - 1 = 9$ others items (1–2, 1–3, 2–3 in the matrix shown above). The prime (1) has nine associates and related prime and target (1 and 2) have eight common associates. Related prime and target share a combination of direct Step 1^9 (1–2) and indirect Step 2_8 (1–3–2) relations. In this scenario, there are only two types of relationships between items—they either belong to the same group and then are related together both directly (Step 1) and indirectly (Step 2), or they are unrelated.

B. Nonoverlapping Groups with Prototypes

In this scenario, items belong to groups of p_i items in which one item (the ''prototype'') is directly associated to all of the others, which are not directly associated with each other (Brunel, Carusi, & Fusi, 1998; Parga & Virasoro, 1986). In the case $p = 6$, $p_g = 2$, and $p_i = 3$, the synaptic matrix is

$$M = \begin{vmatrix} 1 & a & a & 0 & 0 & 0 \\ a & 1 & 0 & 0 & 0 & 0 \\ a & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & a & a \\ 0 & 0 & 0 & a & 1 & 0 \\ 0 & 0 & 0 & a & 0 & 1 \end{vmatrix} \quad (5)$$

In the simulations, we use $p = 100$, $p_g = 10$, and $p_i = 10$. Simulations with Scenario B enable to discriminate between Step 1^1 (2–1), Step 1^9 relation (1–2), and Step 2_1 relation between items having one common associate (2–1–3). In this scenario, there are three types of relationships between items: (i) direct (Step 1) relationship between a group member and the ''prototype'' of the group; (ii) indirect (Step 2) relationship between two group members (through the prototype); (iii) no relationships between members of two different groups.

C. Indirect Relationships through Various Numbers of Associates

We also use a synaptic matrix in which items can be associated indirectly through more than one indirect associate (Brunel, 1996; Amit et al., 1994; Griniasty, Tsodyks, & Amit, 1993). In this matrix, each item is again associated with p_i other items, the synaptic structure is as follows (shown in the case $p = 6$ and $p_i = 2$):

$$M = \begin{vmatrix} 1 & a & 0 & 0 & 0 & a \\ a & 1 & a & 0 & 0 & 0 \\ 0 & a & 1 & a & 0 & 0 \\ 0 & 0 & a & 1 & a & 0 \\ 0 & 0 & 0 & a & 1 & a \\ a & 0 & 0 & 0 & a & 1 \end{vmatrix} \quad (6)$$

Again, we use in simulations $p = 100$ and $p_i = 10$. For such parameters, there exist Step 1^{10} relations (e.g., 1–2), Step 2_n (where $0 < n < 6$) relations (e.g., 1–5–9) and Step 3, 4,... relations (e.g., 1–5–9–13) (Table 1).

Protocol

Experimental protocols used in humans were simulated in the model to investigate how the wealth of priming

Table 1. Parameters of the Model

p	Number of selective populations	100
τ	Time constant of rate dynamics	10 msec
J_E	Average excitatory synaptic strength	3
J_S	Strength of synaptic potentiation	3.65
J_1	Intrapopulation efficacy	$J_E + J_S$
a	Association strength between associated items	$\frac{J_a - J_0}{J_1 - J_0}$; 0.001–0.02
J_a	Synaptic efficacy between populations representing associated items	$J_E + J_S \frac{a(p-p_i+1)-1}{(1-a)(p_i-1)+p-p_i}$
J_0	Synaptic efficacy between populations representing nonassociated items	$J_E - J_S \frac{a(p_i-1)+1}{(1-a)(p_i-1)+p-p_i}$
J_I	Inhibitory synaptic efficacy	J_E
v^θ	Threshold for reaction time	27 Hz
v_S	Spontaneous activity	5 Hz
I_{ext}	Nonselective external currents	set in order to obtain v_S = 5 Hz
I_{sel}	Selective external currents	0.15

effects emerges from the dynamics of these interconnected populations. Priming effects were tested according to the following experimental protocol: first 50 msec without any input; then the prime was presented for t_1 = 200 msec, followed by variable delay periods t_d with no selective input (interstimuli interval). The (variable) SOA was defined as SOA = t_1 + t_d. Finally, the target was presented for 200 msec, followed by 50 msec with no input before the end of trial (Figure 2C).

In the model, a trial begins with the network in a state of spontaneous activity. When the prime is presented, the corresponding neuronal population reaches an elevated activity (''visual response'')—after prime removal, the excitatory connectivity is strong enough so that these neurons do not come back to spontaneous activity, but rather exhibit elevated (retrospective) persistent activity. The elevated activity of such neurons leads, in turn, to activation of populations of neurons coding for related stimuli. Hence, at the time of the presentation of the target, neuronal populations that code for associated Step 1 and Step 2 targets exhibit increasing firing rates corresponding to prospective activity. Recognition or response times to a given item are usually computed as proportional to its level of activation in memory (Randall et al., 2004; Bullinaria, 1995; Masson, 1995; Plaut, 1995; Masson et al., 1991). Electrophysiological studies have reported that spike rates of neurons coding for a given response are correlated to response times (Roitman & Shadlen, 2002). Based on these experimental data, many modeling approaches take the reaction time to be the time at which the mean spike rate of a population of neurons reaches a prescribed threshold (Wong & Wang, 2006; Wang, 2002), similar to classical diffusion models of reaction time (Ratcliff, 1978, 2006; Ratcliff, Gomez, & McKoon, 2004). Then, when a target is presented to the network, its recognition time T^θ is the time elapsed from target onset to the time at which the mean firing rate of the corresponding population first crosses a threshold v^θ. For a given target, T^θ depends on the level of prospective activity of the neurons population coding for this target at target onset, itself assumed to depend on the synaptic matrix and preceding prime. The target can follow a related (R), unrelated (U), or no (neutral, N) prime, leading to specific recognition times $T_R{}^\theta$, $T_U{}^\theta$, and $T_N{}^\theta$, respectively. These response times enable to quantify the activatory (Equation 7) and inhibitory (Equation 8) components of priming effects (Equation 9):

$$\mathrm{PE}_{\mathrm{act}} = T_N^\theta - T_R^\theta \tag{7}$$

$$\mathrm{PE}_{\mathrm{inh}} = T_N^\theta - T_U^\theta \tag{8}$$

with the global priming effect calculated as:

$$\mathrm{PE} = \mathrm{PE}_{\mathrm{act}}\mathrm{PE}_{\mathrm{inh}} \tag{9}$$

RESULTS

When a stimulus is shown to the model network, it elicits a visual response during its presentation, followed by persistent activity of the population coding for that stimulus (see Figure 3A). This activity elicits an increase in the firing rate of the populations associated to the stimulus (see Figure 3A), similar to prospective activity observed in monkey experiments, because of the

