Synaptic Theory of Working Memory for Serial Order

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Abstract

Working Memory (WM) enables the temporally-ordered maintenance of sequences of stimuli over short periods of time. This ability is critical for many cognitive and behavioral tasks. Despite its importance, however, how WM encodes, stores and retrieves information about serial order remains a major outstanding problem. Here, we extend our previously-proposed synaptic theory of WM to include synaptic augmentation, as experimentally observed at the same synapses that feature short-term facilitation. We find that synaptic augmentation leads to the emergence of a primacy gradient that can be used to reconstruct the order of presentation at recall, by an appropriate control of the background input to the WM network. The model reproduces prominent features of the behavior of human subjects recalling lists of items and makes a series of experimentally-testable predictions. Intriguingly, the model suggests that WM capacity limitations could result from a failure in retrieving, rather than encoding, information.

Keywords: serial order, working memory, synaptic augmentation, recurrent network model

Introduction

Adaptive behavior requires storing and updating relevant information over multiple time scales. Over short time scales, this ability is supported by the Working Memory (WM), a specialized component of the memory system (Cowan, 2001; Baddeley, 2003). The guidance of behavior, decision-making and, indeed, practically any cognitive function rely critically on WM function.

A defining feature of WM is its surprisingly small capacity, conventionally estimated to be 4 items or chunks (Cowan, 2001). For comparison, people can store in the visual long-term memory thousands of pictures with an astonishing detail (Standing, 1973; Brady et al., 2008). The encoding of serial order information is another defining feature of WM (Lewandowsky and Farrell, 2008; Hurlstone et al., 2014). This is not surprising; the information in WM has, typically, a temporal component. For instance, to reach the closest coffee place we just asked directions to, we have to turn left at the next corner, walk one block, and then turn right. We'll get no espresso following the directions in the *wrong* order. Experimentally, the encoding of serial order in WM is studied with the *serial recall* task (Kahana, 2012). In serial recall, a list of randomly chosen items (e.g. words) is presented, one at a time, to subjects that have to recall them in the presented order. For lists within capacity (typically up to 4 items), people usually perform without errors; for longer lists, subjects tend to omit the items late in the list (Lewandowsky and Farrell, 2008; Hurlstone et al., 2014).

Interestingly, people almost invariably recall short lists of up to 4 items in the presented order even without explicit instructions to do so, as in *free recall* experiments (Dimperio et al., 2005; Ward et al., 2010; Grenfell-Essam and Ward, 2012). For longer lists, people gradually forget more and more items, and report the recalled items in the "wrong" order, typically beginning from the end of the list. We illustrate this

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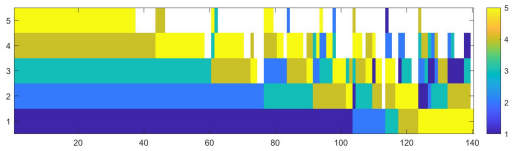

Figure 1: ***Spontaneous* emergence of serial order during free recall of 5-word lists.** For each of the 140 subjects, the words recalled are shown from bottom to top in the order they were recalled. The color indicates the serial position of the corresponding word in the presented list, from blue (first) to yellow (last). White indicates omissions. Data courtesy of G. Ward.

phenomenon by showing the results of free recall experiments for lists of 5 words that most subjects cannot recall completely (see Fig. 1; data courtesy of G. Ward). As can be seen in the figure, only about 30% of the subjects recalled the list without omissions (i.e., the list was within their WM capacity) and almost all of them recalled the 5 words in the order they were presented, even though the subjects were instructed to recall the words in an arbitrary order. On the other hand, the subjects that could not recall the full list (i.e., the list was above their WM capacity) exhibited significant variability in the recall order. Thus, it appears that WM inherently stores items together with information about the order in which they were presented, and only when WM is overloaded this information cannot be retrieved.

This suggests that the mechanisms responsible for capacity limitations and those responsible for the encoding of serial order are closely related. However, the models originally proposed for the computational architecture of WM did not provide any account for the encoding of serial order (Cowan, 2001; Baddeley, 2003). This shortcoming has been extensively addressed in subsequent work, reviewed in (Lewandowsky and Farrell, 2008; Hurlstone et al., 2014).

One class of models relies on rapid, *Hebbian*-like synaptic plasticity to form associations between the (neural representations of the) items or between the items and some *independent*, pre-existing representations that encode serial order, such as list positions or a temporal context signal, e.g., (Lewandowsky and Murdock Jr, 1989; Burgess and Hitch, 1999; Brown et al., 2000; Botvinick and Plaut, 2006). Another class of models relies on the notion of encoding strength in WM, which, in turn, is assumed to affect recall so that the stronger the encoding of an item, the larger its probability of being recalled. Then, if one further assumes that the encoding strength of an item decreases with its position in the list, one obtains a primacy gradient that leads to a recall in the presented order, e.g., (Grossberg, 1978; Henson, 1998; Page and Norris, 1998). In all these models, the storage of the items and the encoding of their order rely on separate computational substrates whose neurophysiological underpinnings are left unspecified.

