Determinants of Multiple Semantic Priming: A Meta-analysis and Spike Frequency Adaptive Model of a Cortical Network

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Abstract

■ Recall and language comprehension while processing sequences of words involves multiple semantic priming between several related and/or unrelated words. Accounting for multiple and interacting priming effects in terms of underlying neuronal structure and dynamics is a challenge for current models of semantic priming. Further elaboration of current models requires a quantifiable and reliable account of the simplest case of multiple priming resulting from two primes on a target. The meta-analytic approach offers a better understanding of the experimental data from studies on multiple priming regarding the additivity pattern of priming. The meta-analysis points to the effects of prime–target stimuli onset asynchronies on the pattern of underadditivity, overadditivity, or strict additivity of converging activation from multiple primes. The modeling approach is then constrained by results of the meta-analysis. We propose a model of a cortical network embedding spike frequency adaptation, which allows frequency and time-dependent modulation of neural activity. Model results give a comprehensive understanding of the meta-analysis results in terms of dynamics of neuron populations. They also give predictions regarding how stimuli intensities, association strength, and spike frequency adaptation influence multiple priming effects. ■

INTRODUCTION

Language comprehension involves contextual recall of knowledge in memory in real time. This fundamental cognitive function relies on the increased availability of concepts in memory due to activation of other concepts related to the words being processed. Such semantic priming effects correspond, at the behavioral level, to shorter RTs in processing target words (e.g., "butter") when a preceding prime word was related (e.g., "bread") than when it was unrelated (e.g., "tree") (Meyer, Schvaneveldt, & Rudy, 1972; Meyer & Schvaneveldt, 1971). Investigating the determinants of priming effects and specifying their underlying cognitive processes has become a fast growing field of research (Hutchison, 2003; Neely, 1991). Current models of semantic priming aim to account for single priming effects—in terms of whether processing of related word pairs has activating effects or inhibiting effects on the status of the corresponding concepts in memory (for a discussion, see Brunel & Lavigne, 2009). However, processing of sequences of more than two words triggers multiple and interacting priming effects between several words presented at different times. How the cerebral cortex performs sequential and simultaneous semantic priming processes is poorly understood. Answering this question implies understanding the dynamics of the cortical network that underlie the complex interactions between multiple priming processes. Taking up the challenge requires identification of cognitive, procedural, and cellular factors that determine the balance between priming from previous and from current words in a sequence and that direct semantic activation within the semantic space.

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Pattern of Additivity of Multiple Priming

The simplest case of multiple priming involves a target that is related (R) or unrelated (U) to two preceding primes (labeled 1 and 2). The experimental operationalization of the relatedness between two primes and a target generates four conditions: RR, RU, UR, and UU—wherein the first and second letter correspond to the type of relationship between the target and the first and second prime, respectively. A given target (e.g., "tiger") can then be preceded by two primes related to it (RR condition, e.g., "lion" and "stripes"), two primes unrelated to it (UU condition, e.g., "fuel" and "shutter"), or one prime related and one prime unrelated (RU condition, e.g., "lion" and "shutter" or UR condition, e.g., "fuel" and "stripes"). In the previous examples, the two primes are not related to each other, but primes can be related in some cases (e.g., "copper" and "bronze" are related to the target "metal" and are also related to each other) without affecting significantly the magnitude of RR priming effects (See Balota & Paul, 1996, Table 1 for examples and Experiment 1 for results). When primes are labeled in such a way that differentiates

Table 1. Parameters of the Model

p	Number of selective populations	99
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	0.00675–0.00825
J_a	Synaptic efficacy between associated populations	$J_E + J_S \frac{a(p-p_i+1)-1}{(1-a)(p_i-1)+p-p_i}$
J_0	Synaptic efficacy between nonassociated populations	$J_E - J_S \frac{a(p_i-1)+1}{(1-a)(p_i-1)+p-p_i}$
J_I	Inhibitory synaptic efficacy	J_E
v^θ	Threshold for RT	21 Hz
v_s	Spontaneous activity	5 Hz
I^{ext}	Nonselective external currents	set for $v^S = 5$ Hz
I^{sel}	Selective external currents	10–200 μA
I^A_{max}	Maximum adaptation current	0–500 nA
τ^A	Time constant of passive recovery	200 msec
dt	Variable integration time step	0–3 msec

the RU and the UR conditions, these labels are defined according to their order of presentation or position in the visual field. Multiple priming effects are calculated by subtracting RTs in a given condition of prime–target relatedness (RR, RU, UR) from RTs in the UU baseline condition (see Equation 6). Regarding the RR condition, the target is assumed to receive converging sources of activation from the associated primes (Balota & Paul, 1996), in accordance with studies that report an increasing magnitude of priming in conjunction with an increasing number of primes related to the target (Abad, Noguera, & Ortells, 2003; Chwilla & Kolk, 2003; Faust & Lavidor, 2003; Faust & Kahana, 2002; Nievas & Mari-Beffa, 2002; Ortells, Abad, Noguera, & Lupianez, 2001; Beeman, Friedman, Grafman, & Perez, 1994; Brodeur & Lupker, 1994; Klein, Briand, Smith, & Smith-Lamothe, 1988; Schmidt, 1976). A synthetic way of describing multiple priming effects is to compare the effects of two related primes (RR)—assumed to trigger converging facilitation—with the sum of the effects of one related and one unrelated prime (RU + UR; Equation 7).

Three patterns of RR effects can then be defined depending on the ratio of facilitation/inhibition of target-processing in the RU and UR conditions: Strictly additive effects correspond to equal magnitudes of priming effects in the RR and RU + UR conditions, underadditive effects correspond to priming in the RR condition that has a smaller effect than that in the RU + UR conditions, and overadditive effects correspond to priming in the RR condition that has a larger effect than that in the RU + UR conditions. In fact, all three of these patterns of results have been reported in the literature (e.g., strictly additive: Chwilla & Kolk, 2003; Lavigne & Vitu, 1997, Experiments 1 and 4; Balota & Paul, 1996; underadditive: Angwin, Chenery, Copland, Murdoch, & Silburn, 2005; Chenery, Copland, McGrath, & Savage, 2004; and overadditive: Chwilla & Kolk, 2003, Experiment 2; Lavigne & Vitu, 1997, Experiments 2 and 3; see Deacon et al., 2004). As for the different types of multiple priming effects, the apparent discrepancies in how the magnitudes of these effects determine the pattern of additivity then point to the importance of identifying variables that could change these effects.

In multiple priming studies as well as in single priming studies, the magnitude of priming effects are considered to result from a combination of rapid automatic activation and slower inhibition (Deacon, Uhm, Ritter, Hewitt, & Dynowska, 1999; Neely, 1991; for a discussion, see Brunel & Lavigne, 2009). These processes are reported as being highly dependent on SOA—defined as the sum of prime duration and ISI (or prime–target delay). SOA can be assumed to roughly match the stages of automatic and controlled processes in single priming (see Hutchison, 2003; Hutchison, Neely, & Johnson, 2001; Deacon et al., 1999; Neely, 1991) and has been proposed to influence multiple priming (see Kandhadai & Federmeier, 2007; Lavigne & Vitu, 1997). The effects of SOA on multiple priming will therefore be tested in the meta-analysis of the experimental data. Another variable widely reported to strongly influence the time course and magnitude of single priming effects is the strength of the prime–target association (Frishkoff, 2007; Hutchinson, Whitman, Abeare, & Raiter, 2003; Coney, 2002; Abernethy & Coney, 1993). In addition, its effects interact with those of SOA. When targets are strongly associated with the prime, single priming effects arise at shorter SOAs and are of larger magnitude (for

reviews, see Brunel & Lavigne, 2009; Chiarello, Liu, Shears, Quan, & Kacinik, 2003). Given that a modeling approach requires the definition of precise values of SOA and of association strength for simulations, the combined effects of these two variables will be investigated in the research proposed here.

Neural Correlates of Priming

Electrophysiological experiments on behaving monkeys provide information on neuronal activities during priming protocols that are very similar to protocols used in experiments conducted with humans involving a single prime preceding a target (see Brunel & Lavigne, 2009). The majority of protocols used in studies on monkeys involve a delayed pair associate (DPA) task on a target that, after a delay, follows a prime that can be associated or not with the target depending on prior learning. To obtain a reward, the monkeys are required to hold (or release) a bar if the prime and the target are associated (e.g., Erickson & Desimone, 1999; Rainer, Rao, & Miller, 1999). Another type of task requires the monkeys to choose the item associated with the prime among two targets presented by touching it on the screen (e.g., Naya, Sakai, & Miyashita, 1996; Sakai & Miyashita, 1991). In both tasks, during the delay period neurons specific to the prime exhibit "retrospective" activity—that is, increased spike rates compared with spontaneous activity (for a discussion, see Brunel & Lavigne, 2009). It is reported in inferior temporal (IT), perirhinal, and prefrontal (PF) cortices and is believed to underlie short-term or working memory of the prime stimulus (Miyashita, 1988; Miyashita & Chang, 1988; Fuster & Alexander, 1971). When additional items unrelated to the prime and target were presented to the monkeys, retrospective activity of PF neurons but not of IT neurons was found to resist interference (Miller, Erickson, & Desimone, 1996). Another type of neuronal activity is reported between prime and target presentations: IT and PF neurons specific to the associated target show "prospective" activity—that is, increasing firing rate during the delay period (Naya, Yoshida, & Miyashita, 2001, 2003; Yoshida, Naya, & Miyashita, 2003; Fuster, 2001; Erickson & Desimone, 1999; Rainer et al., 1999; Miller et al., 1996; Sakai & Miyashita, 1991; Miyashita, 1988; Miyashita & Chang, 1988). Prospective activity is believed to underlie priming processes of activation of the target associated with the prime before target presentation. Once generated by the related prime, prospective activity of IT neurons carrying information about the associated target is reported to resist processing of an unrelated item interposed between the associated prime and the target, which corresponds to the RU condition of multiple priming (Takeda, Naya, Fujimichi, Takeuchi, & Miyashita, 2005). Combined recordings of electrophysiological and behavioral data show that spike rates of neurons coding for a given response correlate with the time required to give this response (Roitman & Shadlen, 2002) and allow us to relate prospective activity to the kinds of priming-like effects found in monkeys' RTs (e.g., Erickson & Desimone, 1999).

Studies in monkeys provide us with very helpful data to improve the biophysical realism of cerebral cortex models by satisfying constraints on neuron properties and dynamics, network architecture, spike rates of neurons, and behavioral response times. This allows us to elaborate in further detail on computational models that aim to bridge the gap between neuronal and behavioral effects of multiple priming.

Models of Priming

The modeling approach provides us with mathematically tractable descriptions of cognitive processes. It provides descriptions of the correlational structure of experimentally tested independent and dependent variables in terms of causal relations governing the inner system. Current connectionist models are based on network architectures connecting elementary processing nodes. Most have focused on lag0 priming effects, when there is no word interposed between the prime and the target. They have been challenged by data on multiple priming, especially regarding the lag1 RU condition where one unrelated word is interposed between the related prime and target (for a discussion, see Becker, Moscovitch, Behrmann, & Joordens, 1997). Indeed, priming effects where the lag > 0 require that models take into account the possibility for active interference, usually assumed to be based on inhibitory processes. Localist models account for lag0 priming by describing how activation from a node coding for the prime is propagated to several nodes coding for related concepts activated in parallel (Anderson, 1976, 1983a, 1983b; Collins & Loftus, 1975; Collins & Quillian, 1969; see Ratcliff & McKoon, 1988). They can account for lag1-RU priming with variable levels of "resistance" to interference depending on the time constant of passive deactivation, which can correspond to active interference if set as being dependent on the number of items activated. Distributed models encode items as attractor states in which a single item can be fully present (McRae & Ross, 2004; Randall, Moss, Rodd, Greer, & Tyler, 2004; Cree, McRae, & McNorgan, 1999; Becker et al., 1997; Bullinaria, 1995; Masson, 1995; Plaut, 1995; Moss, Hare, Day, & Tyler, 1994; Sharkey & Sharkey, 1992). They account for lag0 priming between a prime and a target sharing units in the same state—activated or inactivated—by the reduced time needed by the network to settle in the attractor encoding the target, when units are already in the corresponding state due to previous processing of the prime. Regarding RU priming, the model of Masson (1995) and Masson, Besner, and Humphreys (1991) shows interference-based canceling of priming. Given that the network can be on only one state at a time that corresponds to the full encoding of one item only, the network shifts from an attractor state encoding the prime 1 to a state encoding the prime 2. The change in the pattern of activated/

inactivated units then induces a canceling of priming. Plaut's (1995) model shows that RU priming is possible using a less stringent criterion for the degree to which the network output must settle before it responds—that is, a greater tolerance of target recognition. This allows for the detection of some units still in the state of encoding the target after processing of the unrelated Prime 2. The determinants of RU priming in terms of conceptual structure in memory or procedural parameters are still to be understood at the network level. However, current connectionist models do not account for behavioral data on multiple priming in terms of properties of cortical neurons and cortex architecture, which make it difficult for them to capture physiological mechanisms of information processing.

We propose here a comprehensive understanding of the different types of multiple priming and of their variability. This is achieved through computational modeling of both behavioral data from studies in humans and electrophysiological data from studies in monkeys. Under this perspective, models of cortical networks are mathematically described according to neurophysiological data recorded during behavioral tasks that require monkeys to process sequences of items. These models allow us to account for network behavior as a function of properties of different types of neuron and of dynamics of different types of receptors related to biophysically based neural network models such as networks of integrate and fire neurons (see Lavigne & Darmon, 2008; Mongillo, Amit, & Brunel, 2003; Brunel & Wang, 2001). In addition, the link to network behavior respects the reported relationship between electrophysiological and behavioral data—that is, the relationship between spike rates and RTs (Roitman & Shadlen, 2002; see Erickson & Desimone, 1999). Rate models permit a mean field approach on the basis of a Wilson–Cowan formalism which, although presenting a simplification of the dynamics of neuron populations, can reproduce the dynamics of networks of more realistic Hodgkin–Huxley neurons (see Roxin, Brunel, & Hansel, 2005). Both types of cerebral cortex models account for retrospective activity in working memory in monkeys (Amit, Bernacchia, & Yakovlev, 2003; Brunel & Wang, 2001; Renart, Moreno, de la Rocha, Parga, & Rolls, 2001; Amit & Brunel, 1997; Amit, 1995). Recent models also account for the maintenance of sequences of items as a function of semantic associations in humans (Haarmann & Usher, 2001) and for prospective activity (Lavigne, 2004; Mongillo et al., 2003; Lavigne & Denis, 2001, 2002; Brunel, 1996), determining a variety of semantic priming effects observed in humans (Brunel & Lavigne, 2009; Lavigne & Darmon, 2008; Brunel, 1996). These models are formal theories that attempt to unify a large variety of behavioral and neurophysiological data. They link fundamental cognitive functions such as working memory and semantic priming to the behavior of large neuron populations and to neuron properties. Brunel (1996) proposed a realistic cortical network model of priming on the basis of the inhibitory regulation of activation between neural populations that encode items. Results brought in evidence that cortical network models are able to keep several items simultaneously activated, according to a capacity-limited view of WM (see Cowan, 2001; for a review, see Haarmann & Usher, 2001). Mongillo et al. (2003) propose a model that accounts for retrospective activity of the neurons, maintained through positive feedback between neurons coding for the stimulus. Their model also accounts for the amount of time taken up by prospective activity of the associated neurons that code for the expected target due to feedback from associated neurons that are coding for the prime. Other models describe how two converging sources of activation generate multiple priming (Lavigne, 2004; Lavigne & Denis, 2001). Recent computational models of cortical networks have been proposed that account for a rich phenomenology of lag0 priming in humans as a function of the SOA, various types of semantic relations, values of association strength, and levels of dopamine neuromodulation (Brunel & Lavigne, 2009; Lavigne & Darmon, 2008). This makes these models good candidates for a unified approach to multiple priming in the cerebral cortex.

