The Journal of Experimental Biology 202, 1365–1375 (1999)
Printed in Great Britain © The Company of Biologists Limited 1999
JEB2078

NEURAL CIRCUITRY FOR COMMUNICATION AND JAMMING AVOIDANCE IN GYMNOTIFORM ELECTRIC FISH

WALTER METZNER*

Department of Biology, University of California at Riverside, Riverside, CA 92521-0427, USA

*e-mail: walter.metzner@ucr.edu

Accepted 25 January; published on WWW 21 April 1999

Summary

Over the past decade, research on the neural basis of communication and jamming avoidance in gymnotiform electric fish has concentrated on comparative studies of the premotor control of these behaviors, on the sensory processing of communication signals and on their control through the endocrine system, and tackled the question of the degree to which these behaviors share neural elements in the sensory–motor command chain by which they are controlled. From this wealth of investigations, we learned, first, how several segregated premotor pathways controlling a single central pattern generator, the medullary pacemaker nucleus, can provide a large repertoire of behaviorally relevant motor patterns. The results suggest that even small evolutionary modifications in the premotor circuitry can yield extensive changes in the behavioral output. Second, we have gained some insight into the concerted action of the brainstem, the diencephalon and the long-neglected forebrain in sensory processing and premotor control of communication behavior. Finally, these studies shed some light on the behavioral significance of multiple sensory brain maps in the electrosensory lateral line lobe that long have been a mystery. From these latter findings, it is tempting to interpret the information processing in the electrosensory system as a first step in the evolution towards the 'distributed hierarchical' organization commonly realized in sensory systems of higher vertebrates.

Key words: *Eigenmannia*, *Apteronotus*, premotor control, neuronal oscillator, multiple sensory maps.

Introduction

Although weakly electric fish are potentially able to use visual, tactile and acoustic information for orientation or prey capture, they nonetheless rely mainly on their active orientation system by emitting electric organ discharges (EODs) and monitoring the feedback from these discharges. In wave-species, on which this paper will focus, EODs follow a quasi-sinusoidal time course at a normally extremely constant rate (Bullock, 1970; Moortgat et al., 1998). During social encounters, and sometimes spontaneously, most species can modulate the frequency of their EODs, which gives them the acoustic quality of chirps. This behavior is, therefore, also called 'chirping' (Larimer and McDonald, 1968; Bullock, 1969; Hopkins, 1974; Hagedorn and Heiligenberg, 1985). In addition, most gymnotiforms adjust their EOD frequency to avoid jamming by signals that originate from neighboring conspecifics (Watanabe and Takeda, 1963; Bullock et al., 1972; Heiligenberg, 1973, 1977). *Eigenmannia*, for instance, lowers its own EOD frequency in response to neighboring signals of slightly higher frequency and raises its EOD frequency when the neighbor's frequency is slightly lower. This 'jamming avoidance response' (JAR; Bullock et al., 1972) can improve the electrolocation performance (Heiligenberg, 1973, 1991) and possibly also aid in social identification in the context of communication (Kramer, 1987, 1990, 1999).

Over the past almost three decades, many of the computational rules that explain sensory processing and sensorimotor integration during the JAR have been elucidated (for a review, see Heiligenberg, 1991). More recent work has focused on the premotor control of the JAR and communication behavior (Kawasaki et al., 1988; Dye et al., 1989; Keller et al., 1990; Zupanc and Heiligenberg, 1989, 1992; Metzner, 1993; Heiligenberg et al., 1996; Wong, 1997a,b; Zupanc and Maler, 1997; Juranek and Metzner, 1997, 1998), on sensory processing of communication signals (for reviews, see Metzner and Viete, 1996a,b; see also Wong, 1997a,b) and on the segregation of these behaviors on the sensory side (Shumway, 1989a,b; Metzner and Heiligenberg, 1991; Turner et al., 1994, 1996; Metzner and Juranek, 1997a).

In the present paper, we will attempt to summarize these recent findings and integrate them into a more general scheme that might allow us to make assumptions about the possible evolution of the neural substrates underlying these behaviors in gymnotiform electric fish. Because of space constraints, we will concentrate on two of the best-studied and closely related

(Alves-Gomes et al., 1995) gymnotiform 'wave species', *Eigenmannia* sp. and *Apteronotus* sp.

