Recurrent Feedback in the Mormyrid Electrosensory System: Cells of the Preeminential and Lateral Toral Nuclei

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Sawtell, Nathaniel B., Claudia Mohr, and Curtis C. Bell. Recurrent feedback in the mormyrid electrosensory system: cells of the preeminential and lateral toral nuclei. J Neurophysiol 93: 2090–2103, 2005; doi:10.1152/jn.01055.2004. Many sensory regions integrate information ascending from peripheral receptors with descending inputs from other central structures. However, the significance of these descending inputs remains poorly understood. Descending inputs are prominent in the electrosensory system of mormyrid fish and include both recurrent connections from higher to lower stages of electrosensory processing and electric organ corollary discharge (EOCD) signals associated with the motor command that drives the electric organ discharge. The preeminential nucleus (PE) occupies a key position in a feedback loop that returns information from higher stages of electrosensory processing to the initial stage of processing in the electrosensory lobe (ELL). This feedback reflects the integration of ascending electrosensory input from ELL, descending input from the lateral toral nucleus (torus), and EOCD inputs to PE. We used intracellular recording and axonal tracing of stained cells to characterize EOCD and electrosensory responses of several cell types in PE and the torus. PE and toral cells exhibit prominent EOCD responses that are not due to EOCD inputs from ELL. PE cells giving rise to a direct feedback projection to ELL respond to electrosensory stimuli with rapid, precisely timed spikes that will affect ELL neurons early during the same EOD cycle. EOCD and electrosensory responses in toral cells are similar to those observed in PE and may be important in shaping feedback to ELL. These results provide an initial description of electrosensory feedback to ELL as well as information about how ascending, descending, and EOCD inputs are combined at higher stages of electrosensory processing.

I N T R O D U C T I O N

Descending inputs are a prominent but poorly understood feature of vertebrate sensory systems. Descending inputs may allow sensory systems to function flexibly and adaptively by altering neural responses based on sensory contexts (Allman et al. 1985; Chacron et al. 2003; Lee et al. 2002), ongoing motor actions (Bell et al. 1992; Suga and Schlegel 1972; Toyama et al. 1984), and past experience (Bell 2001; Churchland et al. 1994; Rao and Ballard 1999). Significant progress toward understanding how various classes of descending inputs contribute to adaptive sensory processing has been made in the electrosensory systems of fish (Bastian 1986; Bell 2001; Bodznick et al. 1999; Doiron et al. 2003).

Weakly electric fish rely on active electrolocation to navigate, find prey, and avoid predators in the dark. Electroreceptors on the skin respond to an electric organ discharge (EOD) generated by an electric organ in the body of the fish. The responses of electroreceptors to the self-generated EOD current are modulated by nearby objects, creating an electrical image of the world on the body of the fish. Afferents from electroreceptors terminate centrally in the cerebellum-like electrosensory lobe (ELL). Principal cells of ELL integrate ascending electrosensory input from electroreceptors with several classes of descending inputs including: proprioceptive inputs, electric organ corollary discharge (EOCD) inputs associated with the motor command that drives the EOD, and recurrent inputs from higher stages in the electrosensory system.

One function of these descending inputs is to convey information about the fish’s own motor actions that can be used to remove predictable features from the sensory inflow. Anti-Hebbian spike-timing-dependent synaptic plasticity at parallel fiber synapses carrying EOCD inputs cancels out the predictable sensory consequences of the EOD in neurons of the mormyrid ELL (Bell 1981; Bell et al. 1997b). Plasticity at parallel fiber synapses also appears to cancel expected electrosensory reafference due to body movements and respiratory rhythms in gymnotiform and elasmobranch fish (Bastian 1996a; Bodznick et al. 1999). Additional functions for recurrent electrosensory inputs to ELL have been identified in wave-type gymnotiform electric fish (Bastian 1986, 1996b; Doiron et al. 2003).

The mormyrid electrosensory system offers a number of advantages for understanding the roles of descending input to sensory structures. In mormyrid fish, active electrosensory processing occurs at discrete times following the fish’s pulsatile EOD. Because the timing of EOCD inputs and sensory responses at various stages of the electrosensory system can all be related to the arrival of reafferent sensory inputs, it may be possible to characterize and eventually model the dynamics of recurrent loops. Accordingly, a primary goal of the present study was to characterize the timing of electrosensory responses in higher stages of the electrosensory system that project back to ELL. The mormyrid electrosensory system also provides a unique opportunity to observe motor corollary discharge effects both in isolation and in combination with sensory reafference at multiple stages of sensory processing. Accordingly, we have characterized the integration of electrosensory and EOCD inputs in a number of cell types in preeminential nucleus (PE) and the torus.

M E T H O D S

All experiments that were performed in this study adhere to the American Physiological Society’s Guiding Principles in the Care and Use of Animals and were approved by the Institutional Animal Care...
and Use Committee of Oregon Health and Sciences University. A brief overview of our general approach is followed by more detailed descriptions of our surgical, physiological, and histological methods.

Overview

Mormyrid fish of the species *Gnathonemus petersii* were used in these experiments. Surgery was performed under anesthesia, and curare was given after the surgery. Curare blocks the effect of electromotorneurons on the electric organ, preventing the EOD, but the motor command signal that would normally elicit an EOD continues to be emitted by the electromotorneurons at a variable rate of 2–5 Hz. The curare makes it possible to examine the EOCD responses in isolation from the EOD that normally follows the motor command and to control the electroosensory input that the cells receive. Responses to the motor command alone, to electroosensory stimuli alone, and to the motor command plus an electroosensory stimulus delivered at various delays were examined. EOCD plasticity was examined by delivering electroosensory stimuli at a fixed delay after the EOD command signal for 1–3 min and comparing the EOCD responses before and after such pairing.

Cells were recorded intracellularly from both the preeminential nucleus and from the lateral toral nucleus (torus) of the mesencephalon. Only those intracellular recordings with stable membrane potentials less than −50 mV were analyzed. Juxtacellular recordings were sometimes used to characterize spiking responses of various cell types. Characteristic EOCD field potentials were used to locate PE and the torus and to estimate approximate depth of the recording electrode (von der Emde and Bell 1996). In most cases, the field potentials were recorded just outside a cell after intracellular recording, using the same electrode. In some cases, the field potentials were averaged and subtracted from averaged intracellular recordings to determine the true transmembrane potential changes evoked by electroosensory stimuli and the EOCD. Cells were injected with biocytin for morphological identification after being studied intracellularly. In many cases, the axons of the cells could be traced in the tissue sections, allowing us to determine the projection sites of individual cells.

