Dendritic spiking accounts for rate and phase coding in a biophysical model of a hippocampal place cell

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Abstract

Hippocampal place cells provide prototypical examples of neurons firing jointly phase and rate coded spike trains. We propose a biophysical mechanism accounting for the generation of place cell firing at the single neuron level. An interplay between external theta-modulated excitation impinging the dendrite and intrinsic dendritic spiking as well as between frequency modulated dendritic spiking and somatic membrane potential oscillations was a key element of the model. Through these interactions robust phase and rate coded firing emerged in the model place cell, reproducing salient experimentally observed properties of place cell firing.

Key words: phase precession, tuning curve, active dendrite

1. Introduction

Hippocampal place cells code the spatial position of the animal both by their firing rate and the precise timing of their firings. A place cell fires only when the rat traverses the particular portion of the environment preferred by the cell, the 'place field' of the cell [1]. Rate coding implies that during a single traversal, firing frequency of the cell increases in the early and decreases in the late portion of the place field [2,3]. Phase coding was demonstrated as a monotonic decrease in the phase of firings relative to the ongoing theta oscillation during a single traversal [2,4,5,3].

In most of the models that have been suggested to account for these two phenomena, hippocampal area CA3, where pyramidal cells are known to be densely interconnected by recurrent collaterals, played a key role [6–8]. However, experimental studies reported that place cell activity could be maintained in CA1 even when input from CA3 was severely impaired [9,10]. Recently, Lengyel et al. [11] has shown both by analytical calculations and numerical simulations of a simplified single neuron model that the doubly coded firing pattern of place cells can be generated in a cell receiving periodic perisomatic inhibitory input and dendritic excitatory input from the perforant path without the need for recurrent excitatory connections. Here we present a biologically more realistic version of this model which was extended in two ways. First, a bio-

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physical conductance-based model was adopted, and thus the dynamics of intrinsic dendritic membrane potential oscillations (DMPOs) were studied in detail. Second, theta modulation of perforant path transmission [12,13] was taken into account. The interaction of these two oscillations in the dendrite generated dendritic spiking that in turn interacted with the periodic hyperpolarization of the soma. Through these interactions robust phase and rate coded firing emerged in the model place cell.

2. Methods

Following our earlier work [11], the present model consisted of a somatic and dendritic compartment, and received two inputs. (1) The membrane potential of the soma was modulated by a somatic oscillating current (SOC) with a constant theta frequency, in phase with the ongoing theta field potential oscillation [14–16]. (2) Dendritic postsynaptic current represented perforant path input from the entorhinal cortex and was a sum of two terms: (i) a sinusoidal term oscillating with the same frequency but in anti-phase with SOC [16], and (ii) a speed-dependent term that was proportional to the speed of the rat and was switched on when the animal was inside the place field of the cell. Although experimental evidence of the presence of velocity-dependent postsynaptic depolarization is lacking, a theoretical study has recently shown that realistically timed theta-modulated perforant path-specific inhibition [17] transforms position-dependent activity of entorhinal cells [18] into speed-dependent postsynaptic excitation in hippocampal place cells [19].

The dendrite sustained DMPOs within the place field that were frequency modulated by the speed-dependent term, and were in anti-phase with SOC [16,20] at the beginning of the traversal of the place field due to the oscillatory term of the input. The net excitation received by the soma of the hippocampal place cell was the sum of SOC and the current flowing from the dendrite due to DMPO.

The model cell was a modified Pinsky-Rinzel neuron [21]. Passive parameters of the model were: $C_{\mathrm{m}} = 1\ \mu\mathrm{F/cm}^2$ membrane capacitance, $g_{\mathrm{L}} = 0.3\ \mathrm{mS/cm}^2$ leak conductance, $V_{\mathrm{L}} = -60\ \mu\mathrm{V}$ leak reversal potential, $g_{\mathrm{c}} = 0.01\ \mathrm{mS/cm}^2$ axial conductance between the soma and the dendrite and $p = 0.2$ soma/cell surface area ratio. Equilibrium potentials (in mV) and maximal channel conductances (in $\mathrm{mS/cm}^2$) of active conductances were $V_{\mathrm{Na}} = 60$, $V_{\mathrm{K}} = -75$, $V_{\mathrm{Ca}} = 80$, and $g_{\mathrm{Na}} = 30$, $g_{\mathrm{K-DR}} = 15, g_{\mathrm{Ca}} = 10$, $g_{\mathrm{K-C}} = 15$, $g_{\mathrm{K-AHP}} = 0$. In order to shorten somatic action potentials, the exponents of the m and n gating variables were increased from 2 to 3, and from 1 to 4, respectively.

