A Learning rule for Place Fields in a Cortical Model: Theta phase precession as a network effect

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Abstract

We show that a model of the hippocampus introduced recently by Scarpetta, Zhaoping & Hertz ([2002] Neural Computation 14(10):2371-96), explains the theta phase precession phenomena. In our model, the theta phase precession comes out as a consequence of the associative-memory-like network dynamics, i.e. the network’s ability to imprint and recall oscillatory patterns, coded both by phases and amplitudes of oscillation. The learning rule used to imprint the oscillatory states is a natural generalization of that used for static patterns in the Hopfield model, and is based on the STDP synaptic plasticity observed experimentally. In particular, long-term potentiation and depression of the synaptic efficacies depends on the relative timing of the pre- and post- synaptic activities during learning. A further step is introduced to allow imprinting of correlated patterns properly.

The second part of this paper shows how the model accounts for theta phase precession observed in rat hippocampus. During learning of each spatial location, our model imprints an oscillatory pattern of specific amplitude and phase relationship between neurons by modifying neural synaptic connection strengths. After learning, the network’s response to external inputs reveals the encoded phase information, and thus phase precession, even though the external inputs do not have the phase information. The phase precession effect comes out from the crucial ability of our model to store both the amplitudes relationship and the phases relationship of the oscillatory patterns in the synaptic connections. Both the bell-shaped firing tuning curves (receptive fields) and the phase precession phenomena are a result of the network synaptic connections. In particular, the phase precession is an effect of the overlap between the imprinted place fields: if adjacent receptive fields do not overlap the firing tuning curve of each place cell is preserved but the phase precession does not occur.

The model agrees with the observation (Skaggs et al 1996) that the first spikes, as the animal enters the receptive field, come near the phase of the minimal pyramidal population average activity. The total phase precession of the place cells can be close to 360°, but not larger than 360° as found by O’Keefe Recce (1993) and Skaggs et al (1996). In our model, the theta phase of the place cell is a function of the animal location, almost insensitive to the animal’ running speed (for speeds that do not exceed a critical value related to the network connections and pa-
rameters), and therefore the theta phase shows a better
correlation with the animal location than with time, in
agreement with the experimental results of O’Keefe Recce
(1993) and Huxter et al. (2003).

Numerous computational models (Burgess et al 1994, 
Tsodyks et al 1996, Wallenstein et al 1997, Jensen and
Lisman 1996, Bose et al 2000, Booth et al 2001, Yam-
aguchi 2003, and Lengyel et al 2003) have attempted to
account for the mechanisms that underlie the phase pre-
cession observation, but a fully-satisfactory explanation
is still lacking. Recently, to contrast the predictions of dif-
ferent models of phase precession, Zugaro et al (2005) have
transiently turned off neuronal discharges for up to 250 ms
and reset the phase of theta oscillations by stimulating the
commissural pathway in rats. After recovery from silence,
phase precession continued: the theta phase of the place
unit and is the synaptic strength from excitatory unit
neurons that share common input, so the number of neu-
rious firing rates) are given by

denote vectors by bold fonts.) The unit outputs, repre-
senting the probabilities of the cells firing (or instanta-

taneous firing rates) are given by

g_{\alpha v}(v_1, \ldots , v_n) where \( g_a \) and \( g_i \) are sigmoidal activa-
tion functions that model the neuronal input-output rela-
tions. The equations of motion are

\[
\begin{align*}
\dot{u}_i &= -\alpha u_i - \beta_i v_i + \sum_j J_{ij}^0 u_j + I_i, \\
\dot{v}_i &= -\alpha v_i + \gamma_i^0 u_i + \sum_{j \neq i} W_{ij}^0 u_j.
\end{align*}
\]  

where \( \alpha^{-1} \) is the membrane time constant (for simplicity
assumed the same for excitatory and inhibitory units), \( J_{ij}^0 \)
is the synaptic strength from excitatory unit \( j \) to excita-
tory unit \( i \), \( W_{ij}^0 \) is the synaptic strength from excitatory
unit \( j \) to inhibitory unit \( i \), \( \beta_i^0 \) and \( \gamma_i^0 \) are the local
inhibitory and excitatory connections within the E-I pair
\( i \), and \( I_i(t) \) is the net input from other parts of the brain.
The model units represent local populations of biological
neurons that share common input, so the number of neu-
rons represented by an excitatory unit may be different
from the number of neurons represented by an inhibitory