Premotor control

The pacemaker nucleus of gymnotiform fish represents a neuronal network in which separate modulatory inputs generate distinct behavioral motor patterns by altering the discharge rate of its neuronal components (Fig. 1; for recent reviews, see Heiligenberg, 1991; Metzner and Viete, 1996a,b). The pacemaker nucleus is composed of two types of neuron, pacemaker and relay cells. Both fire in a one-to-one manner with the electrocytes of the electric organ. Relay cells project to spinal motoneurons that either, as in *Eigenmannia*, innervate the electrocytes of the electric organ, or, as in *Apteronotus*, comprise the electric organ itself (for a review, see Bass, 1986). Pacemaker cells are connected with each other and with relay cells *via* mixed chemical and electrical synapses (Szabo and Enger, 1964; Bennett, 1971; Tokunaga et al., 1980; Ellis and Szabo, 1980; Elekes and Szabo, 1982, 1985; for a review, see Dye and Meyer, 1986). A series of pharmacological and anatomical studies has revealed, so far, three afferent synaptic inputs to the pacemaker nucleus: two from a diencephalic prepacemaker nucleus (the CP/PPn complex; Heiligenberg et al., 1981; Kawasaki et al., 1988; Zupanc and Heiligenberg, 1992; Wong, 1997a) and one from a mesencephalic, sublemniscal prepacemaker nucleus (SPPn; Keller et al., 1991; Metzner, 1993; Heiligenberg et al., 1996). In *Eigenmannia* and *Apteronotus*, all inputs to the pacemaker nucleus and its intrinsic connections are excitatory (Kennedy and Heiligenberg, 1994) and presumably use glutamate as their natural transmitter. One input generates chirp-like communicatory signals and arises from the ventrolateral portion of the diencephalic prepacemaker nucleus (PPnC; Heiligenberg et al., 1981; Rose et al., 1988; Kawasaki et al., 1988; Dye et al., 1989; Metzner, 1993; Juranek and Metzner, 1997, 1998). Its role in the control of communication behavior is discussed below in more detail. The functional significance of the two remaining inputs differs between *Eigenmannia* and *Apteronotus*, and this difference is probably linked to differences in their JAR: while *Eigenmannia* both raises and lowers its EOD frequency in response to jamming signals that are lower and higher in frequency, respectively, *Apteronotus* can only actively increase its EOD frequency (Bullock et al., 1972). Nevertheless, these two genera discriminate the sign of the frequency difference between their own EOD and that of a jamming neighbor using the same sensory computational rules (Heiligenberg, 1986, 1991). Hence, we assumed that differences in their JAR behavior are mainly due to differences in the premotor circuitry (Metzner, 1993; Heiligenberg et al., 1996).

Jamming avoidance response (JAR)

In *Eigenmannia*, EOD accelerations and decelerations during the JAR appear to be controlled by distinctly separate pathways (Metzner, 1993). As illustrated in Fig. 1A, the pathway that controls EOD accelerations in response to jamming signals with slightly lower frequencies involves projections from the torus semicircularis dorsalis (TSd) in the midbrain to the dorsal portion of the nucleus electrosensorius complex (nE↑) in the diencephalon (Keller, 1988; Keller and Heiligenberg, 1989; Keller et al., 1990; Metzner, 1993; for reviews, see Heiligenberg, 1991; Metzner and Viete, 1996b). The nE↑, in turn, innervates the dorsomedial portion of the