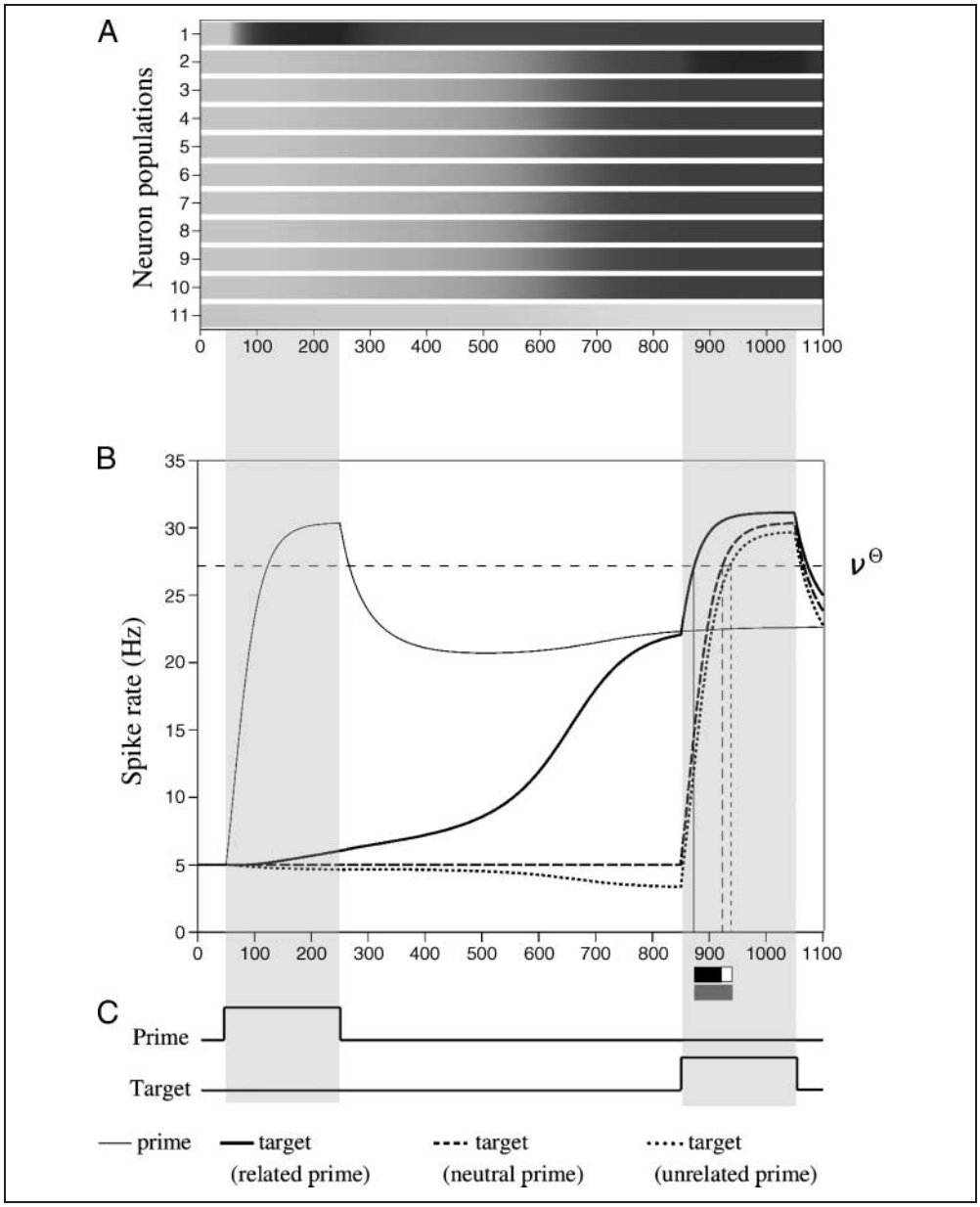

Figure 2. (A) Spike rates of 11 neurons populations (for clarity, Items 12 to 100 and inhibitory population are not displayed) as a function of time for $a = 0.005$, according to Matrix 4 and to Protocol C: Spontaneous activity for 50 msec, prime input for 200 msec (gray area), variable delay or interstimuli interval (the case of an ISI of 150 msec is displayed) defining a variable SOA (prime duration + ISI), target input for 200 msec (gray area) and posttarget delay for 50 msec. Spike rates are indicated by gray levels from 0 Hz (white) to 35 Hz (black). Prime is encoded by Population 1, associated items are encoded by Populations 2 to 10, target is encoded by Population 2, and a representative nonassociated item is encoded by Population 11. (B) Spike rates of neurons populations coding for a prime (Item 1: thin solid curve) in different trials where the target followed a related prime (Target Item 2 in associated condition: thick solid curve), no prime (e.g., Target Item 2 in neutral condition: thick dashed curve), or an unrelated prime (Target Item 11 in nonassociated condition: thick dotted curve). Vertical lines indicate response time T^{θ} from target onset for target population activity to reach threshold $v^{\theta} = 27$ Hz (horizontal thin dashed line). Horizontal black, white, and gray bars indicate the magnitude of activatory ($PE_{act} = 49$ msec), inhibitory ($PE_{inh} = -13$ msec), and global ($PE_{main} = 62$ msec) priming effects, calculated as the difference between reaction times in the related ($T^{\theta}_{R} = 73$ msec), neutral ($T^{\theta}_{N} = 122$ msec), and unrelated ($T^{\theta}_{U} = 135$ msec) conditions (see Equations 7, 8, and 9).

increased connection strength (as measured by the parameter a) between these populations. Hence, when the second stimulus shown is associated to the first through Step 1 and/or Step 2 associations, the corresponding population has initially a higher firing rate than if the first stimulus had not been shown. It then reaches the threshold for recognition faster: The network exhibits a large activatory priming effect (Figure 2B) of magnitude quantitatively similar to priming reported in human (i.e., tens of milliseconds). This basic effect is consistent with reduced reaction times reported in human literature (see, e.g., Hutchison, 2003; Neely, 1991), prospective

