Storage of a sensory pattern by anti-Hebbian synaptic plasticity in an electric fish

(corollary discharge/electrosensory system/inhibition/cerebellum)

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ABSTRACT   Synaptic plasticity occurs in several regions of the vertebrate brain and is believed to mediate the storage of behaviorally significant information during learning. Synaptic plasticity is well demonstrated in most cases, but the behavioral meaning of the relevant neural signals and the behavioral role of the plasticity are uncertain. In this paper we describe a case of synaptic plasticity which involves identifiable sensory and motor signals and which appears to mediate the storage of an image of past sensory input. Corollary discharge signals associated with the motor command that drives the electric organ are prominent in the electrosensory lobe of mormyrid electric fish. Some of these corollary discharge signals elicit a negative image or representation of the electrosensory input pattern that has followed recent motor commands. When the temporal and spatial pattern of sensory input changes, the corollary discharge effect also changes in a corresponding manner. The cellular mechanisms by which the corollary discharge-evoked representation is stored were investigated by intracellular recording from cells of the electrosensory lobe and pairing intracellular current pulses with the corollary discharge signal. The results indicate that the representation of recent sensory input is stored by means of anti-Hebbian plasticity at the synapses between corollary discharge-conveying fibers and cells of the electrosensory lobe. The results also suggest that dendritic spikes and plasticity at inhibitory synapses are involved in the phenomenon.

Many sensory regions of the brain are affected by signals of central origin that are associated with motor commands (1–3). These signals are known as efference copy (4) or corollary discharge (5) signals and prepare the sensory regions for the (re)afferent (4) input that follows a commanded motor action. Optimal interaction between reafferent and corollary discharge inputs requires an approximate match in the timing and spatial distribution of the two signals. This requirement for matching, and the likelihood of variation in reafferent input, suggests that some corollary discharge effects may be plastic. Plastic corollary discharge effects are in fact present in the mormyrid electrosensory lobe (6–8).

The electrosensory lobe is the termination site for afferent fibers from the three classes of electroreceptors in mormyrid fish: knollenorgans, ampullary organs, and mormyromasts (9). This paper is concerned only with the regions that receive ampullary and mormyromast input. Ampullary afferents project to the ventrolateral zone of the electrosensory lobe cortex, and mormyromast afferents project to the medial and dorsolateral zones (Fig. 1A). The projections are somatotopically organized. The histological structure of the cortex is like that of the cerebellum and contains Purkinje-like cells
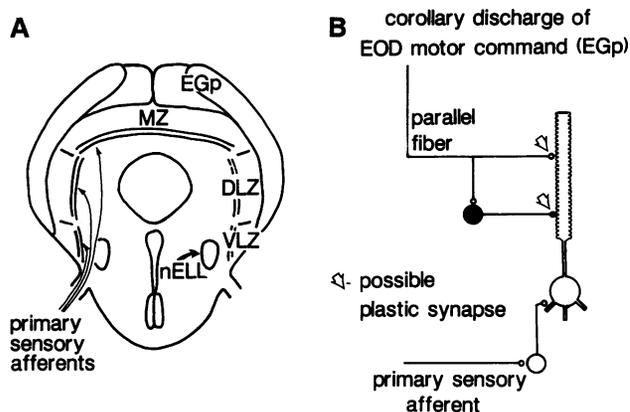

FIG. 1. Anatomical relationships and possible sites of plastic change. (A) Drawing of a transverse section through the medulla showing the electrosensory lobe and the eminentia granularis posterior. The cortex of the lobe is divided into three zones: medial (MZ), dorsolateral (DLZ), and ventrolateral (VLZ). The molecular layer lies just external to the cell layers, which are indicated by a pair of lines. nELL, nucleus of the electrosensory lobe. (B) Diagram showing some basic circuitry and suggested sites of plastic change. The diagram shows a Purkinje-like cell that is excited by primary sensory afferents acting via an excitatory interneuron. The apical dendrites of the cell are excited by parallel fibers from the eminentia granularis posterior (EGp) and inhibited by inhibitory interneurons.

(9, 10) with cell bodies in the deeper layers and apical dendrites in the molecular layer.

Each electric organ discharge (EOD) evokes reafferent responses in ampullary and mormyromast fibers (11). The reafferent responses of mormyromast afferents inform the fish about the impedance of nearby objects, whereas responses of ampullary afferents are only minimally affected by nearby objects.