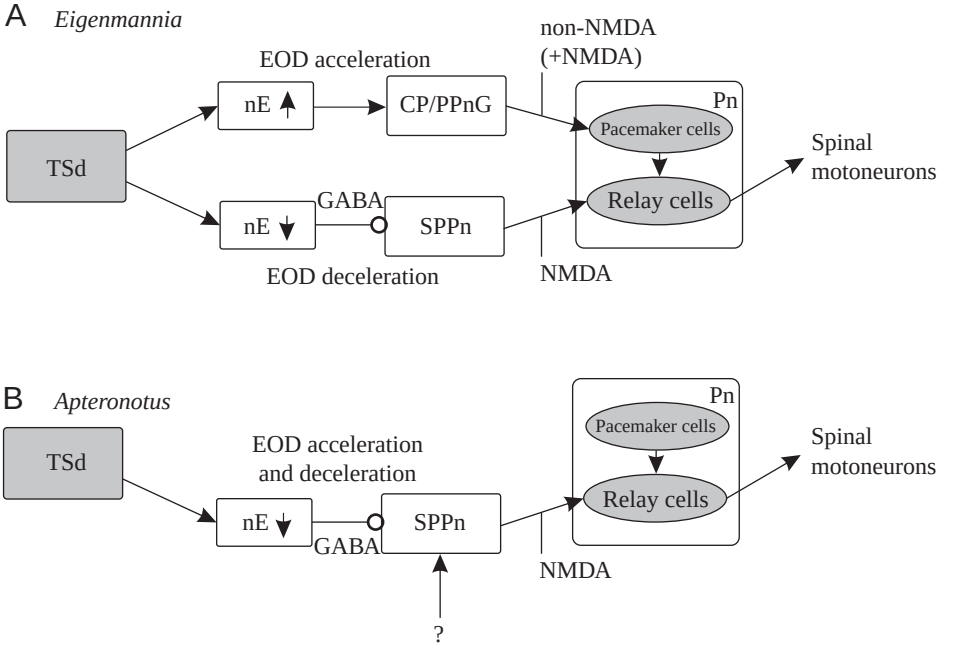

Fig. 1. Flow diagrams of premotor control of the jamming avoidance response (JAR) in *Eigenmannia* (A) and *Apteronotus* (B). Arrowheads indicate excitatory and open circles inhibitory connections. For details, see text. (After Kawasaki et al., 1988; Dye et al., 1989; Keller and Heiligenberg, 1989; Metzner, 1993; Heiligenberg et al., 1996; Juranek and Metzner, 1997, 1998.) CP/PPnG, dorsomedial portion of the diencephalic prepacemaker nucleus complex; nE↑, dorsal portion of the diencephalic nucleus electrosensorius complex whose stimulation causes gradual rises in electric organ discharge (EOD) frequency; nE↓, ventral portion of the diencephalic nucleus electrosensorius complex whose stimulation causes gradual reductions in EOD frequency; Pn, medullary pacemaker nucleus; SPPn, sublemniscal prepacemaker nucleus; TSd, mesencephalic torus semicircularis dorsalis; ?, postulated but unidentified excitatory input; GABA, γ-aminobutyric acid; NMDA, *N*-methyl-D-aspartate.

diencephalic prepacemaker nucleus (parvocelluar division of the CP/PPn), where, much as in the nE↑, electrophoretic applications of L-glutamate elicit gradual EOD accelerations identical to those occurring during the rising phase of the JAR (Kawasaki et al., 1988; Keller and Heiligenberg, 1989). This subnucleus is therefore also called PPnG. Conversely, lesions of either the nE↑ or PPnG selectively abolish this phase of the JAR (Keller and Heiligenberg, 1989; Metzner, 1993). The PPnG finally projects to the medullary pacemaker nucleus, where it terminates on pacemaker cells (Kawasaki et al., 1988; Dye et al., 1989; Metzner, 1993; Spiro et al., 1994; Juranek and Metzner, 1997, 1998).

EOD decelerations in *Eigenmannia*, in contrast, are controlled *via* a pathway running from the TSd through the ventral portion of the nucleus electrosensorius complex (nE↓) and the mesencephalic SPPn primarily to relay cells in the pacemaker nucleus (Fig. 1A). Neurons in the nE↓, when stimulated with L-glutamate, cause gradual decreases in EOD frequency similar to those occurring during the falling phase of the JAR (Keller, 1988; Keller and Heiligenberg, 1989; Keller et al., 1990). Conversely, the falling phase of the JAR is eliminated after lesions of the nE↓ (Keller and Heiligenberg, 1989). However, lesions of the SPPn, the target of the nE↓, both reduce the 'resting frequency' (i.e. the EOD rate produced when no jamming signal is present) and obliterate EOD decelerations during the JAR. Hence, we concluded that neurons in the SPPn appear to be tonically active and inhibited by input from the nE↓. Indeed, γ-aminobutyric acid (GABA) injections into the SPPn mimic nE↓ stimulations, while injections of the $GABA_A$ antagonist bicuculline block any effects of glutamate stimulation of the nE↓. The reduced level of SPPn activity normally caused by activation of the nE↓, in turn, results in a lower level of excitatory drive to relay cells and hence EOD decelerations (Metzner, 1993; Juranek and Metzner, 1997, 1998).