Mechanistic models of WM, on the other hand, have largely focused on the neurophysiological substrate of active maintenance and the ensuing capacity limitations. Early electrophysiological recordings pointed to persistent spiking activity as the neuronal correlate of active maintenance (Fuster, 1973; Miyashita and Chang, 1988; Goldman-Rakic, 1995; Amit, 1995). Subsequent work, however, has questioned the necessity of persistent activity for maintenance (LaRocque et al., 2014; Constantinidis et al., 2018; Lundqvist et al., 2018). We have proposed a theory – the synaptic theory of WM – that does not require persistent activity for maintaining information in WM (Mongillo et al., 2008). The theory is broadly compatible with multiple experimental observations and motivated further experiments aimed at disentangling persistent activity and information maintenance (Rose et al., 2016; Wolff et al., 2017).

According to the synaptic theory of WM, the information is stored in the level of short-term synaptic facilitation within neuronal populations that code for the items. Short-term facilitation is an experimentally well-characterized transient enhancement of the synaptic efficacy that is quickly induced by pre-synaptic spiking activity and can last for up to several seconds (Zucker and Regehr, 2002; Markram et al., 1998). In particular, short-term facilitation was reported at inter-pyramidal connections in the prefrontal cortex, a region heavily implicated in WM (Hempel et al., 2000; Wang et al., 2006). In the framework of the synaptic theory, the maintenance of information in WM can be achieved via different regimes of neuronal activity, depending on the background input to the network; at increasing levels of the background input, the regimes are: (i) activity-silent regime, where the information is transiently maintained without enhanced

spiking activity; (ii) low-activity regime, where the information is periodically refreshed, at low rate, by brief spontaneous reactivations of corresponding neuronal populations (i.e., population spikes, PSs); (iii) persistent-activity regime, where the information is maintained by tonically active neuronal populations. In a subsequent study, we clarified the origin of the capacity limitations in the low-activity regime (Mi et al., 2017). The storage capacity predicted by the theory, using experimental measures of short-term plasticity at cortical synapses, is consistent with typical memory spans reported in behavioral studies. However, similarly to other neurophysiologically-grounded theories of WM (e.g., (Amit and Brunel, 1997; Edin et al., 2009)), the synaptic theory does not provide an account for the encoding of serial order information.

In the present contribution, we propose that transient synaptic enhancement on multiple time scales provides a plausible mechanism to encode serial order information within the framework of the synaptic theory of WM. Specifically, we extend the theory to include synaptic augmentation: an enhancement of the synaptic efficacy that slowly builds up with repetitive pre-synaptic activity and that, once induced, persists over tens of seconds in the absence of activity (Fisher et al., 1997; Thomson, 2000; Fioravante and Regehr, 2011). Importantly, experiments reveal that augmentation is observed at the same synapses in the prefrontal cortex that exhibit significant short-term facilitation (Hempel et al., 2000; Wang et al., 2006). We find that, when the network operates in the low-activity regime, synaptic augmentation naturally leads to the emergence of a primacy gradient that encodes the presentation order of the items.

Results

To illustrate the putative role of synaptic augmentation in the encoding of serial-order information, we consider the simplified setting used in (Mi et al., 2017). To recapitulate, the network is composed of P distinct excitatory populations, that represent the memory items, and one inhibitory population, that prevents simultaneous enhanced activity in the excitatory populations. The recurrent synaptic connections within each excitatory population display short-term synaptic plasticity according to the Tsodyks-Markram (TM) model (Markram et al., 1998). The population-averaged synaptic input to population a ($a = 1, \ldots, P$), h_a, evolves in time according to

$$\tau \dot{h}_a = -h_a + I_a(t) + A_{EE} u_a x_a r_a - A_{EI} r_I \tag{1}$$

where τ is the neuronal time constant; $I_a(t)$, the external input to population a, is the sum of two components: a background input, to control the activity regime of the network, and a selective input, to elicit enhanced activity during the presentation of the corresponding item; A_{EE} is the average strength of the synapses within an excitatory population; r_a, the average activity of population a, is a smoothed threshold-linear function of h_a, i.e.,

$$r_a = \phi\left(h_a\right) \equiv \alpha \log\left(1 + \exp\left(\frac{h_a}{\alpha}\right)\right) \tag{2}$$

where $\alpha > 0$ is a parameter controlling the smoothing; u_a and x_a are, respectively, the levels of short-term facilitation and depression of the recurrent synapses within population a; A_{EI} is the strength of the synapses from the inhibitory population to any excitatory population; $r_I = \phi\left(h_I\right)$ is the average activity of the inhibitory population, and

$$\tau \dot{h}_I = -h_I + I_I + A_{IE} \sum_{a=1}^{P} r_a \tag{3}$$

where I_I is the constant background input to the inhibitory population and A_{IE} is the strength of the synapses from any excitatory population to the inhibitory population.