With each EOD, the electrosensory lobe receives both reafferent input and corollary discharge input associated with the motor command that elicits the EOD (11, 12). The corollary discharge input arises from three or four different central sources (8, 9, 13), and the effects are both excitatory and inhibitory (7, 8). The latency of corollary discharge activity in individual fibers varies from 0 to 100 msec after the EOD motor command. The overall effect of the corollary discharge input is thus a mixture of excitation and inhibition that is distributed over time.

Some corollary discharge effects in the ampullary (6, 7) and mormyromast (8) regions of the lobe are plastic and depend on the pattern of sensory input that follows the EOD motor command. This plasticity shows temporal specificity, in that pairing an electrosensory stimulus with the EOD motor

Abbreviation: EOD, electric organ discharge.
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command, at delays of 0–100 msec after the command, results in an altered corollary discharge response at that same delay. The change is opposite in sign to the effect of the paired stimulus. Pairing with an excitatory sensory stimulus leads to a reduction in corollary discharge excitation or to corollary discharge inhibition. Pairing with an inhibitory sensory stimulus leads to an enhancement of corollary discharge excitation. The plasticity shows spatial specificity, in that pairing is effective only if the electrosensory stimulus affects the neuron—i.e., if the stimulus is within the neuron's receptive field on the skin.

The modifiable corollary discharge effect may be viewed as a negative image of the temporal and spatial pattern of sensory input that has recently followed the motor command. The modifiable corollary discharge may serve in detecting novelty in the mormyromast region (8) and in minimizing a potentially disruptive reafferent response in the ampullary region (7).

The altered corollary discharge effect disappears within 2–6 min when *the EOD motor command continues without an associated sensory stimulus*. The rapid decline is due to an active updating process. If updating is prevented by temporarily silencing the motor command after pairing, then the plastic change lasts at least 30 min (14).

The present experiments were directed toward determining the site of plastic change. The site could be either outside the electrosensory lobe, resulting in an altered corollary discharge input to the lobe, or within the lobe, resulting in an altered response to unchanging corollary discharge input (Fig. 1B).

The hypothesis of synaptic plasticity within the lobe was tested by intracellular recording from electrosensory lobe cells while pairing an intracellular current pulse with the EOD motor command (and accompanying corollary discharge input). Thus, an intracellular current pulse to a single cell was substituted for a sensory stimulus that affects many cells. If pairing with intracellular current pulses results in an altered synaptic response to the corollary discharge, and if the current pulse affects only the recorded cell, then synapses on the recorded electrosensory lobe cell must be a site of plastic change. Our results strongly support this conclusion.

METHODS

Details about the methods may be found elsewhere (7, 13). Briefly, all 35 experimental animals were of the mormyrid species *Gnathonemus petersii*. Fish were anesthetized with MS-222 (1:25,000) and the brain was exposed caudally. Fish were then given curare (0.08 mg, i.m.) to prevent the EOD by blocking the electric organ. Fresh, aerated water was passed over the gills for respiration. The isolated EOD motor command that elicits an EOD in non-curare-treated fish was recorded at the tail.

Micropipettes for intracellular recording were filled with 2 M potassium methyl sulfate, with or without a 2% solution of biocytin (120–220 MΩ). Most recordings were from the ampullary zone (ventrolateral zone) but some were from the mormyromast zones (medial and dorsolateral zones). Most recordings were probably from Purkinje-like cells (membrane potentials of 55–65 mV).

Depolarizing and hyperpolarizing current pulses were injected into the cell through a bridge. Pulses were 10–40 msec in duration and 0.3–3.0 nA in amplitude. Such pulses were paired with the EOD motor command at delays of up to 100 msec for periods of 1.5–3.0 min. Control periods of unpaired stimulation were also given in which the intracellular pulses were delivered independently of the command at fixed rates of 2–4/sec. EOD motor commands are emitted spontaneously at irregular intervals of 150–400 msec. Changes in membrane potential during the pairing period, if present,

were usually less than 2 or 3 mV. Brief electrosensory stimuli to the skin were also sometimes paired with the corollary discharge (7, 13).