Spike Frequency Adaptation and Sequence Processing

To account for simple associative priming, cortical networks models are required to activate one or two items at a time—as in, the prime *followed* by the target or the prime and the target together (Mongillo et al., 2003; see also Deco & Rolls, 2005). In these models, the presentation of an item generates a strong visual response that cancels retrospective activity of the preceding items. This effect makes these models unable to simultaneously hold retrospective activity of a Prime 1 and prospective activity of its associated target against an unrelated intervening Prime 2 (lag1-RU condition). Accounting for lag priming requires models to keep several items activated at a time (several primes and the target) during a whole trial. A capacity-limited working memory dealing with several simultaneously activated items could theoretically account for the canceling of a given item activation when maximum capacity is reached because of inhibitory feedback that determines proactive interference and, more importantly, retroactive interference of previous items (see Haarmann & Usher, 2001). Models with a working memory capacity for several items can account for lag0 priming effects involving the simultaneous activation of several items associated with the prime (Brunel & Lavigne, 2009; Lavigne & Darmon, 2008). They could prove to be necessary and sufficient in accounting for RU priming effects at lag1 compared with lag0 in terms of increasing retroactive interference that cancels priming effects. However, they would have difficulties in accounting for variations in RU priming as a function of SOA at fixed lag. Accounting for the presence or absence of RU priming requires models to simultaneously hold retrospective activity

of the primes and prospective activity of the RU associate not only as a function of the sequence of inputs but also as a function of their precise timing. The time-dependent processing of stimuli has been linked to the neural property of spike frequency adaptation (SFA) regarding perceptual phenomena of visual adaptation (Sanchez-Vives, Nowak, & McCormick, 2000a, 2000b), repetition blindness (see Morris, Still, & Caldwell-Harris, 2009), forward masking, and selective attention (Wang, 1998). SFA is reported to determine the level of activity of neurons as a function of the order of presentation of the stimuli they code for. At the phenomenological level, SFA is defined by a progressive decrease in neuronal firing rate in response to repeated (Miller & Desimone, 1994; Miller, Li, & Desimone, 1993; Baylis & Rolls, 1987) or constant input (Puccini, Sanchez-Vives, & Compte, 2006), which leads to time-varying spike rates during retrospective activity of a neuron population that is coding for an input (Markram & Tsodyks, 1996). Deco and Rolls (2005) have proposed SFA as a determinant of noncontinuous spike rate dynamics—an increase followed by a decrease of activity, determining sequential priming of several items—one at a time in a sequence—but with a single-item capacity in the working memory that does not allow for combinations of items to generate multiple priming.

SFA has been observed in many types of neurons and in many species (see Puccini et al., 2006; Fuhrmann, Markram, & Tsodyks, 2002), including human neocortical cells (Lorenzon & Foehring, 1992; Foehring, Lorenzon, Herron, & Wilson, 1991; Avoli & Olivier, 1989). At the neurophysiological level, SFA diminishes the excitability of a neuron because of increased afterhyperpolarization (AHP). The action-potential–dependent hyperpolarized potential I_i^A progressively builds-up with successive spikes after current-induced repetitive firing (Schwindt, Spain, Foehring, Chubb, & Crill, 1988; Madison & Nicoll, 1984) and influences interspike intervals to produce SFA in slices of human cortex (Avoli, Hwa, Lacaille, Olivier, & Villemure, 1994; Lorenzon & Foehring, 1992). The ionic mechanisms underlying AHP have been suggested to be produced by fast Ca^{2+}-activated K^+ currents (Schwindt et al., 1988; Madison & Nicoll, 1984; Connors, Gutnick, & Prince, 1982; Hotson & Prince, 1980), slow-activating and noninactivating voltage-sensitive potassium current (M-current; McCormick, Wang, & Huguenard, 1993; Madison & Nicoll, 1984), and slow Na^+ inactivation (Schwindt & Crill, 1982; Michaelis & Chaplain, 1975). Thus, most pyramidal neurons in the neocortex exhibit SFA on various time scales (Sanchez-Vives et al., 2000a; McCormick, Steinmetz, & Thompson, 1985; see Puccini et al., 2006; Varela et al., 1997). At the neuron level, the nonlinearity of SFA enhances the sensitivity of single neurons to changes in stimuli intensities (Puccini et al., 2006) and enables them to detect new inputs while disregarding previous inputs, depending on their respective intensities (Wang, 1998). At the network level, the mechanism of SFA has been reported to enable a wave of activation to propagate from a single input prime to its associates one after the other, although only one item can be activated at a time (Deco & Rolls, 2005). Given that current cortical models of semantic priming do not embed SFA, their ability to account for all conditions of multiple priming and for their precise dynamics is still an open question. The behavioral effects of SFA suggest that a network of adaptive neurons can generate time-varying spike rates of different neuron populations that are coding for successive inputs, enhancing the ability of neuron populations to detect changes in input-switching by time-modulating their spike rate. We propose here that during a protocol of multiple semantic priming, SFA could generate nonlinear time-variations of the retrospective activity levels of the primes and of the related prospective activity level of the target. This would lead to differential priming effects, depending on the positions of successive primes in a sequence and probably also depending on their respective processing times defining prime–target SOAs.

Purpose of the Present Research

The purpose of this study is to provide a comprehensive understanding of the current research on the determinants of multiple semantic priming. It is therefore of importance to identify if, to what extent, and how multiple priming effects are additive, underadditive, or overadditive. The meta-analytic approach is proposed to search for moderator variables regarding the questions of "if" and "to what extent." A further goal of this study is to provide a unified explanation of "how" the experimental conditions, protocols, and neuron properties such as SFA determine the behavior of a computational model of a cortical network. This would provide us with a model accounting for an extended range of semantic priming effects in the cerebral cortex.

METHODS

Meta-analysis

The experimental approach provides us with essential information on the different types of multiple priming effects across many studies. However, data from different studies are gathered using various protocols, numbers of participants, and experimental materials, making hazardous the global assessment of the effects reported. The meta-analytic approach is a statistical method for investigating the magnitude of an effect on the basis of the combined results from several independent studies (Glass, 1976) and seeks to explain inconsistencies in the results of separate studies by examining the relationship between study outcomes and characteristics of the individual studies (Durlack, 1995). Comparison of priming effects from separate studies is made possible through the use of effect sizes, which are statistical standardized representations of the magnitude of an effect, as a function of the amplitude of the effect

reported in each study, weighted by the standard deviation of the effect. The standardized effect sizes are then a common metric through which different studies may be compared, making possible to address whether differences in study characteristic or sampling errors are responsible for variations in effect sizes across studies. During analysis, the effect sizes become the dependent variable and study features become the independent variables.

The first step in the meta-analytic procedure is to formulate the research questions that will guide the study (Wampler, Serovich, Sprenkle, & Moon, 1996; Durlack, 1995; Johnson, 1989) to define the literature search. They rely on multiple priming in word sequence processing involving at least an RU condition compared with a UU condition. To capture most studies with relevant data, we carried out a computer-based search of all studies providing data on multiple priming, using Web databases, articles references lists, achieved by a keywords search using various combinations of terms such as "semantic priming," "multiple primes," "lag priming," and "word sequence." The literature review allowed selecting published articles that tested for lag priming effects and whose experimental protocols met the following inclusion criteria (see Riby, Perfect, & Stollery, 2004; Lucas, 1999; Rosenthal, 1995): (1) results provided behavioral data on processing time of the target (RTs), (2) words lists involved semantic relatedness and no syntactic relations, (3) one prime was related and one prime was unrelated to the target (RU condition), (4) no cues or task could bias processing of the primes or prime–target relation (see Fuentes, Vivas, & Humphreys, 1999), (5) a baseline condition was tested involving unrelated primes (UU condition; see Neely, 1991, for a discussion), (6) healthy participants were considered, and (7) adequate data were provided to compute effect sizes from either descriptive or inferential statistics.

The coding criteria were then set up to accurately capture information on the variables under study: lag, SOAs, and ISIs between each prime and the target, condition of multiple priming, number of subjects, experimental task, and RTs in the RR, RU, UR, and UU conditions for effect sizes calculation. Some parameters were not reported or were present among too few studies to be analyzed beyond simple counting. This was the case of the type of semantic relation (association vs. feature overlap), the association strength between the primes and target (strong vs. weak), and the relation between the two primes (related vs. unrelated). We then investigated as main moderator variables parameters the most likely involved in modulating multiple priming: condition of multiple priming and SOA. A total of 239 effect sizes were then calculated for the different meta-analyses on the basis of data of 63 experiments meeting the aforementioned criteria and selected for consideration from 25 articles (labeled by asterisks in the reference list).

The question of multiple semantic priming was assessed following the meta-analytic strategies outlined by Hedges and Olkin (1985). An effect size was calculated for each condition of multiple priming, examined for each group of participants. When some studies reported separate effects for different conditions (e.g., SOAs), the corresponding d values were calculated on the basis of priming effects for each experiment of each study, as the difference between the average RT in the UU baseline condition and the related condition of interest (RR, RU, and UR). To combine the results of the different studies, we converted means and standard deviations from each experiment into Hedges' g. This standardized mean difference represents the mean difference between the conditions of multiple priming, divided by a pooled standard deviation, which results in less variance and less bias than the use of the control condition's standard deviation (Hedges, 1981). When no means or standard deviations were reported, effects size estimates were calculated from statistics of the data (Wolf, 1986). The g values were then converted to d values to remove the small sample bias inherent in this statistic (Hedges & Olkin, 1985). This provided a less variable and less biased estimate of the population effect sizes (Hedges, 1982) regarding the different conditions of multiple priming.

Cortical Network Model

To investigate multiple priming effects, we constructed an elaboration of the model of a local network of an area of association cortex used by Brunel and Lavigne (2009), in which neurons were spike frequency adaptive. Populations of excitatory neurons code for concepts in memory and exhibit simultaneous persistent activities following presentation of the corresponding items. We study a simplified "rate model" in which dynamical variables represent average firing rates of populations selective to the same concept. Parameters of the model are presented in Table 1. We assume for simplicity nonoverlapping populations of neurons coding for p distinct stimuli (Brunel & Lavigne, 2009; Brunel & Wang, 2001; Amit & Brunel, 1997), shown schematically in Figure 1A.

We use a mean-field–type description of a firing-rate model that includes multiple associated sets of neural populations. Each population $i = 1,\ldots, p$ is described by an average firing rate v_i whose dynamics is described by a standard Wilson–Cowan type equation:

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

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^{sel} represents the selective input to population i, I_{ext} represents the global external inputs to all populations, and I_{inh} represents a global inhibitory current regulating the activity of all populations,

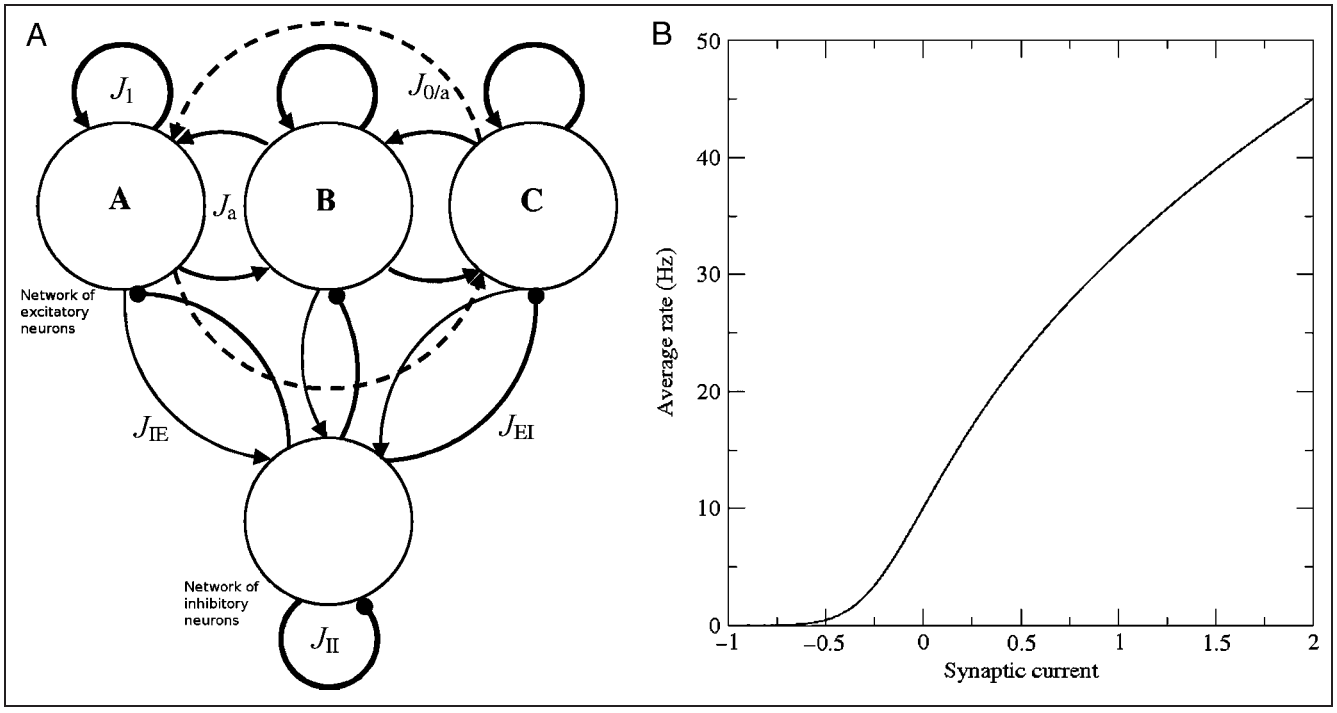

Figure 1. (A) Architecture of the excitatory–inhibitory network: Excitatory neurons are divided in p subpopulations of neurons selective for distinct stimuli (for clarity, $p = 3$ in the schema: A, B, and C). Inhibitory neurons are nonselective. Synaptic strength is indicated by line thickness and type (precise values of the parameters are given in Table 1). (B) *I–f* curve described by the transfer function Φ.

which is here for the sake of simplicity proportional to the average activity of all populations:

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

Strength of inhibition is described by J_I. This description of inhibition corresponds to a scenario in which inhibitory neurons have linear *f–I* curves and an instantaneous time constant. The external input in absence of stimuli is chosen such that all populations in the network have some prescribed level of background activity $v_0 = 5$ Hz. J_1, J_0, and J_I are chosen so that both nonselective background state and selective attractors corresponding to single or multiple activated items are present in the network.