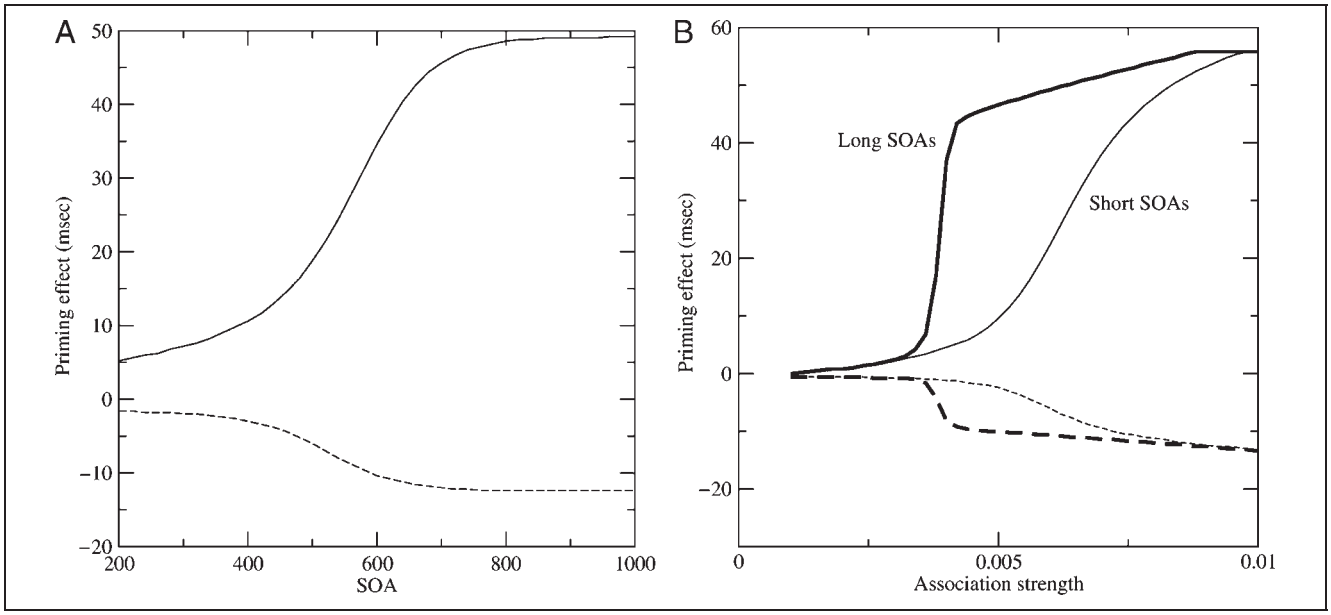

Figure 3. (A) Priming as a function of SOA (Matrix 4). The association strength is $a = 0.005$. Full curve: Activatory component of priming effect for associated stimulus (Equation 7). Dashed curve: Inhibitory component of priming effect for unrelated stimulus (Equation 8). (B) Priming as a function of association strength at short SOAs (400 msec; thin curves) and long SOAs (800 msec; thick curves). Full curves: Activatory component (Equation 7). Dashed curves: Inhibitory component (Equation 8).

activity observed in neurophysiological experiments on monkeys (e.g., Sakai & Miyashita, 1991) and previous modeling studies (e.g., Mongillo et al., 2003).

Priming Effects as a Function of SOA and Association Strength

In Scenario A (Matrix 4), a given target is related to a prime through combined direct Step 1^9 and indirect Step 2_8 associations. Figure 3A shows that the magnitude of priming effects depends in a pronounced way on the SOA (Figure 3A) and on the parameter a of association strength (Figure 3B). Conversely, if an unrelated item is shown first (a stimulus belonging to another category), the corresponding population starts with a firing rate that is below its baseline firing rate because of the global inhibition. Hence, there is an inhibitory priming effect that is smaller than activatory priming, but increases with SOA (Figure 3A) and whose magnitude again depends on a (Figure 3B).

The fact that activatory effects are larger than inhibitory ones is in accordance with the activation dominance of priming reported in the literature in humans (Smith et al., 1987; den Heyer et al., 1985; Favreau & Segalowitz, 1983; Neely, 1977). Furthermore, the model predicts that the effect becomes only weakly dependent on association strength at long SOAs.

Step 1 vs. Step 2 Priming Effects

The next question we investigate in Scenario B is whether the magnitude of priming effects depends on step, that is, the nature of association between prime and target. We do this by comparing Step 1^1, Step 1^9, and Step 2_1 priming. Scenario B extends simulations by Deco and Rolls (2005) on the processing of sequences of three associated items (i.e., Step 2_1 priming). However, in their study, only one item was activated at a time so the prime was deactivated when the target was activated, and priming could then not depend on the simultaneous activation of several associates in working memory to distinguish between different types of step. The present results show that all types of associations lead to priming when processed following an associated prime compared to when following an unrelated prime. The magnitude of priming increases with SOA (Figure 4A) and associative strength a (Figure 4B). The asymptotic magnitude of priming at long SOA decreases with number of associates, Step 1^1 priming being stronger than Step 1^9 priming that involves the activation of more associates. This is in accordance with findings of reduced priming when memory load is increased (Sabb, Bilder, Chou, & Bookheimer, 2007), suggesting common neural system resources for priming and working memory. The model shows that such result can be due to the fact that items with more associates activated in memory lead to increased inhibition that, in turn, leads to decreased priming of such associates. Priming magnitude also depends on the nature (direct or indirect) of the association, Step 1 priming being stronger than Step 2 priming (Figure 4A). The fact that Step 1 effects are larger than Step 2 can be easily understood that in Step2, target activation has to go through the activation of common associates of both items. This has the consequence that Step 2 priming is

