An Interaural Time Difference Map Resulting from Axonal Selection through Non-Specific Learning

Christian Leibold [a] Richard Kempter [a,b] Hermann Wagner [c]
J. Leo van Hemmen [a]

[a] *Physik Department, Technische Universität München, D-85747 Garching bei München, Germany*

[b] *present address: Keck Center for Integrative Neuroscience, UCSF, San Francisco, CA 94143-0732, USA*

[c] *Institut für Biologie II, RWTH Aachen, D-52074 Aachen, Germany*

Abstract

An orderly spatial representation of the azimuthal position of a sound source has been observed in many animals. Barn owls, e.g., derive the azimuth from a neuronal map of interaural time differences (ITD) with a temporal precision of a few microseconds. We present a model of how an ITD map can develop in an array of spiking neurons in the barn owl's *nucleus laminaris*. We have combined homosynaptic spike-based Hebbian learning with presynaptic propagation of synaptic modifications. The latter is a feasible interaction mechanism between neurons and may be orders of magnitude weaker than the former. It is a key to explaining the widely assumed place code proposed by Jeffress (1948).

Key words: spike-based learning; Jeffress model; Hebbian learning; polysynaptic modification; interaural time difference tuning

Introduction

In 1948 Jeffress [7] proposed a wiring scheme that combines axonal delay lines from both ears and neuronal coincidence detectors so as to convert interaural time differences (ITDs) into a place code (Fig. 1a). A map resembling the circuit envisioned by Jeffress has been found in the nucleus laminaris (NL) of the barn owl [2,3] and many other animals, whereas the mechanisms that underlie map formation have remained unresolved.

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In the owl, the first binaural interaction occurs in the tonotopically organized NL. Information is conveyed via phase-locked spikes for frequencies up to 8 kHz [2]. For a successful localization, the temporal precision of spike encoding and transmission from the ear to the NL must be at least of the order of $50\,\mu$s, a quarter of the period of a 5 kHz tone [6]. Taking into account that delays from the ear to the border of NL ('NL delay') are between 2.5 and 3.4 ms [2] (Fig. 1b), the $50\,\mu$s precision requires some fine tuning of the neuronal architecture.

Experiments [4] have shown that ITD tuning evolves during a critical period of development. Computational approaches [6,8] demonstrate that ITD tuning of a *single* laminar neuron can be explained by means of a spike-based Hebbian learning rule. The orderly development of tuning in an *array* of NL neurons, however, requires an interaction mechanism between them. Here we suggest that presynaptic spreading of synaptic modifications [1,5] is appropriate. It results in a selection of *axons* to NL with proper delays (Fig. 1c) and suffices to explain map formation. The interaction mechanism can be orders of magnitude weaker than homosynaptic Hebbian learning.

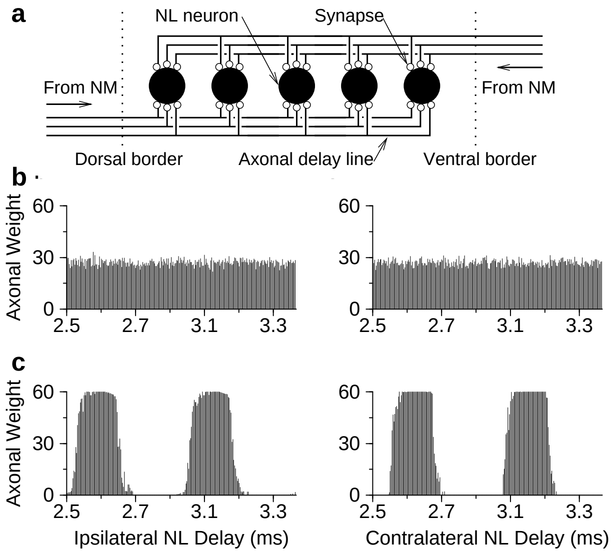

Fig. 1. Schematic diagram of the architecture of the *nucleus laminaris* (NL), i.e., part of a single frequency layer. **a**, 250 contralateral afferents (full lines, only 3 shown) enter NL through its ventral border (arrow and vertical dotted line) while ipsilateral axons enter NL dorsally. They have synaptic contacts (small open circels) with all 30 laminar neurons (large filled circles, only 5 shown). Synaptic weights are assumed to be modifiable. **b**, In young owls, the distribution of NL delays (from one papilla to the border of NL) is assumed to be broad with respect to the best frequency f (here $f = 3\,\mathrm{kHz}$) of the considered NL layer. The 'axonal weight' is the sum of all synaptic weights of an axon. **c**, After applying the learning algorithm presented in this paper, NL delays roughly differ by multiples of the period $T = f^{-1}$ (cf. [2], Fig. 9C). In other words, the learning rule selects axons with suitable delays.

