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Roles for short-term synaptic plasticity in behavior

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Abstract

Short-term synaptic plasticity is phylogenetically widespread in ascending sensory systems of vertebrate brains. Such plasticity is found at all levels of sensory processing, including in sensory cortices. The functional roles of this apparently ubiquitous short-term synaptic plasticity, however, are not well understood. Data obtained in midbrain electrosensory neurons of *Eigenmannia* suggest that this plasticity has at least two roles in sensory processing; enhancing low-pass temporal filtering and generating phase shifts used in processing moving sensory images. Short-term synaptic plasticity may serve similar roles in other sensory modalities, including vision.

Keywords: *Eigenmannia*; Gymnotiformes; Depression; Facilitation

1. Introduction

Short-term synaptic plasticity is ubiquitous in ascending sensory systems. The mechanisms underlying this use-dependent modification of synaptic efficacy have been intensively studied, but the roles of plasticity in sensory processing and behavior are not well understood. We have used behavioral and neurophysiological experiments to examine the function of short-term synaptic plasticity in awake, behaving *Eigenmannia*. In this review we will show how short-term synaptic depression is used in behaviorally relevant low-pass temporal filtering [7,8,20].

Neurons with low-pass temporal filtering properties are common in vertebrate sensory systems. These neurons are characterized by vigorous responses to ongoing low temporal-frequency stimulation and weak or no responses to ongoing higher temporal-frequency stimulation [15–17]. Many neurons with low-pass filtering properties, however, have what was believed to be paradoxical responses to certain time-varying stimuli; brief stimuli or the onset of high temporal-frequency stimuli can also elicit vigorous responses [5,7,12,20]. Such transient responses were, until recently, considered paradoxical from an engineering point of view because these stimuli are composed of temporal frequencies that would be rejected by a static low-pass filter.

Recent theoretical and experimental data suggest that these response profiles may be a result of short-term synaptic plasticity—particularly short-term synaptic depression [5]. Data obtained in *Eigenmannia* demonstrate a role for short-term synaptic depression in generating temporal filtering; short-term depression is used to significantly increase the magnitude of low-pass filtering [7]. This is intriguing because previous work has shown that the passive and active membrane properties of neurons can generate significant low-pass temporal filtering [6]. Theoretically these membrane properties could be used in CNS circuits to produce any magnitude of low-pass filtering.

If additional mechanisms are available to enhance low-pass temporal filtering, why does the nervous system employ short-term synaptic depression for this function? One of the consequences of short-term synaptic plasticity is a shift in the phase of peak responses relative to neurons with no such plasticity. A model of synaptic plasticity suggests that these shifts may be used in the processing of moving sensory images [5]. Preliminary evidence from *Eigenmannia* supports this view—phase shifts appear to generate time disparities that may underlie directional selectivity [8].

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2. Temporal filtering in *Eigenmannia*

Fish of the genus *Eigenmannia* are ideal for studying the mechanisms for low-pass filtering because they exhibit a robust behavior that requires low-pass filtering of electrosensory information for its control. This behavior is the jamming avoidance response, or JAR. In *Eigenmannia*, ongoing low temporal-frequency interference of 3–8 Hz impairs electrolocation whereas ongoing high temporal-frequency interference, e.g. above 20 Hz, has little effect [9,14]. In the JAR, fish avoid detrimental low temporal-frequency interference by changing the frequency of their own electric organ discharges (EODs). Low temporal-frequency interference elicits maximal JARs [2,4,10,16] whereas higher temporal-frequency information elicits weak or no JAR (see Fig. 1).