unit. In this minimal model we omit inhibitory connecti-
between pairs, since the real anatomical long-range
connections appear to come predominantly from excita-
tory cells. The sensory input \( I_i(t) \) to the system, which
drives the excitatory units, has a static part \( I_i \) and an
oscillatory part \( \delta I_i(t) \) modulated at the theta frequency.
The static part \( I \) of the input determines a fixed point
\((\bar{u}, \bar{v})\), given by the solution of equations \( \bar{u} = 0, \bar{v} = 0 \)
with \( I = 1 \).

Linearizing the equations (1) around the fixed point
leads to

\[
\begin{align*}
\dot{u}_i &= -\alpha u_i - \beta_i v_i + \sum_j J_{ij} u_j + \delta I_i, \\
\dot{v}_i &= -\alpha v_i + \gamma_i u_i + \sum_{j \neq i} W_{ij} u_j.
\end{align*}
\]  

where \( u_i \) and \( v_i \) are now deviations from the fixed point,
\( \delta I \equiv I - I \) is the oscillatory part of the sensory input,
\( \beta_i = g_a(\bar{u}_i) \beta^0_i, \gamma_i = g_i(\bar{u}_i) \gamma^0_i, \)
\( W_{ij} = g_a(\bar{u}_j) W_{ij}^0, J_{ij} = g_i(\bar{u}_j) J_{ij}^0 \). Henceforth, for simplicity, we assume \( \beta_i = \beta, \gamma_i = \gamma, \) independent of \( i \).

Eliminating the \( v_i \) from (2), we have the second order
differential equations

\[
\begin{align*}
\ddot{u} + (2\alpha - J)\dot{u} + [\alpha^2 - \alpha J + \beta(\gamma + W)]u = (\partial_t + \alpha)\delta I. 
\end{align*}
\]  

(We use sans serif to denote matrices) or, equivalently,

\[
[(\partial_t + \alpha)^2 + \beta \gamma] u = Mu + (\partial_t + \alpha)\delta I. 
\]  

(4)

The terms in the square bracket describe the local E-
I pair dynamics, while \( M \) gives an effective coupling be-
tween the oscillating E-I pairs. Learning imprints pat-
terns into \( M \) through the long range connections \( J \) and \( W \).
After learning, \( u \) depends on how \( I \) decomposes into the
eigenvectors of \( M \). Thus the network can selectively am-
plify or distort \( I \) in an imprinted-pattern-specific manner
and thereby functions as input mapping into the subspace
spanned by the imprinted patterns.

Following Scarpetta et al (2002), we distinguish a learn-
ing mode, in which the oscillatory patterns are imprinted
while the connections \( J \) and \( W \) are ineffective, from a re-
call mode, in which connection strengths are effective and
fixed. With appropriate learning kernel, the network dur-
dering recall responds strongly (resonantly) to inputs simi-
lar to those learned or linear combinations of them, and
weakly to other inputs not related to the imprinted pat-
tterns. The phases and the amplitudes of the oscillatory
response will be a linear combination of the phases and
the amplitudes, respectively, of the imprinted oscillations
recalled by the input.

2.1 Learning oscillatory patterns

First, let’s imprint a single oscillatory pattern, \( P^\mu(t) =
\xi^\mu e^{-i\omega t} + \text{c.c.} \) where c.c. denote complex conjugate, and
\( \xi^\mu \) is a complex vector with components \( \xi_j^\mu = |\xi_j^\mu|e^{i\phi_j^\mu} \). Following Scarpetta et al (2002), the synaptic strength
\(J_{ij}\) is shaped by the correlations of the activities, \(u_j(t')\) and \(u_i(t)\), in pre-synaptic and post-synaptic cells through

\[
J_{ij} = \frac{1}{NT} \int_0^T dt \int_{-\infty}^{\infty} dt' u_i(t) A_j(t-t') u_j(t')
\]  