Model

We have modelled $N = 30$ laminar neurons as integrate-and-fire units. An input spike at time t at synapse i evokes an excitatory postsynaptic potential (EPSP) of the form $J_i(t)\, t\, \tau^{-2} \exp[-t/\tau]$ for $t > 0$, with $\tau = 100\,\mu$s, $1 \leq i \leq 500$, and $J_i(t)$ being the time-dependent synaptic strength. A neuron fires, if the membrane voltage, i.e. the sum of all EPSPs, reaches a threshold which is chosen to be 96 times the EPSP-amplitude for $J_i = 1$. After firing, the membrane voltage is reset to zero. At the beginning of learning, synaptic efficiacies J_i are randomly distributed in the interval $[0.57, 1.23]$.

Laminar neurons are positioned regularly in a row with a distance of $27\,\mu$m between neighbors (Fig. 1a). Conduction velocities of spikes within NL are assumed to be constant (4 m/s) for all axons [2]. Laminar neurons are receiving input through 500 afferents, 250 from each side. The individual latencies ('NL delays') through axons from the papilla to the NL border are uniformly distributed in an interval between 2.5 ms and 3.4 ms (see Fig. 1b). Input spike trains are generated independently for each afferent by an inhomogeneous Poisson process with mean rate 667 Hz. Unless stated otherwise, the Poisson rate is T-periodic ($T = 333\,\mu$s) with a temporal jitter of $40\,\mu$s; see, e.g., [6]. To simulate changing spatial positions of statistically independent sound sources, we randomly initialize the mean phase (independently for each ear) of the stimulating 3 kHz tone every 100 ms. As a consequence, we find ipsi- and contralateral synaptic weights evolve almost independently of each other.

In spike-based Hebbian learning [6,8,12], synaptic strengths are increased (decreased), if presynaptic spikes arrive before (after) the postsynaptic neuron fires. The temporal constraint is implemented by means of a learning window [6]; cf. Fig 2. Its amplitude is measured in units of the learning parameter $\eta = 1/4000$, adjusting the speed of synaptic change. Furthermore, we use a weight increment of $w^{\mathrm{in}} = \eta/50$ following every presynaptic spike and a decrement $w^{\mathrm{out}} = -\eta/4$ of all synaptic strengths after each postsynaptic spike [5]. There is a lower bound 0 and an upper bound 2 for each individual synaptic weight. We assume that the width of the learning window in laminar neurons

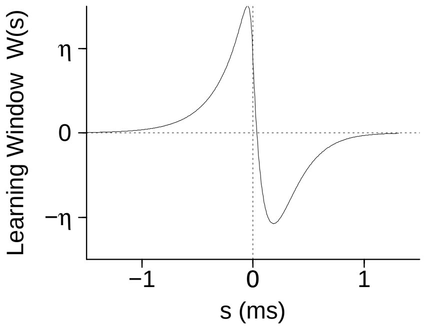

Fig. 2. Homosynaptic alterations of synaptic strengths are governed by the time difference s between pre-, and postsynaptic spike. The learning window $W(s)$ represents the idea of temporally sensitive synaptic change.

is in agreement with the membrane time constants [11] so that it is significantly smaller than that observed in tectal neurons [12]. This spike-based learning rule leads to a normalization of the mean output rate (about 200 Hz) of a single postsynaptic neuron and a selection of *synapses* in a competitive self-organized process [6,8,9].

A coordinated development of ITD tuning along the array of NL neurons requires an interaction mechanism between them. 'Non-specific axonal learning' [1,5] serves this purpose. We assume that the change ΔJ of a synaptic efficacy through homosynaptic learning gives rise to a factor that 'propagates' along the presynaptic axon and induces changes in all other synapses of the same afferent by an amount $\rho\,\Delta J$ where the interaction parameter ρ is small and positive.

Formation of a Temporal-Feature Map

After the above learning rule has been applied until the synaptic weights have reached their bounds (depending on η; [8]) with interaction parameter $\rho = 0.017$, each of the laminar neurons exhibits ITD tuning (example in Fig. 3a) and the 'best ITD' varies in an orderly manner throughout NL. Figure 3b shows how ITD $= 0$ is converted into a place code as proposed by Jeffress [7]. Moreover, the final distribution of axonal weights is clearly structured (Fig. 1c). NL delays of axons with nonvanishing weights differ by multiples of the period T of the stimulating tone. This distribution of NL delays is a necessary prerequisit for a map of ITD-tuned neurons.

The quality of the final map depends on the interaction parameter ρ. For $\rho = 0$ during learning there is no map. The order of the final map increases with increasing ρ and approaches an upper limit at about $\rho = 0.017$, where we obtain an almost perfect ITD map (cf. Fig. 1c). That is to say, non-specific axonal learning being two orders of magnitude weaker than homosynaptic Hebbian plasticity is sufficient for map formation in the laminar nucleus.