The levels of short-term facilitation and depression, u_a and x_a, evolve in time according to

$$\dot{u}_a = \frac{U_a - u_a}{\tau_F} + U_a (1 - u_a) r_a \tag{4}$$

$$\dot{x}_a = \frac{1 - x_a}{\tau_D} - u_a x_a r_a \tag{5}$$

where U_a is the baseline release probability of the recurrent synapses within population a ($a = 1, \ldots, P$); τ_F and τ_D are the facilitation and depression time constants, respectively. In words: Activity in the population induces both facilitation, i.e., it increases u_a, and depression, i.e., it decreases x_a, while, in the absence of activity (i.e., $r_a = 0$), facilitation and depression decay to their respective baseline levels, $u_a = U_a$ and $x_a = 1$.

In (Mi et al., 2017), the U_a's in Equation (4) are time-independent parameters with the same value for all the excitatory populations. By contrast here, to model synaptic augmentation, the U_a's are activity-dependent dynamic variables that increase with the r_a's according to

$$\dot{U}_a = \frac{U_0 - U_a}{\tau_A} + K_A (1 - U_a) r_a \tag{6}$$

where U_0 is the basal release probability (i.e., following a long period of synaptic inactivity), τ_A is the augmentation time constant, and the parameter K_A controls how fast the baseline release probability increases with the activity.

The physiological mechanisms responsible for synaptic augmentation are poorly understood. Nevertheless, the empirical evidence suggests that augmentation results from an increase in the release probability rather than an increase in the number of release sites and/or an increase in the unitary quantal response (Fisher et al., 1997; Thomson, 2000; Fioravante and Regehr, 2011). Equation (6) provides a minimal phenomenological description of such an increase in the release probability, in the spirit of the original TM model (Markram et al., 1998). The description of augmentation requires only two additional parameters (i.e., K_A and τ_A) as compared to the TM model. However, as it will become clear in the following, our results do not critically depend on this modeling choice. For instance, one would obtain the same results by modeling augmentation as an activity-dependent increase in the synaptic strength A_{EE}.

Facilitating synaptic transmission observed at inter-pyramidal synapses in the prefrontal cortex is well reproduced by the above model with the following choice of synaptic parameters: $U_0 \sim 0.2$, $\tau_F \sim 1$s, $\tau_D \sim 0.1$s, $\tau_A \sim 10$s and $K_A \ll 1$ (Hempel et al., 2000; Wang et al., 2006; Barri et al., 2016). In Fig. 2, we illustrate the model described by Equation (1) (with $I_a(t) = 0$, $r_I = 0$ and $A_{EE} = 1/U_0$) and Equations (4)-(6), when driven by a train of 10 spikes at 50Hz, followed by 1 spike 500ms after the end of the train and 1 spike 10s after the end of the train (top panel). The equations are solved with substituting the firing activity, $r_a(t)$, with a sum of delta functions corresponding to the pre-synaptic spikes: $r_a(t) = \sum_{k=1}^{12} \delta(t - t_k)$, where the sum is over all spike times, t_k.

Repetitive synaptic activation at a rate much larger than $1/\tau_D$ induces significant short-term depression (i.e., the decrease of x; middle panel), as can be seen by comparing the response to the first spike in the train with the response to the last spike in the train (bottom panel). However, high-rate activity also induces short-term facilitation and augmentation (i.e., the increase of u and U; middle panel). Due to the difference in time scales (i.e., $\tau_D \ll \tau_F \ll \tau_A$), both short-term facilitation and augmentation are still present after a period of inactivity long enough to allow the almost-complete recovery from short-term depression. This can be seen in the responses to the two isolated spikes. Note that the response to the last spike, 10s after the end of the train, is still slightly larger than the response to the first spike due to augmentation.

The full set of network and short-term plasticity parameters used in the simulations is summarized in Fig. 3. It is instructive to first understand why the model without augmentation ($K_A = 0$) *cannot* encode serial order. For illustration, in Fig. 3A we show the response of the network to a list of 3 items. The interval between presentations is 1.5 seconds, a typical rate of presentation in the experiments. At the end of the presentation, the neuronal populations that have been stimulated reactivate in a repeating cycle, indicating that the corresponding items have been stored in WM. This regime of activity, which results in a constant

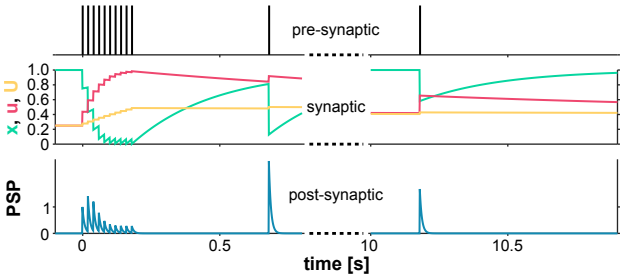

Figure 2: **Synaptic augmentation.** In response to pre-synaptic spiking activity (upper panel), the depression level, x, decreases while the facilitation and augmentation levels, u and U respectively, increase (middle panel). The slow decay of U produces an enhanced post-synaptic response long after x and u are back to their baseline levels (bottom panel). Parameters: top panel – train of 10 spikes at 50Hz followed by 1 spike 500ms after the end of the train, and 1 spike 10s after the end of the train; middle panel – $U_0 = 0.25$, $K_A = 0.0375$, $\tau_D = 0.3$s, $\tau_F = 1.5$s, $\tau_A = 20$s; bottom panel – Post-synaptic response are obtained by integrating the Equations (1), (5)-(6) with $\tau = 8$ms (see main text for details). The post-synaptic responses are normalized with the response to the first spike in the train.