(6)

where \(A_j(t-t')\) is the learning kernel that measures the strength of the synaptic change at time delays \(\tau = t-t'\) between the pre-synaptic and post-synaptic activities. In the case when \(A_j(t-t') = \delta(t-t')\) the learning rule becomes the conventional Hebbian learning with \(\dot{J}_{ij} \propto \int dt u_i(t) u_j(t)\) used in Li and Hertz (2000). To model the experimental results of STDP (Markram et al 1997, Bi & Poo 1998) the kernel \(A(\tau)\) was taken as an asymmetric function of \(\tau\), mainly positive (LTP) for \(\tau > 0\) and mainly negative (LTD) for \(\tau < 0\). Analogously for the synaptic strength \(W_{ij}\) we write

\[
W_{ij} = \frac{1}{NT} \int_0^T dt \int_{-\infty}^{\infty} dt' v_i(t) A_W(t-t') u_j(t').
\]  

(7)

Since the connections \(J\) and \(W\) are ineffective during learning, the responses \(u_i\) and \(v_i\) are proportional to the imprinting input \(P_i(t)\). Substituting them into eqs. (6) and (7) yields connections

\[
J_{ij}^\mu = \frac{2}{N} \Re \left[ \tilde{A}_j(\omega_0) \xi_i^\mu \xi_j^{*\mu} \right]
\]

\[
W_{ij}^\mu = \frac{2}{N} \Re \left[ \tilde{A}_W(\omega_0) \xi_i^\mu \xi_j^{*\mu} \right]
\]

(8)

where

\[
\tilde{A}_{J,W}(\omega) = |\chi_0(\omega)|^2 \int_{-\infty}^{\infty} d\tau A_{J,W}(\tau)e^{-i\omega\tau}
\]

(9)

with \(\chi_0(\omega) = \frac{\alpha - i\omega_0}{\alpha^2 + \beta\gamma - \omega_0^2 - 2i\omega_0\alpha}\)

(10)

Note that \(\Im \tilde{A}_{J,W}(\omega) = 0\) if \(A_{J,W}(\tau)\) is symmetric in \(\tau\) and that \(\Re \tilde{A}_{J,W}(\omega) = 0\) if \(A_{J,W}(\tau)\) is antisymmetric.

The dependence of the neural connections \(J\) and \(W\) and the oscillator couplings \(M\) on \(\xi_i^\mu \xi_j^{*\mu}\) is just a natural generalization of the Hebb-Hopfield factor \(\xi_i^\mu \xi_j^{*\mu}\) for (real) static patterns. This becomes particularly clear for kernels satisfying the following matching condition:

\[
\tilde{A}_j(\omega_0) = \frac{\beta\gamma}{\alpha^2 + \omega_0^2} \tilde{A}_{W}(\omega_0)
\]

at \(\omega = \omega_0\).

(11)

Then the oscillator coupling simplifies into a familiar outer-product form for complex vectors \(\xi\):

\[
M_{ij}^\mu = -2i\omega_0 \tilde{A}_j(\omega_0) \xi_i^\mu \xi_j^{*\mu}/N
\]

(12)

In the following we will always consider the case of matching kernels (11).

We are interested in generalizing eq. (8) and (12) to the case in which multiple correlated patterns have to be imprinted. To construct the corresponding matrices for multiple patterns, \(\xi^\mu, \mu = 1, \ldots, N\), we use the rule proposed by Personnaz et al (1985) and Diederich and Oppen (1997) (see also Hertz et al 1991) for learning static correlated patterns without errors in the Hopfield model. Thus we write, for \(N\), linearly independent, correlated patterns \(\xi^\mu\),

\[
J_{ij} = \Re \left( \sum_{\mu\nu} \tilde{A}(\omega_0) \xi_i^\mu (Q^{-1})_{\mu\nu} \xi_j^{*\nu} \right)
\]

(13)

where the \(N \times N\) matrix \(Q\), defined by

\[
Q_{\mu\nu} = \frac{1}{N} \sum_j \xi_j^{\nu*} \xi_j^{\mu*}
\]