SOC represented various sources of synaptic inputs modulated at theta frequency [13]: $I_{\mathrm{s}}(t) = A_{\mathrm{s}}/2\ \cos(2\pi f_\theta t) + I_{0-\mathrm{s}}$, where $f_\theta = 8$ Hz, $A_{\mathrm{s}} = 4\ \mu\mathrm{A/cm}^2$ and $I_{0-\mathrm{s}} = -0.5\ \mu\mathrm{A/cm}^2$. The only input to the dendrite arrived from entorhinal cortex, and it had two components: $I_{\mathrm{d}}(t) = I_\theta(t) + I_v[v(t)]$. I_θ was a sinusoid depolarizing current in antiphase with SOC: $I_\theta(t) = A_{\mathrm{d}} \cos(\pi + 2\pi f_\theta t) + I_{0-\mathrm{d}}$, where $A_{\mathrm{d}} = 0.5\ \mu\mathrm{A/cm}^2$ and $I_{0-\mathrm{d}} = 1.9\ \mu\mathrm{A/cm}^2$. Velocity-dependent current arose only when the rat was within the afferent place field of the cell (i.e., the area covered by all the place fields of its presynaptic entorhinal partners), and there it was a linear function of the rat's running speed: $I_v[v(t)] = kv(t) \cdot \mathcal{H}[x(t) - x_{\mathrm{in}}] \cdot \mathcal{H}[x_{\mathrm{out}} - x(t)]$, where $k = 0.01\ \left(\mu\mathrm{A/cm}^2\right) / (\mathrm{cm/sec})$, $x(t)$ is the position of the rat, and x_{in} and $x_{\mathrm{out}} = x_{\mathrm{in}} + l$ are the entry and exit points of the afferent place field, l is the length of the afferent place field, and $\mathcal{H}$ is the Heaviside function.

Phase response curves (PRCs) of the separated dendrite were calculated in response to pulse and periodic perturbations. Regular dendritic spiking was elicited by $I_{0-\mathrm{d}} = 1.82\ \mu\mathrm{A/cm}^2$ current injection. Perturbations were timed at different phases (τ) of the spiking cycle, and consisted of current pulses of 0.5 msec duration and variable amplitude starting at τ msec after a spike (Fig. 5A), or one cycle of sinusoid current injection of $A_{\mathrm{d}} = 0.5\ \mu\mathrm{A/cm}^2$ amplitude and f_θ frequency starting with its peak τ msec before the expected time of the next spike (Fig. 5B). Phase response was measured as a phase advancement of the dendritic spiking oscillation in response to the perturbation compared to the unperturbed case: positive or negative values showed shortening or lengthening of the spiking cycle, respectively. As a control, PRCs for peri-

odic perturbation were also calculated from pulse PRCs by integrating the product of the pulse PRC with the sinusoid perturbation.

Moving direction of the animal was always from left to right (Fig. 3,4).

3. Results

First we analyzed the behavior of the separated dendrite ($g_c = 0$) in response to constant current injection (data not shown). Depending on the level of depolarization, the dendrite either settled to a resting membrane potential (stable fixed point) or sustained an intrinsic membrane potential oscillation. Amplitude of oscillation decreased while frequency increased with increasing external current amplitude. In the relevant frequency regime, that is close to the 8 Hz theta frequency, these oscillations were high amplitude dendritic calcium spikes (see Fig. 2B).

The interaction between a theta frequency (8 Hz) sinusoid external current oscillating around a given mean value ('offset') and intrinsic membrane potential dynamics was also studied in the separated dendrite, so that the offset of the external oscillation was varied while its amplitude (minimum to maximum depth) was kept constant (Fig. 1A). At lowest current intensities ($< 1.4\ \mu\mathrm{A/cm^2}$) no spikes or subthreshold oscillations were generated. After a short transition of dendritic oscillations at half theta frequency, dendritic spikes appeared and were driven by the external oscillation at 8 Hz in a wide range of medium current intensities (between $1.5 - 1.9\ \mu\mathrm{A/cm^2}$). At higher levels of injected current the offset component of external depolarization dominated, eliciting intrinsic membrane potential oscillations in the dendrite at frequencies corresponding to the offset level (frequency modulation). Due to interaction with the sinusoid component of the external current these oscillations were not perfectly regular: interspike intervals (ISIs) varied, but this variation was small enough not to mask the frequency modulation effect.