Surgical methods

A total of 62 fish with body lengths between 8 and 15 cm were used. Fish were anesthetized (MS:222, 1:25,000), and a plastic rod was cemented to the anterior skull to hold the head rigid. A portion of the skull was removed laterally to expose the valvula cerebelli that covers most of the dorsal and lateral brain surface. The valvula was exposed between the medulla and the mesencephalon where it covers PE and the torus. The anterior and posterior extero-lateral nuclei of the torus semicircularis are visible at the ventrolateral edge of the brain without reflecting the valvula. These nuclei are located anterior and ventral to PE and could be used as landmarks. Curare (d-tubocurarine, 10 µg/cm of body length) was given at the end of the surgery, the anesthesia was removed, and aerated water was passed over the fish’s gills for respiration.

Physiological methods

The EOD command signal was recorded with a Ag-AgCl plate placed over the electric organ. The command signal is the synchronized volley of electromotorneurons that would normally elicit an EOD in the absence of curare. The command signal lasts ~3 ms and consists of a small negative wave followed by three larger biphasic waves (Fig. 3, A and B, EOCD + ES, bottom). The latencies of central EOCD responses were measured with respect to the negative peak of the first large biphasic wave in the command signal (time 0 or *t*<sub>c</sub>). In the absence of curare, the EOD occurs 4.5 ms after *t*<sub>c</sub>. Intracellular recordings were made with sharp microelectrodes filled with 2% biocytin in 2 M potassium methyl sulfate (140–250 MΩ). Biocytin was injected into recorded cells by passing depolarizing intracellular current pulses at 1 Hz with a duty cycle of 50% and amplitudes of 1–1.2 nA for 5–12 min.

Electroosensory responses were evoked either by local stimulation of restricted areas of the skin or by global stimulation of the entire fish. Local stimuli were delivered by means of a bipolar stimulating electrode consisting of two small Ag-AgCl balls 6 mm apart. The electrode was held with the axis of the dipole perpendicular to the skin. Individual electroreceptors can be easily distinguished on the skin surface with an operating microscope, and the stimulating electrode could be placed close to individual receptors. Brief pulses of current (100 µs, 5–50 µA) were delivered through the electrode to activate electroreceptors. Global stimuli were delivered by passing current between a small chlorided silver ball inserted through the mouth into the stomach of the fish and a second electrode placed in the water near the tail of the fish. This stimulus geometry activated all submerged electroreceptors and resembled the current flow during the actual EOD. All cells were tested with sensory stimuli at the EOD delay of 4.5 ms. Effects of the sensory stimulus alone were examined by delivering stimuli either independently of the motor command or at long delays of 60–100 ms.

Plasticity was induced by delivering an electroosensory stimulus at the EOD delay for 2–3 min. Both global and local electroosensory stimuli were used for pairing experiments. The fish’s spontaneous EOCD rate (2–5 Hz in our curarized preparation) determine the number and temporal pattern of EOCD + stimulus pairings. Although we have observed substantial effects of EOCD rate on ELL neurons in experiments in which electromotor behavior is varied across its entire natural range (1–60 Hz) (Sawtell, unpublished observations), the small variations in EOCD rate characteristic of our curarized preparation have little effect on EOCD responses or on plasticity of these responses.

Response latency in mormyromast electroreceptor primary afferents decreases by 9–11 ms as intensity is increased from threshold intensity to the intensity that gives a maximum response (Bell 1990a; Szabo and Fessard 1974). The latencies reported in this study for the electroosensory responses of different cell types are minimal latencies, obtained by increasing stimulus intensity until no further reduction in latency was observed. We used minimal latencies because such measurements are relatively insensitive to small cell-to-cell variations in stimulus intensity due to the positioning of dipole stimuli within the receptive field and allow for comparisons of timing relationships across different cell types and different structures.

Data were recorded on tape and analyzed off-line with a Cambridge Electronic Design interface and with the same company’s software. Statistical data are presented as means ± SE. Statistical comparisons were made using the *t*-test.

Histology

After the experiment, fish were anesthetized in concentrated MS: 222 (1:10,000) and perfused through the heart with teleost Ringer solution, followed by a fixative, consisting of 2% paraformaldehyde and 2% glutaraldehyde in 0.1 M phosphate buffer. The brains were postfixed overnight and cryoprotected with 20% sucrose. Cryostat sections (50 µm) were reacted with avidin-biotin complex and diaminobenzidine to reveal the biocytin. Sections were mounted on slides and counterstained with Richardson’s stain. Reconstructions of cells were made with a camera lucida attachment to the microscope.

Results

The central electroosensory pathways of mormyrid fish, including the major efferent connections of ELL and the recurrent feedback loops that return electroosensory information from
higher centers are illustrated in Fig. 1A. Efferent fibers from ELL travel by way of the lateral lemniscus and send collaterals to PE before terminating in the torus. These fibers are glutamatergic and their effects on PE and toral cells are presumed to be excitatory. Input to PE from higher centers, most notably the torus and valvula cerebelli, is extensive, suggesting rich interactions between ascending and descending components of the electro sensory system in PE (Fig. 1B). Three feedback pathways to ELL originate from separate cell populations in PE (Bell et al. 1981). 1) A large, reciprocal and topographically precise feedback pathway from PE enters ELL dorsally, descending to the deep molecular layer to make type 1 excitatory contacts with interneurons and efferent cells on the proximal portion of their apical dendrites (Meek et al. 1999). Tracer studies indicate that these direct projecting PE neurons provide feedback to the same local region of ELL from which they receive input (Bell et al. 1981). 2) A second class of PE cells project indirectly to ELL via the eminentia granularis posterior (EGp), a large granule cell mass that covers most of ELL. Axons of EGp granule cells form the parallel fibers of the molecular layer of ELL, terminating on the spiny apical dendrites of ELL interneurons and efferent cells. 3) Large GABAergic cells located medially and ventrally in the hilar region of PE give rise to a sparser direct projection to ELL (not shown in Fig. 1A). The direct projection to ELL from GABAergic hilar cells reaches ELL by traversing the medial octavolateral nucleus and enters the deep layers of ELL cortex from below, a path quite different from that taken by the other direct feedback projection from PE to ELL. A separate population of large, GABAergic hilar cells in PE project bilaterally to the torus.