(14)

is the natural generalization of the correlation matrix \(Q\) used by Personnaz et al (1985) and Diederich and Oppen (1997) for the Hopfield model. The rule (13) is not local and not iterative. However it has been proved in Diederich and Oppen (1997) that, in the Hopfield framework, there exists a local rule that uses only successive presentation of one pattern at a time that converges exactly to the synaptic efficiencies obtained with the Q-rule. Work is in progress to generalize their procedure, and obtain the same result also in our case. In the following, we have used the rule (13). With the rule (13), we imprint \(N\) oscillatory patterns \(P^\mu = \xi^\mu e^{i\omega_0 t}\), all with the same frequency \(\omega_0\). Then the \(M\) matrix becomes:

\[
M_{ij} = \frac{1}{N} \sum_{\mu\nu} -2i\omega_0 \tilde{A}_j(\omega_0) \xi_i^\mu (Q^{-1})_{\mu\nu} \xi_j^{*\nu}
\]

(15)

### 2.2 The recall mode

In the recall mode the network dynamics is governed by the matrix \(M\), learned during the learning mode. Keeping in mind eq. (15), it is clear that the imprinted oscillations (even though not-orthogonal) are eigenvectors of the connection matrix \(M\),

\[
M \xi^\gamma = -2i\omega_0 \tilde{A}(\omega_0) \xi^\gamma
\]

(16)

Thus, after the transient (which is governed by the network reaction time that depends both on the membrane time constant of the neurons, \(\alpha\), and the network synaptic connections), the response \(u\) to an input that perfectly match one of the imprinted vector is

\[
u = \chi I
\]

(17)

with the linear response coefficient (or susceptibility)

\[
\chi = \frac{\alpha - i\omega_0}{\alpha^2 + \beta\gamma - \omega_0^2 - 2i\omega_0\alpha}
\]

(18)

A properly chosen learning kernel \(A(t)\) satisfying

\[
\alpha^2 + \beta\gamma - \omega_0^2 + 2i\omega_0 \Re [\tilde{A}(\omega_0)] \rightarrow 0
\]

\[
\alpha - \Im [\tilde{A}(\omega_0)] \rightarrow 0
\]

(19)

would produce a very strong susceptibility \(\chi\) and thus a resonant response to match input. Meanwhile, an input pattern \(I\) outside the subspace of the imprinted vectors evoke a weaker response \(u = \chi_0 I\). In general, an input
that we will call place cells, will be associated with a re-
and the unit $2J2\mu$.

This feature enables the system to interpolate between
imprinted patterns, and to perform an elementary form of
generalization from the learned set of patterns. As it will
be shown in the next section, the phase precession as the
animal moves from one location to the other is related to
this interpolation mechanism.

An example of learning kernel $A(\tau)$ that satisfy the re-
sonance conditions (19) at frequency $\omega_0 = 10$ Hz (theta
range) is shown in Fig. 1. This learning kernel is able to
imprint oscillations with frequency $\omega_0 = 10$ Hz, and to
make the network resonant at theta frequencies. It could
be of interest to note that the kernel $A(\tau)$ shown in Fig.
1 satisfies the resonance condition also at $\omega_0 = 40Hz$, i.e.
in the gamma range. However we will not exploit this last
property in this paper.

3 Imprinting locations and the
theta phase precession

Let's consider $N$ locations, $x_{\mu}$ arranged in a 1-dimen-
sional space with periodic boundary conditions (such as in a cir-
cular or triangular track). For each of these $N$ locations
$x_{\mu}$, $\mu = 1, ..., N$ we imprint, in the synaptic strengths
of the network, an oscillating pattern $P_{\mu}^{x}$, with specific am-
plitude and phase relationship between its components. Using
eq (13) in order to imprint $N$ (linearly independent)
(patterns properly). In all simulations we used
$N = 10$ locations in the net
with $n = 20$ excitatory units, we simulate the network