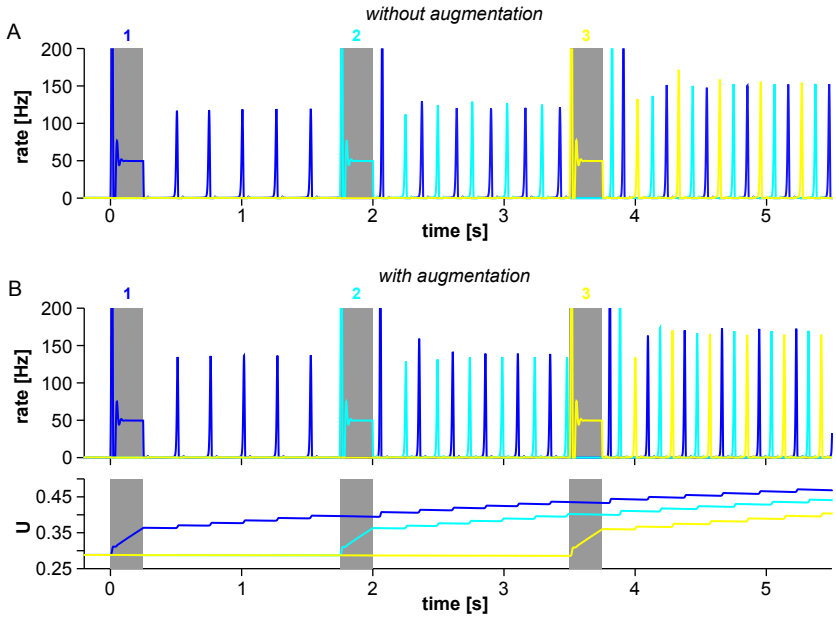

Figure 3: **The level of synaptic augmentation encodes serial order.** Network responses to 3 sequentially presented items without (A) and with synaptic augmentation (B). The bottom panel in (B) shows the level of synaptic augmentation in the corresponding synaptic populations. The presentation of an item is simulated by 14-fold increasing of the background input selectively to the corresponding neuronal population for 250ms (gray areas). The background input to the remaining populations is kept constant at its baseline level. The onsets of 2 consecutive presentations are separated by 1.75s. The population spikes in response to the onset of the stimulation are clipped for clarity of presentation (see Fig. 5). Network parameters: $P = 16$, $\tau = 8$ms, $\alpha = 1.5$Hz, $A_{EE} = 8.0$, $A_{EI} = 1.1$, $A_{IE} = 1.75$, $I_{bkg} = 8.0$Hz; Short-term plasticity parameters: $U_0 = 0.25$, $K_A = 0.0075$, $\tau_D = 0.3$s, $\tau_F = 1.5$s, $\tau_A = 20$s.

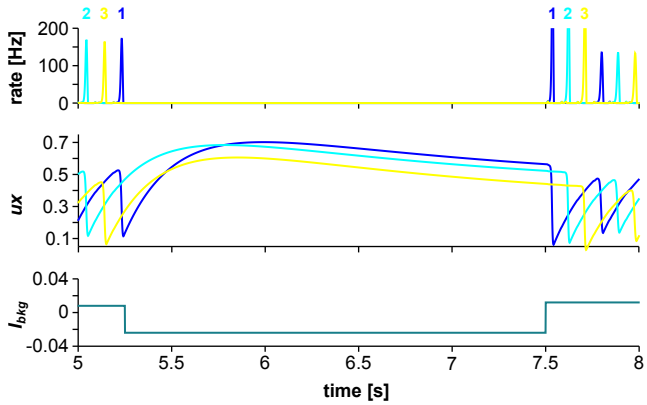

Figure 4: **Readout of serial order by background input.** Top panel: Response of the network in Fig. 3 to the background input depicted in the bottom panel. The background input undergoes a 4-fold decrease compared to its baseline level for $T_{supp} = 1.5\tau_F$ followed by 40% increase compared to its baseline level. The middle panel shows the resulting time course of ux in the corresponding synaptic population. Immediately before the background input is increased again, $ux \simeq U$.

order of reactivation (i.e., $1 \to 3 \to 2 \to 1 \to \cdots$ in Fig. 3A), is an attractor of the network dynamics. By symmetry, there is one attractor for each possible (cyclical) order of reactivations. In our specific example, there is only one other such attractor, corresponding to the order $1 \to 2 \to 3 \to 1 \to \cdots$. With 3 items, however, there are 6 possible orders of presentation. After the presentation, the network dynamics converge to one of the two attractors. In other words, the network dynamics will necessarily map *different* order of presentations onto the *same* attractor; the information about the order of presentation is asymptotically lost. At least in principle, information about the order of presentation could be extracted from the transient dynamics. In the model, however, the time to converge to the attractor(s) is on the order of a second (i.e., $\sim \tau_F$). Thus, transient effects are too short-lived to encode serial order on the time scales relevant to the experiments (i.e, tens of seconds).