In the complete model with soma and dendrite coupled through an axial conductance, the amount of current received by the dendrite during a traversal of the environment was in the medium to high ranges. When the rat was outside the place field, offset of dendritic current injection was low, thus its relative depth of modulation (peak-to-trough amplitude relative to peak level) was high, effectively driving DMPO at theta frequency (Fig. 1B). As the rat crossed the boundary of the afferent place field, the velocity-dependent component of dendritic excitation was switched on, increasing its offset to a level where it induced intrinsic dendritic oscillations and also reducing its relative depth of modulation. Consequently, intrinsic dynamics of the dendrite shaped the frequency of DMPO similarly to that seen in the case of the separated dendrite (Fig. 1B). Note, that the dendritic frequency-current tuning curve in the complete model was slightly shifted towards higher current intensities compared to the separated dendrite. This can be explained as a weak shunting of the dendritic membrane potential by the somatic compartment through the axial conductance. Positive axial current to the dendrite

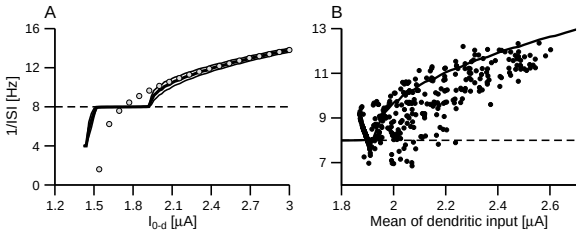

Fig. 1. Frequency modulation of dendritic spiking in response to sinusoid current injection. Offset level of external current was changed, while its peak-to-trough amplitude was kept constant. A. Firing frequency tuning curve of the separated dendrite at different offset levels of external current ($I_{0-\mathrm{d}}$). ISIs were transformed to instantaneous firing frequency values. Thick solid line shows average firing frequency, thin solid lines shows range of ± 1 standard deviation. Open circles show frequency-current tuning curve in response to constant current injection for comparison. Dashed line shows frequency of external stimulation at 8 Hz. B. Dendritic firing frequency with soma attached. Offset level was continuously changing in time. Dots show instantaneous firing frequencies calculated from individual ISIs as a function of average external current entering the dendrite during the corresponding ISI. Solid line shows average ISIs expected from simulations of the separated dendrite (solid line in A) for reference. Dashed line shows 8 Hz theta frequency.

flowed only during somatic action potential peaks, but these were too short to have any measurable effect on DMPO (data not shown). Therefore somatic shunting can be considered as a practically constant negative current that decelerates phase precession but has no effect on the derivative of phase precession speed.

As intradendritic firing frequencies inside the afferent place field were higher than theta frequency, the phase of dendritic spiking gradually precessed relative to SOC reflecting field theta oscillation (Fig. 2). The effect of dendritic spikes on somatic firing depended on the phase of SOC at which they were fired (Fig. 2B). Dendritic spikes arriving near the troughs of SOC did not cause somatic firing, because somatic hyperpolarization was strong enough to prevent them reaching firing threshold in the soma. However, as dendritic spikes appeared at increasingly higher levels of somatic depolarization, they were able to trigger bursts consisting of an increasing number of somatic action potentials. This mechanism together with the phase precession effect ensured that firing frequency as a function of position formed a unimodal, approximately symmetrical curve (Fig. 3A).

Earlier work on a simplified neuron [11] predicted that, in line with experiments [2], correlation between phase of somatic spikes and position would be higher than between somatic spikes and time, as observed in experiments. To test this prediction we simulated 50 traversals of the place field and calculated respective correlation coefficients. We found that firing phase correlated significantly stronger with the position of the animal than with time spent in the place field (Fig. 3A and B).

Place cells often show nearly 360° phase precession [2,4]. In order to reproduce this in the model (Figs. 2,3) two parameters had to be carefully tuned: l, the length of afferent place field, i.e. the area containing place fields of all presynaptic entorhinal cells, and k, the current/velocity conversion factor being proportional to the synaptic strength of perforant path synapses. Both of these parameters were proportional to the cumulative velocity-dependent excitation received by the dendrite during a traversal of the place field, and thus to the phase at which dendritic spikes were emitted on exit from the place field (see also Methods).