The cell types and circuitry of ELL have been extensively characterized (Bell et al. 1997a; Meek et al. 1999; Mohr et al. 2003a). However, little is known about the nature of feedback signals returned to ELL from higher stages of the electro sensory system via PE and how ELL output is processed at higher stages of the electro sensory system. Intracellular labeling and axonal tracing allowed us to determine the projections of several types of recorded cells including: PE cells projecting directly to ELL, PE hilar cells projecting to the torus, PE interneurons, and cells of the torus projecting to PE (Fig. 2). We did not succeed in labeling PE cells projecting to EGp or PE hilar cells projecting to the deep layers of ELL, although it is likely that some of our recordings were made from cells of these types.

Direct feedback to ELL

Fifteen cells projecting directly to the deep molecular layer of ELL were identified morphologically after physiological recording. These cells have spiny dendrites and compact dendritic arbors (150–250 μM in maximal extent; Fig. 2A). Their axons exit the dorsocaudal pole of the nucleus without branching and enter the preeminential tract (PEET) ipsilaterally at the rostral margin of ELL. These cells are morphologically similar to those stained in PE after biocytin injections into the deep molecular layer of ELL (unpublished observations). The morphology of these cells is similar as well to the stellate cells that give rise to a direct feedback projection to ELL described in the preeminential nucleus of gymnotiform electric fish (Bratton and Bastian 1990; Sas and Maler 1983).

Although terminal arboris were not completely stained, direct projecting PE cells could be identified on the basis of fine axon branches descending into the molecular layer of ELL. Terminal branches were commonly observed both ipsilaterally and contralaterally in the medial (MZ) and dorsolateral (DLZ) zones of ELL (Fig. 1A). Some PE cells recorded and stained in this study projected to ELL both ipsilaterally and contralaterally and to both the MZ and the DLZ, in confirmation of previous tract-tracing experiments (Bell et al. 1981). No correlations were observed between dendritic morphology and axon projection patterns in this study. The MZ and DLZ receive afferent input from mormyromast electoreceptors involved in active electrolocation, whereas the ventrolateral zone (VLZ) receives input from ampullary receptors involved in passive electrolocation. As described in the following text, the physiology of the 14 cells projecting to the MZ and DLZ were consistent with input from mormyromast receptors. The physiology of the lone cell projecting to the VLZ was consistent with ampullary input and quite distinct from other recorded cells.
Like the efferent cells of ELL, PE neurons feeding back directly to ELL can be classified as I cells that are inhibited by electrosensory stimuli within their receptive field center or E cells that are excited by such stimuli. We did not observe any clear morphological differences between E and I cells (compare E and I cells in Fig. 2A).

**I cells projecting to the molecular layer of ELL**

Consistent with the results of a previous study using extracellular recordings (von der Emde and Bell 1996), many cells in PE exhibit a burst of spikes in response to the EOCD that is reduced or silenced by an electrosensory stimulus within the cell’s receptive field. Thirty-nine cells were classified as I cells based on their responses to the EOCD and electrosensory stimuli. The three cells of this type that were morphologically identified projected directly to the molecular layer of ELL (Fig. 2A). In most I cells, the EOCD evoked a stereotyped burst of 2–7 (4.3 ± 0.2; n = 35) spikes with an onset latency of 9–13 ms after \( t_0 \) (10.7 ± 0.2 ms; n = 35; Fig. 3, A and B). The timing of spikes evoked by the EOCD was extremely precise in these I cells with a jitter that was often <1 ms (rasters, Figs. 3, A and B, and Fig. 6, A and B). In other I cells, the EOCD evoked an excitatory postsynaptic potential (EPSP) (onset latency 9.8 ± 0.4 ms after \( t_0 \); n = 4) that occasionally gave rise to a single spike (Fig. 3C). The prominent EOCD responses characteristic of I cells recorded in PE are surprising in light of the comparatively weak and less precisely timed EOCD responses of ELL efferent cells (Bell et al. 1997a) (Fig. 13B). The EOCD evokes an IPSP in most E-type ELL efferent cells, known as large fusiform (LF) cells. In I-type efferent cells, known as large ganglion (LG) cells, EOCD responses are variable and often subthreshold (Bell et al. 1997a; Mohr et al. 2003a). Thus one major conclusion of this study is that EOCD responses of higher centers in the mormyrid electrosensory system are due to separate EOCD inputs rather than to the EOCD responses of ELL cells. The anatomical basis for these EOCD inputs is not known.

A local electrosensory stimulus delivered at the EOD delay strongly inhibited EOCD responses in I cells (Fig. 3, A–C, EOCD + ES). Receptive field dimensions were variable,
The absence of sensory-evoked IPSPs could be explained if the inhibitory interneuron that is itself gated by the EOCD accounts for the absence of sensory-evoked IPSPs at long delays. Another possibility is that inhibition in PE I cells is not due to direct inhibitory inputs but rather the removal of an excitatory input that is also driven by the EOCD (Fig. 3E, right). Toral I cells recorded in this study are excited by the EOCD and exhibit clear sensory-evoked IPSPs. At least some of these cells project to PE and could be responsible for EOCD responses in PE I cells. Excitatory effects of electrosensory stimuli delivered at long delays relative to the EOCD observed in some I cells suggest that these cells may also receive direct excitatory inputs that are not gated by the EOCD (not shown in Fig. 3E).

**E cells projecting to ELL**

Three main types of E cells could be distinguished on the basis of their responses to the EOCD and electrosensory stimuli, termed here E1, E2, and E3 cells (Fig. 4). Two E1 cells and nine E2 cells were morphologically identified and could be shown to project directly to the deep molecular layer of ELL. An additional 29 E1 cells and 15 E2 cells were recorded but not stained. The axon of one E3 cell could be traced in the preeminential electrosensory tract, but it could not be determined whether its axon terminated in the ELL molecular layer or in the overlying granule cell mass (EGp). Two additional E3 cells were recorded but not stained. One E cell that did not fit into any of our defined physiological classes (E1–E3) projected directly to the ventrolateral zone (VLZ) of ELL. The physiology of this cell resembled cells in the VLZ of ELL that receive input from ampullary receptors that are involved in passive electrolocation (Fig. 7F).

The EOCD evoked an EPSP in E1–E3 cells. Onset latencies following \( t_0 \) ranged from 7 to 13 ms and did not differ significantly across cell types (E1 cells 9.7 \( \pm \) 0.3 ms, \( n = 15 \); E2 cells 10.6 \( \pm \) 0.4, \( n = 9 \); E3 cells 11.2 \( \pm \) 0.5, \( n = 3 \)). The EOCD often evoked 1–2 spikes in E2 cells (Fig. 7, A and B). The EOCD response in E3 cells was a small EPSP that occasionally gave rise to a broad, all-or-none voltage response (Fig. 4C).