Many cells did not discharge spontaneously, and averages of synaptic responses could be calculated without contamination by action potentials. Other cells discharged so rarely that traces without spikes could be easily selected for averaging. Averages of 10–15 corollary discharge responses were calculated at 15-sec intervals before and after pairing to assess the presence of plastic change. Responses of cells with many action potentials were assessed with superimposed traces or histograms.

RESULTS

Intracellularly injected current pulses were paired with the corollary discharge in 53 cells. Twenty-eight of these cells (20 from the ampullary region, 8 from the mormyromast region) showed two types of spikes—large, broad spikes (>30 mV, 10–20 msec) and small, brief spikes (<15 mV, <1 msec; Fig. 2 A and C). Two cells (both from the ampullary region) showed only the large, broad spikes (Fig. 2B). Depolarizing synaptic potentials due to sensory stimuli or the corollary discharge also elicited broad spikes. The broad spikes are presumed to be dendritic, because of the similarity to dendritic spikes in other systems (15–17) and because they were obtained only in the molecular layer [as judged by field potentials (13) and depth], which contains the apical dendrites of cells with somas in the deeper layers. The small spikes are presumed to be somatic or axonal.

Twenty-three cells (21 from the ampullary region and 2 from the mormyromast region) recorded in the deeper layers of the lobe showed only large, brief spikes (>30 mV, <1 msec). These spikes are presumed to be somatic. Biocytin injections were made into cells that showed the large broad spikes and small narrow spikes, as well as into cells that showed only the large narrow spikes. Morphological examination of such cells showed that the same Purkinje-like cells were labeled regardless of spike type (C.C.B., A.C., K.G., unpublished work).

Pairing of depolarizing current pulses with the corollary discharge was effective in altering the synaptic effect of the corollary discharge in 25 of the 30 presumed dendritic recordings. However, pairing was effective only if the current pulse evoked a broad spike. Thresholds for the small spikes in these recordings were always lower than thresholds for the large spikes. Pairing with current pulses that evoked only small spikes yielded no detectable change in the synaptic response to the corollary discharge (Fig. 2 Aa and Ca). In the same cell, however, pairing with more intense current pulses, sufficient to evoke a broad spike, resulted in a clear change in the synaptic response (Fig. 2 Ab and Cb). This same result was obtained in all seven of the cells which were tested above and below threshold for the broad spike.