In our model the external inputs in the recall mode
resembles but are not exactly the imprinted patterns.
When the animal is exactly at location $x_{\mu}$, the input pat-
tern $\delta I$ is given by $\delta I = L^{x}e^{-i\omega_{0}t}$ where $L^{x}$ is a vector
with components $L_{\mu}^{x} = \delta_{\mu,2\mu}$. This pattern conveys the
animal location in the amplitude modulation but has no
phase coded information. When the animal is at position
$x$ between location $x_{\mu}$ and location $x_{\mu+1}$, $x_{\mu} < x < x_{\mu+1}$,
we take the input vector as linear combination of $L^{x}$ and
$L^{\mu+1}$,

$$L^{x} = (1 - |x - x_{\mu}|)L^{\mu} + |x - x_{\mu}|L^{\mu+1} \quad (21)$$

$L$ is then normalized. Thus, if the animal moves from
location $x_{\mu}$ at time $t = 0$ to the adjacent one $x_{\mu+1}$ with
speed $v(t)$, the current location will be a function of time
$x(t) = x_{\mu} + \int_{0}^{t} v(t)dt$, and the input vector will be ex-
pressed by eq. (21) where $x$ is replaced by $x(t)$. The
vector $L$ is always normalized. Fig. 2B shows the input
amplitude $L_{\mu}^{x}$ when animal moves along the track with
constant speed.

A animal at location $x$ receives an input pattern $L^{x}$
that resembles imprinted pattern $\xi^{x}$ for $x_{\mu} \approx x$. Hence, the
network response to input $L^{x}$ would amplify the im-
printed pattern $\xi^{x}$ that resembles $L^{x}$ most (see previous
section), and thus exhibits in the responses the non-trivia-
lar phase relationship among units that was stored in the
couplings. When animal is in between two stored locations,
the response will be a combination of the two imprinted
patterns (see eq. (20)), and in particular, the phase of the
response will be a combination of the phases of the two
imprinted overlapping patterns which depends from the
animal position. So, even though the input carries infor-
mation about current location only in the amplitudes modu-
lation, the associative memory nature of our model,

having phase-coded locations imprinted in the synaptic
couplings, allows the network to show both amplitude and
phase coded informations in the response.

Since the theta rhythm is the local field potential, in
our model it is simply the mean field. We compute the
theta rhythm doing the average of all the excitatory units
activities $M.F. = 1/2N \sum_{j=1}^{2N} u_{j}(t)$. This choice allows us
to compare the the absolute phase of theta at which place
cells fire during the place field traversal, with the experi-
mental findings of Skaggs et al. (1996). Indeed, the phase
zero of the theta rhythm in the experimental work of Sk-
aggs et al 1996 is defined to be the point in the theta cycle
that overlaps with several imprinted patterns
$L^{x}$ that are functions of the distance between the unit
$\mu$ and the imprinted pattern associated with location
$\phi_{\mu}$. In particular, the imprinted pattern associated with location
$\phi_{\mu}$ has components
$P_{\mu}^{x}(t) = \xi^{x}_{\mu}e^{-i\omega_{0}t} + c.c., \mu = 1, 2, ..., N, j = 1, 2, ..., 2N$, where
$\xi^{x}_{\mu} = A(2\mu - j)e^{i\phi(2\mu - j)}$; the am-
plitudes $A$ and the phases $\phi$ of the pattern $\xi^{x}_{\mu}$ on each
unit $j$ are functions of the distance between the unit $j$ and
the unit $2\mu$ that is associated with location $x_{\mu}$. In
particular, $A()$ is an even function decreasing quickly with
distance, while $\phi()$ is an odd function. This means,
when imprinting a location $x_{\mu}$, only units located close to unit
$2\mu$ are excited by the imprinting inputs. In such a man-
ner, after the learning process, the N excitatory units $u_{2\mu}$,
that we will call place cells, will be associated with a re-
cceptive field centered at $x_{\mu}$, for $\mu = 1, 2, ..., N$. The other
N units (with odd index, called auxiliary units) are neces-
sary in order to allow the network to store the N desired
patterns properly. In all simulations we used
$A(0) = 1, A(\pm 1) = 0.3 A(\pm 2) = 0.3, A(x) = 0$ otherwise, and
$\phi(0) = 0, \phi(\pm 1) = \pm 2.4 rad$ and $\phi(\pm 2) = \pm 2.5 rad$.
The network has a quite robust behavior with respect to the
choice of the functions $A$ and $\phi$.