Electrosensory stimuli evoked excitatory responses from E cells within local receptive fields. The size of E cell receptive fields were similar to those described for I cells. The majority of E cells lacked obvious surround effects and responded vigorously to global as well as to local electrosensory stimuli. The interaction between EOCD and electrosensory responses was highly nonlinear with responses to the EOCD plus an

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**FIG. 3.** Electric organ corollary discharge (EOCD) and electrosensory responses in I cells projecting to ELL. A and B: typical I cells recorded in PE, the cell in A was stained and reconstructed (shown in Fig. 2). Top (EOCD): superimposed intracellular recordings of EOCD responses consisting of a brief burst of spikes. Rasters, above illustrate the precise timing of successive EOCD responses. Each line of the raster is triggered by the EOCD. Middle (EOCD + ES): an electrosensory stimulus (\( \bullet \)) at the EOCD delay inhibits spikes. Bottom: the command signal as recorded over the electric organ. The 1st negative wave of the command signals is defined as time 0 and is marked by open triangles in this and subsequent figures. Bottom traces (ES), a strong electrosensory stimulus delivered at a long delay following the EOCD has no effect. C: atypical I cell recorded and stained in PE. Top (EOCD): the EOCD evokes an excitatory postsynaptic potential (EPSP) and 0–1 spikes. Spikes are truncated. Middle (EOCD + ES): an electrosensory stimulus at the EOCD delay inhibits spikes and reduces the EOCD-evoked EPSP. Bottom (ES): averaged response to an electrosensory stimulus given at a long delay is a small EPSP. D: typical I cell in which an intracellular current pulse was delivered at a long delay relative to the EOCD. An electrosensory stimulus preceding the current pulse (\( \bullet \), bottom traces) increased the probability of a spike (9 of 12 pulses evoked a spike). E: 2 hypothetical circuits that could account for the EOCD and electrosensory responses of I cells.

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ranging from a few square millimeters to several centimeters, with the smallest fields being located on the chin appendage where the density of electroreceptors is highest. Large fields were often elongated in the rostro-caudal dimension. No excitatory responses were observed when the stimulus dipole was placed just outside the cluster of receptors where stimulation caused an inhibition. In addition, effects of a global electrosensory stimulus were similar to those of a local stimulus. Thus there was no evidence for an opponent, excitatory surround outside of the region of inhibition. This is in contrast to ELL efferent cells which exhibit prominent opponent receptive field surrounds (Bell et al. 1997a).

Surprisingly, most I cells were unaffected by a strong electrosensory stimulus delivered at a long delay (\( n = 11 \); Fig. 3, A and B, ES), and in four cells, an electrosensory stimulus delivered at a long delay evoked an EPSP rather than the expected inhibitory postsynaptic potential (IPSP; Fig. 3C, ES). The absence of sensory-evoked IPSPs could be explained if the cell resting potential was near the IPSP reversal potential and the inhibition evoked by a sensory stimulus was due to a change in membrane conductance. To address this possibility, we tested the effects of a strong electrosensory stimulus delivered at a long delay relative to the EOD motor command on spikes evoked by an intracellular current injection. In six of the seven cells tested, an electrosensory stimulus resulted in an increase, rather than the expected decrease, in the number of spikes evoked by a near-threshold current pulse (Fig. 3D).
electroreceptive stimulus being much larger than would be expected from a simple summation of the independent responses. Thus as in the efferent cells of ELL, the EOCD facilitates the excitatory effects of electroreceptive stimuli. The three types of E cells could be clearly distinguished by their responses to electroreceptive stimuli. Increases in stimulus intensity above threshold resulted in an increase in spike number (maximum: 8.3 ± 0.9 spikes per stimulus) and a decrease in first spike latency in E1 cells (Fig. 5A, left). In contrast, the main effect of increases in stimulus intensity on E2 cells was a decrease in first spike latency, and the response of these cells to a maximal intensity was a single spike time-locked to the stimulus (1.4 ± 0.2 spikes/stimulus, n = 9; Fig. 5A, right). Difference in intensity coding in these cells is likely due to inhibitory inputs to E2 cells which followed the initial excitatory responses. At more depolarized membrane potentials, we often observed such EPSP–IPSP sequences in response to an electroreceptive stimulus in E2 cells (Fig. 5B). In E3 cells, a gradual increase in stimulus intensity was accompanied by an abrupt transition from a small EPSP to a large, broad, all-or-none voltage response (Fig. 4C, EOCD + ES). The latency of the all-or-none response decreased further with increasing stimulus intensity.

By observing the latency of electroreceptive responses in identified PE neurons, we can estimate the timing of feedback modulation in ELL. Rapid feedback loops could modulate the responses of ELL cells during the same EOCD cycle while slower feedback loops could modulate responses during the subsequent cycle (Fig. 13). Minimal first spike latencies in response to a local electroreceptive stimulus were short in both E1 and E2 cells (E1: 8.9 ± 0.3 ms, n = 22; E2: 12.9 ± 1.0 ms, n = 6). Responses to global stimuli were even more rapid (E1: 7.6 ± 0.1 ms, n = 6; E2: 9.7 ± 0.2 ms, n = 7). Conduction times from PE to ELL can be estimated based on responses recorded in ELL evoked by electrical stimulation of PE. Stimulation of PE evokes spikes in PE axons recorded in ELL with delays as short as 0.7 ms and EPSPs recorded in ELL neurons that peak as early as 2 ms (Mohr et al. 2003). Thus we expect that direct PE feedback will modulate ELL efferent cell responses early during the same EOCD cycle. Although activity in E1 and I cells is almost entirely gated by the EOCD, many E2 cells showed sensory-evoked activity at longer delays relative to the EOCD (Fig. 5C). This second phase of the excitatory electroreceptive response was separated from the initial command-related burst by an apparent inhibition lasting 100–200 ms and often immediately preceded the response to the next EOCD (Fig. 5C, top). Effects of a prior sensory stimulus on the subsequent EOCD response could be observed directly when a late EPSP summated with the EOCD evoked EPSP, often evoking a spike (Fig. 5C, inset).

**Plasticity of corollary discharge responses**

The responses of ELL efferent cells to the EOCD are plastic in that pairing the EOD motor command for a few seconds to a few minutes with electroreceptive stimuli in the receptive field of the cell leads to a change in the EOCD response that opposes the effects of the sensory stimulus (Bell and Grant 1992; Bell et al. 1997a). This plasticity may act to minimize responses to predictable features of reafferent sensory input (Bell 2001).
Many ELL efferent cells also exhibit marked adaptation in response to an unchanging electrosensory stimulus due probably to a combination of plastic EOCD inputs and adaptation in primary electrosensory afferents (Bell, unpublished observations). Thus it was of interest to test the effects of pairing an electrosensory stimulus with the EOCD in PE neurons projecting to ELL.