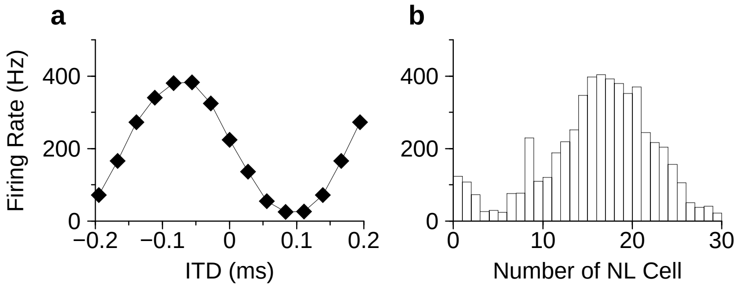

Fig. 3. Firing rates of NL neurons after learning. **a**, ITD-tuning of a single neuron with 'best ITD' of about $-70\,\mu$s. **b**, For fixed ITD $= 0$, the firing rate varies as a function of the spatial position of neurons along the array.

The observed result is very robust against variation of various parameters. A similar map has been obtained by stimulating, e.g., with a 5 kHz tone (Fig. 4a). In Fig. 4b, stimulating again at 3 kHz, we have changed the axonal conduction velocity from 4 m/s to a distribution of conduction velocities (4 ± 0.5 m/s). Results do not change substantially either as compared to Fig. 1c, if we stimulate with a 4th-order linearly filtered white noise (centered at 3 kHz, filer width 1 octave) and use the half-wave rectified and normalized filter output as Poisson intensity for the input process (Fig. 4c). Neither does a restriction of non-specific propagation on 8 neighbouring cells (200μm) prevent map formation (Fig. 4d).

Conclusion

We offer a solution to the longstanding problem of how a map of ITD-tuned neurons in the barn owl's NL can be formed. It has been shown here that the evolution of an ITD map might be based on an interplay of local, time-resolved,

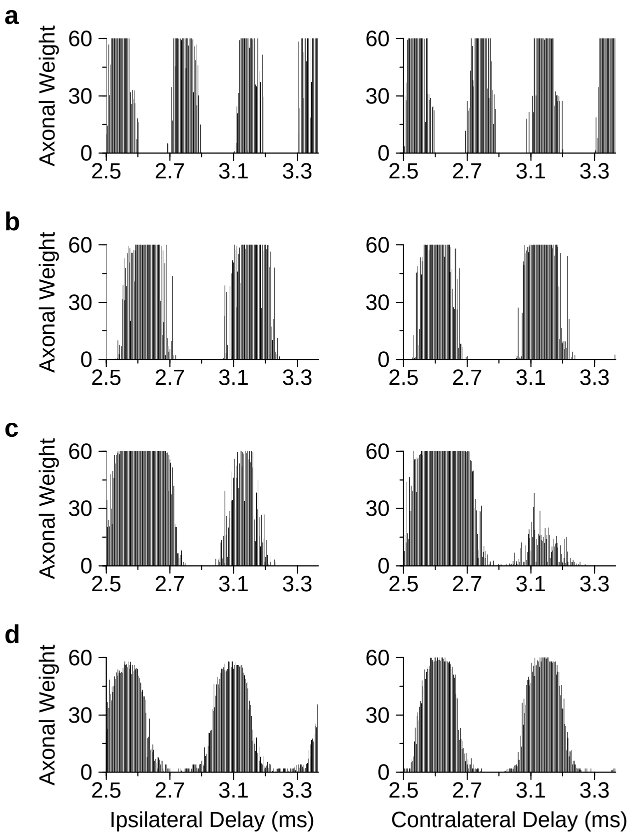

Fig. 4. A wide range of parameters and initial conditions yields a selection of NL delays and, thus, to a *map* of ITDs. **a**, 5 kHz stimulus. **b**, 3 kHz stimulus and a distribution of axonal velocities (4 ± 0.5m/s). **c**, Papillary filtered noise. **d**, Restriction of non-specific propagation to the neighboring 8 cells.

Hebbian learning [6,12,8] and its non-specific axonal propagation [1,5]. The strength of the interaction can be orders of magnitude weaker than long term potentiation at the level of a single neuron. Other interaction mechanisms can be ruled out [10]. Presynaptic propagation of synaptic modifications might be a key mechanism to the formation of computational maps whenever time plays a key role.

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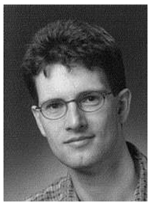
Christian Leibold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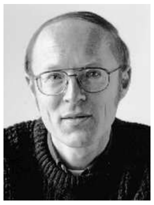
J. Leo van Hemmen