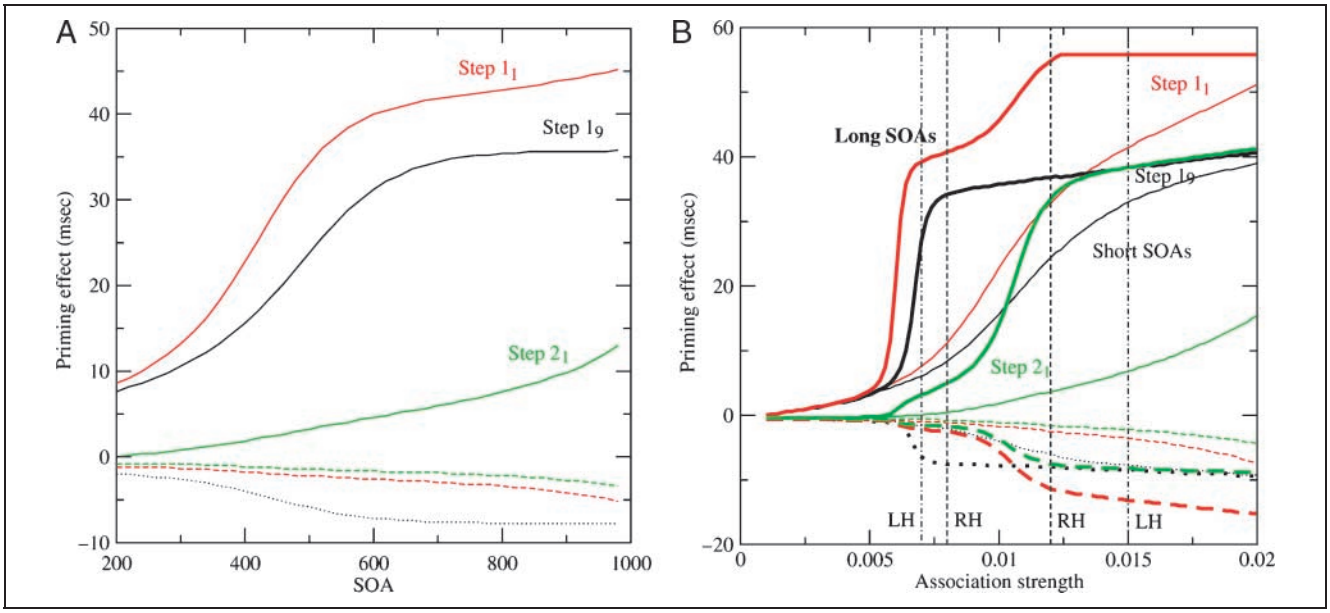

Figure 4. (A) Priming as a function of step and SOA for $a = 0.01$ (Matrix 5). Red curves: Step 1_1; black curves: Step 1_9; green curves: Step 2_1. Full curves: Activatory component. Dashed curves: Inhibitory component. (B) Priming as a function of step and association strength at short SOAs (200 msec; thin curves) and long SOAs (800 msec; thick curves). Red curves: Step 1_1; black curves: Step 1_9; green curves: Step 2_1. Full curves: Activatory component. Dashed curves: Inhibitory component. Vertical dashed lines correspond to possible values of a accounting for priming effects in the left (LH) and right (RH) hemispheres ($a = 0.007$ and $a = 0.015$ for weak and strong associations in the LH; $a = 0.008$ and $a = 0.012$ for weak and strong associations in the RH).

much weaker at short SOA, and that it increases more slowly with SOA. In addition, when Step 2 target becomes activated, the feedback inhibition is stronger because both the prime and Step 1 target are already activated. This accounts for lower asymptotic Step 2 priming at long SOA. Step 2 priming arises at long SOA for all values of a, and at short SOA for high values of a only (Figure 4B). When the prime, Step 1, and Step 2 associates are sufficiently strongly associated and the SOA is long enough, the three items can activate each other strongly enough to further increase target activation (e.g., the timed relation between the sharp transitions visible on

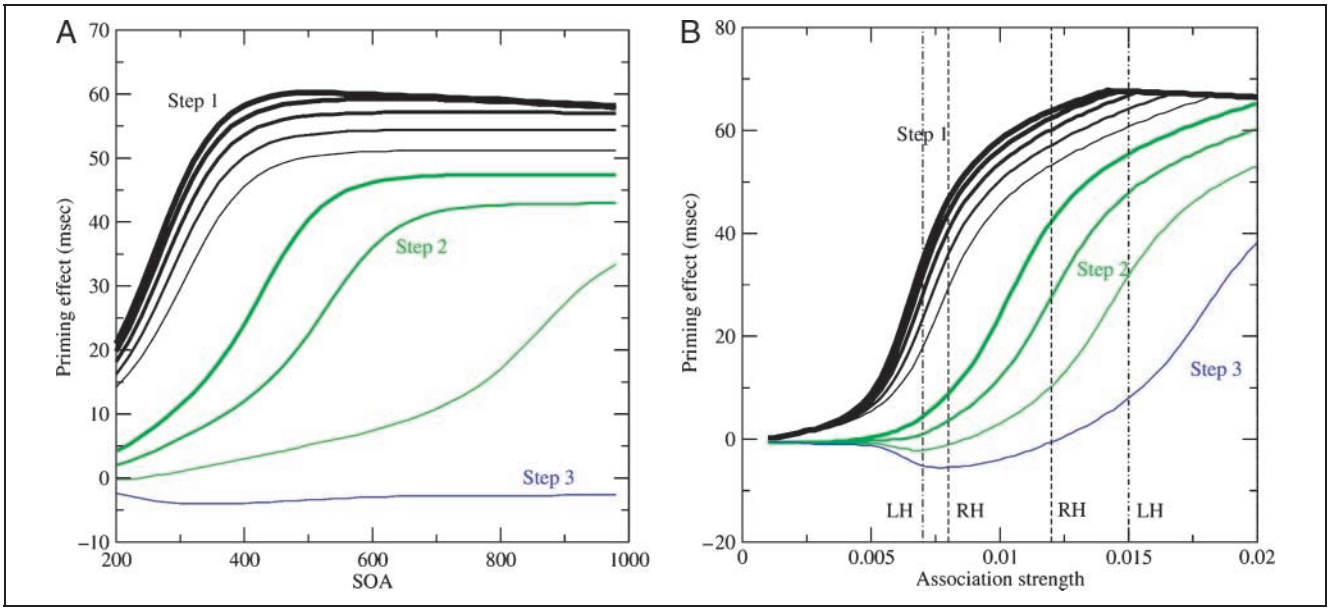

Figure 5. (A) Step 2_n priming as a function of n and SOA for $a = 0.01$ (Matrix 6). From strongest to weakest effects (black, green, and blue curves, respectively): Step 1_{10} + Step $2_{8,7,6,5,4}$ (line thickness decreases with decreasing number of Step 2 associates); Step $2_{3,2,1}$ and Step 3 relations. (B) Step 2_n priming as a function of n and a at 400 msec SOAs. From strongest to weakest effects: Step 1_{10} + Step $2_{8,7,6,5,4}$; Step $2_{3,2,1}$ and Step 3 relations. Vertical dashed lines correspond to possible values of a accounting for effects in the left (LH) and right (RH) hemispheres (same values as in Figure 4).

the red and green curves for $a = 0.01$ and long SOAs). At short SOAs, even though Step 2 effects are weak they are, nonetheless, present, and therefore the present model accounts for the possibility reported in the experimental literature in humans of an early onset of Step 2 priming (Yochim, Kender, Abeare, Gustafson, & Whitman, 2005; Kiefer et al., 1998; Richards & Chiarello, 1995).