Low temporal-frequency information e.g., modulations in signal amplitude and phase, can result from the mixing of the EODs of neighboring fish that have similar EOD frequencies. We use the term "temporal-frequency" in this manuscript to differentiate the carrier frequency of EOD signals, which in *Eigenmannia* ranges between 200 and 450 Hz, and the frequency of modulations of EODs, which carries information for the JAR and other behaviors. The term "temporal-frequency" is derived from the fact that, in the relevant electrosensory system (P-type tuberous), AM rate is represented using a temporal code. All P-type tuberous electroreceptors encode AMs of the fish's EOD of rates up to about 50 Hz with little decline in average spike rate. The temporal structure of modulations is encoded almost entirely in the temporal organization of stimulus-related spikes. There is no spatial map of AM frequency in the ascending electrosensory system.

Primary electrosensory afferents terminate in the electrosensory lateral line lobe (ELL) and, in the case of P-type tuberous units, innervate three zones in parallel to form three somatotopic maps. Temporal filtering appears to begin in the ELL, with some neurons (particularly 'I' units) in the centromedial somatotopic map showing low-pass filtering properties [21]. It is presently unclear to what degree these filtering properties are due to mechanisms resident in the ELL versus those imparted by descending feedback from the midbrain [1]. Projections from the three tuberous maps converge in the contralateral torus semicircularis to form a single map of the electroreceptive body surface. The density of ELL projections is greatest in the dorsal layers of the torus. It is in the dorsal torus that population-level selectivity for temporal frequencies of 3–8 Hz is first seen [16]. The mechanisms that generate this behaviorally relevant selectivity have been the focus of our research in the past few years. It is in this context that we have studied the roles of membrane properties and of short-term synaptic plasticity in the generation of temporal filters.

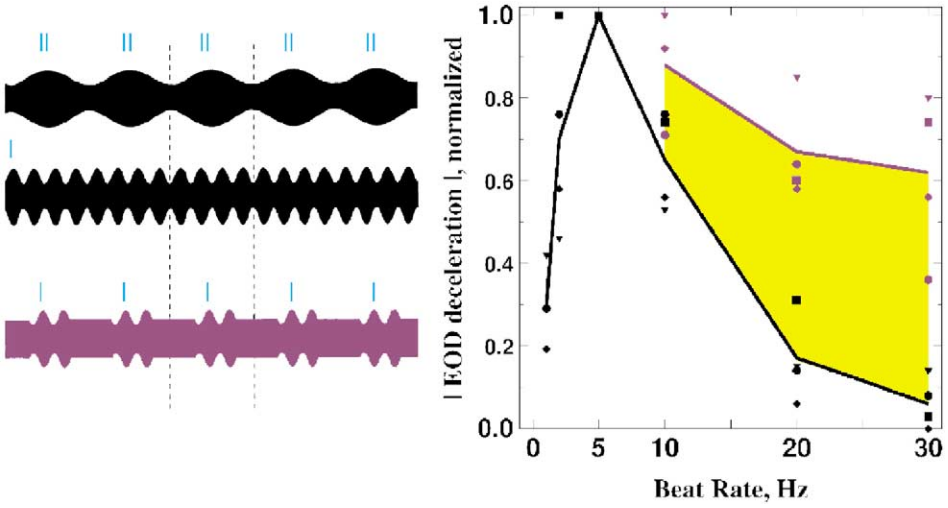

Fig. 1. Behavioral evidence for plasticity-enhanced low-pass filtering. Left, oscillograms of sensory stimuli—black are continuous AM rates of 5 and 20 Hz. Purple is a discontinuous stimulus with an AM rate of 20 Hz gated at a rate of 5 Hz. Vertical dotted lines are 200 ms apart, representing the duration of one cycle of a 5 Hz signal. Blue ticks represent responses of a toral neuron with short-term synaptic depression to these stimuli. Such a neuron would respond strongly to each cycle of the 5 Hz stimulus, and only to the first one or two cycles of a continuous 20 Hz stimulus. The neuron would respond to each burst of a stimulus gated at a rate of 5 Hz. Right, behavioral responses of four fish to the electrosensory AM stimuli. Magnitude of EOD decelerations (normalized for each fish to the maximum deceleration evoked from that fish) are plotted versus stimulus AM rate. Each symbol type—e.g. triangles, diamonds—represents data from an individual fish. Black symbols are data from continuous electrosensory stimuli, purple symbols are data from gated stimuli. Gated stimuli include 10, 20, and 30 Hz AMs gated at a rate of 5 Hz. Lines are mean responses to each stimulus type across fish. The yellow area highlights the enhancement of low-pass filtering presumed to be due to short-term synaptic depression (modified from Ref. [20]).