The effect of pairing was temporally specific, depending systematically on the delay at which the depolarizing current pulse was given following the EOD motor command. Different responses were obtained following pairings at different delays in all 15 of the cells that were tested at two or more delays. In cells of the ampullary region, pairing with a current pulse that evoked a broad spike, at delays between 25 and 100 msec, resulted in the development of a hyperpolarizing corollary discharge response which reached a peak at the approximate delay of the previously paired broad spike (Figs. 2 Ab, B, and Cc). In mormyromast region cells, pairing at delays of 20–30 msec resulted in a reduction in the corollary discharge-driven depolarization of these cells (Fig. 3Aa). Pairing with current pulses at delays of 20 msec or less resulted in a slight hyperpolarization or reduction of depolarization at the delay of the previously paired broad spike,
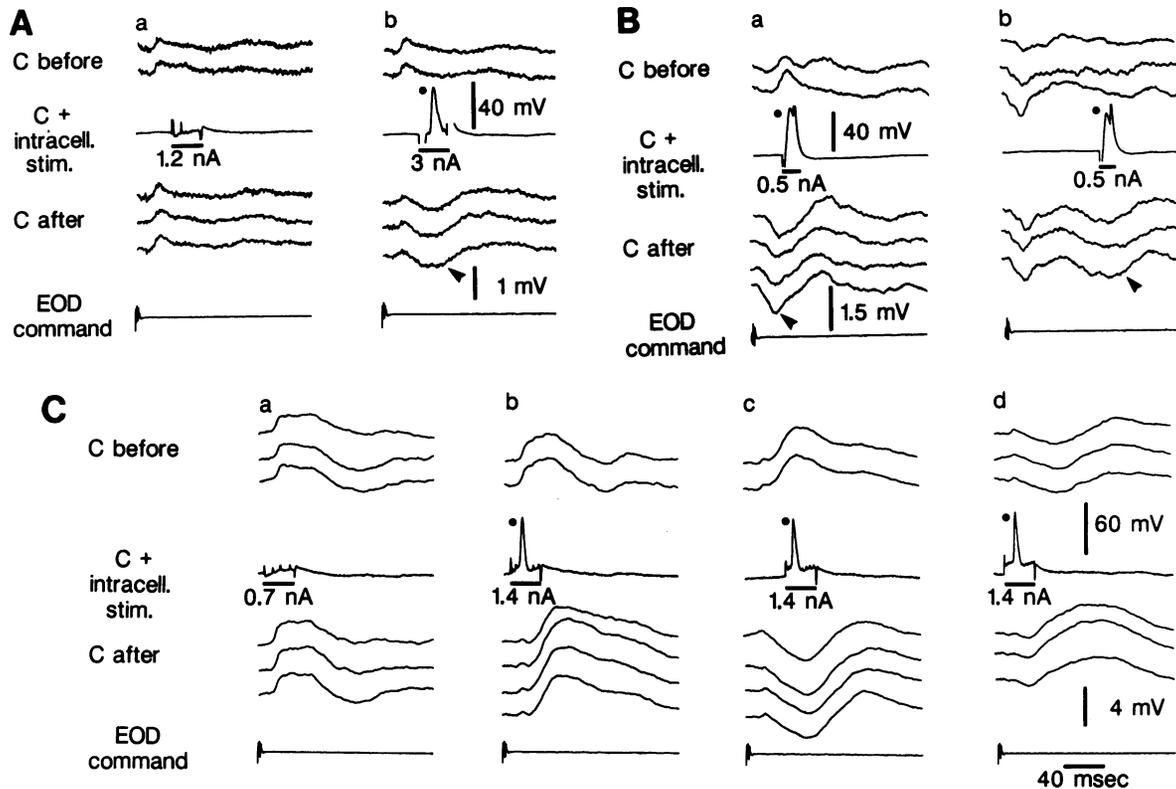
FIG. 2. Pairing with intracellular current pulses: Cells in the ampullary region paired with current pulses of different intensities and at different delays. Each group of intracellular records includes (i) a set of averages showing the synaptic effect of the corollary discharge before pairing (C before), (ii) a single sweep at a lower gain showing the effect of the intracellular current pulse (C + intracell. stim.), and (iii) a set of averages showing the synaptic effect of the corollary discharge after pairing for the stated period (C after). All sweeps were initiated by the EOD motor command, as indicated by a final trace at the bottom of each group of records. All averages were based on 15 sweeps, taken at successive 15-sec intervals just before and just after pairing. The time base is the same for all records (C, bottom right). In this figure and in Fig. 3, the gains for synaptic responses before and after pairing are shown with the bottom traces in each part of the figure. Gains for the responses to paired intracellular current pulses are shown with the single middle trace. Command signal amplitudes were between 100 and 200 μV and are not indicated. (A) Effects of pairing with intracellular current pulses that were below and above threshold for a broad spike. (a) A 1.2-nA pulse evoked small narrow spikes but not a broad spike. Pairing with this current pulse for 2.5 min had no effect on the response to the corollary discharge. (b) A more intense pulse, of 3.0 nA, evoked both small narrow spikes and a broad spike. (The broad spike is indicated by a dot in this figure and in Fig. 3.) Pairing with this pulse resulted in a hyperpolarizing response (arrowhead) to the corollary discharge at about the delay of the previously paired broad spike. (B) Effects of pairing with current pulses at different delays. A different ampullary-region cell from that shown in A was used. (a) Pairing for 2 min with a current pulse that evoked a broad spike at 30 msec resulted in an early hyperpolarizing response to the corollary discharge (arrowhead). (b) Pairing with the same pulse but at 100-msec delay resulted in a later hyperpolarizing response (arrowhead) roughly centered on the time of the previously paired broad spike. Records at the top in b were taken 3 min after those in the bottom in a. (C) Effects of pairing with current pulses at different intensities and different delays. A different ampullary-region cell from those shown in A and B was used. (a) Pairing with a 0.7-nA pulse that evoked only small narrow spikes had little effect on the response to the corollary discharge. The delay of the pulse was 10 msec and the duration of pairing was 1.6 min. (b) Pairing at the same delay and duration but with a more intense pulse, 1.4 nA, that evoked a broad spike resulted in a clear change in synaptic effect. Early excitation was reduced and later excitation was enhanced. (c) Pairing with the same current pulse but at a delay of 40 msec (again for 1.6 min) resulted in a large hyperpolarizing response to the corollary discharge. The top record in c was recorded 6 min after the bottom record in b. (d) A second pairing of the same current pulse at a delay of 10 msec resulted in a broad excitatory effect of the corollary discharge that was roughly similar to that obtained in the previous pairing at the same delay shown in b.

but this was followed by a large depolarizing potential (Fig. 2 Cb and Cd; Fig. 3Bc).