Here we use a mean field approach describing population rates effective to account for a large set of single priming effects (Brunel & Lavigne, 2009). We embed in the model the neuron property of SFA, described at the level of neuron population i as a function of the mean firing rate v_i averaged over this population. The dynamics of the mean AHP current I_i^A are described using the equation of Fuhrmann et al. (2002) derived from the integrate-and-fire model of an adapting neuron:

$$\frac{dI_i^A}{dt} = -I_i^A/\tau^A + \alpha I_{\max}^A v_i \qquad (3)$$

The effects of SFA on the dynamics of the mean firing rate v_i of population i are described by applying the delayed negative feedback due to AHP I_i^A to population i according to Equation 1. $I_{\max}^A$ approximates the relation between maximal conductance for the adaptation current and cell membrane conductance (with $\alpha = 0.02$; see Fuhrmann et al., 2002), and τ^A is the tau of passive recovery (when $v_i = 0$). The choice for parameters values was guided by the results of Fuhrmann et al. (2002) and by experimental results showing that adaptation induces changes in the response magnitude of neurons (Gutnisky & Dragoi, 2008; Sharpee et al., 2006; Felsen et al., 2002; Dragoi, Sharma, & Sur, 2000) within a range of hundreds of milliseconds (Dragoi, Sharma, Miller, & Sur, 2002; Muller, Metha, Krauskopf, & Lennie, 1999).

Finally, Φ describes the static current-to-rate transfer function (or *f–I* curve, Equation 4, Figure 1B). As in Brunel and Lavigne (2009), we take this function to be the transfer function obtained analytically for quadratic integrate-and-fire neurons in presence of strong background noise, which is expected to be qualitatively and in some condition quantitatively similar to the one of cortical excitatory neurons (Brunel & Latham, 2003). The function Φ is given by

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

where $\sigma = 0.5$.

Synaptic Matrix

We consider $p = 99$ items stored in memory, organized in $p_g = 33$ groups of $p_i = 3$ semantically associated items within a group ($p = p_i p_g$). 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. For the sake of simplicity, we present an example with $J_1 = 1$ and $J_0 = 0$. In the case of a subset of the network with $p = 9$, $p_g = 3$, and $p_i = 3$, the synaptic matrix has the following form:

$$M_{i,j} = \begin{pmatrix} 1 & a & a & 0 & 0 & 0 & 0 & 0 & 0 \\ a & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & a & a & 0 & 0 & 0 \\ 0 & 0 & 0 & a & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & a & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & a & a \\ 0 & 0 & 0 & 0 & 0 & 0 & a & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a & 0 & 1 \end{pmatrix} \quad (5)$$

Hence, for simulations, a target item (e.g., 1) is associated to two primes (e.g., 2 and 3), and each prime is related to the target by a direct relation. It corresponds to a step1[1] relation defined in Brunel and Lavigne (2009) that is each prime is directly associated to the target, which is their only associate. This allows analysis of conditions of multiple priming corresponding to different sequences of items: 2-3-1 for the RR condition, 2-5-1 for the RU condition, 8-3-1 for the UR condition, and 5-8-1 for the UU condition.

Protocol

Simulations emulated in the model used the same experimental protocols as those used in humans studies (Figure 2B). The equations for the population spike rate v_I and AHP current I_i^A are integrated using an extension of the Runge–Kutta fourth-order numerical integration method with variable time step dt given by an estimation of the error by a comparison to the Runge–Kutta third-order method. During numerical simulations, a trial begins with the network in a state of spontaneous activity (Figure 2A). At Prime 1 onset, the corresponding neuron population reaches an elevated activity ("visual response"). In response to a depolarizing current, the neuron population initially fires at a high frequency that decreases according to SFA. The level of activation decreases after prime offset but remains above the level of spontaneous activity because of the strong excitatory feedback through J_1. Then Prime 1 exhibits time-varying level of retrospective activity during Delay 1 due to adaptation. This leads in turn to SFA-dependent prospective activation of the population of neurons coding for its associate through J_a. At Prime 2 onset, the level of activity of neurons coding for Prime 2 and its associate increases. This triggers an increase in feedback inhibition proportional to activation and then to SFA-dependent activation of Prime 2. Hence, at the time of the presentation of the target the corresponding neuronal population can exhibit variable firing rate ranging from above (prospective activity) to under (prospective inhibition) level of spontaneous activity. The precise level of activity for a given association strength a depends on the experimental condition (Figure 2A): RR (strong prospective activity), UU (prospective inhibition), and RU and UR (ranging from low prospective activity to prospective inhibition).

On the basis of electrophysiological studies reporting correlation between spike rates and response times (Roitman & Shadlen, 2002), modeling approaches of cortical networks take as the RT the time at which the mean spike rates of a population of neurons reaches a prescribed threshold (Brunel & Lavigne, 2009; Lavigne & Darmon, 2008; Wong & Wang, 2006; Wang, 2002), similar to classical diffusion models of RT (Ratcliff, 1978; see Randall et al., 2004; Plaut & Booth, 2000; Masson, 1995; Plaut, 1995; Masson et al., 1991). In the mean field model, when a target is presented to the network, its recognition time T^θ is the time elapsed from target onset to the instant at which the mean firing rate of the corresponding neurons population crosses a threshold v^θ for the first time. T^θ depends on the level of prospective activity of the neurons population coding for the target at target onset, itself assumed to depend on the synaptic matrix and protocol. The four experimental conditions led to specific recognition times T^θ_{RR}, T^θ_{RU}, T^θ_{UR}, and T^θ_{UU} that enable to quantify the magnitudes of multiple priming effects of two Primes 1 and 2 either related or unrelated to the target

$$PE_{12} = T^\theta_{UU} - T^\theta_{12} \quad (6)$$

and the resulting pattern of additivity:

$$\begin{aligned} ADD &= PE_{RR} - (PE_{RU} + PE_{UR}) \\ &= T_{RU} + T_{UR} - T_{RR} - T_{UU} \end{aligned} \quad (7)$$

RESULTS

Types of Multiple Priming

We first investigate the relative magnitudes of the different types of multiple priming through a meta-analysis of effect sizes in the three conditions RR, RU, and UR. The meta-analysis of data gives a good overview of the effects through a test of heterogeneity of each condition (Hunter & Schmidt,

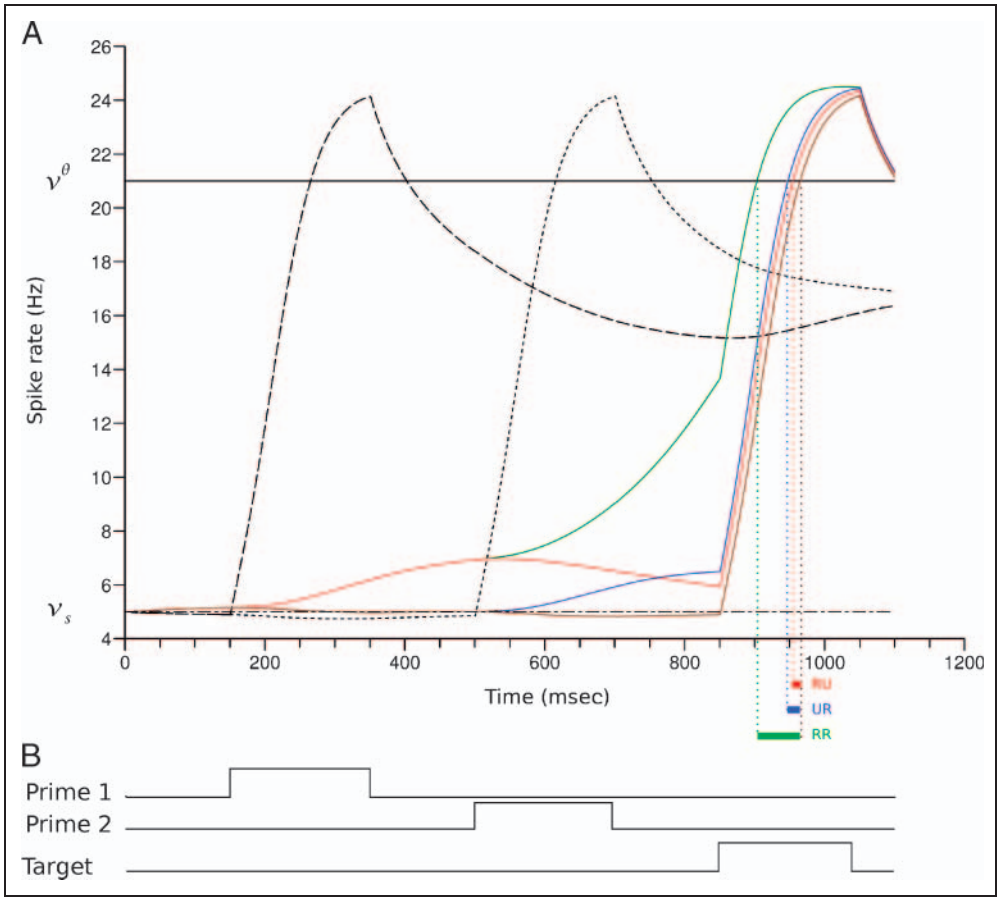

Figure 2. (A) Spike rates of six neurons populations (for clarity, the 94 other excitatory populations and inhibitory population are not displayed) as a function of time defined in protocol B and Equation 5 ($a = 6.75 \times 10^{-3}$, $I^{\text{sel}} = 50$ μA, and $I^A_{\max} = 420$ nA). Prime 1 (black dashed curve) and Prime 2 (black dotted curve) successively exhibit visual response during prime presentation, followed by retrospective activity above spontaneous activity (v_s = 5 Hz; horizontal black dash-dotted line). Targets in the RR (green solid curve), RU (red solid curve), UR (blue solid curve), and UU (brown solid curve) conditions exhibit prospective activity at variable level before target presentation. Vertical dotted lines indicate response times T^θ from target onset for target population activity to reach threshold v^θ = 21 Hz (horizontal solid line). Horizontal color bars indicate the magnitude of RR, RU, and UR priming effects, calculated as the difference between RTs in the unrelated (T^θ_U) and related conditions (T^θ_{RR}, T^θ_{RU}, and T^θ_{UR}, respectively; Equation 6). (B) Example of a particular experimental protocol starting with neuron populations at spontaneous activity for 50 msec, preceding Prime 1 presented for duration t_{P1} (here 200 msec) followed by delay period t_{d1} with no selective input (interstimuli interval, ISI) defining variable SOA (SOA1 = $t_{P1} + t_{d1}$), then the Prime 2 was presented for duration t_{P2} (here 200 msec) followed by delay period t_{d2} with no selective input (SOA2 = $t_{P2} + t_{d2}$; here $t_{d1} = t_{d2} = 150$ msec); finally, the target was presented for 200 msec followed by 50 msec with no selective input before the end of trial.

1990), which indicates whether the variation in effect sizes is significantly greater than expected by chance. The greater the value of the Q statistic—which measures the heterogeneity of the sample of individual effect sizes constituting the composite effect size—the greater the heterogeneity in effects sizes distribution. It allows for a χ^2 test of significance of the null hypothesis that all of the effect sizes are equal (Hedges & Olkin, 1985). In the case of a significant value of the Q statistic showing heterogeneity of the distribution, the group can then be further subdivided and the process repeated (Gurevitch, Morrow, Wallace, & Walsh, 1992).

Meta-analysis 1 (MA1, Sample1): Comparison of RR, RU, and UR Priming Effects

Understanding the pattern of multiple priming effects requires investigation of the relative magnitudes of priming between the three conditions RR, RU, and UR. To make comparisons as accurate as possible, we first compared these conditions as within-study variables. Indeed, different studies often vary greatly in their particular experimental protocols (defined by primes' durations and interstimuli intervals), the task given to subjects (lexical decision, naming, semantic matching), and the type of prime–target relation (direct association, feature overlap, mixed semantic relations). Selecting studies testing each of the three conditions as within-study variables provided us with a value of effect size for each condition. This ensured that a particular protocol, task, or type of relation from any given study was not overrepresented or underrepresented in any condition.