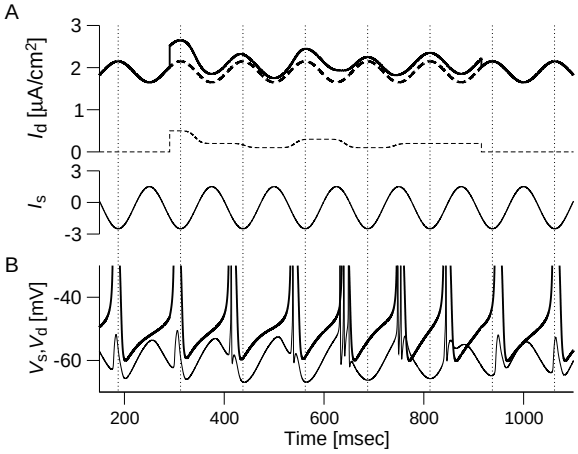

Fig. 2. A typical traversal of the place field. A. External inputs to the somatic (lower trace) and dendritic (upper traces) compartments. Net dendritic synaptic excitation (thick solid line) consisted of a persistent theta frequency (thick dashed line) and a place field-specific velocity-dependent (thin dashed line) component. Current injected to the soma (thin solid line) was in antiphase with dendritic periodic input. Vertical dotted lines show troughs of SOC for reference. B. Somatic (lower thin line) and dendritic (upper thick line) membrane potential. Note the somatic depolarization bumps caused by dendritic spikes not reaching firing threshold in the first and last cycles shown, but giving rise to a graded number of action potentials in intermediate cycles. Dendritic and somatic spikes were clipped for better visibility of subthreshold events.

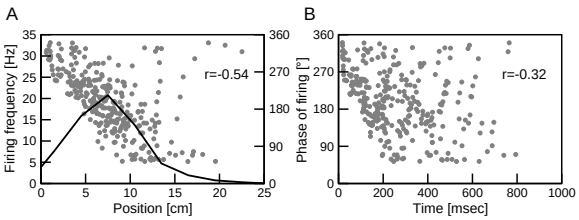

Fig. 3. Firing phase and rate during multiple traversals. A. Firing phase was monotonically decreasing (dots), while average firing rate was a unimodal function of position (solid line). Firing phase was better correlated with position (A) than with time spent in the place field (B). Each dot represent a single somatic spike.

More importantly, had dendritic spiking phase not been in antiphase with SOC on exit, it would also have corrupted the firing frequency tuning curve of the cell, not just the pattern of phase precession, as firing would have continued on indefinitely.

Contrary to these ominous expectations, the

model showed robust behavior even when k and l were set to non-optimal values: after exit from the afferent place field, phase of dendritic firing precessed or recessed until it got in antiphase with SOC (Fig. 4). This was achieved through the following 'branching behavior': if dendritic spiking phase on exit did not reach a $\sim 220°$ threshold value then dendritic spiking recessed (reversed precession), otherwise, if place field-specific input was able to cause sufficient precession to get past this threshold phase, then dendritic spiking finished precession up to a full theta cycle independent of the place field-specific term of the entorhinal input. By analyzing phase response curves of the dendritic compartment, the source of this branching behavior became apparent: DMPO was only sensitive to perturbations in the part of the cycle immediately preceding spiking (Fig. 5A). As a consequence, in response to periodic excitation, dendritic spiking had one stable fixed point at 330° (approximately in phase with dendritic excitation, thus in antiphase with SOC), and an unstable fixed point at 220° (Fig. 5B). Because the length of the animal's trajectory within the afferent place field may never be exactly the same, especially in two-dimensional environments, it seems inevitable that on some traversals phase recession instead of precession will occur in the late portion of the place field. We predict that this branching behavior may be the source of the decreased correlation of firing phase with position in the second half of the place field as observed in experiments [4,22,23].