The occurrence of different corollary discharge effects in the same cell after pairing at different delays shows that the plasticity is not a generalized consequence of the depolarizing pulse alone. We examined this issue further by using unpaired current pulses. In five presumed dendritic recordings in which pairing of depolarizing current pulses with the corollary discharge altered the synaptic response, giving the same pulses at a fixed rate of 3 or 4/sec for the same amount of time had no effect (Fig. 3Ab).

Very different current intensities were needed to ensure that pulses were either always below or always above threshold for the broad spike (Fig. 2 A, Ca, and Cb). Thus, changes in the postsynaptic response to the corollary discharge could be dependent on the intensity of the paired depolarizing pulse rather than on the occurrence of a broad spike. In two cases,

however, hyperpolarizing pulses evoked broad spikes at the "off" of the pulse (anodal break response). Pairing with such pulses was effective in these cells (Fig. 3Bb), whereas pairing in the same cells with hyperpolarizing pulses that evoked either no response or only a small, narrow spike was not effective (Fig. 3Ba). These results strengthen the hypothesis that plasticity depends on the occurrence of the presumed dendritic spike.

Plastic changes were less frequently observed in recordings with only large narrow spikes. A change in corollary discharge effect was observed in only 10 of 23 recordings of this type, when depolarizing current pulses that evoked spikes were paired with the corollary discharge input. Following pairing at delays of >25 msec, the 10 affected cells developed hyperpolarizing corollary discharge responses at the approximate delay of the previously paired depolarizing pulse.
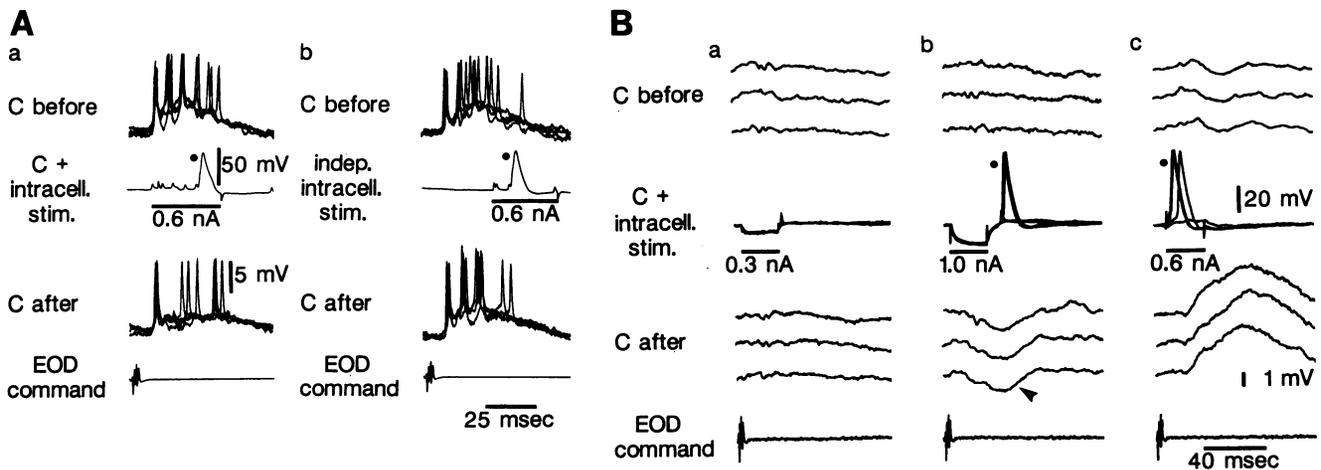
FIG. 3.   Pairing with intracellular current pulses: Unpaired control and effect of hyperpolarizing current pulses. (*A*) Pairing in a mormyromast region cell with an intracellular current pulse together with unpaired control. (*a*) Effects of pairing. As with many cells in the mormyromast region, the corollary discharge alone elicits a strong burst in this cell (C before). The corollary discharge was paired for 2 min with an intracellular pulse that evoked a broad spike at 30- to 40-msec latency (C + intracell. stim.). Such pairing results in a reduction of the corollary discharge-evoked depolarizing response and in the number of spikes per burst (C after). Four superimposed traces are shown before and after pairing, and a single trace at a low gain is shown during pairing. (*b*) Effects of control period with unpaired stimuli. Six minutes after the pairing shown in *a*, the corollary discharge effect had returned to the prepairing level (C before). The same intracellular current pulse was then given independently of the command at a fixed rate of 3/sec for 2 min (indep. intracell. stim.). This period of unpaired stimulation did not affect the corollary discharge-evoked depolarizing response, although there was some reduction in the number of spikes (C after). The reduction in spike number may have been due to a 2- to 3-mV increase in membrane potential that often follows a series of intracellular current pulses that evoke a broad spike. Four superimposed traces are shown before and after the period of independent stimulation. A single trace at a low gain shows the effect of the independent stimulus (this trace was *not* triggered by the command). (*B*) Pairing in an ampullary-region cell with a hyperpolarizing pulse that evokes a broad spike at stimulus "off." As in Fig. 2, averages of 15 corollary discharge-evoked responses were taken at 15-sec intervals before and after pairing with the intracellular pulses. (*a*) Pairing for 2 min with a stimulus that evoked only a small narrow spike at current "off" had no effect. (*b*) In the same cell, pairing with a larger hyperpolarizing pulse that evoked a broad spike at current "off" resulted in a hyperpolarizing effect of the corollary discharge. (*c*) In the same cell, pairing with a depolarizing pulse that evoked a broad spike at a latency of 10 msec resulted in a slight reduction in excitation followed by a large and prolonged depolarization.