In addition, retroactive interference (involved in the RU condition) is reported to be stronger than proactive interference (involved in the UR condition) (Cowan, 2001; for a review and model, see Haarmann & Usher, 2001). This suggests that although the RU and UR conditions would both involve facilitation of the target (from the related prime), they would involve different types of retroactive versus proactive interference, respectively. To clearly assess for RU and UR priming effects, we therefore considered for MA1 cases where primes were presented sequentially and not cases where they were presented simultaneously. We then included in this first meta-analysis 78 effect sizes

from 26 studies, all using protocols of sequential presentation of the primes and target and all reporting an effect for each of three conditions RR, RU, and UR (Sample 1). The distribution of effect sizes was partitioned into three subgroups corresponding to the three multiple priming conditions. This allowed for calculation of the two-way between-group homogeneity Q_B that measures the variation in effect sizes between the three groups, which is explained by the moderator variable (Gurevitch & Hedges, 1993; Johnson, 1989) (i.e., the condition of multiple priming). The significant between-group Q_B value, $Q_B(2) = 10.97$, $p < .01$, indicates that there is a main effect of the type of priming studied as a moderator variable on the magnitude of effect sizes—that is, effect sizes significantly differ between RR, RU, and UR conditions. The within-group homogeneity Q_W measures the residual error variance that is not explained by the moderator variable (condition of priming). The nonsignificant Q_W value, $Q_W(75) = 8.4$, $p = .99$, does not indicate global within-group heterogeneity when the three groups are considered together. The total fit statistic $Q_T = Q_B + Q_W$ measures if the mean weighted effect size is a representation of the distribution of effect sizes among studies. The nonsignificant Q_T value, $Q_T(77) = 19.4$, $p = 1$, does not indicate that the global distribution of the three conditions was heterogeneous—that is, variation in the whole sample of RR, RU, and UR priming effect sizes is not greater than would be expected by chance alone (Durlack, 1995). This suggests that the heterogeneity between RR, RU, and UR conditions revealed by the significant two-way Q_B value does not correspond to pairwise differences between all three subgroups but only between some of the subgroups. To further differentiate between the three conditions of priming, we therefore tested the pairwise heterogeneity between pairs of priming conditions by calculating Q_B values for each one-way comparison. Results show significant main differences between RR priming ($d'_{RR} = 0.51$) and RU priming ($d'_{RU} = 0.26$), $Q_{B\text{-}RR\text{-}RU}(1) = 10.3$, $p < .01$, and between RR priming and UR priming ($d'_{UR} = 0.33$), $Q_{B\text{-}RR\text{-}UR}(1) = 5.3$, $p < .05$. The nonsignificant Q_T values, $Q_{T\text{-}RR\text{-}RU}(51) = 24.9$, $p = .99$ and $Q_{T\text{-}RR\text{-}UR}(51) = 4.8$, $p = 1$, and Q_W values, $Q_{W\text{-}RR\text{-}RU}(50) = 14.5$, $p = .99$ and $Q_{W\text{-}RR\text{-}UR}(50) = 0.5$, $p = .99$, do not indicate that the distributions of pairs of effects are heterogeneous. In addition, results do not show a significant difference between RU and UR priming: $Q_{T\text{-}RU\text{-}UR}(51) = 3.7$, $p = 1$; $Q_{W\text{-}RU\text{-}UR}(50) = 2.8$, $p = .99$; and $Q_{B\text{-}RU\text{-}UR}(1) = 0.8$, $p = .37$. MA1 then shows the following pattern of effects: RR > RU; RR > UR; RU ≃ UR (wherein the symbol "≃" indicates a nonsignificant difference between the effects).

Modeling of Results from MA1 (MOD1, Same Protocols as Sample1): Comparison of RR, RU, and UR Priming Effects

Turning to the modeling account of the different types of multiple priming effects, different response latencies to the target stimulus are possible in the different conditions (RR, RU, UR, and UU), allowing us to systematically study the different types of multiple priming effects (RR, RU, and UR) (Figure 2). Regarding the relations between the primes and the target defined in Equation 5, the value of association strength is defined to permit each of the three types of priming effects to arise. The level of association strength could not be estimated from the experimental data because its value was not sufficiently reported and was not manipulated. Association strength can be seen as an unknown "free" parameter in most experimental studies, and its manipulation as a free parameter in the model could optimize the consistency of its results with those of experiments for each protocol. However, to clearly understand the effects of other variables that are explicitly given from experimental studies (e.g., SOA) and to later analyze the global effects of association strength ($MOD_{a\text{-}SOA}$ and Figure 5), we did not consider it as a free parameter in the model. The first model simulations (MOD1–MOD7) do not then investigate the interstudy variability in association strength, and the corresponding simulations used a fixed value ($a = 7.50 \times 10^{-3}$). Once multiple priming effects are better understood for fixed values of association strength, we will propose a description of its interactive effects with SOA on the different types of multiple priming conditions ($MOD_{a\text{-}SOA}$).

To compare results from the model and from MA1, we made simulations in MOD1 using the same values of primes durations and interstimuli intervals as in the experimental protocols and the same conditions (RR, RU, UR, and UU) as those used for each of the 26 studies. This allowed for the calculation of effects in the 78 conditions (26 RR, 26 RU, and 26 UR) corresponding to the experimental protocols of the Sample 1 used for MA1. A first qualitative result is presented in Figure 2, corresponding to a particular protocol taken as an example that shows the activity of the target in the conditions of multiple priming (RR, RU, UR, and UU) and the relative magnitudes of the three types of multiple priming effects. The difference in magnitudes go in the same direction as the average values calculated over the 78 conditions tested in the model and also in the same direction as the average values calculated in MA1 (RR > RU; RR > UR; and RU ≃ UR; the discussion of the precise magnitudes determining the pattern of additivity will be presented in Figure 5 in relation to the interactions between SOA and association strength). The cortical network model exhibits the three types of multiple priming effects as follows: When a sequence of two primes is presented to the network (Figure 2B), spike rates diagrams (Figure 2A) show that each prime elicits retrospective activity of the population coding for itself as well as prospective activity of its associate. It corresponds to the coexistence of retrospective and prospective activities of several items in the network working memory (Brunel & Lavigne, 2009; Lavigne, 2004; Amit et al., 2003; Haarmann & Usher, 2001; Brunel, 1996). Such ability for simultaneous activation of items allows the network to simultaneously activate both primes and their associates at various levels at target onset. Activity of the neuron population coding for the target can then

reach the threshold for recognition at variable times after target onset, leading to variable RTs and priming effects.

To provide us with a quantitative account of the fit of results from MA1 by the model, we compared averaged effects from the 78 simulations. MA1 showed that RR priming was greater than RU and greater than UR priming, RU and UR priming being not significantly different. The *t* tests between the three groups of 26 effects obtained from simulations show that RR priming (55 msec) is larger than RU priming (24 msec; $p < .01$) and larger than UR priming (17 msec; $p < .01$) and that RU and UR priming differ marginally from each other ($p = .065$) (RR > RU; RR > UR; and RU ≃ UR). This indicates the effects in the experimental studies that the model captures the overall pattern of.

Meta-analysis 2 (MA2, Subgroup LDT of Sample1): Comparison of RR, RU, and UR Priming Effects in the Lexical Decision Task

MA1 included studies using different experimental tasks: lexical decision task (LDT; 18 experiments), naming task (NT; 6 experiments), and semantic matching task (SMT; 2 experiments). The different tasks used led to different mean response times and to different magnitudes of priming effects, smaller effects in the NT than that in the LDT, and larger effects in the SMT than that in the LDT (for a review and discussion, see Neely, 1991). In the multiple priming studies analyzed here, the three types of tasks resulted in the same patterns of multiple priming but with different magnitudes of averaged effect sizes that were smaller in the NT than that in the LDT and larger in the SMT than that in the LDT ($d'_{NT}RR = 0.27$, $d'_{LDT}RR = 0.54$, $d'_{SMT}RR = 0.95$; $d'_{NT}RU = 0.13$, $d'_{LDT}RU = 0.27$, $d'_{SMT}RU = 0.62$, $d'_{NT}UR = 0.15$, $d'_{LDT}UR = 0.37$m and $d'_{SMT}UR = 0.55$). Given that there were a large enough number of studies using the lexical decision task (18 studies each testing all of the three conditions of multiple priming), it was possible to make a meta-analysis of the subgroup of 54 effects sizes from these 18 studies. Results show significant between-group Q_B values, $Q_{B\text{-}RR\text{-}RU}(1) = 8.8$, $p < .01$ and $Q_{B\text{-}RR\text{-}UR}(1) = 5.8$, $p < .05$, respectively, which indicate main differences between RR and RU and between RR and UR priming. The nonsignificant Q_W values, $Q_{W\text{-}RR\text{-}RU}(34) = 5.8$, $p = .99$ and $Q_{W\text{-}RR\text{-}UR}(34) = 7.32$, $p = .99$, and the Q_T value, $Q_{T\text{-}RR\text{-}RU}(35) = 14.6$, $p = .99$ and $Q_{T\text{-}RR\text{-}UR}(35) = 4.1$, $p = .99$, fail to indicate that the distributions are heterogeneous. When comparing RU and UR priming effects, results do not show a significant difference between these effects: $Q_{T\text{-}RU\text{-}UR}(51) = 6.5$, $p = 1$; $Q_{W\text{-}RU\text{-}UR}(50) = 5.1$, $p = 1$; and $Q_{B\text{-}RU\text{-}UR}(1) = 1.4$, $p = .24$. MA2 then replicate the pattern of effects obtained from MA1: RR > RU; RR > UR; RU ≃ UR. This enables us to rule out the possibility that the NT and SMT could modify the pattern of results obtained with LDT but leaves open the question regarding the pattern of multiple priming effects from studies using the NT and SMT.

Modeling of Results from MA2 (MOD2, Same Protocols as Subgroup LDT of Sample1): Comparison of RR, RU, and UR Priming Effects in the Lexical Decision Task

To compare results from the model and from MA2, we compared averaged effects from the subgroup of simulations corresponding to studies using the LDT with the results of MA2 (same protocols as in the subgroup LDT of Sample 1; fixed value of $a = 7.50 \times 10^{-3}$ same as MOD1). The *t* tests between the three effects of multiple priming obtained from simulations using the protocols of LDT studies show that RR priming effects (54 msec) are larger than RU priming (22 msec; $p < .01$) and larger than UR (15 msec; $p < .01$) priming and that RU and UR priming effects do not differ from each other ($p = .17$) (RR > RU; RR > UR; RU ≃ UR).

The model accounts for results of MA2 involving only data from studies using the lexical decision task. Further data on multiple priming effects in other tasks (naming task, semantic matching task, etc.) would be necessary to investigate the task dependency of the effects and to further elaborate the model regarding task specificities—such as average RTs and effect sizes.

Modeling of the Effect of Interprimes Relatedness (Protocols and Variable ISIs of Balota and Paul, 1996)

The model proposed here exhibits a pattern of effects in the RR, RU, and UR conditions that is very similar to the pattern of results from MA1 and MA2. These results are from conditions of multiple priming where the two primes are unrelated to each other. Balota and Paul (1996) explicitly compared cases where the primes were either unrelated or related by manipulating the interprime relatedness within the same study: Experiment 1 involved related primes that were coexemplar of a same category target ("copper" and "bronze" for "metal"), and Experiment 2 involved unrelated primes that had the target as a common associate ("lion" and "stripes" for "tiger"). Their results, although not testable in the meta-analysis, do not show effects of interprime relatedness: For related primes (Experiment 1) compared with unrelated primes (Experiment 2), RR priming is 10 msec smaller, UR priming is 3 msec smaller, and RU priming is 2 msec larger. We then tested the model in these two conditions using the same protocol (presentation durations and delays) as in Balota and Paul's study and for four values of interprime association strength (0, 6.75×10^{-3}, 7.50×10^{-3}, and 8.25×10^{-3}; fixed value of $a = 7.50 \times 10^{-3}$ between the primes and target, same as MOD1, MOD2, and MOD3). To this, we used matrices from Brunel and Lavigne (2009) that permitted to test priming of a target (Population 1) in groups of three populations, when the two primes (Populations 2 and 3) were related (M4) or unrelated (M5) together. Results show that the magnitude of multiple priming effects varies marginally as a function of interprime relatedness for each of the RR (2-msec variation), RU (1-msec variation), and UR (1-msec

variation) conditions. We further tested if longer IS11 and ISI2 could influence the pattern of multiple priming by permitting stronger interprime priming (varying ISIs from 0 to 300 msec, corresponding to SOAs from 133 to 433 msec). Again the interprime relatedness did not influence the magnitudes of RR, RU, and UR priming effects (with a maximum of 6-msec variation in the RR condition at 300-msec ISIs). This result relies on several mechanisms. In the RR condition, the interprime relation permits each prime to receive activation from the other in addition to from other sources of activation (external currents and their associates if activated). However, this increased activation leads to an increase in feedback inhibition that reduces overall activity, including the one of the associated target. The target is then at the same time activated (directly) and inhibited (indirectly) by the preceding primes. Within the range of association strengths and SOAs tested, the two effects almost compensate for each other, with a maximum effect of interprime association being a 6-msec increase in the magnitude of RR priming. Then, although activation of the target by the second related prime increases when it is related to the first prime, its effect is compensated by an overall increase of inhibition.

Meta-analysis 3 (MA3, Sample 1): Pattern of Additivity of Multiple Priming

We then meta-analyzed the precise pattern of additivity from studies using sequential presentation of items and providing values of SOAs that were either explicitly reported or calculable from word presentation durations, ISIs, and subjects RTs. We then included in the analysis all 26 studies reporting an effect for each of the three sequential RR, RU, and UR conditions (the same 78 effect sizes as for MA1). Effect sizes in the three conditions of multiple priming allowed us to calculate 26 effect sizes corresponding to the RU + UR priming effects to be compared with the 26 effect sizes in the RR condition (two groups of effects). Results from the overall meta-analysis do not show between-group differences, $Q_B(1) = 0.1, p = .75$, with the RR condition ($d'_{RR} = 0.51$) not being significantly different from the sum of priming effects in the RU and UR condition ($d'_{RU + UR} = 0.59$) (RR ≃ (RU + UR)). This does not rule out the additivity hypothesis of priming effects (Balota & Paul, 1996). However, the overall heterogeneity, $Q_T(51) = 68.8$, $p < .05$, and the within-group heterogeneity, $Q_W(50) = 68.6, p < .05$, are associated with a heterogeneous distribution of the RU + UR condition, $Q_{T,RU + UR}(25) = 63, p < .01$, suggesting that variability in the magnitude of RU + UR priming could generate variability in the pattern of additivity.