Divided visual field experiments have pointed to the importance of the direct associative strength on Step 1 priming in the right visual field–left hemisphere (RVF–LH) and in the left visual field–right hemisphere (LVF–RH) (see Chiarello et al., 2003, for a review). At short SOA, Step 1 priming is reported when both hemispheres are involved (Frishkoff, 2007; Hutchinson, Whitman, Abeare, & Raiter, 2003), or when the RVF–LH is primarily involved (Bouaffre & Faita-Ainseba, 2007) more reliably on strong associates (Yochim et al., 2005; Coney, 2002; Abernethy & Coney, 1993; Nakagawa, 1991). Trends for priming of strong associates are reported when the LVF–RH is primarily involved (Hutchinson et al., 2003; but see Nakagawa, 1991). Priming of weak associates at short SOA is reported when primes are presented to the LVF–RH (Hutchinson et al., 2003), and weaker than priming of strong associates when both hemispheres are involved (Frishkoff, 2007; Coney, 2002). Turning to long SOAs, Step 1 priming effects are reported to involve both hemispheres (Khateb et al., 2003) stronger on strong associates (Frishkoff, 2007; Coney, 2002; Abernethy & Coney, 1993; Nakagawa, 1991; but see Hutchinson et al., 2003 for an absence of effect when both prime and targets are presented to the RVF–LH). At long SOA, priming of weak associates is reported when solely the LVF–RH is involved (Hutchinson et al., 2003), weaker than priming of strong associates when reported in both hemispheres (Frishkoff, 2007; Coney, 2002). Regarding Step 2 priming, it is reported at short SOA when both hemispheres are involved, although smaller than Step 1 priming (Yochim et al., 2005; Kiefer et al., 1998; see Richards & Chiarello, 1995 for right hemisphere effects when primes are centrally presented) and less reliably when only one hemisphere is primarily involved (Yochim et al., 2005). At long SOAs, Step 2 priming is reported as equivalent to Step 1 priming from primes in the LVF–RH, and weaker from primes in the RVF–LH (Yochim et al., 2005; Richards & Chiarello, 1995). Taken as a whole, these hemispheric differences in semantic priming effects have been described as ''fine'' or focused semantic coding in the left hemisphere, in which strong Step 1 associates are activated, and ''coarse'' or extended semantic coding in the right hemisphere, in which strong and weak Step 1 and Step 2 associates are activated (Beeman, Bowden, & Gernsbacher, 2000; Beeman, Friedman, Grafman, & Perez, 1994; see Chiarello et al., 2003; Beeman & Chiarello, 1998).

Differential hemispheric priming of weak and strong associations can be accounted for in our model through different values of the association strength a: extreme values of a in the left hemisphere ($a = 0.007$ and $a = 0.015$), and intermediate values of a in the right hemisphere ($a = 0.008$ and $a = 0.012$) (Figure 4B). Results obtained with such values of a in the case of Step 1 priming (black curves) are consistent with effects reported in the literature when one hemisphere is primarily addressed. At short SOA, Step 1 priming is larger in the left hemisphere than in the right hemisphere on strong associates, and larger in the right hemisphere than in the left hemisphere on weak associates. At long SOA, Step 1 priming arises in both hemispheres on strong associates, and is stronger in the right hemisphere on weak associates. Relative to Step 2 priming, the mean field model exhibits Step 2 priming (green curves) in the right hemispheres for all association strengths, and in the left hemisphere for strong associations only. This supports the hypothesis that differential synaptic potentiation can account for differential semantic coding in the hemispheres. Although association strength has not been manipulated in experiments testing for Step 2 priming, the model predicts that the respective increases of Step 1 and Step 2 priming with SOA should depend markedly on a.

Step 2_n Priming Effects

Scenario C (Matrix 6) permits us to investigate the question of how the number of shared associates influences the magnitude of Step 2_n priming. Results show increasing magnitude of priming effects with SOA for Step 1 as well as Step 2. However, priming effects depend strongly on the type of prime–target relationship, especially at short SOAs: They are much stronger for Step 1 than Step 2; and they are also stronger when the number of common associates n is large (Figure 5A). At long SOAs, priming effects reach a maximum inversely proportional to the number of steps, and proportional to n for Step 2_n relations (McRae et al., 1999; see Fischler, 1977). Indeed, n determines the amount of activation received by a target—The larger the n, the larger its prospective activity. These results are in accordance with the literature on Step 2_n priming (Sanchez-Casas et al., 2006; Grose-Fifer & Deacon, 2004). In addition, for all types of steps, the magnitude of priming effects is proportional to the association strength a (Figure 5B), whose effect is larger at short than at long SOAs (Figure 5A). Step 3 associates can also be primed (McNamara, 1992) for high values of a, but remote associates are reported as inhibited at long SOAs in the left hemisphere (see Ince & Christman, 2002; Nakagawa, 1991). The model shows that for low values of a both Step 2 and Step 3 remote associates can be inhibited, which would correspond to left hemisphere processing (Figure 5B, thin green and blue curves). This is due to the fact that Step 1 items are directly activated by the prime and reach enough activation to trigger inhibitory

feedback. The model predicts the possibility for inhibition of Step 3 associates for low values of a corresponding to right hemisphere processing. To our knowledge, step and association strength have not been cross-manipulated in the experimental literature. Their synergistic effects can therefore be considered as an experimental prediction of the model.

Divided visual field experiments have shown that the effect of overlap (number of shared associates) on Step 2_n priming strongly depends on the cerebral hemisphere primarily involved. At short SOAs, Step 2_n priming occurs more reliably in the RVF–LH (Korsnes & Magnussen, 2007; Collins, 1999; Koivisto, 1997, 1998; Abernethy & Coney, 1990, 1996) than in the LVF–RH (Bouaffre & Faita-Ainseba, 2007; Koivisto, 1998), for which it is reported between strongly but not weakly overlapping co-exemplars (Grose-Fifer & Deacon, 2004). Step 2_n priming is also reported at long SOAs (Rossell, Price, & Nobre, 2003; Hines, Czerwinski, Sawyer, & Dwyer, 1986; Flores d'Arcais, Schreuder, & Glazenborg, 1985; Lupker, 1984; Seidenberg, Waters, Sanders, & Langer, 1984; Huttenlocher & Kubicek, 1983; see also McKoon & Ratcliff, 1992), the effect being larger on strongly than on weakly overlapping co-exemplars (McRae & Boisvert, 1998; Hines et al., 1986). It is reported in both the LVF–RH (Collins, 1999; Koivisto & Laine, 1999; Koivisto, 1997; Chiarello & Richards, 1992; Chiarello, Richards, & Pollock, 1992; Chiarello, Burgess, Richards, & Pollock, 1990; see Shears & Chiarello, 2003) and the RVF–LH (Khateb et al., 2003; Shears & Chiarello, 2003; Koivisto & Hamalainen, 2002; Koivisto & Revonsuo, 2000; Koivisto & Laine, 1999; Koivisto, 1998; Abernethy & Coney, 1996; Chiarello et al., 1992), for which a tendency for priming effects between strongly overlapping co-exemplars only is reported (Chiarello & Richards, 1992).