Although, to our present knowledge (Heiligenberg et al., 1996), the same diencephalic and mesencephalic inputs to the pacemaker nucleus are also present in *Apteronotus*, there are some striking differences in their functional significance between the two genera. Most noticeably, in *Eigenmannia*, the premotor control of the rising and falling phases of the JAR is shared equally between the diencephalic (PPnG) and mesencephalic prepacemaker nucleus (SPPn) (Fig. 1A). In contrast, it is solely the mesencephalic SPPn that controls this behavior in *Apteronotus*, resulting in its asymmetrical JAR (Fig. 1B): lesions of the PPnG leave the JAR unaffected, whereas lesioning the SPPn eliminates the entire JAR. Similarly, lesions of the nE↓ completely abolish the JAR, whereas inactivations of the nE↑ have no effect on the JAR (unlike in *Eigenmannia*, however, these lesions fail to affect the fish's resting frequency). These lesioning results are particularly surprising since, much as in *Eigenmannia*, stimulation of the nE↑ causes rises and nE↓ stimulation causes reductions in EOD frequency. In contrast to *Eigenmannia*, however, these frequency rises are often brisker and sometimes superimposed with chirp-like transients, and the frequency reductions are smaller. Moreover, in both genera, nE↑ projects to the PPnG (although in *Apteronotus*, it also innervates the PPnC; see below and Fig. 2) and the nE↓ projects to the SPPn. Therefore, extrapolating from what we have learned in its sister species *Eigenmannia*, the nE↑/PPnG pathway in *Apteronotus* would be expected to play the primary or even sole role in mediating its asymmetrical JAR. Instead, the experimental results leave us with a puzzling question: how can the nE↓/SPPn pathway alone control the JAR of *Apteronotus*? How is this pathway, which in *Eigenmannia* controls reductions in EOD rate during JAR, in *Apteronotus* able to raise the EOD rate in response to jamming signals with lower frequency? Even more surprisingly, SPPn input from the nE↓ appears to be mediated by GABA in both genera: GABA application to the SPPn lowers the EOD rate, much like the corresponding sensory input does. In addition, application of the $GABA_A$ antagonist bicuculline blocks nE↓ input, much as in *Eigenmannia*, but also enhances the EOD frequency rises occurring in response to jamming signals with lower frequency, signals that in *Eigenmannia* are processed by the nE↑/PPnG pathway. Since we know from the aforementioned lesioning experiments that, in *Apteronotus*, this pathway is not involved in any premotor control of the JAR, the most plausible explanation is that EOD frequency increases during the JAR are produced by a release of inhibition originating from the nE↓ onto the SPPn: when no jamming signals are present, the SPPn is under tonic inhibition from the nE↓ and is, therefore, quiescent. This idea is consistent with all observations except that lesions of the nE↓ fail to result in rises in EOD frequency. Hence, we postulated an additional, excitatory and so far unidentified, input to the SPPn (which cannot be ruled out by any of the results achieved so far; Heiligenberg et al., 1996).

Whereas the SPPn in *Eigenmannia* appears to be involved only in the JAR, it plays an additional role in *Apteronotus*: excitation of the SPPn in *Apteronotus* results in a swift rise of the EOD rate and in some cases even yields interruptions to the EOD that occur naturally during courtship (Heiligenberg et al., 1996). Similar effects of SPPn stimulation have been observed in *Hypopomus* (Kawasaki and Heiligenberg, 1989, 1990) and *Sternopygus* (Keller et al., 1991), but never in *Eigenmannia*. Moreover, histological analysis of the SPPn in *Apteronotus* revealed two cell types, whereas in *Eigenmannia* there is only one (Heiligenberg et al., 1996). It is therefore tempting to ascribe the two cell types in the *Apteronotus* SPPn to its two different functions, the JAR and communication behavior.