The above discussion suggests two possible solutions: (i) having as many attractors as the possible orders of presentation; (ii) having suitably slow dynamics that transiently carries information about the order of presentation. To implement the first solution one would need, for instance, 24 different attractors to encode the possible orders of presentation of a list of 4 items. The coexistence of so many attractors, however, makes the dynamics extremely sensitive to the initial conditions (Pisarchik and Feudel, 2014). In this case, the attractor asymptotically reached would depend on the exact timing of the presentations rather than on their order. Regardless of the possible robustness issues, there is no obvious way of implementing this solution in our model without dramatically altering the underlying theoretical framework. Instead, as we now show, incorporating synaptic augmentation provides a natural implementation of the second solution.

In Fig. 3B, we show the response of the network with augmentation ($K_A > 0$) to the same protocol as in Fig. 3A. As before, the stimulated neuronal populations cyclically reactivate at the end of the presentation (Fig. 3B, top panel). Unlike before, however, this regime of activity does not correspond to a steady state (attractor) of the network dynamics. This is evident from the levels of synaptic augmentation in the reactivating neuronal populations, shown in the bottom panel of Fig. 3B, which are still changing with time. Similarly, as can be seen in the top panel of Fig. 3B, the amplitudes of PSs in each population are slightly different. Clearly, in a steady state the levels of synaptic augmentation as well as the amplitudes of the PSs are stationary and will have the same value for all the reactivating populations. This transient regime is long-lived because K_A is small and, hence, the level of augmentation grows rather slowly with each reactivation. Furthermore, the decay of the level of augmentation between two consecutive reactivations of the same population ($\sim \tau_D$) is negligible, because $\tau_D/\tau_A \ll 1$. Therefore, the longer an item has been in WM – that is, the larger the number of reactivations – the larger the corresponding level of augmentation.

In summary, the simulation shows that synaptic augmentation transiently induces a *primacy* gradient; the neuronal populations encoding items earlier in the list have larger augmentation levels. The duration of