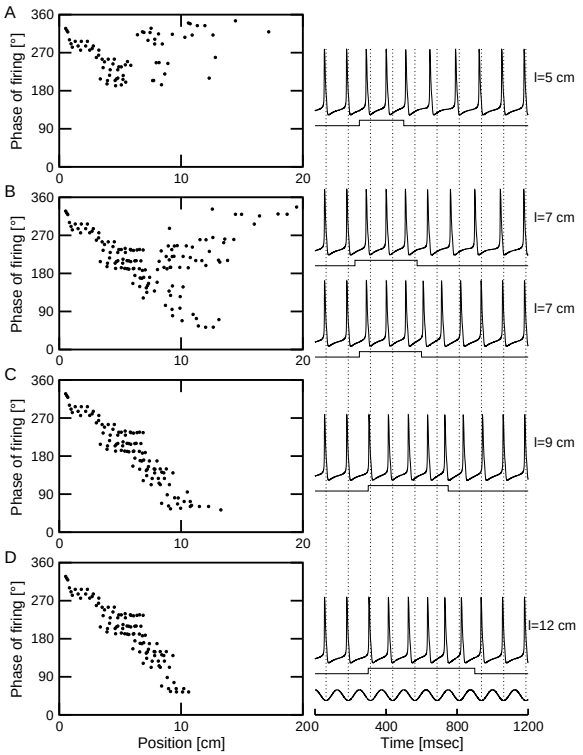

Fig. 4. The effect of changing the length of the afferent place field, l, while keeping the current/velocity conversion factor, k, constant. If the afferent place field is too short and dendritic spiking on exit does not reach a $\sim 220°$ threshold value, phase recession follows the break off of place field-specific excitation (A). If the afferent place field is longer and thus phase precession continues longer in it, past the threshold value, precession after exit continues 'automatically' until reaching 360°(C,D). For intermediate afferent place field lengths, some traversals may result in recession while in others a full cycle of precession is produced depending on the exact phase on exit from the place field (B). Panels on the right show dendritic membrane potential traces during individual traversals (upper lines) with square wave traces showing when the animal was within the afferent place field (lower lines), and vertical dotted lines showing troughs of SOC. Panels on the left show phase-position plots of multiple traversals (with different running velocities and entry phases), each dot corresponding to a single somatic spike.

4. Discussion

Place cells provide prototypical examples of neurons firing jointly phase and rate coded spike trains [2,5,22,3]. Many theories have tried to explain this phenomenon, but most of these models used only abstract neuron [2,22,11], relied on implausible assumptions about the connectivity of the network encompassing place cells [6–8,24,25], or failed to reproduce both the phase and rate code appropriately [6–8,24,26,5,22].

In this study, we have proposed a biophysical mechanism accounting for the generation of place cell firing at the single neuron level. An interplay between external theta-modulated excitation impinging the dendrite and intrinsic dendritic spiking (Fig. 1) as well as between dendritic spiking and periodic somatic hyperpolarization (Fig. 2) was a key element of the model. Indeed, hippocam-

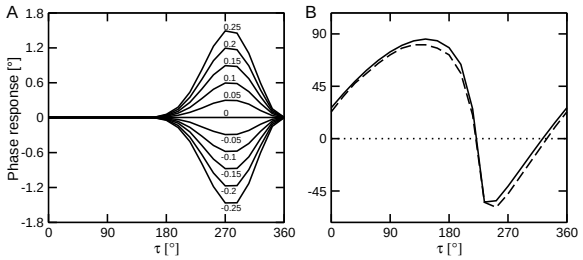

Fig. 5. Phase response curves of the dendritic compartment. A. The separated dendrite received constant current that triggered repetitive dendritic firing with approximately theta frequency. The effect of the injection of a short extra current pulse depended on the phase of the intrinsic oscillation at which this perturbing current was applied ($0 = 360°$: peak of dendritic spike without perturbation). In the first half of the cycle (0-180°) perturbations had no effect; in the second half (180-360°) the sensitivity of the dendritic oscillation increased until about 270° and decreased afterwards. Numbers on different phase response curves denote the amplitudes of perturbations in μA/cm^2. Negative current caused lengthening of the cycle (dendritic spike was delayed), whereas positive current accelerated the cycle (dendritic spike occurred earlier). The extent of this decelerating/accelerating effect increased nearly linearly with the amplitude of the applied current. B. The separated dendrite received one cycle of extra sinusoid current injection at theta frequency. Solid line shows the result of simulations with sinusoid excitation, dashed line shows prediction from instantaneous phase response curves in A for comparison (see Methods for details). The horizontal axis shows phase lag of dendritic spiking to the periodic excitation. Intersections with the horizontal axis show fixed points of this phase lag in response to continued sinusoid excitation: intersection with negative slope at 220° is unstable, intersection with positive slope at 330° is stable.

pal pyramidal cells were shown to be capable of sustaining rhythmic firing of dendritic spikes close to theta frequency in antiphase with somatic action potentials [16,20], and the frequency of these spikes was modulated by the amount of injected current [16]. Moreover, extracellular currents in the somatic and dendritic layer of the hippocampus proper are theta modulated and in antiphase with each other [16], just as assumed by the model.

The symmetry of place cell firing rate tuning curves is still hotly debated: while several some studies found an average negative skewness of place fields [27,22], others found symmetric and both negatively and positively skewed pace field with no significant differences in their number [2,3]. The model produced spike trains conforming experimental result (Fig. 3), in which firing rate was found to be a graded, unimodal, essentially symmetric function of position [2,3], firing phase was a monotonically decreasing function of position [2,4,3] and was better correlated with position than with time [2,3].

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Biosketches

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