3. Mechanisms for low-pass temporal filtering: membrane properties

Our recent work has demonstrated that temporal filtering is due to mechanisms resident in the torus [7]. Following the finding that low-pass temporal filtering is correlated with dendritic spine density [19], we have systematically identified and quantified the magnitude of the contributions of particular mechanisms present in the torus to low-pass filtering using intracellular recordings and anatomical analyses. Classes of neurons with larger dendritic arborizations and greater spine densities have, on average, greater low-pass temporal filtering to behaviorally relevant sensory stimuli than classes of neurons with small, smooth dendritic arborizations [18,19].

We tested the hypothesis that these morphological characteristics contribute to differences in the passive electrical properties of the neuron. To determine the behaviorally relevant contribution of passive electrical properties, sinusoidal current, 0.1 nA peak to peak, was passed through the recording electrode (Fig. 2A). The range of frequencies, 2–30 Hz, matched the range of temporal-frequencies used in sensory stimuli. Because the recordings were achieved in intact animals, direct comparisons between sensory responses and the biophysical measurements were possible. The data demonstrated a correlation between magnitude of low-pass filtering due to passive membrane properties and dendritic morphology [6,19]. Classes of neurons with larger dendritic arborizations and more spinous dendrites showed up to 6 dB of low-pass filtering, measured as the decline in the amplitude of voltage responses to current injection over the frequency range of 2–30 Hz (Fig. 2). In contrast, classes of neurons with smaller dendritic arborizations and few or no spines showed less passive electrical filtering, <2 dB, over this range of frequencies.

Neurons, however, could show up to 18 dB of low-pass filtering in response to sensory stimuli over the same range of temporal-frequencies. Indeed, in few neurons did passive membrane properties account for all of the low-pass filtering seen in response to sensory stimuli [20] (see Fig. 2). Additional mechanisms must therefore contribute to the low-pass filtering. Behavioral and early neurophysiological evidence suggested that plasticity may contribute to low-pass temporal filtering in this system.

4. Behavioral evidence of plasticity-enhanced low-pass filtering

As mentioned earlier, the JAR is strongest for beat rates of 3–8 Hz. This behavior results from the simultaneous evaluation of modulations in signal amplitude and differential phase. To focus on the filtering of just amplitude modulation information, we utilized a related electrosensory behavior, the deceleration response to amplitude modulations [22].

In this behavior *Eigenmannia* lowers its EOD frequency in response to slow modulations of the amplitude of an electrosensory stimulus [22]. Largest decelerations are elicited by modulation rates of about 5 Hz (up to 10 Hz drop in EOD frequency) whereas modulation rates of 20 Hz and above generate weak or no decelerations in EOD frequency. This band-pass behavioral response is shown in Fig. 1 (black). The deceleration response to AMs alone accounts, in many fish, for most of the deceleration of the EOD frequency that fish produce when jammed by a higher frequency neighbor in the JAR [22]. These data constitute a behavioral correlate of the strong preference of midbrain neurons for slow AMs.