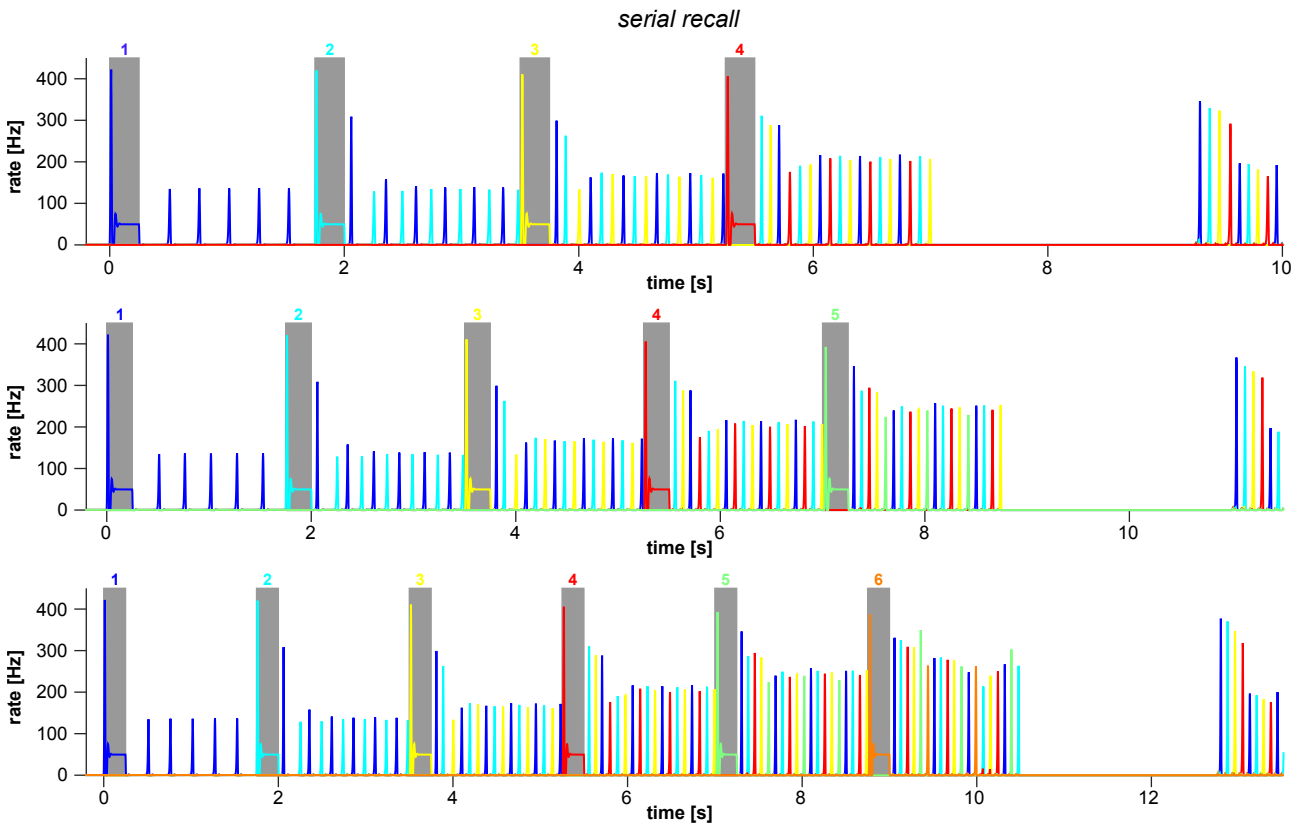

Figure 5: **Serial recall is primacy-dominated.** Network response to lists with an increasing number of items (from top to bottom). The background input is set to a level that allows the reactivation of the loaded items in between the presentations (i.e., the same level as in Fig. 3). Serial order is read-out by using the same control of the background input as in Fig. 4.

these transient effects is compatible with the time scales relevant to the experiments. This primacy gradient, however, has no major effect on the neuronal dynamics and is, hence, largely *hidden* in the levels of synaptic augmentation of the different populations. Can such a primacy gradient be used to reconstruct the order of presentation at recall?

We suggest a plausible read-out mechanism, which relies on the order-of-magnitude difference between the decay times of facilitation and augmentation. It works as follows (see Fig. 4). Recall is initiated by decreasing the level of background input to the network for a time $T_{supp} \sim \tau_F$. This prevents further reactivations and the synaptic variables start decaying toward their baseline levels (see Fig. 4, middle panel). After T_{supp}, the background input is then raised again to its original level, or possibly to a larger level. The levels of augmentation, i.e., the U_a's, have hardly changed because $T_{supp} \ll \tau_A$. However, both depression and facilitation will be close to their corresponding baseline levels. That is,

$$x_a \simeq 1; \quad u_a \simeq U_a; \quad (a = 1, \ldots, P) \tag{7}$$

The primacy gradient in the augmentation levels has been *copied* into the facilitation levels. For the activated neuronal populations, the steady, low-rate state of activity is unstable, once the background input is raised again. Hence, they will start reactivating, with the most *unstable* one (i.e., with the larger u_a) reactivating first, the next most unstable one reactivating second, and so on (Mi et al., 2017). As a result, the reactivations follow the primacy gradient encoded in the augmentation levels (Fig. 4, top panel).

The proposed read-out mechanism is just one possible way of reconstructing the order of presentation from the primacy gradient. Another, less parsimonious, possibility is that a dedicated read-out network has access to the primacy gradient via the augmentation level of the synaptic connections it receives *from* the

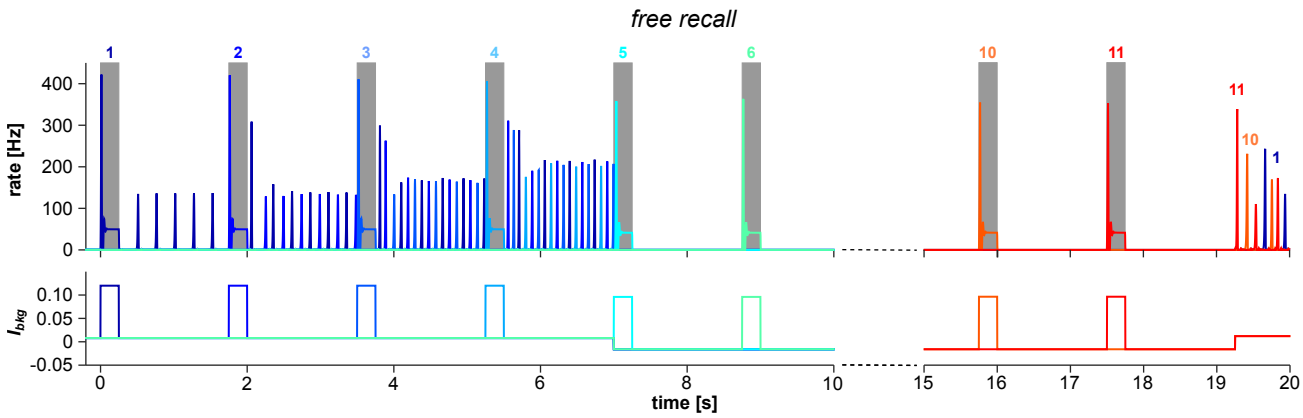

Figure 6: **Free recall is recency-dominated.** Top panel: Network response to a list of 11 items, largely exceeding the storage capacity. Bottom panel: Control of the background input. The background input is initially set at the same level as in Fig. 5 and, upon the presentation of the fifth item, it is reduced to a level preventing further reactivation (4-fold decrease compared to the baseline level). Recall is initiated 1.75s after the presentation of the last item by a 40% increase of the background input as compared to its baseline level.

neuronal populations in the memory network. In this case, the order of presentation could be reconstructed by a competitive queuing mechanism as originally proposed in (Grossberg, 1978).