Modeling of Results from MA3 (MOD3, Same Protocols as Sample 1): Pattern of Additivity of Multiple Priming

To compare results on additivity from the model and from MA1, we used simulation data of the 26 RR effects and calculated the 26 sums of RU + UR effects from the 52 RU and UR simulation data (these data came from the modeling of results from MA1; simulations used the same protocols and conditions of multiple priming as those of data from Sample 1 used for MA1 and MA2). Regarding prime–target association strength, precise values were seldom given in experimental protocols and were not manipulated, so a fixed value of association strength identical to the one used for MOD1–MOD3 was set for simulations ($a = 7.50 \times 10^{-3}$). MA3 does not rule out the possibility of additive effects. However, model simulations using fixed association strength indicate 14 msec of overadditivity, with t tests between the two groups of 26 RR effects and 26 RU + UR effects, indicating that RR priming (55 msec) is larger than RU + UR (41 msec) ($p < .05$). Although the model exhibits relative magnitudes of RR, UR, and RU priming going in the same direction as those shown by MA1–MA2, the overall pattern of overadditivity corresponds to an underestimation of UR and/or RU priming and/or an overestimation of RR priming. This is to be related to the heterogeneity of the RU + UR condition indicated by the MA3. This heterogeneity points to the effect of moderator variables that modulate the precise magnitudes of some conditions of multiple priming and of the resulting patterns of additivity. Values of SOA (primes durations and ISIs) were given or calculable from the experimental protocols of studies so that simulations could be run using these exact values. However, we note here that the values of association strength were seldomly provided and probably varied from experiment to experiment—that is, from SOA to SOA, which varied between experiments. In addition, association strength often varied within a given experiment between the conditions of RU and UR priming (i.e., between the target and the first and second prime, respectively). As a consequence of these two sources of variability in association strength (between SOA and between conditions of multiple priming), the different protocols and conditions tested in the meta-analysis most likely corresponded to different values of association strength. These effects of association strength are of importance in that they are known to influence the magnitude of single lag0 priming and also known to interact with those of SOA. In the case of sequential processing of items such as those involved in multiple priming, the effects of proactive and retroactive interference in the UR and RU conditions also depend on association strength (see Haarmann & Usher, 2001, Figure 5). Then the use of fixed values of association strength in the model in MOD1–MOD3 could have led to underestimation or overestimation of the precise magnitude of RU and UR effects. Therefore, not only do the effects of SOA need to be better understood from the experimental data, but the effects of association strength and their interactions with SOA need to be clarified as well in the model by combining these two variables. After the analysis and modeling of the different types of multiple priming, it is then important to analyze and model the effects of determinants of the magnitudes of multiple priming effects.

Determinants of Multiple Priming

Variables of importance that come to mind with regard to the determinants of priming effects include the prime–target delay (SOA) and the association strength (a) (for a review and model of single priming, see Brunel & Lavigne, 2009). In addition, SFA defined at the neuron level is reported as being central to the question of time-dependent processing of stimuli (see Morris et al., 2009; Wang, 1998). This neuron property can then interact with interstimuli delays and possibly with association strength. A first step toward analyzing the effects of SOA and association strength therefore requires a better understanding of their interactions with SFA to modify the levels of processing of the two successive primes. These effects should be visible on the relative magnitudes of priming from the first and from the second prime, that is, of RU and UR priming effects.

In the absence of SFA, Brunel and Lavigne's (2009) model accounts for a variety of single lag0 priming effects in terms of varying levels of target activation. The priming effects depend on the balance between activation received from the associated prime—and from items associated to both the prime and the target—and depends as well on overall feedback inhibition. The lag0 condition is similar to the UR condition, with additional prospective interference generated by the unrelated prime preceding the related prime and the target. In most studies on lag0 priming, a response is required on the target and not on the prime letter string, but the target of the preceding trial could generate proactive interference. For example, a few studies on multiple priming require subjects to make a response on each word in the sequence (McNamara, 1992a). In this case where each word is a trial from the point of view of the participant, the unrelated trial preceding a related prime (2) corresponds to the unrelated Prime 1 and is assumed to generate proactive interference. However, in studies on lag0 priming, a warning signal is presented before the prime. This warning signal does not activate a particular item in memory. It corresponds to a nonselective input that has been shown in a cortical network model to generate a peak of global feedback inhibition that "resets" the network by canceling retrospective activities of previous items (Brunel & Wang, 2001). In fact, a warning signal is used in studies on lag0 priming to "reset" the system and prevent from intertrial interferences. Such a "resetting" effect of the warning signal would therefore predict UR priming effects to be weaker than lag0 priming effects. On this basis, we analyze the effects of SFA on multiple priming using classical protocols of sequential presentation of two primes and a target (see legend of Figure 3).

Modeling of the Effects of SFA on Multiple Sequential Priming (MODSFA)

The current model reports lag0-UR priming for the nonadaptive case ($I^A_{\max} = 0$), although prospective activity of the related target is weaker than that in Brunel and Lavigne's (2009) model. This is to be related to the absence of "reset" of the network before presentation of the related Prime 2 in protocols of multiple priming, which permits proactive interference generated by the first unrelated prime (Haarmann & Usher, 2001). Indeed, feedback inhibition is weaker at the first prime onset than at the second prime onset. As a result, although both populations coding for the primes exhibit retrospective activity, priming from the second prime (UR) is weaker than priming from the first prime (RU), for the three values of a (Figure 3A) and SOA (Figure 3B) tested.

Considering the effects of SFA in the adaptive model, Figure 3A shows that although prospective activity of the UR target is stable across SOAs, association strength, and SFA, prospective activity of the RU target is more variable, being stronger for high values of association strength a and low values of $I^A_{\max}$. This is in accordance with the heterogeneity of RU priming shown by MA4. What results is a time-dependent cross between a previously activated RU associate and the last activated UR associate, which increases with $I^A_{\max}$. Figure 3B shows that the magnitude of the shift between prospective activities of associates to the first and/or second prime increases with SOAs and with $I^A_{\max}$. With very short SOAs, both RU and UR targets are weakly activated and show limited effects of SFA. With increasing SOAs, the effect of SFA becomes larger and switches the ratio of prospective activities of an RU associate on a UR associate. Then SFA can account for the larger variability in RU than that in UR priming by making RU priming more sensitive to the effects of SOA and association strength. SFA is then an important factor interacting with a and with SOA to improve the neural network's ability to switch from prospective activity of associates to previously processed inputs (Prime 1) to prospective activity of associates to new inputs (Prime 2).

Meta-analysis 4 (MA4, Sample 2 of All Effects Sizes on lag1-RU Effects, Including Subgroup of lag1-RU Effects from Sample 1): Effects of SOA on lag1-RU Priming

The analysis of the effects of SFA on multiple priming puts forward that in an adaptive network the magnitude of RU priming can greatly vary with SOA. It is to be related with the fact that RU priming is the less reliably reported effect of multiple priming in the literature. Given the sufficiently larger number of data on RU priming available in the literature, we will then focus on the meta-analysis of SOAs as moderator variables of RU priming, although not overlooking the RR and the UR conditions in the modeling approach. Considering RU priming when the words are processed sequentially permits us to clearly distinguish between SOA1—between Prime 1 and Prime 2 onsets—and SOA2—between Prime 2 and target onsets. The effects of SOAs have been extensively studied in research on single lag0 semantic priming, and SOAs have also been

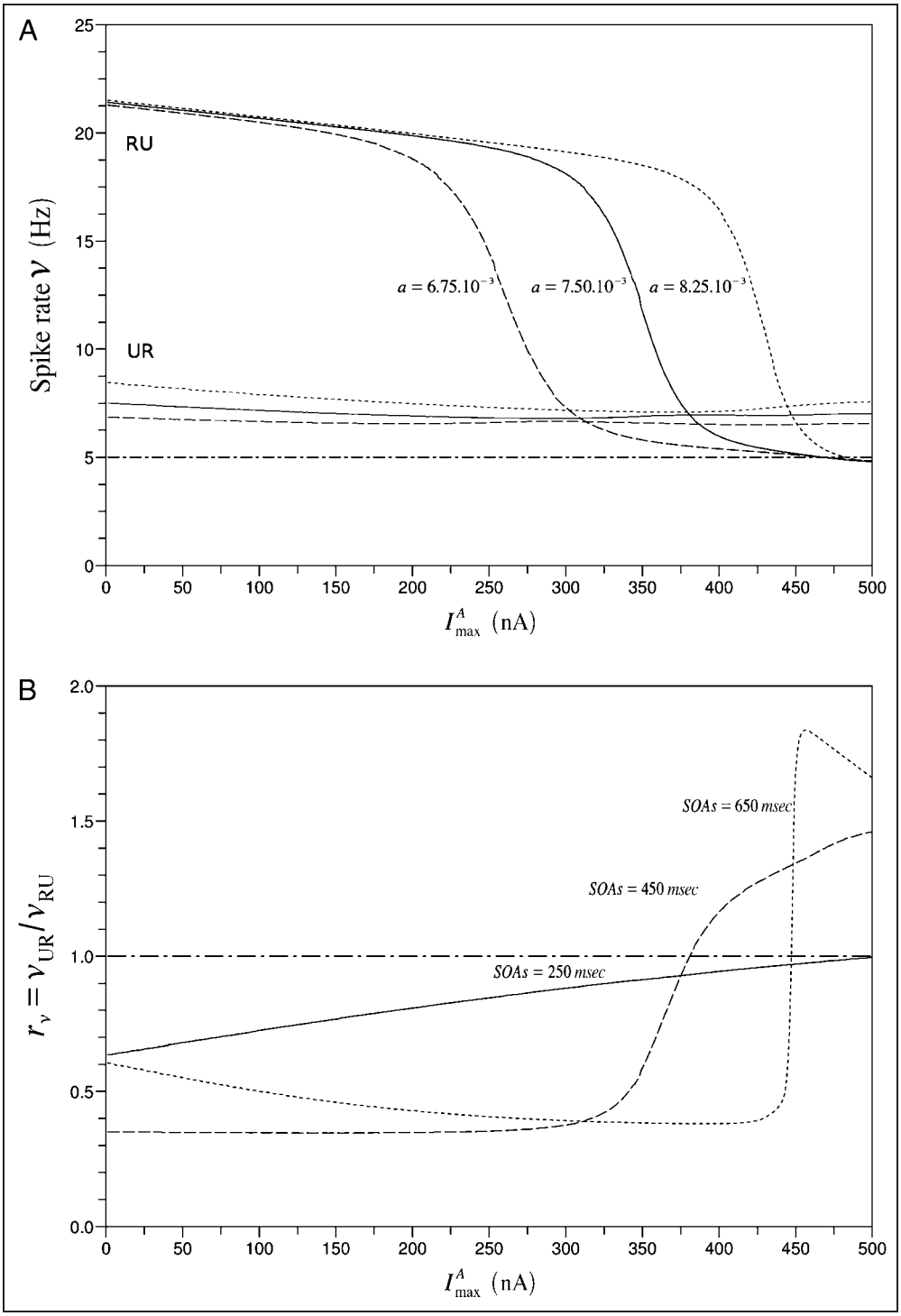

Figure 3. (A) Prospective activities (Hz) at target onset of populations coding for items associated to the first (RU, black curves) or second (UR, gray curves) prime as a function of I^A_{max} describing the strength of SFA every 1 nA. Results are displayed for three values of association strength a (6.75×10^{-3}, dashed curves; 7.50×10^{-3}, solid curves; and 8.25×10^{-3}, dotted curves) between the target and its associated prime and for SOA1 = SOA2 = 450 msec (I^{sel} = 50 μA). The level of spontaneous activity (v_s = 5 Hz; horizontal dash-dotted line) is displayed for reference. (B) Ratio of prospective activities of populations coding for items associated to the second (UR) over the first (RU) prime, $r_v = v_{UR}/v_{RU}$, indicating the shift from RU to UR priming as a function of I^A_{max}, for three values of SOA1 = SOA2 (250 msec, solid curve; 450 msec, dashed curve; and 650 msec, dotted curve; $t_{d1} = t_{d2}$ = 150 msec) (I^{sel} = 50 μA and $a = 7.5 \times 10^{-3}$). The line of equal effects is displayed for reference (r_v = 1; dash-dotted line).

manipulated in multiple priming studies (see Chenery et al., 2004; Balota & Paul, 1996). However, only one study by Lavigne and Vitu (1997) cross-manipulated SOA1 and SOA2 in a multiple sequential priming procedure. This allowed to investigate the respective time courses of facilitation by the related Prime 1 and of retroactive interference by the unrelated Prime 2. Lavigne and Vitu's results show that RR and UR priming effects do not depend on SOAs, but with regard to RU priming at short SOA1, SOA2 is a relevant parameter that accounts for apparently contradictory data in the literature: RU effects decrease with increasing SOA2, switching the pattern of multiple priming from being additive to being overadditive.

Generally, studies report priming effects in the RU condition for a variety of SOAs (e.g., Chenery et al., 2004; Balota & Paul, 1996). However, SOA1 and SOA2 are neither distinguished nor cross manipulated in these studies. Yet they can involve different priming processes that interact

with variable levels of proactive and retroactive interferences generated by the first and second prime. To assess for the combined effects of SOA1 and SOA2 on sequential lag1-RU priming, we selected for the meta-analysis the set of all 53 effect sizes corresponding to sequential lag1-RU priming. These studies used sequential presentation of items and provided values of SOAs that were either explicitly reported or that were calculable from word presentation durations, ISIs, and subject RTs. The set of all sequential lag1-RU effect sizes (Sample 2) was partitioned into two subsets according to the hypothesis that RU priming effects could be canceled under the double condition that (i) the target's activation by a related Prime 1 varies between short SOA1 (sSOA1) and long SOA1 (lSOA1), and (ii) it can be inhibited by an unrelated Prime 2 at long SOA2 (lSOA2) compared with short SOA2 (sSOA2). We then partitioned the distribution of lag1-RU priming effect sizes into two subgroups, defined as (1) short SOA1 and long SOA2 ($RU_{s/l}$) versus (2) short SOA1 and short SOA2 or long SOA1 ($RU_{s/1\text{-}l/s\text{-}l/1}$). (To fit the actual SOAs reported in the experiments, we set short SOA1 ≤ 500 msec and long SOA2 ≥ 450 msec.)

We then calculated the fit statistic Q_T for the distribution of lag1-RU effects to measure if its mean weighted effect size d' was a good representation of the distribution of effect sizes among studies. The significant fit statistic, $Q_{T,RU\text{-}SOA}(52) = 95.9$, $p < .01$, indicates a heterogeneous distribution of sequential lag1-RU priming effects. Between-group heterogeneity, $Q_{B,RU\text{-}SOA}(1) = 6.33$, $p < .05$, shows that RU priming is significantly weaker in the "short SOA1–long SOA2" condition ($d' = 0.13$) than that in other conditions of SOAs ($d' = 0.38$). Regarding the dependence of priming effects on SOA, it is assumed that postlexical processing of the prime–target pair occurs after the target is processed and is less sensitive to the pretarget SOA (see Neely, 1991). In the case of multiple priming, three experiments did not present subjects with the RR or UR condition (Lavigne & Vitu, 1997, Experiment 5; Joordens & Besner, 1992, Experiments 2 and 3). Lavigne and Vitu (1997) showed that when the Prime 2 was never related to the target in the experiment, RU priming was reliable independently of the SOAs. In this case, subjects could have more easily used strategies of processing the Prime 1–target relation and left aside the Prime 2 that was never related, minimizing the effect of retroactive interference and SOAs. When calculated on the remaining 50 effect sizes, the significant fit statistic, $Q_{T,RU\text{-}SOA}(49) = 72$, $p < .05$, and between-group heterogeneity, $Q_{B,RU\text{-}SOA}(1) = 11$, $p < .01$, confirms the sensitivity of lag1-RU priming effects on the combination of SOA1 and SOA2. In addition, MA4 reveals a significant within-group heterogeneity, $Q_{W,RU\text{-}SOA}(51) = 89.6$, $p < .01$, associated with significant heterogeneities of each subset, $Q_{RUs\text{-}l}(39) = 56.8$, $p < .05$ and $Q_{RUs\text{-}s/1\text{-}s/1\text{-}l}(12) = 32.7$, $p < .01$. This suggests that although SOAs account for some of the variability in RU priming, SOAs alone do not account for all of the variability in RU priming.