4 Simulation Results and compar-
isons with experimental findings

In all simulations reported here we used the kernel $A(t)$
shown in fig. 1 that satisfy the constraints (19) when
$\omega_{0} = 10Hz$, i.e. when the frequency is in the theta range.

After having imprinted $N = 10$ locations in the net
with $n = 20$ excitatory units, we simulate the network
dynamics while the animal moves continuously in space. Fig. 2 shows in red the activity of the two place cell units \((u_{2u}, \text{with } \mu = 5, 6)\), as a function of time, while the animal moves with constant running speed. Black curves show the theta rhythm computed as the mean field of all the excitatory units of the network.

The simulation is done at a constant animal speed \(v = 1/(400ms)\) (note that space is adimensional in our framework, and a unitary distance correspond to the distance between the centers of two adjacent receptive fields). The oscillatory input on each unit \(j\) is \(I_j(t) = L_j(t)\cos(\omega_0 t)\), where the amplitude \(L_j(t)\) is shown in fig. 2B as a function of time. The figure shows that, for each place cell, the oscillation amplitude of the firing rate increases as the animal approaches the center of its place field and decrease as the animal left the center (firing tuning curve), while the place cell phase advanced at consistently earlier phases of the theta cycle as the animal pass through the cell's receptive field (phase precession). To quantify the phase precession phenomena, we compute the phase shift between the theta rhythm and the place cell activity in each cycle, and we plot in fig. 2 the theta phase as a function of the animal position during running, together with the amplitude modulation.

The model dynamics is in agreement with the experimental evidence of theta phase precession in two main points: 1) the total phase precession is less than 360° (O'Keefe Recce 1993, Skaggs 1996); 2) as the animal enters the receptive field the absolute phase of the cell activity with respect to the phase of maximal excitatory units population activity (theta rhythm) is slightly less than 180°, in agreement with experimental finding of Skaggs et al (1996).

Effects on nonlinearity in our general model have been analyzed in Scarpetta et al (2002), where we distinguished two class of nonlinearity. For the nonlinearity of class I (as the sigmoidal function shown in fig. 3B) we expect that the nonlinearity do not change critically the linear results. For the nonlinearity of class I (as analyzed in Scarpetta et al (2002), where we distinguished (1996).)

The model dynamics is in agreement with the experimental results of O'Keefe Recce (1993) and Huxter et al (2003).

Finally, in order to test our model, we compare it with the recent experiments of Zugaro et al 2005. We have simulated the effect of a perturbation that silences all units (both excitatory and inhibitory ones) and reset the phase of theta rhythm. The activity of two place cells, before during and after the network perturbation, is shown in fig. 5. The perturbation last for 200 ms. Despite the theta phase reset and the transient interruption of firing, the theta phase of the first cycle after the recovery is more advanced than the theta phase of the last cycle before the perturbation, as in the experiments (Zugaro et al 2005). The decrease of the peak firing rate is stronger in our simulations than in the experimental findings of Zugaro et al (2005), that reported only a small but not-significant decrease in peak firing rate. To quantify the degree of conservation of the phase precession and the effect on the amplitudes of oscillation we shows in fig. 5 the plot of the theta phase as a function of the position, and the amplitude of the oscillation versus the animal position. Figure 5C should be compared with fig. 2 in Zugaro et al (2005).

Figs. 6 and 7 show the synaptic connections \(J_{ik}\) and \(W_{ij}\) learned in our model, as a function of pre-synaptic cell index \(k\), for a post-synaptic place cells (\(i\) even) and an auxiliary units (\(i\) odd). As expected, the synaptic connections are translation invariant, so \(J_{ij}\) depend only on the difference \(i - j\) and on the parity of \(i\) (even or odd). We note that connections have both positive and negative values. Since they are supposed to be projections from excitatory pyramidal cells, the negative connections weights means effectively inhibitory connections, which could be implemented by additional inhibitory interneurons with very short membrane time constant. Note that the synaptic weights are highly asymmetric.