Altered corollary discharge responses following pairing with intracellular current pulses appeared to decay more slowly than those following pairing with sensory stimuli (7, 8). The altered responses were reduced but still present 6–8 min after a paired intracellular current pulse was turned off (compare the bottom traces of Fig. 2*Cc* and the top traces of Fig. 2*Cd*), whereas altered responses induced by pairing with sensory stimuli disappeared within 2–6 min.

As described previously (refs. 6–8 and above), pairing with excitatory sensory stimuli resulted in inhibitory corollary discharge effects, whereas pairing with inhibitory sensory stimuli resulted in excitatory corollary discharge effects. Such symmetry was not observed with intracellular current pulses. Pairing with depolarizing pulses at delays >25 msec yielded inhibitory corollary discharge effects, as described above, but pairing with hyperpolarizing intracellular pulses (0.6–2.5 nA) was largely ineffective. Fourteen cells were tested. Two of these cells were those described above in which the hyperpolarizing pulse evoked a broad spike at "off." Changes in the corollary discharge response were observed in only 2 of the remaining 12 cells in which a broad spike was not evoked at "off."

Our demonstration of the development of hyperpolarizing synaptic responses as a result of pairing is of particular interest because most reported examples of synaptic plasticity involve excitatory synapses (18–23). Only a few examples of plasticity at inhibitory synapses have been described (24–26). Plasticity could take place at the terminals containing γ-aminobutyrate, which are present on the dendrites of Purkinje-like cells (J. Meek and J. P. Denizot, personal communication; Fig. 1*B*).

Alternatively, the hyperpolarizing responses could be due to reduction of excitation rather than enhancement of inhibition. This issue was investigated by injecting hyperpolarizing dc currents of up to 1 nA into the recorded cells with the

expectation that hyperpolarization would enhance excitatory responses and reduce or invert inhibitory responses. Hyperpolarizing dc currents were injected before any pairing and also immediately following pairing with intracellular current pulses and with sensory stimuli. Hyperpolarizing dc current had little or no effect on newly developed hyperpolarizing synaptic responses in three cells which were tested following pairing with depolarizing *intracellular current pulses*. However, such currents did reduce the amplitude of newly developed hyperpolarizing responses to the motor command in six out of seven cells that were tested following pairing with excitatory *sensory stimuli*. The results as a whole are thus suggestive but not conclusive concerning plasticity at inhibitory synapses.