Differential Step 2_n priming effects in the two hemispheres can be accounted for by the model in Scenario C through different values of a in the two hemispheres in a similar way as in Scenario B (compare Figures 4B and 5B). Experimental studies testing for the effect of overlap n in humans do not manipulate the association strength a. However, the model predicts that the effect of n highly depends on a. Step 2_n priming effects are stronger in the LVF–RH for weak values of a and stronger in the RVF–LH for strong values of a (Figure 5B). Differences in priming effects of strong and weak associates decrease with SOA (Figure 5A). The experimental literature also reports that Step 2_n priming is stronger when combined with Step 1 priming than when involved solely (see Lucas, 2000). Such associative boost is also reported as a necessary condition for Step 2_n priming to occur (Abad, Noguera, & Ortells, 2003; Williams, 1996; Shelton & Martin, 1992). Association strength would then be a critical parameter influencing combined Step 1 and Step 2_n priming effects, reported in both hemispheres at short and long SOAs (Audet, Driessen, & Burgess, 1998; Chiarello et al., 1992), although later in the LVF–RH (Chiarello et al., 1992). Simulations results show that both cases are possible depending on SOA (Compare green and black curves in Figure 5A) and on the value of association strength a (compare again green and black curves in Figure 5B). For strong values of a, Step 2_n priming occurs solely and increases when combined with Step 1 priming. For weak values of a, Step 2_n priming does not occur solely and requires combination with Step 1 priming.

DISCUSSION

The present article shows that a cortical network model that reproduces electrophysiological data in monkeys can account, qualitatively and also quantitatively, for priming effects reported in humans. The novelty of the model is to account for priming effects in humans in terms of parameters (like the time constant of firing rate dynamics or the *f–I* curve) that can be related to the biophysics of cortical neurons. Priming effects emerge from the dynamics of simultaneously activated and interacting neuronal populations coding items stored in memory, which are shaped by the synaptic structure that encodes the semantic relationships between the items. The mathematical model presented here accounts for priming effects as a function of the level of prospective activity of neuronal populations, influencing the response time at which its activity reaches a given threshold. Priming effects are due to activation of the target-related neurons by prime-related neurons after presentation of the prime, similar to the observed prospective activity in monkey experiments. The results shown in this article have been obtained with a simple rate model. These results are robust to changes in parameters of the model, provided synaptic connectivity is chosen such as both spontaneous and selective states are stable. The rate model is clearly an oversimplification of the dynamics of populations of real neurons, but it has been shown in some instances to reproduce qualitatively the dynamics of networks of more realistic Hodgkin–Huxley neurons (see e.g., Roxin, Brunel, & Hansel, 2005). Preliminary simulations with networks of leaky integrate-and-fire neurons show qualitatively similar results (Lavigne & Darmon, 2008). The choice of three simple matrices permitted to clearly analyze and disentangle the various types of priming effects. It was not a necessary condition for the effects to arise for our results are robust to the details of the synaptic matrix: For instance, the difference in priming effects between direct and indirect relationships are qualitatively similar in Matrices B and C (Compare Figures 5 and 6). The robustness of priming effects to local changes in the synaptic matrix is also supported by a model of integrate and fire neurons using a single ''unified'' matrix encoding various types of relationships (Lavigne & Darmon, 2008). Then, Matrices A, B, and C can be considered as local synaptic structures encoding ''local'' semantic fields that

are part of a larger matrix encoding a whole set of semantic fields. A local semantic field would be defined by a set of items more associated together than with other items. Such heterogeneous connectivity guaranties that when a local structure is involved in a priming effect, that is, when the corresponding neuron populations are activated at a level above spontaneous activity, corresponding to visual response, retrospective activity, and prospective activity, the resulting increase in inhibitory feedback prevents runaway excitation to propagate to remote local fields. Remote neuron populations are then weakly activated at the level of spontaneous activity or even inhibited (see Figure 5) and do not significantly modify ongoing priming effects.

The cortical network model exhibits working memory activation of simultaneous neuronal populations in stable attractor states (Lavigne, 2004; Amit et al., 2003; Haarmann & Usher, 2001; Brunel, 1996), depending on the ratio of activation/inhibition received by activated neuronal populations (Brunel, 1996). A working memory capacity of several items (Cowan, 2001; Luck & Vogel, 1997) is necessary for step priming involving a prime, several step associates, and a target. Prospective activity of items associated to a prime stimulus has been reported in several areas: in prefrontal (Rainer et al., 1999), inferotemporal and perirhinal cortices (Naya, Yoshida, & Miyashita, 2003; Naya, Yoshida, Takeda, et al., 2003; Yoshida et al., 2003; Naya et al., 2001; Erickson & Desimone, 1999; Sakai & Miyashita, 1991; see Buckley & Gaffan, 1998a, 1998b; Murray, Baxter, & Gaffan, 1998). Likewise, semantic processing in humans has been observed in frontal cortex (Gough, Nobre, & Devlin, 2005; Khateb et al., 2003) and in the anterior temporal lobe (Mummery, Shallice, & Price, 1999; Nobre & McCarthy, 1995; Nobre, Allison, & McCarthy, 1994; see Henson, 2003). This suggests that the neuronal substrate for priming in humans involves widespread cortical areas including prefrontal and temporal networks.