Finally, note that in our model the activity of all the units, both excitatory and inhibitory units, is modulated at the theta frequency. The network shows resonance in the theta range, and the inhibitory units, similarly to the place cells, exhibit phase precession (not shown), while the auxiliary units have a very weak place field with very little phase precession (not shown).

5 Conclusions and Discussion

In this paper we propose a theoretical model for theta phase precession in the hippocampus by applying a framework (Scarpetta Zhaoping Hertz 2002) that is a generalization of the Hopfield model to oscillatory patterns. The learning rule is a generalization of the Hebb prescription, inspired to the STDP synaptic plasticity.

The associative memory behaviour of our model, allows the network to show both amplitude and phase coded informations in response to inputs that carry information about current location only in the amplitudes modulation.

Our model dynamics is in agreement with the experi-
mental evidence of theta phase precession in 3 main points:

1) the model accounts for the observations that after a perturbation that transiently turned off neuronal discharges for a couple of theta cycles and reset the phase of the theta oscillations, the phase precession continued (Zugaro et al. 2005). This happens in our model, even if the external input to the network is silenced during the perturbation. This comes naturally from the fact that in our model, the oscillation phase of a place cell depends on both the network connections and the external input strength. Hence, when the external input strength represents the spatial location (relative to the center of the place field), the oscillation phase of the place cell will, by the influence of the network connections, code the location relative to the center of the place field.

2) In our model the theta phase depends on current position of the animal, and is only slightly sensitive to the animal speed (almost insensitive at low speeds). Therefore in our model the theta phase is better correlated with position than with time spent in the place field, in agreement with experimental data (O’Keefe Recce 1993, Huxter et al. 2003). This is achieved without need of any speed-dependent input, in contrast with the recent model of phase precession (Lengyel et al. 2003) that assumes a differential input proportional to the instantaneous velocity of the rat.

3) Both the initial theta phase as the animal enters the place field and the total phase precession generated by our network dynamics, are in quantitative agreement with experimental evidence. In our model the total phase precession is less than 360°, and the absolute phase of the cell activity w.r.t. the average pyramidal cells activity, when the animal enters the place field, is slightly less than 180°, in agreement with experiments of Skaggs et al. (1996). Note that our definition of the theta rhythm as the average activity of all the excitatory units is in agreement with the definition used by Skaggs et al. (1996).

Finally note also that our model predicts both a monotonous phase precession and a unimodal firing tuning curve with a large amplification factor when the animal is in the center of the place field. This is in contrast with some other models. For example, models in which both firing rate and phase are linear or monotonic functions of the same parameter (Booth and Bose 2001; Magee 2001) produce a monotonically increasing firing rate inside the place field if monotonic phase precession is preserved, or non-monotonic phase precession if unimodality of the firing profile is to be preserved.

Although there is some controversy as to whether theta phase precession is linear (O’Keefe Recce 1993) or if theta phase is an accelerating function of position (Skaggs et al. 1996, Yamagushi et al. 2002), and if it’s smooth or clustered, its trend is undoubtedly monotonic (O’Keefe and Recce 1993; Skaggs et al. 1996, Yamagushi 2002), since there is a systematic progressive phase retardation as the animal passes through the place field.

In contrast, experimental data indicates that firing rate changed in a non-monotonic waxing-waning manner (O’Keefe Recce 1993, Skaggs et al. 1996), even though there is some controversy as to whether phase precession accompanies skewed firing profiles (Mehta et al. 2002; Harris et al. 2002) or not (Huxter et al. 2003).

Our model shares some similarities to the one of Tsodyks et al. 1996, since both are based on the associative memory properties of the network. In contrast with the Tsodyks et al. (1996) work, here the couplings are computed using a mathematical learning rule and the network dynamics is studied analytically in a framework (Scarpetta et al. 2002) that is the generalization of the Hopfield model to the case of not-static but oscillatory patterns.

A point of our model that still needs investigation is the origin of the imprinted patterns used during the learning mode. Of course, if there is no phase differences between units in the imprinted patterns, there would be no phase precession despite of learning and network dynamics. We are currently investigating how such phase differences arise in the imprinted patterns during learning, i.e. if they are generated by an initial asymmetry of the connections that produces the phase shift among units when the animal is moving, or if they are inherited from earlier stages input during learning. Anyway, what we have proven here is that when the network is equipped with such a synaptic connectivity the network dynamics shows phase precession and phase-coded response to sensory input that do not have any phase difference between units activities.