We tested the effects of pairing the EOD motor command with electrosensory stimuli in 10 I cells. Nine of 10 cells exhibited adaptation during the stimulus period (i.e., a gradual decrease in the inhibitory effect of a sensory stimulus; Fig. 6, A and B). Adaptation could be a result of decreased inhibitory drive or a direct result of the opposing actions of plastic EOCD inputs. Evidence for EOCD plasticity was observed in some I cells immediately after turning the electrosensory stimulus off (Fig. 6A). Pairings with local electrosensory stimuli delivered to the cell’s receptive field and with global electrosensory stimuli yielded qualitatively similar results. In 6 of 10 cells tested, we observed a significant decrease in the latency of the first spike in the EOCD burst and an increase in the number of spikes in the EOCD burst after pairing ($P < 0.05$). This plasticity is consistent with the pattern of change observed in large ganglion cells of ELL in that the excitatory EOCD response was stronger after pairing with an inhibitory electrosensory stimulus (Bell et al. 1997a). We cannot yet determine whether plasticity observed in the EOCD responses of PE I cells is due to plasticity occurring in ELL efferent cells that project to PE or to plasticity occurring at the PE cell itself. The gradual decrease in the inhibitory effect of a sensory stimulus in I cells could be due either to the increased EOCD response in these cells or to decreased inhibitory input or to a combination of both.

The effects of pairing the EOD motor command with local electrosensory stimuli in E cells projecting to ELL were diverse. Electrosensory responses of E2 cells adapted during pairing as evidenced by an increase in response latency and a decrease in spike number (Fig. 7, A–C). Paradoxically, the EOCD response was consistently enhanced after pairing ($n = 8$; Fig. 7, A–C). Increased EOCD responses after pairing were not accompanied by a change in cell membrane potential. EOCD response enhancement was transient and returned to prepairing levels over the course of 30–60 s. A similar pattern of adaptation and EOCD enhancement has been observed in EOCD- and sensory-evoked field potentials recorded in PE (von der Emde and Bell 1996). The increased EOCD response observed in E2 cells would be expected to oppose adaptation in these cells. Consistent with this possibility, in some E2 cells an initial period of adaptation was followed by a gradual recovery of the sensory response (Fig. 7C). Effects of pairing on EOCD responses in E2 cells are opposite to those occurring in ELL large fusiform cells, the E cells that are efferent from ELL. This provides further evidence that EOCD responses in PE are shaped by influences other than ELL.

In contrast, the responses of E1 cells to an unchanging electrosensory stimulus were remarkably constant ($n = 6$; Fig. 7, D and E). E1 cells showed little or no change in EOCD responses after pairing (data not shown). The lack of adaptation in E1 cells is again surprising in light of the adaptation observed in ELL efferent cells. A nonadapting efferent pathway projecting to PE has been described in gymnotid fish (Bastian et al. 2004) although no evidence for such a pathway has yet been discovered in mormyrid fish.

Although uncommon, the type of adaptation observed in ELL, that is, adaptation of the sensory response accompanied by a decrease in the EOCD response after pairing, was observed in several E cells. The effects of pairing in an E cell projecting to the VLZ of ELL is shown in Fig. 7F. The loss of a prominent EOCD response and a high level of spontaneous activity are consistent with this neuron receiving input from the ampullary zone of ELL. After pairing, the EOCD response resembles a negative image of the effect of the sensory stimulus on the cell. This pattern of response is similar to that observed in the ampullary cells of ELL (Bell 1981).

**Hilar cells**

Two general regions of PE may be recognized histologically: 1) a hilar region at the rostral, medial, and ventral corner of the nucleus where the collaterals of lemniscal fibers enter PE and 2) a surrounding cortical region in the dorsal, lateral, and caudal parts of the nucleus. Efferent cells in the cortical region give rise to the direct and indirect feedback pathways to ELL as described in the preceding text. Large GABAergic cells in the hilar region are retrogradely labeled after tracer injections both in ELL and the torus (Bell et al. 1981). The cells projecting to the torus and those projecting to ELL appear to be distinct populations (unpublished observations). Eight hilar
cells were identified morphologically after physiological recording. These cells had large cell bodies (15–20 μM) and several thick smooth dendrites that extended several hundred microns, often stretching along the medial border between PE and the lateral lemniscus (Fig. 2). The axons of seven of these cells exited PE medially and ventrally, passed beneath the lateral lemniscus and coursed rostrally, before branching at the anterior border of PE. Two axon branches, one ipsilateral and one contralateral, continued rostrally, ending in the torus. Hilar cells could be divided into two physiological types on the basis of their EOCD and electrosensory responses. Cells of both types were labeled, but it was not clear from our stained material whether these physiological differences correlated with any morphological differences between the cell types. Consistent with the large negative field potentials recorded in this region of PE (von der Emde and Bell 1996), type 1 hilar cells responded to the EOCD with a large (15.9 ± 2.3 mV, n = 7), steeply rising EPSP, which often gave rise to a burst of spikes (Fig. 8, A and B). The latency of the EOCD response in these cells was 9.5 ± 0.3 ms after t₀. The EOCD response of type 2 hilar cells was a similarly timed (9.8 ± 0.8 ms, n = 4), but much smaller EPSP (5.0 ± 1.0 mV, n = 4) that seldom evoked spikes (Fig. 8C, top).

Both type 1 and type 2 hilar cells were excited by electrosensory stimuli. As in ELL efferent cells, the effects of an electrosensory were facilitated by the EOCD. Type 1 hilar cells responded maximally to electrosensory stimuli delivered within a local receptive field on the skin. No response was observed when the stimulus electrode was placed just outside the cluster of receptors where stimulation caused an excitation. Maximal responses to a global stimulus were similar to those for a local stimulus (Fig. 8B). In contrast, type 2 hilar cells were weakly excited by local electrosensory stimuli positioned over well separated regions of the body surface (Fig. 8C, right). Global electrosensory stimuli evoked a much larger excitation than any single local stimulus (Fig. 8C, left), suggesting that these cells integrate sensory inputs over a wide region of the body surface. Pairing the EOCD with an electrosensory stimulus in hilar cells resulted in modest adaptation accompanied by little or no change in the EOCD response after pairing (data not shown; n = 6).