Communication behavior

Various types of communication signal are produced during courtship and aggressive encounters by both *Eigenmannia* and *Apteronotus*. These signals mostly consist of sudden and strong rises in EOD frequency, often followed by interruptions to the EOD cycle (Larimer and McDonald, 1968; Bullock, 1969; Hopkins, 1974; Hagedorn and Heiligenberg, 1985). Since these interruptions have the acoustic quality of chirps, this behavior

is also called 'chirping'. Chirps are produced in response to the EOD signal of another conspecific; they can probably be triggered by input from other sensory modalities, such as olfactory or visual cues, and/or they can also occur 'spontaneously', i.e. without an obvious external releasing stimulus, primarily under endocrine control (Hopkins, 1974; Hagedorn and Heiligenberg, 1985; Dye, 1987; Maler and Ellis, 1987; Heiligenberg et al., 1991; Zupanc and Maler, 1993; Dulka and Maler, 1994).

Reflecting the behavioral complexity of chirping, the underlying neural circuitry is also quite intricate. In gymnotiform fish, the ventrolateral, magnocellular subdivision of the diencephalic prepacemaker nucleus (CP/PPn) appears to play a central role in the control of chirping. Since stimulation of this portion results in chirps that resemble those produced under natural conditions, it has been termed the PPnC (Kawasaki and Heiligenberg, 1989; Kawasaki et al., 1988). Increased activity in cells of the PPnC rapidly depolarizes relay cells in the pacemaker nucleus *via* non-*N*-methyl-D-aspartate (NMDA)-type receptors (Dye, 1988; Dye et al., 1989; Kawasaki and Heiligenberg, 1990; Metzner, 1993; Spiro et al., 1994; Juranek and Metzner, 1997, 1998).

Numerous connectional (Heiligenberg et al., 1981; Kawasaki et al., 1988; Keller et al., 1990; Heiligenberg et al., 1991; Johnston et al., 1990; Stroh and Zupanc, 1995; Wong, 1997a,b) and immunohistochemical (Sas and Maler, 1991; Zupanc et al., 1991; Johnston and Maler, 1992; Weld and Maler, 1992; Yamamato et al., 1992; Dulka et al., 1995; Richards and Maler, 1996; Wong, 1997b; for a review, see Zupanc and Maler, 1997) investigations have established a wealth of connections linking the PPnC to electrosensory, mechanosensory and possibly olfactory structures and/or reproductive centers (Fig. 2). However, since the PPnC represents an 'open' nucleus, i.e. the dendrites of its neurons extend far beyond the boundaries of the PPnC proper, as defined in Nissl-stained material (Kawasaki et al., 1988), and also since intrinsic connections from the PPnG to the PPnC appear to exist (Wong, 1997a), the projection patterns of the CP/PPn are difficult to ascribe to only one or other of its subdivisions. The following, therefore, summarizes primarily the connectivity of the entire CP/PPn complex but will, wherever possible, delineate the exact relationship to certain of its subdivisions.

Electrosensory and mechanosensory information reaches the CP/PPn *via* five different pathways from the various subdivisions of the diencephalic nucleus electrosensorius (nE) (for reviews, see Wong, 1997a; Zupanc and Maler, 1997). The most prominent input arises from the nE$\uparrow$ which, in *Eigenmannia*, projects mainly to the dorsomedial CP/PPn, i.e. the CP and PPnG, whereas in *Apteronotus*, it is connected mainly with the PPnC (Bastian and Yuthas, 1984; Keller, 1988; Keller and Heiligenberg, 1989; Keller et al., 1990; Heiligenberg et al., 1996). The nE$\uparrow$ controls the rising phase of the JAR in *Eigenmannia* (Metzner, 1993) but is not necessary for the JAR in *Apteronotus*, in which its stimulation instead leads to brisk rises in EOD frequency on which chirping is often superimposed (Heiligenberg et al., 1996). Hence, the nE$\uparrow$/PPn pathway in *Apteronotus* appears to be involved in chirping behavior rather than in the JAR. [It also seems to control another electrosensory behavior, the 'non-selective response' (Dye, 1987), which consists of a smooth rise in EOD rate when jamming signals are presented, irrespective of whether the jamming signals are lower or higher in frequency (Heiligenberg et al., 1996).] A second possible, although minor, pathway from the nE to the CP/PPn originates from its acousticolateralis region (nEAR), which processes mechanosensory and low-frequency ampullary electrosensory information (Keller et al., 1990; Heiligenberg et al., 1991; for a review, see Metzner and Viete, 1996a). A third pathway involves projections from a subdivision that contains neurons responding to beat modulations in the electrosensory feedback (nEb), such as those produced when two EOD signals interfere (Keller, 1988; Heiligenberg et al., 1991). Although not necessary for the JAR, this pathway might aid in the recognition of conspecific EODs (Heiligenberg et al., 1991). It projects, however, mainly to the PPnG. There are two indirect pathways from the nE to the CP/PPn. One of them passes from the nEb through the anterior tuberal nucleus of the hypothalamus (TAd) and terminates in the PPnC (Keller et al., 1990; Heiligenberg et al., 1991; Wong, 1997a). Its functional significance is unclear. The second indirect route involves projections from chirp-sensitive neurons in the nEAR to the inferior lobe (CE), which in turn projects to the PPnG (Keller et al., 1990; Heiligenberg et al., 1991; Wong, 1997a).