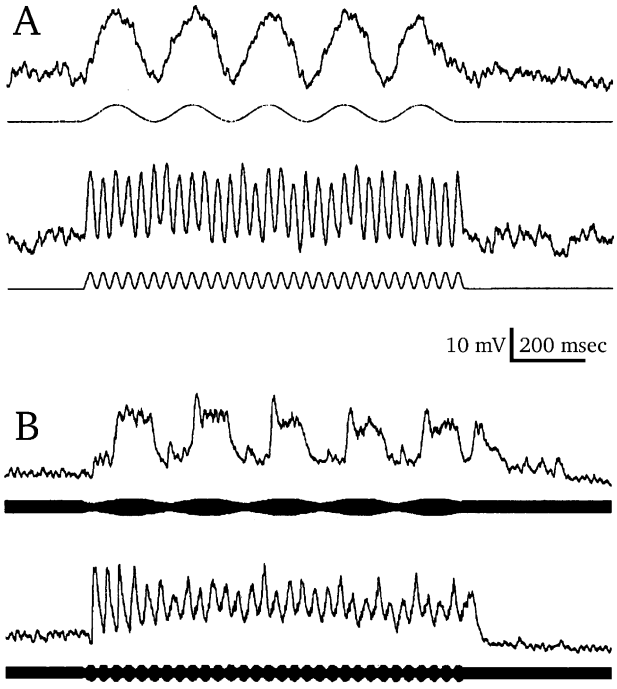

Fig. 2. Contributions of passive membrane properties and short-term plasticity to low-pass temporal filtering. (A) Voltage responses to 0.1 nA positive-going current injection. There is about a 6 dB reduction in the amplitude of voltage responses elicited by the 5 and 30 Hz stimuli. Voltage responses were of consistent amplitude throughout the duration of sinusoidal current injection. (B) Responses to sensory stimulation, 5 and 30 Hz AM rates. Roughly 6 dB of PSP depression, the reduction in PSP amplitude from the first few cycles in comparison to the last cycles, is evident in the response to the 30 Hz stimulus. Much weaker PSP depression was elicited by the 5 Hz stimulus. Also, current injection did not elicit depression at either rate. The total reduction in PSP amplitude from the maximum response to 5 Hz sensory stimulation to the minimum response to 30 Hz stimulation is much greater than the reduction in voltage responses to current injection over the same frequency range (A). Holding current was −0.1 nA in both A and B (modified from Ref. [20]).

To test the hypothesis that dynamic processes, e.g. short-term synaptic depression, contribute to this filtering, we measured deceleration responses to a stimulus in which a 20 Hz AM was gated on and off at a rate of 5 Hz. In the gated stimulus, the amplitude of the stimulus is alternately modulated at 20 Hz for 100 ms (two modulation cycles) and then held constant for 100 ms. The gated stimulus elicited responses that are nearly as large as those to 5 Hz AM (Fig. 1, purple) [20]. These results are consistent with the model that the low-pass neural filters are excited initially by fast AMs, but that this responsiveness declines with maintained stimulation; the 100 ms separation between pairs of pulses evidently was sufficient for limiting the development of this attenuation process. In this model, strong transient neural responses at the onset of each burst in the gated stimulus elicit large behavioral responses.

These behavioral responses to gated stimuli are, however, less than those to a continuous 5 Hz stimulus. The larger responses to the 5 Hz AM stimulus are expected because active membrane properties can augment neural responses to low temporal frequencies, and passive membrane properties attenuate transient responses to the onset of high-temporal frequency bursts [6].

5. Synaptic plasticity in midbrain electrosensory neurons

A neural correlate of the behavioral results exists in the torus where frequency dependent declines in PSP amplitude to sustained electrosensory stimuli were observed in at least 60% of neurons [20]. The time course of depression was well-fit using double exponentials; the first order time-constant was on the order of tens of milliseconds and the second on the order of seconds. Nearly complete recovery from depression could occur in less than 150 ms. The magnitude of temporal-frequency dependent PSP depression was significant, up to 12.5 dB (mean 4.5 dB). For most toral neurons, all of the low-pass filtering of sensory information was explained by the combination of membrane properties and temporal-frequency dependent plasticity [20]. In the few toral neurons with unexplained low-pass temporal filtering, direct stimulation of afferents demonstrated that the additional filtering was a result of low-pass filtering in ELL afferents (see below).