Modeling of Results from MA4 (MOD4, Same Protocols as Sample 2): Effects of SOA on lag1-RU Priming

We investigated the behavior of the model regarding the exact values of t_P and t_d defining SOAs in experimental protocols. As for the preceding simulations of results (MOD1–MOD4) and because association strength was not known in the sample used for the meta-analysis, MOD4 simulations were again carried out using the same fixed value ($a = 7.50 \times 10^{-3}$) as in the preceding simulations (see $MOD_{a\text{-}SOA}$ and Figure 5B2).

Data from simulation of lag1-RU priming show that the effect is correlated with the combination of SOAs used in the meta-analysis (Figure 4C and D; see also Figure 5B2). Data from simulation are coherent with experimental data in the long SOA2 range, wherein they both show a decrease of lag1-RU priming with increasing SOA2. However, the two sets of data show discrepancies in the short SOA2 range, wherein simulation data show increasing RU priming with increasing SOA1, whereas experimental data reveal no significant relationships. This suggests that the combination of SOAs alone does not account for all the variability in lag1-RU priming and points to the possible involvement of association strength in interaction with SOA. Observing that only two experiments yielded data for SOA1 greater than 1000 msec, and assuming that a parameter such as association strength varied from experiment to experiment, this made it possible for a difference to exist between mean values calculated over two studies at long SOA1 and over 48 studies at short SOA1, whereas this value was fixed in the simulations of MOD4. Thus, the model either faces an intrinsic limitation or predicts effects of association strength that are to be confirmed by further experiments. More experimental data are needed on this point, which led us to carry out additional experiments cross-varying SOA1 and SOA2 with fixed values of association strength (Dumercy, Darmon, Lavigne, & Vitu, in preparation). Results show that in the short SOA2 range of interest here, lag1-RU priming effects are stronger at long SOA1 than at short SOA1. This tends to confirm predictions of the model, although more experiments are needed that manipulate association strength to improve the reliability of the meta-analysis results.

Modeling of the Combined Effects of Association Strength and SOAs on Multiple Priming (MODa-SOA)

The heterogeneities of each subset of RU priming defined by SOAs means that although the combination of SOA1 and SOA2 accounts for a part of the variability in lag1-RU priming, an additional moderator variable still influences the magnitude of the effect. This challenges us to explore the synergistic effects of SOA and association strength on the pattern of multiple priming. However, a meta-analytic approach of the effects of association strength in multiple priming was prevented by two factors: (1) there were too few studies reporting precise values of the association

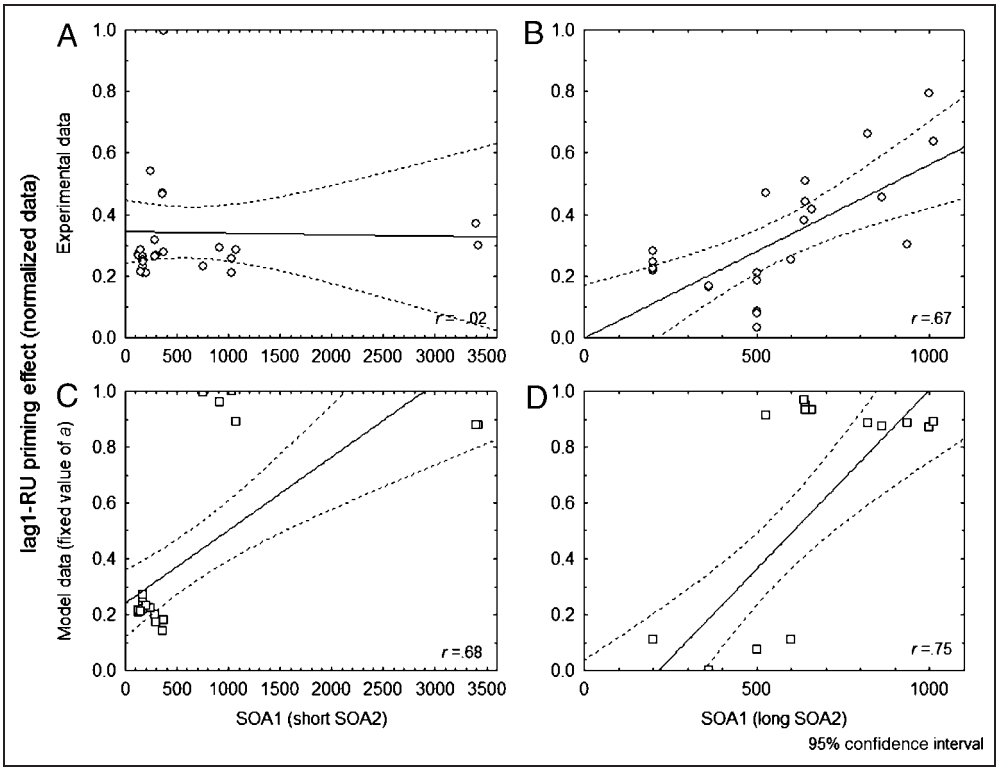

Figure 4. Fifty normalized effects sizes calculated from experimental data included in the meta-analysis of lag1-RU priming (A,B, circles), plotted as a function of SOA1 = t_{P1} + t_{d1} and short (A) versus long (B) SOA2 = t_{P2} + t_{d2} reported in experimental protocols. Fifty normalized RU priming effects calculated from simulation results computed with the same values of t_{P1}, t_{P2}, t_{d1}, and t_{d2} (C, D, squares) are plotted as a function of SOA1 and short (C) versus long (D) SOA2 ($a = 7.50 \times 10^{-3}$, $I^{sel} = 50$ μA, and $I^{A}_{max} = 420$ nA).

strength between each prime and target, and (2) this variable was not manipulated in multiple priming studies. At this point of the meta-analytical approach where groups cannot be partitioned any more, the modeling approach allows us to extrapolate testable predictions of the effects of association strength on multiple priming (Figure 5).

We first investigated all of RR, RU, and UR priming effects and the resulting pattern of additivity of priming within a wide range of the values of SOA1 and SOA2 reported in experimental data (150–650 msec). Simulations were carried out using the value of association strength used in MOD1–MOD7 (Figure 5A, B, C, and D2; $a = 7.5 \times 10^{-3}$), a 10% lower value (Figure 5A, B, C, and D1; $a = 6.75 \times 10^{-3}$), and a 10% higher value (Figure 5A, B, C, and D3; $a = 8.25 \times 10^{-3}$).

For the middle value of association strength (Figure 5A, B, C, and D2), the model interpolates the main differences between RR, RU, and UR effects reported in the experimental data. Regarding RR priming (Figure 5A2), it increases with the coincreasing SOA1 and SOA2 because of the progressive increase of prospective activity of the target generated by both Prime 1 and Prime 2. The effect of SFA on retrospective activity of the prime and on prospective activity of the target is compensated by the strong external current during Prime 1 and Prime 2 presentations, resulting in the increasing RR effect with SOA1 and SOA2. Considering RU priming (Figures 5B2 and 6B2), results show that it increases with increasing SOA1 because of increasing prospective activity of the target generated by the related Prime 1 (the effect of SFA being again compensated by the strong external current during Prime 1 presentation). RU priming decreases with SOA2 because of the combined effects of increased inhibition during Prime 2 input and SFA. Turning to UR priming (Figure 5C2), results reveal a reliable effect whose magnitude only increases with SOA2 because of increasing prospective activity of the target generated by the associated Prime 2. In addition, it is interesting that the resulting pattern of additivity is not stable across combinations of SOAs because of the strong dependence of RU priming on SOAs. Although strict additivity is possible within a large range of SOAs, including the ones tested experimentally (Chwilla & Kolk, 2003; Lavigne & Vitu, 1997; Balota & Paul, 1996), the pattern of multiple priming changes to overadditivity for combinations of short SOA1 and long SOA2 (Lavigne & Vitu, 1997).

Regarding the effects of association strength a on multiple priming, results are straightforward with regard to RR (Figure 5A) and UR (Figure 5C) priming in which retroactive interference is minimized due to the related Prime 2. Decreasing values of a decreases the overall magnitude of priming at a given combination of SOAs—that is, decreasing a delays priming effects, with a larger influence on RR than on UR priming. Focusing on RU priming (Figure 5B), we observe that increasing a leads to increased RU priming effects even in the short SOA1–long SOA2 cases where the effect of retroactive interference is the strongest. Moreover, the model predicts that high values of a can make lag1-RU priming resistant to long SOA2 (Figure 6B3). As a consequence of the resulting ubiquity of RU priming at all combinations of SOAs for high values of a (Figure 5B3), the additive pattern of priming is generalized to a wider

range of combinations of SOAs (Figure 5D3). We note that the generalization of the pattern of additivity is also obtained for low values of a because in this case, RR priming is reduced to a larger amount than RU priming. It then appears that in addition to SOAs, association strength is a crucial parameter influencing the overall pattern of RU priming and additivity, which accounts for the difference between some experimental data and simulations using a fixed value of a (Figure 4). In addition, it points to the importance of testing for the effect of strong association strength

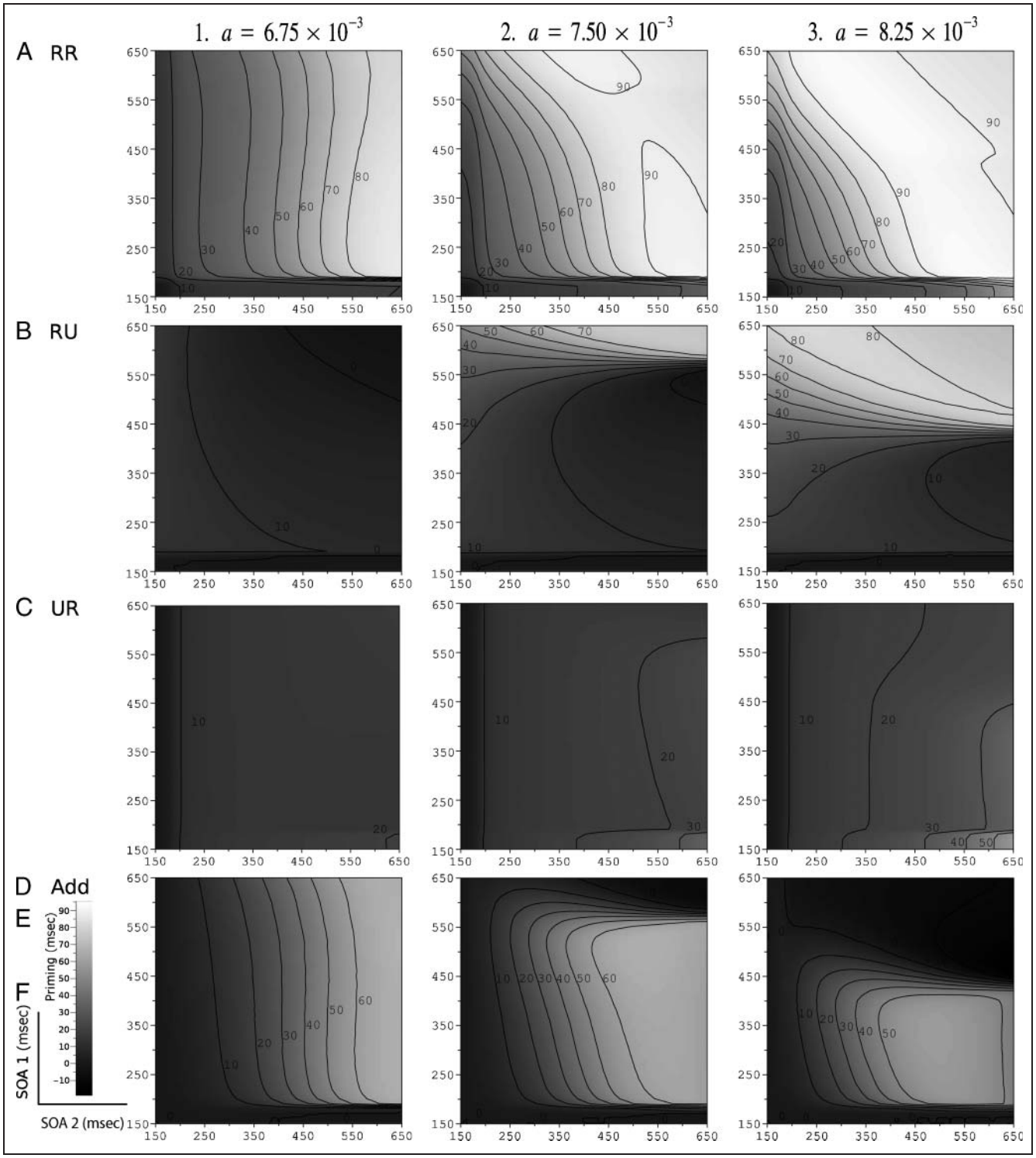

Figure 5. Magnitudes of multiple priming effects (msec) computed for three values of a (1: 6.75×10^{-3}; 2: 7.50×10^{-3}; 3: 8.25×10^{-3}): (A) RR priming; (B) RU priming; (C) UR priming; and (D) additivity (Equations 6 and 7) displayed as a function of F. SOA1 (y-axis) and SOA2 (x-axis) every 10 msec ($t_{d1} = t_{d2} = 150$ msec). (E) Darker (lighter) gray levels correspond to weaker (stronger) priming effects ($I^{\text{sel}} = 50$ μA and $I^{A}_{\max} = 420$ nA).