Priming Dynamics

The cortical network model exhibits priming effects whose magnitudes are quantitatively similar to the ones reported in human studies. These priming effects are calculated on the basis of threshold crossing times of neurons spike rates whose amplitude is similar to the one reported in monkey studies. The dynamics of priming effects emerge from those of neurons populations determined by a biologically realistic *I–f* (current-to-frequency) transfer function. This enables the model to account simultaneously for three dynamical features reported in the literature: effects arising at short SOA (Neely, 1976, 1977; see Valdes, Catena, & Mari-Beffa, 2005, for a discussion); effects that are sustained at long SOAs (Deacon et al., 1999; Stern, Prather, Swinney, & Zurif, 1991; Neely, 1977); and effects arising at long SOAs only (Deacon et al., 1999; Neely, 1976, 1977; see Neely, 1991 for a review). Such effects are usually reported to depend on qualitatively different, although nonexclusive, time-dependent processes of rapid automatic activation producing facilitation, followed by slower inhibition producing selection (Rossell et al., 2003; Rossell, Bullmore, Williams, & David, 2001; Mummery et al., 1999; Neely, 1977, 1991; Keefe & Neely, 1990; Neely et al., 1989; see Posner & Snyder, 1975) as a function of an SOA boundary estimated in humans at about 300 msec (Hutchison, Neely, & Johnson, 2001; see also Burke, White, & Diaz, 1987). The present results show that short SOAs lead to rapid activation of directly related items, together with rapid inhibitory regulation of the level of activation. At long SOAs, already activated items sustain their activation, and indirectly and/or weakly related items become activated on a slower time scale. The increasing global level of excitation at long SOAs leads to a slowly increasing inhibitory feedback induced by coactivation of several populations. Interestingly, this effect determines a limit to the number of coactive populations (Amit et al., 2003) and has been proposed to explain the short-term memory capacity limit (Haarmann & Usher, 2001). In the present model, slowly increasing inhibitory feedback is able to reduce the level of activation of already activated populations, and to inhibit neuronal populations encoding for items not related to the prime (when compared to a baseline in absence of prime). This model can then account for priming effects as a function association strength and type of relation (direct or indirect) on the basis of combined excitation and inhibition that are both automatic and rapid at the neuronal level. Such neuronal mechanisms subtend rapid facilitatory effects between associated items and slower inhibitory effects of leading to deactivation or infrabaseline inhibition of items at the network level, depending on the synaptic structure. In the model described here (see also Plaut & Booth, 2000), slow inhibition does not involve controlled processes such as proposed at long SOAs (e.g., Neely, 1977, 1991), although controlled processes should be taken into account in further development of the model to account for more complex priming effects.

There is a debate in the literature between prelexical and postlexical accounts of priming effects. Prelexical activation of the target by the prime, that is, occurring before presentation of the target, is supported by neurophysiological data in monkeys reporting prospective activity of one (e.g., Sakai & Miyashita, 1991; Miyashita, 1988) to several (Wallis & Miller, 2003) associated objects in memory, and by behavioral data in humans from studies on subliminal priming where either the prime (e.g., Draine & Greenwald, 1998; Greenwald, Draine, & Abrams, 1996) or the target (e.g., Lavigne, Vitu, & d'Ydewalle, 2000) is processed without awareness. It would then not allow conscious postlexical semantic matching processes between the prime and the target, that is, occurring after presentation of the target (Dosher & Rosedale, 1989; Neely et al., 1989; Ratcliff & McKoon,

1988; see Lavigne & Lavigne, 2000; Neely, 1991 for discussions). In the present study, prelexical semantic priming is based on prospective activity of neurons coding for a target that is not yet presented. From a theoretical point of view, this corresponds to an anticipatory activation of concepts in memory on the basis of information (i.e., the prime) actually presented and previously processed. Such anticipations can be considered as probabilistic in the sense that the amount of prelexical activity depends on synaptic strength between populations coding for the prime and the target, and that synaptic strength is a learned parameter depending on the probability of occurrence of the prime and the target close in a time sequence. The benefit of prospective activity could then make the system reading for perceptive processing or motor response as visible on shortened response times on anticipated targets (e.g., Lavigne, 2004). Although different, pre- and postlexical processes are not mutually exclusive. The network model proposed here accounts for a variety of semantic priming effects mainly on the basis of prelexical processes that involve prospective activity of several neurons populations coding for several associates to the prime instead of only one population coding for a single associated target as in monkey studies. In addition, postlexical processes are also involved automatically as backward effects of the target on the prime–target pair during target processing. Again, these effects do not involve attentional control, and further developments are required to account for factors influencing pre- versus postlexical priming, such as the ratio of related pairs (e.g., Keefe & Neely, 1990), experimental task (e.g., Neely, 1991), and lexical ambiguity (e.g., Lucas, 1999).

Semantic Structure in Memory

The cortical network model gives a unified account a variety of priming effects observed experimentally in humans by scanning a wide range of values for several relevant parameters such as associations strength and SOA, and for Step 1 priming (e.g., Hutchison, 2003), Step 2 priming (e.g., Hutchison, 2003, Table 2, p. 792; McNamara & Altarriba, 1988), Step 2_n priming (e.g., Lucas, 2000), and Step 3 priming (Chwilla & Kolk, 2002; McNamara, 1992). The model can exhibit these effects for variables a, SOA, step, and n in combination or independently of each other. In the light of these results, it seems worth discussing the two views of the semantic structure of concepts in memory that have become a central issue in the semantic priming literature (see Hutchison, 2003 for a discussion). Some authors propose localist models of priming effects based on direct associations between concepts encoded by distinct ''units'' (the ''association-based'' view: Anderson, 1976, 1983a, 1983b; Collins & Loftus, 1975). Other authors assume that priming effects are due to overlapping concepts sharing ''units'' (the ''feature-based'' view: McRae & Ross, 2004; Cree & McRae, 2003; Masson, 1995, 1991; Plaut, 1995; Moss et al., 1994). However, normative data from production norms, used to measure association-based or feature-based relations between concepts, quantify associations and feature overlap on the sole basis of verbalizable words without a priori distinction between concepts and features of concepts (Barsalou, 1999; McRae et al., 1997). This raises methodological issues to assess the involvement of direct association or of feature overlap in the semantic organization of concepts (Cree & McRae, 2003; Moss et al., 1994). The association-based view assumes that concepts and features are coded in the same (localist) way in a network. Although this view can easily account for Step>1 priming through the simultaneous activation of several concept units, it does not account for the wide distribution of concept coding across brain regions and the conceptual units are not related to the biophysics of actual neurons. The feature-based view is based on models of concepts that are distributed patterns of activated/inhibited features. Although current distributed models account for the distributed coding of concepts across brain regions, they describe distributed concepts in terms of combinations of localist features (see McRae et al., 1999). The resulting problems are the a priori labeling and attribution of features to concepts, and the learning of associations between features and concepts that are coded differently. Plaut and Booth (2000) addressed this issue using an extension of the Plaut (1995) model that dissociates the lexical and semantic levels of representation in different layers of neurons. This model can learn to associate localist features to distributed concept. However, it still makes strong assumptions on the different coding of features and concepts and on the a priori defined network architecture regarding concepts and features. In the model proposed here, the architecture and distributed coding of the types of semantic relations between concepts and/or features are defined in agreement with known data from animal studies on cortical networks architecture, and are assumed to follow from classical Hebbian learning.