This plasticity was elicited by sensory stimuli, but was not seen in the voltage responses to sustained high frequency current injection (>15 Hz, Fig. 2A). The reduction in PSP amplitude, therefore, was not due to postsynaptic changes induced by rapid fluctuations in the membrane potential, i.e. fluctuations resulting from high temporal frequency stimulation. The plasticity likely results from short-term synaptic plasticity or other mechanisms upstream of the torus. To determine whether this plasticity was due to processes within the torus vs. other mechanisms upstream of the torus, we placed stimulating electrodes into a region of the lateral lemniscus that contains toral afferents from the ELL.

Direct stimulation of toral afferents elicited strong short-term PSP depression (Fig. 3). This result places the mechanism underlying the declines in PSP amplitude in the torus. A detailed analysis of the responses to pairs of stimulus pulses applied to afferents in the lateral lemniscus indicate that the reduction in PSP amplitude is likely due to short-term synaptic depression, not loss of facilitation [7]. This stimulation paradigm also revealed, however, that short-term facilitation could, under specific stimulation regimes, overcome the effects of the short-term depression. This facilitation is triggered by patterns of activity associated with low temporal-frequency stimuli, whereas depression is triggered by patterns resulting from high temporal-frequency stimuli. The combination of facilitation and depression, therefore, appears to maintain desired responses to low temporal-frequency information (facilitation) in the presence of reduced responses to ongoing high temporal-frequency interference (depression).

Stimulation of the lateral lemniscus also revealed that robust EPSPs could be elicited by pairs of pulses delivered at 20 pairs/s even in cells where sensory stimuli of this temporal frequency were ineffective; several neurons failed to respond well to even the onset of fast temporal frequency sensory stimulation. At fast stimulation rates, EPSPs that were elicited by lateral lemniscal stimulation depressed in amplitude, much like those in cells that showed PSP depression to sensory stimulation. These findings are consistent with the finding that some ELL

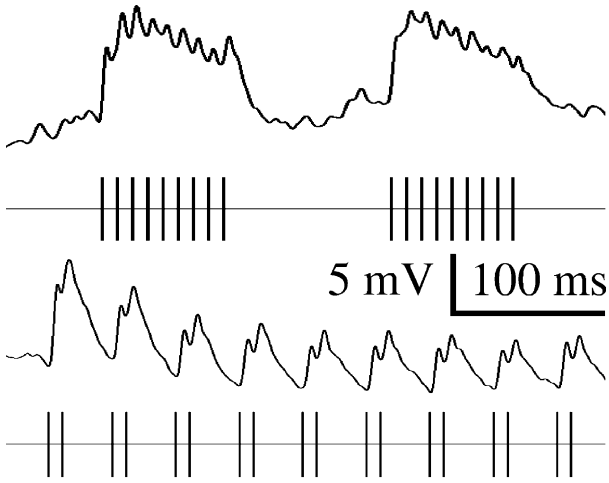

Fig. 3. Temporal-frequency dependent effects short-term synaptic depression. PSPs resulting from two patterns of stimulation of electrosensory afferents in the lateral lemniscus. Each stimulus, 5 Hz-like (top) and 20 Hz-like (bottom), has the same number of pulses. Recordings made with -0.1 nA holding current, data filtered to remove stimulus artifacts. PSP amplitude declines within the first 100 ms of the 20 Hz-like stimulation pattern. The PSPs from 5 Hz-like stimulation are asymmetric—the peak response occurs by the third stimulus pulse (from Ref. [8]).

neurons, particularly those in the centromedial map, exhibit low-pass properties.

These results suggest that short-term plasticity, in the torus and possibly elsewhere, are indeed involved in the generation of the behavioral low-pass filter. Why use plasticity to generate low-pass filtering? One possible answer to this question comes from studies of the mammalian visual system, where synaptic depression has been implicated in a model for computing the direction of image motion [5].