In the simulation illustrated above, the number of items presented is below the capacity, as evidenced by the absence of omissions at recall. What happens when the number of items increases? This is illustrated in Fig. 5. As can be seen in the bottom panel of Fig. 5, up to 6 items are concurrently maintained by the network. Using the same read-out procedure as before, however, the network only recalls the first 4 items. The reason for this discrepancy is that the short-term facilitation level of the fifth item is significantly lower at retrieval than during the maintenance period. Therefore, at retrieval, it is overtaken by the first item (Mi et al., 2017). We note, however, that the augmentation level of the fifth item would allow reactivation in the *absence* of competing items.

In summary, the model reproduces the pronounced *primacy* effect observed in serial-recall tasks, where the subjects are able to recall the first few items (i.e., 3-4) in the correct order, while the probability to recall items later in the list declines as the number of items increases (Ward et al., 2010; Grenfell-Essam and Ward, 2012). In free-recall tasks with long lists, on the other hand, subjects exhibit a *recency* effect: they usually quickly recall the last few items, typically in reverse order, while the probability to recall earlier items declines with the list length (Murdock, 1962; Ward et al., 2010). Can the model also account for this experimental observation? We propose that the switch to recency-dominated recall when ordered recall is not required is an automatic consequence of overloading WM. In general, the strong augmentation levels of the active populations prevent new, potentially relevant, information from entering WM, once the capacity is exceeded. A simple solution to this problem consists in suppressing the reactivations by reducing the background input as soon as WM capacity is exceeded. Additional items can still be stored and passively maintained – by the presentation-induced increase in the facilitation and augmentation levels in the corresponding neuronal populations – and recalled – by increasing the background input to a suitable high level.

The response of the network to a list of 11 items, with the control of the background input just described, is illustrated in Fig. 6. At the end of the presentation, when the background input is increased, the last two items are recalled in the backward order. This is easily understood. In the absence of reactivations, the primacy gradient becomes a *recency* gradient, which is dominated by short-term facilitation effects, shortly after the presentation of the list. However, due to the initial reactivations, the populations enconding the first few items in the list can have significant levels of augmentation. In fact, the first item is still retrieved when the list is not too long.

Discussion

We have extended the synaptic theory of WM to include synaptic augmentation besides synaptic short-term depression and facilitation. We have shown that, in the low-activity regime, where items are maintained by short-lived reactivations of the corresponding neuronal populations, the presence of synaptic augmentation naturally leads to a transient primacy gradient that encodes the order of presentation of the items. This gradient can then be used to reconstruct the order of presentation at recall. The mechanism that generates the primacy gradient is robust, because it relies on the order-of-magnitude differences between the build-up and the decay time of the augmentation and those of short-term depression and facilitation.

Our model allows the storage and retrieval of short sequences of items by relying on synaptic plasticity mechanisms that are well-characterized experimentally, that is, the transient enchancement of synaptic efficacy driven by pre-synaptic activity (Fisher et al., 1997; Thomson, 2000; Fioravante and Regehr, 2011). Alternative models, as already pointed out, rely instead on some form of fast *associative* learning (Lewandowsky and Murdock Jr, 1989; Burgess and Hitch, 1999; Brown et al., 2000; Botvinick and Plaut, 2006). At the physiological level, associative learning is thought to entail long-term synaptic plasticity. This is because the induction of long-term synaptic plasticity is dependent on the joint pattern of pre- and post-synaptic activity, as required for associativity. However, there is presently no evidence that long-term synaptic plasticity can be induced and/or expressed on the relevant time scales, that is, the presentation of a single item during a serial-recall task (Lansner et al., 2023).

A key prediction of our theory is that multiple items are maintained in the low-activity regime. Indeed, if the items are maintained either in the activity-silent regime or in the persistent-activity regime, the proposed mechanism fails. In the first case, because in the absence of reactivations the gradient does not build up; in the second case, because the augmentation levels quickly saturate due to the enhanced firing rates. This prediction is consistent with recent experimental observations (Siegel et al., 2009; Fuentemilla et al., 2010; Lundqvist et al., 2016). In multi-item working memory tasks, the neuronal activity during the maintenance period is characterized by short episodes of spiking synchrony, detected as brief gamma bursts in the local field potential (Siegel et al., 2009; Lundqvist et al., 2016) or in the MEG/EEG signal (Fuentemilla et al., 2010). These episodes, which we identify with the population spikes in our model, are associated with the reactivation of the neural representation of the items, as evidenced by the fact that item's identity can be reliably decoded only during the gamma bursts. Importantly, during a given gamma burst, only information about one of the maintained items can be reliably decoded (Fuentemilla et al., 2010; Lundqvist et al., 2016), suggesting that the items are reactivated one at a time, as required by our theory.