DISCUSSION

Our results indicate that synaptic plasticity takes place at synapses between fibers conveying corollary discharge signals and cells of the electrosensory lobe. This conclusion is supported by the finding that pairing of corollary discharge input with current pulses in single cells altered the postsynaptic responses to that input. Most importantly, axonal action potentials did not seem to be critical for the development of an altered corollary discharge response, indicating that the critical change was not occurring at other (hypothetical) cells that receive input from the recorded cell and which project back to it. Dendritic responses of the recorded cell did seem to be critical, however.

The plasticity observed after pairing the corollary discharge with artificial intracellular current pulses (necessary for indicating the synaptic locus) is very similar to the plasticity observed after pairing with sensory input (6–8). The latter is the natural situation and occurs throughout the fish's life. The close similarity between artificially induced

and normally occurring types of plasticity is a distinguishing feature of the phenomena described here. The relationship to normally occurring plasticity is less direct in most of the studied examples of synaptic plasticity (but see ref. 27).

Some differences were observed, however, between the effects of pairing with intracellular pulses and with sensory stimuli—differences that may be due to network properties which are brought into play with sensory stimuli. Pairing with an inhibitory sensory stimulus, for example, was very effective in altering the synaptic effect of the corollary discharge (6–8), but pairing with a hyperpolarizing current pulse was not. Some electrosensory lobe cells are excited by a stimulus in the center of their receptive fields, whereas other cells are inhibited by the same stimulus. If cells with such opponent properties were coupled by mutual inhibition, then plastic change leading to an inhibitory response in cells of one type would result in a disinhibitory or depolarizing response in cells of the opposite type, even though no plastic change occurred at synapses onto the latter cell type.

Another difference between the effects of pairing with sensory stimuli and with intracellular pulses was the large corollary discharge-evoked excitatory response that developed when depolarizing current pulses were used to evoke broad spikes at delays of <20 msec (Fig. 2 Cb and Cd; Fig. 3Bc). In contrast, hyperpolarizing responses were always the predominant effect of pairing with excitatory sensory stimuli at all delays between 0 and 100 msec. The explanation of this difference may be that the response latency of broad spikes to a sensory stimulus is always >20 msec, and thus the occurrence of broad spikes at shorter delays is not physiological.

Corollary discharge signals enter the electrosensory lobe via parallel fibers in the molecular layer from the eminentia granularis posterior and also via fibers which project directly to the deeper cell layers (8, 9, 13). The fact that dendritic spikes in the apical dendrites appear to be necessary for plastic change suggests the importance of molecular-layer input. This suggestion is supported by preliminary experiments which indicate that altered corollary discharge responses can still be induced after corollary discharge input to the deeper layers has been blocked by lesions (unpublished observations). Thus, our hypothesis is that synaptic plasticity takes place at excitatory and inhibitory synapses onto the apical dendrites of the Purkinje-like cells of the electrosensory lobe (Fig. 1B).

The temporal specificity is a rather remarkable feature of the plasticity in the electrosensory lobe. The temporal specificity can be explained if the corollary discharge-driven activity of different presynaptic fibers occurs at different delays with respect to the EOD command, and if close temporal contiguity of presynaptic impulse and postsynaptic depolarization is required for plasticity to occur. Corollary discharge-conveying fibers with different post-command delays are in fact present in the eminentia granularis posterior (13), where the parallel fibers of the molecular layer of the electrosensory lobe originate (Fig. 1).

The plasticity described here is similar to the long-term depression which has been described in the cerebellum at synapses between parallel fibers and Purkinje cells (19–22). Both types of plasticity are anti-Hebbian (28, 29), in that a correlation between presynaptic impulses and postsynaptic depolarization leads to a change in synaptic effect that reduces the correlation. In the plasticity described here, the net synaptic effect of the corollary discharge (summed excitation and inhibition) can be converted from excitation to inhibition. In this case, the overall correlation between pre- and postsynaptic events not only is reduced but is inverted in sign. Long-term depression in the cerebellum is a reduction in the efficacy of excitatory synapses between parallel fibers and Purkinje cells (19–22). Recent reports indicate that depolarization of Purkinje cells can lead to enhancement of inhibitory inputs to these cells (25, 26). Thus, anti-Hebbian synaptic plasticity may possibly affect both excitatory and inhibitory synapses in the cerebellum and in the electrosensory lobe.

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