Although none of our stained hilar cells projected to ELL, several considerations suggest that the physiological properties of hiliar cells projecting to ELL may be similar to those projecting to the torus. Hilar cells labeled after tracer injections in ELL do not appear to be morphologically distinct from hilar cells projecting to the torus (unpublished observations). In addition to the 4 type 1 hilar cells that were morphologically identified, 20 cells were recorded in the hilar region with similar physiological characteristics. Finally, no other cell types were encountered in the hilar region with the exception of the lemniscal fibers described in the following text.

Interneurons

In addition to the large GABAergic cells in the hilar region of PE, smaller GABAergic cells, presumably interneurons, are also present throughout PE (unpublished observations). Several observations, including IPSPs observed in E2 cells and nonlinear summation of EOCD and electrosensory inputs in I cells, suggest that interneurons may be important in shaping the responses of PE efferent cells. We commonly encountered cells in PE that spiked independently of the EOCD. Effects of
ell and the local ES). The effect of a global stimulus is similar to that of a strong local stimulus (B, EOCD + global ES). C: type 2 hilar cells respond to the EOCD with a smaller EPSP without spikes (left, EOCD). Local electrosensory stimuli moderately enhance the EOCD response at a number of regions on the skin surface (right). Positions of local stimuli are indicated in the schematic above. A global electrosensory stimulus (left, EOCD + global ES) evokes a much larger response than a local stimulus. Spikes are truncated in C.

Electroosensory stimuli were often difficult to detect in these cells. One such cell was morphologically identified as an interneuron. Both the morphology and physiology of this cell were notably distinct from efferent cell types recorded in PE. The cell had a fusiform cell body located far rostrally in PE (Fig. 2, bottom). Smooth dendrites extended to the borders of the nucleus and were far more extensive than those of efferent cells. No cells of this type were stained in PE after extracellular tracer injections either in ELL, EGp, or the torus (unpublished observations). The EOCD evoked a burst-pause pattern in this cell (Fig. 9, A and B). When a global electroosensory stimulus was delivered at the EOD delay, the cell continued to fire spontaneously, but its activity was no longer modulated by the EOCD. The EOCD response of this cell was plastic in that the EOCD-evoked burst-pause pattern was much more pronounced when the electroosensory stimulus was turned off after several minutes of pairing (Fig. 9, EOCD after). Interneurons may play important roles in shaping PE output both in mormyrid and gymnotiform fish. A number of interneuron types have been identified in Golgi preparations of PE in gymnotiforms (Sas and Maler 1983).

**Lemniscal fibers**

ELL efferent axons project in the lateral lemniscus to the torus. Collaterals of these fibers terminate in PE. Three lemniscal fibers were stained after physiological recording; two were located in the hilar region of PE near the border with the lateral lemniscus and one in the torus. The trajectory of these fibers was similar to the efferent cells of ELL. Two of the fibers stained in PE could be traced to the deep layers of the contralateral ELL; however, our staining was incomplete and revealed neither a cell body nor terminal branches. One fiber recorded in PE could be traced in the lemniscus both caudally in the direction of ELL and rostrally in the direction of the torus. ELL efferent cells exhibit a similar projection pattern, sending collaterals to PE on their way to the torus. The physiological characteristics of these elements were distinct from those of other cell types recorded in PE and from those of known ELL efferent cells. Additional elements with similar physiological properties were recorded juxtacellularly both in PE (n = 15) and the torus (n = 8).

The EOCD evoked one to three spikes, the first of which was often quite time-locked (10.2 ± 0.3 ms, n = 15; Fig. 10A, raster). Electroosensory receptive fields were large and often elongated (Fig. 10B, top) and were characterized by an inhibitory center and flanking excitatory regions. A local electroosensory stimulus in the inhibitory region of the receptive field abolished spikes evoked by the EOCD. In intracellular recordings, an inhibitory stimulus revealed a small underlying EPSP with an onset of 8–13 ms after the EOCD (n = 3). Subtraction of the corollary discharge field potentials recorded immediately outside of the cell confirmed the presence of this EPSP. A local electroosensory stimulus in adjacent excitatory regions increased spike number without a change in latency of the first EOCD-evoked spike. Effects of a global electroosensory stimulus were always inhibitory. Likewise, the net effect of stimulating inhibitory and excitatory regions of the receptive field simultaneously with two local dipole stimuli was generally inhibitory.

**FIG. 8.** EOCD and electroosensory responses of hilar cells projecting to the lateral toral nucleus. A and B: type 1 hilar cells. Cell in B was stained and reconstructed (shown in Fig. 2). Type 1 hilar cells exhibit a prominent EPSP often accompanied by a burst of spikes after the EOCD (EOCD), which is enhanced by an electroosensory stimulus within a local receptive field (EOCD + local ES). The effect of a global stimulus is similar to that of a strong local stimulus (B, EOCD + global ES). C: type 2 hilar cells respond to the EOCD with a smaller EPSP without spikes (left, EOCD). Local electroosensory stimuli moderately enhance the EOCD response at a number of regions on the skin surface (right). Positions of local stimuli are indicated in the schematic above. A global electroosensory stimulus (left, EOCD + global ES) evokes a much larger response than a local stimulus. Spikes are truncated in C.

**FIG. 9.** Physiology and plasticity of a PE interneuron. A: representative intracellular traces taken before (EOCD before), during (EOCD + ES), and after (EOCD after) pairing with a global electroosensory stimulus at the EOD delay. B: raster is triggered on the EOCD. Burst-pause pattern evoked by the EOCD is inhibited by the electroosensory stimulus (●) and is markedly enhanced after turning the stimulus off.
The physiology of neurons in the mormyrid torus has not yet been described. We therefore obtained intracellular recordings from neurons in the torus and characterized their responses to the EOCD and electrosensory stimuli.

Many toral cells had response properties nearly identical to those of cells in PE. Sixteen cells resembling E1 cells were recorded in the torus (Fig. 11A). The EOCD evoked a small EPSP in these cells 8–13 ms after \( t_0 \) (10.1 ± 0.5 ms, \( n = 9 \)). These latencies are not significantly different from those of EOCD EPSPs recorded in E1 cells of PE. Local electrosensory stimuli delivered at the EOD delay evoked a burst of 4–14 spikes. As in PE, the effects of the sensory stimuli were enhanced when given at the EOD delay. Both the onset and amplitude of the EOCD-evoked response and the latency and number of spikes evoked by a maximal electrosensory stimulus were very similar to those of E1 cells recorded in PE.