6. Synaptic depression as a mechanism for processing moving sensory images

Following the discovery of direction-selective responses in cat visual cortex [see 11], considerable attention has been focused on determining the mechanisms that underlie this selectivity. Direction selectivity has now been found in numerous sensory systems, including in midbrain electrosensory neurons. One general hypothesis for the generation of direction selectivity involves the use of delay lines from an array, or more simply, a pair, of receptive fields. In this scenario, motion in one direction across the array leads to coincident input at the level where information converges from different parts of the array; motion in the opposite direction results in temporally asynchronous inputs to the motion detector.

Delay lines of this sort have been postulated [3] but have not been found in systems with direction selectivity. Also, there is little evidence that differences in tau (time constant) and lambda (length constant) are widely used to generate delay lines (dramatic examples do, however, exist in the auditory brainstem). A model proposed by Chance et al. [5] suggests that phase advanced inputs, generated by short-term synaptic depression, may be combined with inputs that lack such synaptic plasticity to produce directional selectivity.

The general features of a similar model are presented in Fig. 4. Neurons without short-term synaptic depression (particularly those that show facilitation) will reach peak PSP amplitudes many milliseconds after identical neurons with depression in response to sensory stimulation, e.g. amplitude modulations (Fig. 4A). In the neuron with synaptic depression, depression limits the rise in PSP amplitude over time, such that the maximum PSP amplitude occurs during the first few tens of milliseconds of stimulus onset (Fig. 5). In the neuron without depression, PSP amplitude would continue to rise. In this model, the 'advance' in EPSP peak is a result of depression limiting it from reaching full amplitude i.e., the amplitude maximum that would occur if inputs did not experience synaptic depression. It is important to note that the phase of the initiation of firing of two neurons, one with and one without short-term synaptic depression, may not differ. However, one would expect the distribution of spikes over the entire stimulus cycle would be phase advanced in a neuron with synaptic depression relative to one without such depression. The strength of the short-term synaptic depression, therefore, affects the magnitude of the phase shift. Phase shifts of 20 °, representing more than 25 ms time difference for low-temporal frequency signals, appear plausible (Fig. 5). An advance in peak PSP amplitude, without limiting the maximum amplitude, may be achieved through the interplay of short-term synaptic facilitation and depression. These data appear similar to intracellular responses to moving stimuli recorded from neurons in cat visual cortex [13].