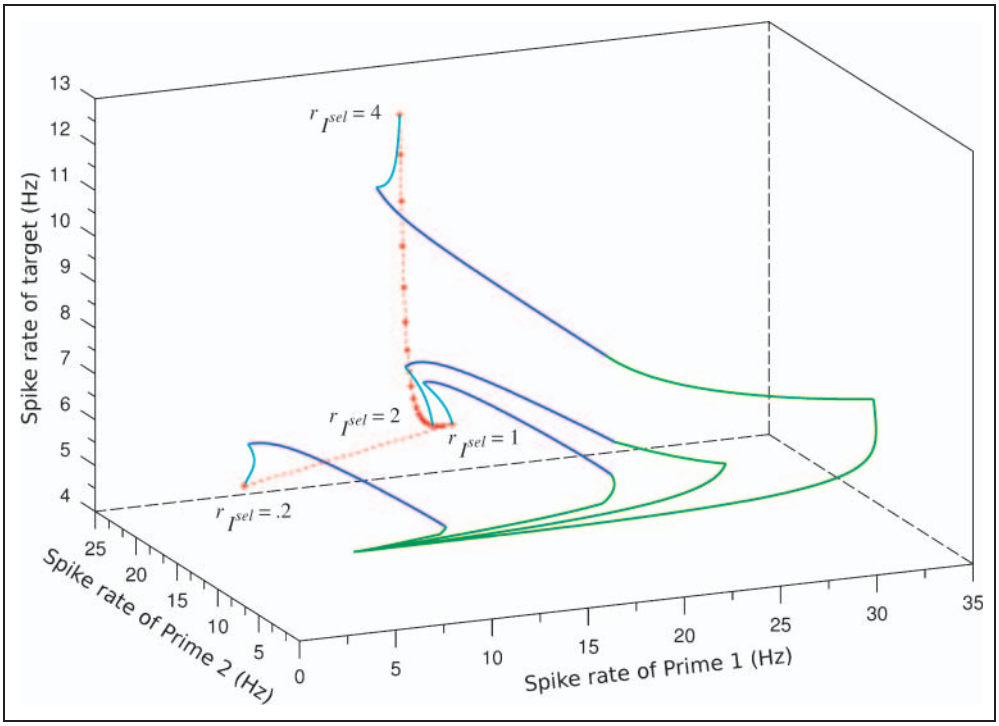

Figure 6. State diagram as a function of spike frequencies (Hz) of neural populations coding for Prime 1 (*x*-axis), Prime 2 (*y*-axis), and target (*z*-axis) during a standard protocol (SOA1 = SOA2 = 350 msec; $t_{d1} = t_{d2}$ = 150 msec) for different ratios $r_I^{sel} = I^{sel}_{prime1}/I^{sel}_{prime2}$. The green curves correspond to SOA1 (dark green curve = Prime 1 input and light green curve = ISI1), the blue curves correspond to SOA2 (dark blue curve = Prime 1 input and light blue curve = ISI2). The red curve shows the level of prospective activity of the neuron population coding for the target at target onset time, for a range of r_I^{sel} from 0.2 to 4 ($a = 7.5 \times 10^{-3}$, I^{sel}_{UR} = 50 μA, and I^{A}_{max} = 420 nA).

on the persistence of lag1-RU effects in the short SOA1–long SOA2 condition.

Modeling of the Effects of Primes' Intensity on lag1-RU Priming (MODpi)

Some data still contrast with the overall pattern of RU priming reported by the meta-analysis. Indeed, RU priming is reported to persist at long SOA2 when processing is oriented to the related Prime 1 because of either attentional cues (Abad et al., 2003; Fuentes et al., 1999) or strategic processing in cases where unrelated Prime 2 was never related to the target during the experiment (for studies in humans, see Becker et al., 1997; Lavigne & Vitu, 1997; Dannenbring & Briand, 1982; for a study in monkeys, see Takeda et al., 2005). RU priming can also be canceled at short SOA2 in cases of visual masking of the primes (Deacon, Hewitt, & Tamny, 1998; Masson et al., 1991). In these experiments, Prime 1 was backward masked by Prime 2 (Masson et al., 1991) or by a visual mask (Deacon et al., 1998), which may have diminished activation of Prime 1, and in turn diminished its priming effect on the target. This would then allow retroactive interference by unrelated Prime 2 to cancel priming (for comments, see Lavigne & Vitu, 1997; Balota & Paul, 1996). These studies suggest that the level of processing of the primes can influence the pattern of multiple priming. This is in accordance with studies reporting that the magnitude of various types of priming decreases when shallow processing of the prime is involved because of the type of experimental task, such as engaging in a letter search during presentation of the prime (Smith, Bentin, & Spalek, 2001; Kaye & Brown, 1985; Henik, Friedrich, & Kellogg, 1983; Smith, Theodor, & Franklin, 1983; for reviews, see Küper & Heil, 2008; Maxfield, 1997) or such as being faced with attentional distraction (Otsuka & Kawaguchi, 2007; Ortells et al., 2001; Fox, 1994, 1996; Ortells & Tudela, 1996; Chiappe & MacLeod, 1995; Tipper & Driver, 1988; Tipper & Baylis, 1987). Diverting a subject's cognitive processing away from the prime would result in reduced depth of processing and in reduced levels of activation of the prime in the subject's memory, which would then decrease if not totally cancel priming effects (Küper & Heil, 2008).

Regarding multiple priming effects, the pattern of lag1-RU priming depends highly on the relative effects of the related and unrelated prime on target activation. This points to the importance of the relative levels of processing of the primes, which are neither manipulated nor controlled in multiple priming experiments. In these protocols, the brightness of the stimuli is fixed. However, stimuli durations and interstimuli delays are different between Prime 1, Prime 2, and the target. As a consequence, at the onset of the stimuli (Prime 1, Prime 2, and target), the intensity of the screen's afterglow varies depending on the duration of the preceding stimulus and depending as well on the duration of the delay from its onset (i.e., warning signal before Prime 1, Prime 1 before Prime 2, and Prime 2 before target). In addition, the level of retinal persistence also varies with stimuli durations and interstimuli delays, leading to variable levels of perceptual processing between the successive items. This can in turn lead to different levels of stimulus saliency processed along the neural pathways, which is approximated in the modeling approach presented here by the fixed parameter of stimulus intensity of items during their presentation (I^{sel} = 50 μA).

In cortical network models, stimulus intensity has been reported to determine the level of visual response and subsequent retrospective activity of neurons coding for that stimulus as well as of the resistance of retrospective activity to new stimuli as a function of their own intensity (Brunel & Wang, 2001). This behavior predicts that stimulus intensity should influence the magnitude of priming effects as a function of SOA and of SFA during the time course of the protocol. It would vary before equilibrium at which the level of prospective activity of the target does not depend on prime intensity anymore but essentially depends on activation received from related Prime 1 through association strength a.

The activity of stimuli in memory can be determined by sensory processing but also by high-level processes that influence the level of activation of concepts in memory. For example, reliable RU priming effects are widely reported during processing of normal sentences whatever the number of words interposed between the related prime and target (lag) (Tree, Hirsh, & Monsell, 2005; Lavigne, Vitu, & d'Ydewalle, 2000; Joordens & Becker, 1997; Deacon, Mehta, Tinsley, & Nousak, 1995; Hess, Foss, & Caroll, 1995; McNamara, 1992a, 1992b; Sereno & Rayner, 1992; Vitu, 1991; Bentin & Feldman, 1990; Kutas, Van Petten, & Besson, 1988; Ratcliff, Hockley, & McKoon, 1985; Reder, 1983; Foss, 1982; Blank & Foss, 1978). This is to be compared with the unreliable RU priming effects when words are presented in scrambled sentences or word sequences (Faust, Bar-lev, & Chiarello, 2003; Faust & Chiarello, 1998; Sharkey & Sharkey, 1992; O'Seaghdah, 1989; Simpson, Peterson, Casteel, & Burgess, 1989; Foss, 1982) because of the fact that RU priming arises at lag 1 but do not span lags > 1 when syntactic processing is not involved (Faust et al., 2003; Foss, 1982). This suggests that in addition to bottom–up effects of sensory processing, top–down effects can influence the level of activation of the primes and modify the magnitude of lag1-RU priming.

We therefore investigated the dependence of RU priming on the relative intensities of the primes at preequilibrium SOAs where effects are reported as being more variable in the experimental literature (SOA1 = SOA2 = 350 msec; Figure 6). Results demonstrate that for a given intensity of the unrelated Prime 2, which triggers proactive interference in RU priming, increasing intensity of the related Prime 1 leads to increasing levels of prospective activity of the target at target onset. This permits RU priming to exhibit stronger resistance to the unrelated Prime 2 for higher intensities of Prime 1. These modeling results predict that the relative values of intensity of Prime 1 and Prime 2 determine the magnitude of RU priming at fixed SOAs. This points to the importance of controlling or manipulating variables in experiments on multiple priming that influence this intensity, such as screen afterglow and backward masking of the primes depending on ISIs. This could be achieved by masking the word stimuli or by presenting them in different positions or by varying their visual intensity and contrast.

Primes' intensity is manipulated in the model by modifying the value of external currents assumed to account for variations in the bottom–up visual signal. Under the assumption that external currents arriving at a population of neurons could have other origins, they could be interpreted to account for the influence of other variables that are exerting top–down effects. This is the case of syntactic processing during sentence comprehension which is reported to make RU effects resistant to lag > 1. Results presented in Figure 6 are in accordance with this assumption: They predict that further activation from syntactic processing that is received by the related Prime 1 could make it possible for RU priming to resist retroactive interference generated by the unrelated Prime 2. To test for the possibility of such a mechanism would require identification of syntactic effects on the activation of the primes generating lag-resistant RU priming during sentence processing.

DISCUSSION

The experimental literature on multiple priming effects showed discrepancies between the patterns of effects reported. Also, consensus in the research was still lacking regarding how the variety of effects are accounted for—effects that are a function of variables such as SOAs and association strength. The present research provides a comprehensive meta-analysis of the determinants of multiple priming effects as well as a model of how the cerebral cortex performs multiple priming as a function of procedural variables and of biophysic properties of neurons. The meta-analytic approach has allowed for the identification of the effects of combinations of SOAs on the pattern of additivity of multiple priming. The cortical network model interpolates the data for experimentally tested values of SOAs and extrapolates to a wide range of SOA combinations. It gives a general framework to interpret the additivity pattern of multiple priming in terms of a balance between prospective activity and retroactive interference in a model of the cerebral cortex. In addition, the model predicts that SOAs modulate priming effects by interacting with parameters embedded at the network level (association strength a, stimulus intensity I_i^{sel}, and SFA $I_{\max}^{A}$). The model does not require fine tuning of these parameters to specific values and exhibits qualitatively similar behaviors within a range of values of SOA, a, and $I_{\max}^{A}$ (Figure 3).

Synergistic Effects of Neural, Perceptive, and Semantic Properties on Multiple Priming

A main conclusion of this research, beyond identifying determinants of multiple priming and their effects on the pattern of additivity, is that these determinants act in close synergy at the network and procedural levels of processing. At the network level, SFA appears to be an important mechanism that prevents overly strong proactive interference from occurring by diminishing the activation of Prime 1

and its associates. In turn, this diminishes the level of feedback inhibition and allows for reliable UR priming. The ability to quickly switch anticipation from words processed in a sequence (as Prime 1 or Prime 2 associates) as a function of input properties (duration and intensity) is improved by mechanisms at the neuronal and network level such as activation, inhibitory feedback, and SFA. The method by which specific values of SFA are met at the population level to allow for behavioral effects could depend on its effects at the single neuron level and on properties of the neuron populations. These include, for instance, within-population connectivity and association strength (J_1), number of neurons, and types of neurons with different types of SFA. SFA is also important in determining the time course of RU priming effects as a function of SOAs defined at the procedural level (see Figure 3B for $I^A_{\max} = 420$ nA, and diagonals of SOA1 = SOA2 of Figure 5B). At short SOAs, the RU associate can be activated without much effect of SFA, and interference is too brief to cancel RU priming; at medium SOAs, the effect of SFA increases and adds to retroactive interference from the unrelated Prime 2, leading to cancellation of RU priming; and at long SOAs, RU associates to Prime 1 have reach a level of activation that is resistant to the combined effects of SFA and interference. Given that lag0 priming can be sustained at very long SOAs, the time course taken up by lag1-RU priming is not a passive phenomenon, in the sense that it requires the combined effects of association strength and SFA (which could be considered to be passive properties of the system, although SFA depends on the level of activation of a given neuron population) but also of the processing time of the unrelated Prime 2 (SOA2).

The switch between RU and UR priming depends not only on the relative processing durations of the primes (SOAs) but also on their relative intensities, which depend in turn on the relative positions of primes in multiple priming protocols. Processing time and intensity would then define the "perceptive" salience of a prime word, and the RU/UR switch could be interpreted as an adaptive mechanism allowing the system to activate words that are associated with either the previous or the subsequent word in a sequence, depending on their respective salience (of bottom–up or top–down origin). The most salient word would be selected in real time to direct recall of word associates and concept representations and would orient semantic anticipations in working memory. Words associated to Prime 1 would either resist interference from a less salient Prime 2 or be deactivated by a more salient Prime 2 to allow its associates to be activated. The relative amounts of proactive and retroactive interference on semantic priming could then depend on the relative salience of the primes.

Types of Semantic Relations

The meta-analyses presented here have included studies manipulating prime–target relatedness according to various types of relations not always reported and sometimes mixed in experimental studies. They include direct Step 1 associations, semantic relations through feature overlap (see Brunel & Lavigne, 2009; Hutchison, 2003 for reviews), and ambiguous target words (see Balota & Paul, 1996) but not cases where the two words are indirectly related (not tested in multiple priming, to our knowledge) or unrelated but part of larger contexts such as scripts (Chwilla & Kolk, 2005). This heterogeneity of the types of prime–target relations points to the possibility that the pattern of results could depend on the precise type of relation. Too few studies tested multiple priming effects using semantic relations such as feature overlap and coexemplars (see Chenery et al., 2004; Fuentes et al., 1999). Few more studies (10) explicitly tested multiple priming effects using primes related to the meanings of ambiguous target words (see Balota & Paul, 1996; Schvaneveldt, Meyer, & Becker, 1976). When tested in the meta-analysis, the pattern of multiple priming effects on unambiguous and ambiguous targets shows that RU priming is of smaller magnitude than RR priming. However, similar patterns of results do not demonstrate similar encoding of unambiguous and ambiguous targets in the matrix of the model, leaving open the question of the encoding of target ambiguity in the synaptic matrix and its effects on multiple priming. More generally, the current literature does provide us with a straightforward account of multiple priming effects as a function of the type of semantic relation involved. Brunel and Lavigne's (2009) modeling approach of single lag0 priming accounts for a large variety of qualitatively different types of lag0 priming effects in terms of quantitatively different time courses and magnitudes within a unified model. In this model, priming effects correspond to the propagation of activation between concepts and features all coded by neuron populations. Various types of semantic relations have been encoded by different synaptic matrices assumed to result from previous learning. Associations and feature overlap are described within a unified synaptic structure of neuron populations—in other words, a structure that codes concepts at the level of populations of neurons. Given that each population is specific to a given word, the coding at the population level resembles localist coding. However, some degree of overlap is possible between neuron populations, corresponding to a distributed coding of concepts. In Brunel and Lavigne's model as in the model proposed here, concepts could be encoded by random subsets of neurons that would generate overlaps between concepts, without qualitatively changing the effects observed (see Romani, Amit, & Mongillo, 2006; Curti, Mongillo, La Camera, & Amit, 2004). Here, we have chosen to study nonoverlapping neuron populations to simplify the description of the model, under the assumption that neuronal coding is sparse (Booth & Rolls, 1998).