Direct associations between items are presumably learned in monkeys on the basis of temporal contiguity (Booth & Rolls, 1998; Yakovlev, Fusi, Berman, & Zohary, 1998; Sakai & Miyashita, 1991; Stryker, 1991; Miyashita, 1988). Consistent with this hypothesis, priming effects in humans are proportional to the lexical distance and frequency of co-occurrence between words encountered in texts (Spence & Owens, 1990; Postman & Keppel, 1970; Deese, 1965; see Prior & Bentin, 2003, 2008). In network models with unsupervised Hebbian learning, synaptic potentiation between neurons coding for two items is proportional to the number of temporally contiguous occurrences of these items (Mongillo et al., 2003; Brunel, 1996) leading to synaptic matrices which are qualitatively similar to the ones used here. In the cortical network model, coded items are assumed to

be learned according to a biologically realistic Hebbian rule and distributed across neuronal populations, unlike in distributed connectionist models using backpropagation algorithms. Hebbian learning does not require a priori attribution of concept or feature labels within a single semantic structure, nor any assumption on their modality and lexical representation (see Hutchison, 2003; Khateb et al., 2003; Lorch, 1982). The present research gives new insights on the link between the two main views of concept coding by accounting for experimental data using associations underlying both associative and category priming. We emphasize here that nonoverlapping populations have been chosen here for the sake of simplicity and under the assumption that neuronal coding is sparse (Booth & Rolls, 1998). A model with concepts encoded by random subsets of neurons would generate overlaps between concepts that would not change qualitatively the effects observed here (Romani, Amit, & Mongillo, 2006; Curti, Mongillo, La Camera, & Amit, 2004). In cortical network models, overlaps are then possible between populations of neurons coding for either concepts or features, and shared neurons do not need to be a priori labeled by explicit semantic feature to generate semantic relations between concepts.

Synaptic Effects on Semantic Coding

Results of the mean field model give a coherent picture of synaptic effects on ''fine'' focused coding in the left hemisphere and ''coarse'' extended coding in the right hemisphere (Chiarello et al., 2003; Beeman et al., 1994, 2000; Beeman & Chiarello, 1998; Bowden & Beeman, 1998; Beeman, 1993; Chiarello, 1988). Given that synaptic values underlying association strengths of strong and weak associates in the left and right hemispheres are not known, we have described priming effects for ad hoc values of the strengths of weak and strong associates in the left and right hemispheres. This provides support for the hypothesis that differential hemispheric synaptic strengths, assumed to rely on differential learning, can account for differential priming in the two hemispheres. When regarding priming of strong and weak associates, extreme upper and lower values of a account for ''focused'' priming in the RVF–LH that strongly activates small semantic fields of strong rather than weak Step 1 associates. Intermediate values of a account for ''extended'' priming in the LVF–RH that weakly activates large semantic fields of Step 1 and Step 2 associates. Both hemispheres would then contribute to semantic processing, though in different ways; the left hemisphere would be more involved in processing of dominant and context-specific information (Hutchinson et al., 2003), whereas the right hemisphere would be more involved in integrating large discourse representations (e.g., McDonald, 2000; Kaplan, Brownell, Jacobs, & Gardner, 1990) such as metaphors (e.g., Brownell, Simpson, Bihrle, Potter, & Gardner, 1990) and subordinate associates to ambiguous words (e.g., Atchley, Burgess, & Keeney, 1999; Atchley, Keeney, & Burgess, 1999). Synaptic effects tested with the mean field model predict increased contrast between priming of strong versus weak Step 1 associates in the left hemisphere, through combined reduced priming of weak associates and increased priming of strong associates. The model can also specifically account for stronger Step 2 priming in the LVF–RH than in the RVF–LH (Yochim et al., 2005; Richards & Chiarello, 1995) in the case of weak associations. Predictions of the model may help conducting further neurophysiological experiments in monkeys, where precise learning protocols could allow testing for the effects of synaptic strength and step on the dynamics of semantic priming in the two hemispheres. Hemispheric differences in semantic priming have been linked to asymmetries in cortical microcircuitry of language areas (Jung-Beeman, 2005; see Hutsler & Galuske, 2003). The right hemisphere has been reported to involve broader connection fields between pyramidal neurons and more branching and dendritic spines, associated with broader overlapping between more densely connected cortical areas than in the left hemisphere (Galuske, Schlote, Bratzke, & Singer, 2000). Alternatively, differential levels of dopamine in the two hemispheres (Slopsema, Van Der Gugten, & De Bruin, 1982) could also lead to hemispheric differences in semantic priming (Lavigne & Darmon, 2008), as L-dopa is reported to change the magnitude of priming in healthy subjects (Roesch-Ely et al., 2006; Kischka et al., 1996). Unbalanced hemispheric lateralization reported in schizophrenic patients (Spitzer et al., 1993) could be linked to disordered semantic priming (Moritz et al., 2001, 2003; Spitzer et al., 1993; Manschreck et al., 1988), proposed as possibly depending on unusual levels of cortical dopamine (Kischka et al., 1996; Spitzer et al., 1993; see Abi-Dargham et al., 2002).

Conclusion

The present article identified mechanisms at the network level which could be responsible for priming dynamics. The results account for a large variety of priming effects and for the modulation of their magnitude as a function of synaptic strength. The model makes also a number of predictions that could be tested in behavioral experiments: the effects of association strength on Step 2 priming; the effects of the number of associates to the prime; the effects of the number of common associates, and the activation/inhibition ratio, in divided visual field experiments. Further developments of the model could then help better understand the interactions between several networks differing at the neuronal and synaptic level and involving different types of semantic structures and priming processes in memory.

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Reprint requests should be sent to Frédéric Lavigne, Laboratoire de Psychologie Cognitive et Sociale, EA1189—Université de Nice Sophia Antipolis, 24 avenue des diables bleus, 06357 Nice Cedex 4, France, or via e-mail: lavigne@unice.fr.

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