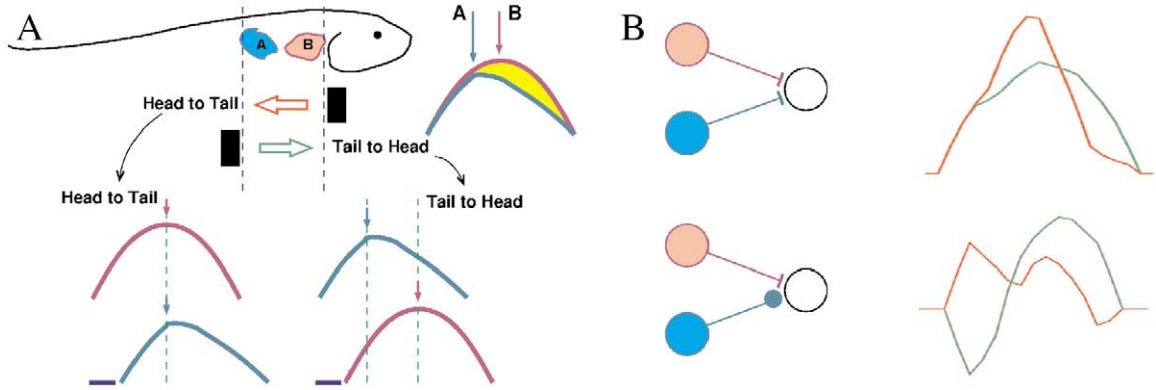

Fig. 4. Direction selectivity using depression-based temporal disparities. (A) Relation of phase shifts and direction of moving sensory images. Synapses made by input A show synaptic depression, those from input B do not. If both receptive fields were activated simultaneously, EPSPs from input A would be advanced in their peak amplitude, relative to those from B (arrows). The peaks would be coincident when objects move from head to tail, not tail to head. (B) Generation of direction selectivity. In this model toral neurons (open circles) receive inputs from ELL afferents that have adjacent receptive fields, A and B, as in (A). Top; both afferents make excitatory synapses onto a toral neuron. Toral neuron will respond maximally when receptive field B is activated prior to A, as in head-to-tail motion of an object; the summated EPSPs for head-to-tail (red) and tail-to-head (green) motion are shown. Bottom; in this model A is an inhibitory receptive field. Note that direction selectivity is maintained, but the maximal response is now in the tail-to-head direction.

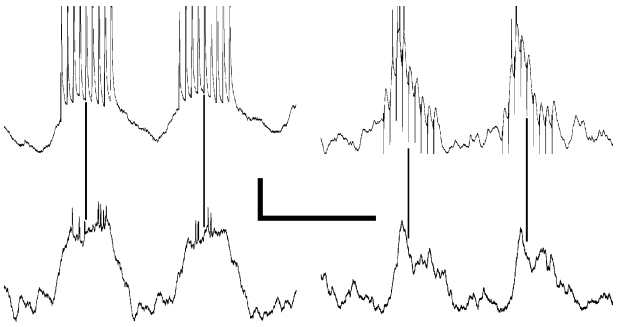

Fig. 5. Recordings showing plasticity-based temporal disparities. Stimulus-related PSPs recorded from neurons that showed little short-term synaptic depression (A) and strong depression (B). Responses were elicited by sensory stimulation (bottom) or direct stimulation of the toral afferents in lateral lemniscus (top). Grey lines indicate the middle (5th pulse of 9) of both types of stimulus. (A) PSPs are relatively symmetric. The large "spikes" on the PSP are not action potentials; they are stimulus artifacts. The downward components of the artifacts have been clipped to reveal the time course of the PSP. (B) PSPs are asymmetric—peak is phase advanced relative to those in (A). The upward components of stimulus artifacts, but not action potentials, have been clipped in the upper trace. Scale bar indicates 5 mV and 50 ms.