Other cells recorded in the torus closely resembled the E2 cells encountered in PE. Seven cells of this type were recorded, three of which were identified morphologically. The axon of one of these cells was well stained and projected caudally in the toro-preeminential tract (the toral feedback projection to PE). The EOCD evoked an EPSP or an EPSP–IPSP sequence in these cells. The EPSP often gave rise to a single spike (Fig. 11B, top). As in PE E2 cells, a local electrosensory stimulus evoked an EPSP–IPSP sequence (Fig. 11B, inset), which gave rise to a single short-latency spike. Some toral cells resembled E2 cells in that electrosensory stimuli evoked an EPSP–IPSP sequence (Fig. 11C, ES); however, the dominant effect of a sensory stimulus paired with the EOCD in these cells was to inhibit spiking (Fig. 11C, EOCD + ES).

Frequent juxtacellular and occasional intracellular recordings were made from I cells in the torus. One morphologically identified I cell projected in the toro-preeminential tract to PE. As in PE, I cells exhibited a stereotyped burst of spikes after the EOCD (Fig. 11D, EOCD). The onset of EOCD-evoked bursts in toral I cells (10.4 ± 0.1 ms, \( n = 16 \)) was not significantly different from the onset of similar responses in PE. Electrosensory stimuli evoked IPSPs in two intracellularly recorded I cells (Fig. 11D, ES). Thus unlike I cell recorded in PE, at least a subset of I cells in the torus receive direct inhibitory input. Inhibitory inputs to toral I cells are likely mediated by a local interneuron as ELL efferent input to the torus is glutamatergic.

Additional toral elements were recorded but not stained. Physiological characteristics of some of these were quite different from cells in PE. The EOCD evoked pure IPSPs in some toral cells. Other toral cells spiked independently of the EOCD and responded to electrosensory stimulation with a late burst of spikes beginning ~25 ms after the command.

Plasticity of EOCD responses was tested in a small number of toral cells by pairing a local electrosensory stimulus with the EOCD for several minutes. As in PE, the effects of pairing were diverse. A gradual weakening of the inhibitory sensory response followed by a slight increase in the EOCD-evoked burst was observed in toral I cells (\( n = 4 \); Fig. 12A). Electrosensory responses of toral cells resembling the E1 cells of PE exhibited little or no adaptation during pairing (\( n = 4 \); Fig. 12B). Toral cells resembling E2 cells of PE exhibited a gradual weakening of the excitatory sensory response accompanied by an enhanced EOCD response after pairing (\( n = 3 \); data not shown). Effects of pairing were tested in the toral cell shown in

**Cells of the lateral toral nucleus**

Neurons in PE integrate ascending electrosensory information from ELL with descending inputs from higher stages of the electrosensory system. The responses of PE cells recorded in this study differ in important respects from those of previously characterized efferent cells of ELL, suggesting that these descending inputs may play significant roles in shaping PE output. The most prominent descending input to PE is from the torus. The torus itself combines ascending input from ELL with feedback from the highest stages of electrosensory processing in the valvula cerebelli and telencephalon (Fig. 1A).
Timing and functions of electrosensory feedback

Understanding the dynamics of recurrent loops is a necessary step in generating hypotheses about the roles of feedback in sensory processing. This task is made easier in the mormyrid electrosensory system by the pulsatile nature of the EOD and the fact that the timing of all events in the electrosensory system may be examined with respect to this single moment in time. Responses to the EOCD and electrosensory stimuli consist of brief bursts of activity, the timing of which can be compared across multiple stages of processing.

DISCUSSION

This study characterizes electrosensory feedback to the mormyrid ELL and provides a description of electrosensory processing in PE and the torus—the major targets of ELL efferent projections. The EOCD and electrosensory responses of PE and toral cells are compared with the properties of ELL efferent neurons, and results are discussed in light of possible functions of electrosensory feedback to ELL.

Figure 13 summarizes information gathered in this study concerning the timing of direct feedback to ELL conveyed by I, E1, and E2 cells. Hilar cells morphologically identified in this study project to the torus, although morphologically, and perhaps physiologically, similar cells are the source of an inhibitory feedback projection to ELL. Aligned traces illustrate typical responses of efferent neurons of PE (Fig. 13A) and ELL (Fig. 13B) to the EOCD and electrosensory stimuli delivered at the EOD delay. The mean and range of minimal latencies for EPSP onsets and first spike onsets after $t_0$ are indicated by rectangles beneath the traces. Previous studies did not consistently obtain minimal latencies for ELL efferent cell responses to stimuli delivered at the EOD delay. Traces for large fusiform (LF) and large ganglion (LG) cells in Fig. 13B are minimal latency responses typical of a small number of such cells recorded in the present study.

Our results suggest two timecourses of feedback to ELL. Rapid responses in PE will affect the early components of EOCD and electrosensory responses in ELL during the same EOD cycle. Delayed electrosensory activity in E2 cells may affect responses of ELL cells to the next EOD (Fig. 5C). The effects of delayed feedback on activity in ELL will depend crucially on the EOD interval, which is under voluntary control and ranges from 10 ms to hundreds of milliseconds depending on behavioral context (Toerring and Moller 1984; von der Emde 1992). Rapid and delayed feedback may have distinct functional importance for electrosensory processing.

The direct feedback projection to the ELL deep molecular layer is believed to be excitatory based on the types of vesicles found in labeled PE terminals (Meek et al. 1999) and on the EPSPs in ELL evoked by electrical stimulation of PE (Mohr et
after pairing. Spikes are truncated. during pairing (EOCD C. Fig. 11 excitatory and inhibitory inputs, similar to the E2 cells of PE. Same cell as in illustrating adaptation and EOCD plasticity in a cell receiving a mix of in response to a constant electrosensory stimulus.

A

B

C

D

Fig. 12. Effects of pairing the EOCD with an electrosensory stimulus in toral cells. A: adaptation and EOCD plasticity in a toral I cell. Note the weakening of the inhibitory effects of a sensory stimulus during pairing (■) and the slight leftward shift in the latency of the EOCD burst immediately after pairing. B: like E1 cells in PE, some toral E cells showed very little adaptation in response to a constant electrosensory stimulus. C: intracellular traces illustrating adaptation and EOCD plasticity in a cell receiving a mix of excitatory and inhibitory inputs, similar to the E2 cells of PE. Same cell as in Fig. 11C. Note marked adaptation in the initially inhibitory sensory response during pairing (EOCD + ES), and the stronger EOCD response immediately after pairing. Spikes are truncated. D: raster for the pairing illustrated in C.

al. 2003b). Most E and I cells in PE give a brief burst of spikes in response to a combination of EOCD and electrosensory input with the number and timing of the spikes dependent on the stimulus. The responses of ELL cells to PE stimulation are markedly facilitated by brief bursts of closely spaced stimuli in both gymnotids and mormyrids (Bastian 1998; Mohr et al. 2003b). As a consequence of this facilitation, ELL cells may be sensitive to small changes in the timing and duration of PE bursts caused by small changes in electrosensory input.