With regard to multiple priming effects—given that the meta-analysis did not permit us to demonstrate an effect of the type of relation on the pattern of effects and because of the quantity of effects requiring investigation in multiple

priming—we have focused on the simplest case of direct association between the primes and the target embedded in a predefined synaptic matrix.

For the models simulations, we have therefore used the simple and generic relationship of direct association between primes and targets, as described in terms of the synaptic potentiations between populations of neurons—shown in Equation 5 in the Methods section of the model. As can be seen in Figure 3A, association strength interacts with SFA to determine the RU/UR switch from activation of previous word associates to activation of subsequent word associates. The strength of the association between these words and the primes would then determine semantic relevance of the associates relative to the sequence of primes. However, although studies on lag0 single priming and predictions from the current model of multiple priming indicate strong effects of association strength on the pattern of multiple priming effects, values of association strength are rarely reported and never manipulated in experiments. In consequence, they probably vary between protocols and even between conditions. In the literature on multiple priming, the lack of studies controlling for the type and strength of the association points to the need for a more thorough experimental investigation of its effects to compare with models developed on the basis of different synaptic matrices. Further research could test for the effects of differential association strength between each prime and its associated targets. This could be explored in human studies by cross-manipulating association strength and prime position, and it could be explored in monkey studies by manipulating the level of pair learning as the number of co-occurrences between each prime and its associate. Combined effects on behavioral and electrophysiological data would be of great help to further develop the model.

Direct associations have been reported to permit lag1 priming in monkeys (Takeda et al., 2005). The lag1 priming effects on retrospective activity of the primes and on prospective activity of the target as well as on RTs have been accounted for by the model. However, the results of Takeda et al. (2005) show that some neurons exhibit strong retrospective and weak prospective activities when their preferred stimulus is presented as prime (cue) or as target (test), respectively (i.e., neurons with "cue holding activity"), whereas some other neurons exhibit weak retrospective and stronger prospective activities when their preferred stimulus is presented as prime (cue) or as target (test), respectively (i.e., neurons with "target recall" activity). These results are very interesting in light of the question of association strength, in that they suggest that the variable levels of retrospective and prospective activities of different neuron populations coding for the prime and target could rely on heterogeneous values of association strengths between these neurons. Both types of neurons are activated when their preferred stimulus is presented as prime because they code for the prime or as target because they code for the target and are thus activated by those coding for the prime. However, their precise level of activity could differ because of different amounts of activation received from other neurons, through variable association strengths at the neuron level. The mean field approach used here suggests that variations in association strength could account for such effects at the population level, but a model of integrate and fire neurons would be more appropriate to test for this possibility at the neuron level. The variability in interneuron association strengths after a given learning protocol asks the question of the learning and precise nature of the variability of interneuron association strengths.

Learning of Association Strength

The modeling approach has permitted us to investigate the effects of association strength a between the primes and the target, defining realistic values of mean synaptic potentiation J_a between neuron populations (Table 1). In the present research, values of association strength could be varied between simulation trials, but they were fixed during trials and assumed to result from previous learning. Associations between items are reported to be learned in monkeys on the basis of temporal contiguity (Booth & Rolls, 1998; Yakovlev, Fusi, Berman, & Zohary, 1998; Sakai & Miyashita, 1991; Stryker, 1991; Miyashita, 1988). This is in agreement with results from human studies showing that priming effects are inversely proportional to the lexical distance and proportional to the frequency of co-occurrence between words in texts (Spence & Owens, 1990; Postman & Keppel, 1970; Deese, 1965; see Prior & Bentin, 2003, 2008). In network models, the Hebbian rule of synaptic potentiation/depression subtending learning at the neuron and population levels leads to associations between items proportional to the number of temporally contiguous occurrences of these items (Mongillo et al., 2003; Brunel, Carusi, & Fusi, 1998; Brunel, 1996), defining synaptic matrices that are qualitatively similar to the one used here (for other types of matrices, see Brunel & Lavigne, 2009). In addition, some studies (e.g., Mongillo et al., 2003) show how learned associations between primes and targets permit prospective activity to happen during simple lag0 priming, such as reported in monkeys (Erickson & Desimone, 1999). In computational models of the cerebral cortex, association strength is slowly learned through unsupervised Hebbian learning. Learning is unsupervised in the sense that neuron activity, taken into account by the Hebb rule, is determined solely by input and not by a "desired" output selected *a priori*. This is a realistic learning procedure at the synaptic level in that it involves a simple and classic Hebb rule related to spike timing-dependent potentiation (Graupner & Brunel, 2007) and does not involve error backpropagation. This learning procedure is also realistic at the behavioral level in that it does not require "intentional" supervision from an external source based on "desired" behaviors. Here, the stimuli themselves are the inputs that "supervise," in the sense that they determine learning of

associations with other co-occurrent or time-correlated stimuli (see Mongillo et al., 2003). It differs from the supervised learning procedures used in most connectionist models, except for the Hopfield-type distributed networks (for discussions, see Becker et al., 1997; Masson et al., 1991).

In models of learning in the cerebral cortex, synaptic weights usually change on a time scale far slower than short-term forms of priming processes, mostly because slow learning is shown to be a condition for memory stability and storage capacity (see Brunel et al., 1998). The possibility for rapid synaptic learning occurring during the experiment has been reported to permit long-term forms of priming at lags > 1 and at long delays of several minutes (i.e., see "long-term priming" in Becker et al., 1997). This effect was modeled by a rapid learning rule in a connectionist network, which increased associations between neurons coding for features of the prime, including certain features of the target, during the experiment. This mechanism of enhanced association between target features during prime processing makes priming resistant to interference for several seconds and up to lag8. Consequently, it makes priming effects independent of prime–target SOAs and lag, which is not in accordance with the meta-analysis of the data on multiple priming reporting no significant priming at lag > 1. An explanation is that in the study of Becker et al. (1997), long-lasting resistance to interference was not reported with the lexical decision task but was reported with the semantic judgment task. The latter was prone to greater effects of strategic processes of recovering the prime in STM during target processing, which is known to enhance priming effects (see Balota & Paul, 1996; Neely, 1991). This points to the central importance of accounting for the differential effects of experimental tasks on the magnitude of priming and on the pattern of multiple priming. This poses a great challenge to models of priming, to be linked with the mechanism of rapid learning during priming trials. It also motivates further research in that direction. Although the current model accounts for a lot of data by assuming that learning is slow compared with a trial duration, fast synaptic potentiation has been considered to be a relevant mechanism of rapid relearning in a cortical network model of monkey behavior. This was explored in experiments where rules of stimuli–responses associations were rapidly switched (Fusi, Asaad, Miller, & Wang, 2007). Short-term synaptic facilitation has also been considered to be a mechanism of activation in working memory (Mongillo, Barak, & Tsodyks, 2008). Rapid and slow learning are not exclusive mechanisms and could then combine to influence the pattern of multiple priming.

Multiple Priming in Working Memory

The multiple priming paradigm involves not only processes of activation and inhibition within the semantic structure but also processes of maintenance of, and of joint priming from, several items simultaneously activated in working memory. Modeling of such phenomena at the neural level provides us with new insights as to how concepts are processed and organized together, on the basis of priming and selection processes in working memory, as a function of their semantic relations in long-term memory and of the sequence of items presented. During the processing of a sequence of items, items that are actually presented as well as their associates are activated in working memory. Nonselective inhibitory feedback sets a limit on the capacity of the working memory system, which prevents runaway excitation and permits selective activation of neuron populations. The number of coactivated populations coding for items depends on their precise levels of activation (see Amit et al., 2003), themselves depending on the synaptic matrix and network dynamics (protocol, SFA, neuron dynamics). It follows then that although working memory capacity is constant in terms of the global level of activation allowed by global feedback inhibition, it can be variable in terms of the number of items activated, depending on their precise levels of activation (Brunel & Lavigne, 2009). The lower the level of activation of items, the more items can be activated simultaneously. Although all three types of multiple priming effects (RR, RU, and UR) involve activation of the target by the associated prime(s), they also involve interference by all the other populations activated during the protocol. This implies that for all types of multiple priming, facilitation and interference do not function as all-or-nothing notions but correspond rather to combinations of activation and inhibition of various origins. On one hand, the RU and the UR conditions correspond to weak facilitation in two ways: (1) lesser activation received by the target from only one associated prime and (2) stronger inhibition due to the activation of two groups of $p_i = 3$ items (two primes and associates in each group, including the target). On the other hand, the RR condition corresponds to strong facilitation in two ways: (1) more activation received from two related primes from the same group and (2) less inhibition because of the activation of $p_i = 3$ items only. Consequently, the effects of two related primes in the RR condition appear to be of greater magnitude when both primes are presented for more than 50 msec than when one of them is presented for less than 50 msec (Figure 5A). In addition, different patterns of additivity are a combination of variable levels of additivity in the RR condition and variable levels of proactive or retroactive interference in the RU and UR conditions, respectively. Strictly additive effects correspond to cases of slight interference by the unrelated prime in the RU or UR conditions (Figure 5D2, short SOA2). Underadditive effects correspond to cases of maximal effects in the RU or UR condition (Figure 5D2, long SOA1 and short SOA1, respectively). Overadditive effects correspond mainly to cases of strong retroactive interference in the RU condition (Figure 5D2, long SOA2). The modeling approach of these processes provides us with new ways of describing resistance to interference and recall performance in terms of activation and inhibition of a target during multiple prim-

ing processes, depending on the timing of presentation, condition of relatedness and synaptic matrix.

The model presented here makes no assumption as to the topographic localization or relative positions of the neurons involved in multiple priming. However, experimental studies allow us to point out some areas reported to be involved in semantic priming. Electrophysiological data from monkeys point to the role of the temporal and prefrontal areas in semantic priming. Prospective activity has been reported in monkeys in the PF (Rainer et al., 1999), AIT, and perirhinal cortices (Naya et al., 2001, 2003; Yoshida et al., 2003; Erickson & Desimone, 1999; Sakai & Miyashita, 1991; see Murray, Baxter, & Gaffan, 1998; Buckley & Gaffan, 1998a, 1998b). Likewise, semantic processing is reported in humans to involve the frontal cortex (Gough, Nobre, & Devlin, 2005; Khateb et al., 2003) and the anterior temporal lobe (Mummery, Shallice, & Price, 1999; Nobre & McCarthy, 1995; Nobre, Allison, & McCarthy, 1994; see Henson, 2003). A recent fMRI study reports that increasing semantic relatedness is associated with increased activation in the left inferofrontal, bilateral mediofrontal, and right mediotemporal gyri ("semantic enhancement"; Raposo, Moss, Stamatakis, & Tyler, 2006; see also Kuperberg, Deckersbach, Holt, Goff, & West, 2007; Tivarus, Ibinson, Hillier, Schmalbrock, & Beversdorf, 2006).

Combined Constraints from Experimental Data and Model Predictions

The approach presented here, which combined a set of experimental data and modeling results, aims to contribute to closing the gap between cognitive and brain processes by linking the behavior of subjects to that of neuron populations. The success of such approaches depends on the reliability of the experimental data and the realism of the model used, but a good fit of the data does not ensure that the model parameters and their values are either the necessary ones or the sufficient ones. That is why the model must be able to tackle larger sets of effects reported in the literature and make clear and straightforward predictions of effects to be tested experimentally. Regarding the statistics of the priming effects to be modeled, a test of not only their magnitude but also their variability would require the addition of stochastic external noise currents as external inputs to the model. In addition, external noise could have effects on the dynamics of multiple priming in that it has been proven to permit sequential deactivation and prevent global forgetting in working memory (Amit et al., 2003). Recent research has shown that the statistics of firing rates during retrospective activity (i.e., spike rates and coefficient of variation of the interspike interval) can be reproduced in a model if synaptic efficacies are a nonlinear function of the presynaptic firing rate because of a short-term depression mechanism (Barbieri & Brunel, 2007). The choice for a noiseless version of the model proposed here was guided by the need for simplicity in the description of the effects and in the interpretation of population dynamics. However, taking short-term synaptic depression into account could be a way to investigate the effects of stochastic noise on retrospective and prospective activities in priming.

The model proposed here fits many of the results of the meta-analysis on multiple priming. On this basis, it predicts the magnitude of the three conditions of multiple priming effects within a wide range of values of SOA and association strength. These two variables having not yet been cross manipulated in experimental studies, additional results would enable testing of the model's robustness. In addition, the model predicts that the relative intensities of activation of populations coding for Prime 1 and Prime 2 also influence the magnitude of lag1-RU. Testing the model could be done by testing, at lag1 and lag > 1, the effects of variables influencing the time during which the population coding for the prime is strongly activated. This could be done by manipulating the prime duration, the type of processing of the prime (i.e., lexical vs. semantic, as defined by the task), and syntactic factors permitting sustained activation of the prime and/or target. Regarding the effects of target ambiguity, an interesting direction that could combine experimental and modeling approaches would be to explore single and multiple priming of ambiguous targets by testing different types of ambiguous coding in the synaptic matrix and by comparing their effects on priming of ambiguous targets. Regarding the effects of association strength and of the type of semantic relation between the related prime and the target, the nonadaptive cortical network model of semantic priming proposed by Brunel and Lavigne (2009) suggests that the type of semantic relation as well as the association strength would modify the pattern of effects. An interesting extension would be to embed Brunel and Lavigne's matrices in the current adaptive model, but further behavioral data are needed for a complete meta-analysis of these variables and to put forth strong predictions of the model behavior. In addition, studies in monkeys could permit certain parameters to be manipulated that are hardly manipulable in humans, such as learned association strength as defined in terms of item co-occurrence and type of relation as defined in terms of the structure of the semantic field of the prime and target (see Brunel & Lavigne, 2009). Electrophysiological recording of neuron activity during multiple priming protocols could then lead to correlation between these parameters, neuron activity, and response times. Data at the behavioral and neuronal levels would therefore enable researchers to test model predictions as well as to propose future developments.

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Note: Studies included in the meta-analysis are not all cited in the text but are marked by asterisks (*) in the Reference list.

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