It has previously been suggested that phase coding occurs in the sensory input to the place cells (Burgess et al. 1994), and it has been shown that under that model individual place cells will show phase precession versus the average place cell activity (Burgess et al. 1993), as in our model. A difference with their model is that, even if we use a phase-coded activity during learning, once the network is equipped with the proper synaptic couplings, the phase precession occurs, when the animal run, without need of any phase coding in the sensory input.

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Figure 2: Simulation of the network dynamics when animal is moving continuously in time with constant running speed \(v = 1/(200 \text{ ms})\). A: The two subplots show the activities (in dotted red) of two place cell units \((u_j, j = 10, 12, \text{ whose place fields are centered at 5 and 6 respectively})\) as a function of time. Black curves show the ongoing theta rhythm activity (computed as the mean field of the excitatory activity of all the network). The external input to the units is \(I_j(t) = L_j(t) \cos(\omega_0 t)\), where the amplitude \(L_j(t)\), shown in B, is maximum when the animal is in the center of the receptive field of that unit \(j\). Note that each place cell unit shows phase precession, i.e., relative phase shift between \(u(t)\) (red) and the theta activity (black), as the animal enters and then leaves the place field of this unit. Same behaviour for all other place cells not shown. C: Theta phase as a function of the current position of the animal, and amplitude of oscillation as a function of current animal position, for the place cell centered in location 5. Theta phase is a bit less than 180° when the animal enters the receptive field, and the total phase precession is less than 360°.

Figure 3: Effects of nonlinearity in the phase precession phenomena dynamics. A. Simulation of the network dynamics using the nonlinear activation function showed in B, when animal is moving continuously in time with constant velocity \(v = 1/(200 \text{ ms})\) as in previous figure. Dotted red curve shows the place cell activity as a function of time. Black curves show the theta rhythm. Results are affected by nonlinearity mainly at large amplitude, but the theta phase precession is preserved, as shown in figure C showing the theta phase as a function of position, and amplitude of oscillation as a function of position for the place cell with receptive field centered in 5.

Figure 4: Theta phase of the place cell as a function of the animal position (A), and as a function of the time spent in the place field (B) at three different animal running speeds. During the place field traversal, for each cycle, we compute the phase of the place cell with respect to the theta rhythm. In each of the three simulations, the animal is moving continuously in time with constant speed. The green circles correspond to speed \(v = 1/200 \text{ ms}^{-1}\), the red triangles to \(v = 1/400 \text{ ms}^{-1}\), and the black squares to \(v = 1/800 \text{ ms}^{-1}\). Figure shows that the theta phase is much better correlated with position than with time.

Figure 6: $J_{ik}$ connection after learning $N=10$ locations, as a function of $k$. On the left, $i = 10$, and on the right, $i = 11$.

Figure 7: $W_{ik}$ connection after learning $N=10$ locations, as a function of $k$. On the left, $i = 10$, and on the right, $i = 11$. 
Figure 5: Phase precession persist after transient intra-hippocampal perturbation. Despite the theta phase reset and the transient interruption of firing, the phases were still correlated with the spatial position of the animal immediately after the recovery, in agreement with experiments (Zugaro et al 2005). A. Simulation of the network dynamics when animal is moving continuously in time with constant velocity \( v = 1/(200 \text{ ms}) \) but a perturbation that silences all units (both excitatory and inhibitory ones) and reset the phase of theta rhythm is applied for 200 ms. Black curves show the theta rhythm. The dotted red line shows the activity of place cells before during and after the network perturbation (place cell \( u_8 \), centered in location 4, in the upper subplot, and place cell \( u_{10} \), centered in 5, in the lower subplot). The theta phase and the firing amplitude are shown as a function of animal position when perturbation was applied, and in control conditions (i.e. without any perturbation), for the place cell centered in 4 (B) and the one centered in position 5 (C).