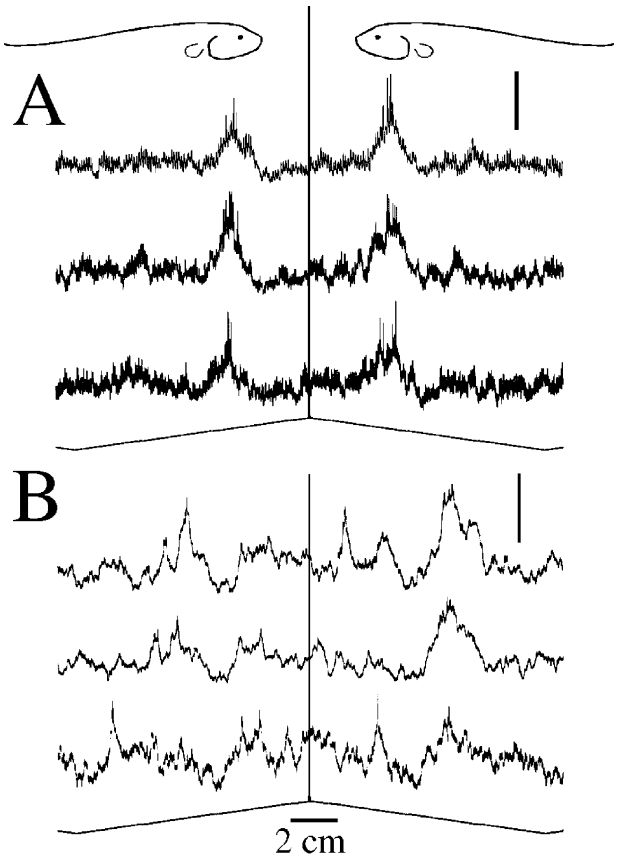

Fig. 6. Responses to moving stimuli. Intracellularly recorded responses of a neuron that showed directional selectivity (B) and another that did not (A). The object, a 1.8 cm stainless steel plate, insulated on one side, was moved linearly on a longitudinal path approximately 1.5 cm lateral to the fish. At its most rostral extent, the caudal edge of the object was 2 cm past the tip of the snout. Objects moved from tail to head, and then reversed direction. Plots show one sweep—tail to head and back. Objects were moved at three different velocities, 6.7 cm/s (top traces), 5.0 cm/s (middle traces), and 4.0 cm/s (bottom traces). Traces in (A) and (B) have been scaled in the time domain so that responses match position on the body surface. As a result, the scale bar represents 300, 400, and 500 ms for the top, middle, and bottom traces, respectively. Notice that the responses shown in (B) head-to-tail (the preferred direction), are also sensitive to velocity, and are similar to the profile shown in the model with an inhibitory synapse (Fig. 3B). Scale bars indicate 10 mV.

The model for generating direction selectivity requires the convergence of information from populations of neurons both with and without short-term synaptic depression. The convergence of information could potentially include a variety of combinations of excitatory and inhibitory inputs. A model with two receptive fields and excitatory synapses shows that direction selectivity emerges as a consequence of coincident arrival of maximal responses on the postsynaptic neuron when objects travel in the preferred direction (Fig. 4B). Alternatively, a combination of excitatory and inhibitory inputs from neurons with and without short-term synaptic depression also could generate direction selectivity. In models with excitatory and inhibitory components, maximal responses occur in the postsynaptic neuron when inputs are not coincident. Directional selectivity using a combination of inhibitory and excitatory inputs may also be enhanced by post-inhibitory rebound.

Preliminary evidence is consistent with the hypothesis that short-term synaptic depression is used as a mechanism for generating time disparities of inputs for direction selectivity. We examined the sensory responses of three toral neurons to moving objects (Fig. 6). One neuron did not show direction-selectivity (Fig. 6A). This neuron had rapid PSPs that did not decline in amplitude to any of the sensory stimuli tested. The neuron showed no apparent synaptic depression and was relatively insensitive to changes in object velocity. Recordings from one of the two neurons that were directionally selective are shown in Fig. 6B. This neuron showed an IPSP prior to a large EPSP in response to an object moving in the preferred direction. The shapes and temporal arrangement of the PSPs appear similar to those predicted by the model that uses a combination of excitatory and inhibitory inputs. Also, the direction selectivity appeared to be velocity dependent, which may result from the 'delay line' mechanism: because delays are fixed, only particular velocities will result in the desired temporal distribution of afferent activity.

7. Conclusions

The mechanisms that underlie low-pass temporal filtering include short-term synaptic plasticity; synaptic depression enhances low-pass temporal filtering. This

role of synaptic plasticity is particularly intriguing when one considers that alternative mechanisms of low-pass filtering e.g., those resulting from passive and active membrane properties, are already implemented in this system. From an engineering perspective, it would appear advantageous to build low-pass filters by employing active membrane properties and several stages of electrical filtering.

Why synaptic plasticity is used for this filtering is best understood in the context of the natural and evolutionary histories of the organism. Short-term synaptic depression probably serves a number of roles, many of which we cannot currently identify. Synaptic depression appears to be phylogenetically widespread in ascending sensory systems, and is likely an ancient mechanism. The detection and processing of moving sensory images is a function common to many sensory modalities in, we imagine, all vertebrate animals. Short-term synaptic depression may therefore be a phylogenetically early solution to a common functional problem. In *Eigenmannia*, synaptic depression used in processing moving sensory images may have served as a pre-adaptation for the evolution of an evolutionarily novel behavior found in this genus, the JAR. Although this particular hypothesis cannot be directly tested, further examination of the roles of synaptic depression in Gymnotiform species with and without JARs may provide some insight into the interesting issue of how neural circuits are modified through evolutionary processes to generate new behaviors.

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