In neurophysiological studies of working memory for sequences, the conjunctive coding of item identity and order information at the single-neuron level has been reported (Barone and Joseph, 1989; Funahashi et al., 1997; Xie et al., 2022). Conjunctive coding refers to the modulation of neuron's activity by both item and order information, so that, for instance, the average firing rate of the neuron during the delay period following different sequences with the same item changes depending on the position of the item in the sequence (Xie et al., 2022). While also in our model the firing rates of neurons are sensitive to the temporal order due to the primacy gradient (Fig. 3B), this effect is very minor compared to observations. In this respect, an important caveat could be that animals in these studies have been extensively trained on the task, with a limited number of sequences, while we are interested in WM representations of novel sequences that may have never been encountered in the past. Extensive training could lead to the emergence of stimulus-adapted neuronal representations but this mechanism is unavailable for processing novel sequences (see above). It remains to be seen whether our model, in a more physiologically-detailed setting, is able to account for some aspects of conjunctive coding or whether additional mechanisms are required, such as, e.g., associative synaptic plasticity (Botvinick and Watanabe, 2007; Gillett et al., 2020; Ryom et al., 2021).

Behavioral data in serial-recall tasks, on the other hand, strongly support the notion that the encoding of serial order relies, indeed, on a primacy gradient that prioritizes recall, and on an additional mechanism that prevents the recall of the items already retrieved (Farrell and Lewandowsky, 2004; Hurlstone and Hitch, 2015, 2018). In computational models, however, those features are essentially *postulated* to account for the behavior. By contrast, our theory makes an explicit proposal as to their neurophysiological substrates: The primacy gradient is encoded by the augmentation levels, its generation depends on a specific interplay of

the synaptic and neuronal dynamics (as described above), and the suppression of the (already) recalled items is a result of the synaptic depression. As such, our theory makes novel predictions that are testable in behavioral experiments. For instance, the primacy gradient builds up gradually with the reactivations of the corresponding neuronal populations between consecutive presentations. This requires a presentation rate that is slow enough for these reactivations to occur in sufficient number. Hence, as the presentation rate is increased, the theory predicts that encoding of the serial order should degrade. Consistently with this prediction, increasing the presentation rate of the items results in a larger number of transposition errors, that is, some items are recalled at the wrong serial position (see, e.g., (Farrell and Lewandowsky, 2004)). Experiments with very rapid serial visual presentation (RSVP) of the items show that the subjects are unable to report the correct order of presentation, even when the number of items is below capacity (Reeves and Sperling, 1986). At the other extreme, if the presentation rate is too slow, or the list is too long, then the primacy gradient will also degrade because of the saturation of the synaptic augmentation. We are not aware of experiments having tested this prediction.

More speculatively, we have shown that the same model is able to account for the switch from primacy-dominated to recency-dominated recall that is observed in free-recall tasks with long lists. We stress that our account is tentative, as it relies on unverified, but not implausible, assumptions about the dynamical control of the background input to the memory network. In fact, there is significant experimental evidence that items can be maintained in WM in different *representational* states with different physiological signatures, e.g., with or without enhanced spiking activity, and that these states can be rapidly altered by task demand (LaRocque et al., 2014; Oberauer and Awh, 2022). Our theory suggests that this could be achieved by regulating, more or less selectively, the background inputs to the memory network.

The explicit modeling of the recall process has revealed an intriguing dissociation between the *storage* and the *retrieval* capacity of the model network; some of the stored items cannot be retrieved (see Fig. 5). In fact, we expect a large storage capacity, because of the long time scales brought about by the synaptic augmentation. The retrieval capacity, on the other hand, is largely determined by the time constant for synaptic depression, τ_D, as shown in Mi et al. (2017). It would seem, hence, that taking a longer τ_D should lead to a better performance (i.e., more items recalled). However, increasing τ_D reduces the augmentation levels of the stored items. In fact, the refresh period (i.e., the interval between two reactivations of the same population) is also controlled by τ_D. A larger τ_D results in slower refresh rates and, therefore, in a slower build-up of the augmentation levels. This, in turn, leads to lower storage capacity, in general, and to a degradation of serial order encoding, in particular. In other words, there is a trade-off between storage capacity (and serial order encoding) and retrieval capacity. This suggests that WM capacity, which is in fact an experimental estimate of the retrieval capacity, could result from the inability to retrieve the information, rather than from the inability to encode and/or maintain it. In this scenario, WM capacity is ultimately determined by the degree of *selectivity* that the background control – that we identify with the "central executive" or the "focus of attention" of cognitive theories – can attain.

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Author contributions

G.M. and M.T. designed the research. G.M. and M.T. developed the mathematical theory. G.M. performed the numerical simulations. G.M. and M.T. wrote the manuscript.

Declaration of interests

The authors declare no competing interests.

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