Effects of E and I cells of PE on ELL output will depend on the details of connectivity in ELL. Direct excitatory feedback to the mormotid ELL may serve to enhance responses to salient stimuli (Berman and Maler 1999; Bratton and Bastian 1990). A similar “searchlight” function was first proposed for mammalian corticothalamic connections (Crick 1984). Positive feedback of this type would require a cell-type specific termination pattern such that E-type efferent cells of ELL that are excited by electrosensory stimuli project to and receive feedback from E cells of PE. The same would have to be true for I cells in the two structures. At present, there is no direct evidence for such specificity. Electrical stimulation of PE also suggests that the direct feedback pathway exerts both monosynaptic excitatory and disynaptic inhibitory effects on neurons in ELL (Mohr et al. 2003b). Thus the precise spatial and temporal patterns of feedback effects in ELL are difficult to predict based on PE responses alone. Future experiments in which direct excitatory feedback to ELL is inactivated will be important for determining the functions of this recurrent pathway for electrosensory processing in ELL.

Diffuse inhibitory feedback to the mormotid ELL has been shown to mediate a switch between oscillatory and nonoscillatory response modes involved in electrical communication versus prey detection (Doiron et al. 2003). Such a function is unlikely in mormyrids where central processing of electrosensation occurs in separate nuclei. Disynaptic inhibition from PE to the deep molecular layer of ELL or direct inhibitory feedback from PE hilar cells could be responsible for an EOCD-evoked inhibition of dendritic broad spikes that is observed in Purkinje-like medium ganglion cells of ELL (Mohr et al. 2003a). This inhibition appears to regulate spike-timing-dependent synaptic plasticity that depends on the occurrence of the dendritic spikes (Roberts and Bell 2002). The timing and response pattern of type 1 hilar cells recorded in this study are consistent with such a function.

Comparison of ELL efferent output with PE and toral cells

Although the cells, circuitry, and synaptic plasticity within the mormyrid ELL are well characterized, little is known about how ELL output is transformed at later stages of processing (Russell and Bell 1978; von der Emde and Bell 1996). Two efferent cell types with opposite response patterns have been identified in ELL. LF cells are excited by an electrosensory stimulus in the center of their receptive field, whereas LG cells are inhibited by such a stimulus. Similarly, two cell classes, termed here E and I cells, were found in PE and the torus. In addition, the present study has revealed a number of differences between the responses of ELL efferent neurons and responses in PE and the torus.

Stereotyped EOCD responses were observed in PE and toral cells. These responses included precisely timed bursts of spikes in I cells, EPSPs or EPSP–IPSP sequences in E cells, and large steeply rising EPSPs in hilar cells. Importantly, these EOCD responses cannot be accounted for by the EOCD responses of LF and LG cells, which are typically synaptic responses that do not evoke spikes (Bell et al. 1997a). Lemniscal fibers recorded in this study may represent an additional input from ELL. However, the latency of EOCD responses in these fibers and the way in which these responses are affected by electrosensory stimuli suggest that these fibers are not the major source
of EOCD input to PE and the torus. A major conclusion of this study is that higher centers in the electrosensory system receive independent EOCD inputs (Fig. 1B).

The mormyrid ELL is unique among vertebrate sensory structures for the prominence and accessibility of motor corollary discharge effects. Several functions have been described for EOCD inputs to ELL including: decoding primary afferent latency (Bell 1990a; Szabo and Hagiwara 1967) and plastic cancellation of expected reafference (Bell 2001). The combination of precisely timed EOCD inputs with electrosensory input is clearly important for shaping responses in PE and the torus as well. Plasticity of EOCD inputs in ELL opposes the effects of a sensory stimulus (Bell et al. 1997a). Changes in EOCD responses after pairing in PE were more diverse and likely serve different functions than those hypothesized for EOCD plasticity in ELL.

Effects of electrosensory stimuli on PE cells also differed in several respects from those observed in ELL efferent cells. Many PE cells lacked the opponent receptive field surrounds characteristic of ELL efferent cells. Spatial integration was striking in type 2 hilar cells, which summed sensory effects over a wide region of the skin. E1 cells of PE and the torus encode electrosensory stimulus intensity in a manner similar to ELL large fusiform cells. In these cells, both response latency and spike number are modulated by changes in stimulus intensity. Intensity coding in E2 cells, on the other hand, is more similar to that observed in electoreceptor primary afferents than in ELL efferent cells. In E2 cells, increases in stimulus intensity are reflected primarily in decreases in stimulus intensity without an increase in spike number. Precisely timed inhibitory inputs to E2 cells appear to truncate the excitatory response such that these cells often fire only a single spike per EOCD cycle.

One explanation for the difference between responses of ELL efferents and PE cells is the influence of descending inputs to PE from the torus. Responses of many E cells of the torus were similar to E cells observed in PE. Toral I cells were also similar, although unlike I cells in PE, some toral I cells responded to electrosensory stimulation with an IPSP. At least a subset of toral E2 and I cells project to PE. The similarity of PE cells and some toral cells could be due to toral cells driving PE cells or to both regions receiving the same EOCD and electrosensory inputs. The timing of EOCD and electrosensory responses responses in PE and the torus was very similar. The observed timing relationships are consistent with both regions receiving the same EOCD and electrosensory inputs but do not exclude the possibility that toral cells play an important role in shaping PE responses. Excitation by toral I cells could explain the EOCD responses of PE I cells and the lack of electrosensory-evoked IPSPs in these cells. Responses of E cells in PE to electrosensory stimuli could be explained, at least in part, by input from toral E cells, but the EOCD-evoked EPSPs in these cells cannot be explained by the subthreshold EOCD responses in E1 and E2 cells recorded in the torus. Inactivation of toral feedback to PE will provide further insight into the functional relationships between PE and the torus.

Although interactions between ascending and descending electrosensory and corollary discharge information described in this study are complex, the temporally discrete nature of the input signals and the relatively simple, precisely timed responses of the mormyrid electrosensory system may facilitate an understanding of the roles of descending inputs in sensory processing.

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FIG. 13. Relative timing of EOCD and electrosensory responses in PE and ELL efferent cell types. A: representative traces for short-latency responses of various cell types in PE (I, E1, E2, hilar) to the EOCD, (left) and electrosensory stimulus at the EOD delay (right). The bar beneath each trace indicates the range of EPSP (Δ) or spike (●) onset times for cell of that type. The line within each bar shows the mean onset time. B: representative traces for short-latency responses of ELL efferent cells.

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GRANTS

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