Developmental and immunohistochemical studies have revealed that many connections of the CP/PPn are not rigid but, instead, can be modulated depending on the animal's reproductive state. For instance, the proximal dendrites of the large cells in the PPnC increase in length by up to 30 % during sexual maturation and invade the area dorsomedial of the PPnC proper ('dorsomedial territory'). This dendritic growth could then respond to new synaptic connections between efferent fibers from the nE (mostly from the nEb), encoding the detection of chirps, and PPnC dendrites (Zupanc and Heiligenberg, 1989; Heiligenberg et al., 1991; Zupanc, 1991). In addition, neuromodulatory substances, such as serotonin (Johnston et al., 1990), catecholamines (Sas et al., 1990) and various neuropeptides (Sas and Maler, 1991; Weld and Maler, 1992; Richards and Maler, 1996; for a review, see Zupanc and Maler, 1997), might be involved in mediating synaptic transmission in the PPnC. The level of some of these substances appears to depend on the reproductive state and sex of the animal. Injections of serotonin, for instance, greatly reduce spontaneous and evoked chirping behavior in *Apteronotus* (Maler and Ellis, 1987). The innervation pattern of substance-P-immunoreactive fibers of the CP/PPn differs between sexes (Weld and Maler, 1992) and also appears to be under androgen control (Dulka et al., 1995).

Additional complications of the circuitry controlling communication behavior originate from numerous connections with brain centers involved in the control of reproductive behavior and those processing other than electrosensory

modalities, such as mechanosensory or olfactory stimuli (Fig. 2). Various partly reciprocal connections exist with such structures in the basal and dorsal forebrain, preoptic area and hypothalamus (Keller et al., 1990; Heiligenberg et al., 1991; Zupanc, 1996; Wong, 1997a,b; Zupanc and Horschke, 1997a–c). Many of these regions, most notably the basal forebrain (ventral subdivision of the telencephalon, Vv), also have connections with the olfactory bulb and/or project to the pituitary (Johnston and Maler, 1992; Sas et al., 1993; Wong, 1997b). The ventral forebrain, for instance, would be a prime candidate for integrating olfactory and ascending thalamic electrosensory information (Wong, 1997b).

Sensory processing

Our current knowledge of the computational rules and neuronal substrates for the sensory processing of interference signals yielding a JAR have been described in detail by Heiligenberg (1991; but see also Kramer, 1999; Takizawa et al., 1999). More recent overviews of various aspects of the electrosensory processing of communication signals have been given by Kramer (1990), Moller (1995) and Metzner and Viete (1996a,b). We will focus, therefore, only on a few topics not covered in those reviews.

Who is chirping?

One such aspect is the problem that a fish faces when it produces chirps in response to detecting another conspecific's chirps. How can the fish distinguish between its own chirps and those produced by other fish?

Several possible solutions have been suggested. Chirps, at least in *Eigenmannia*, contain both a high-frequency component consisting of the fast transients at the beginning and end of an EOD interruption and a low-frequency (quasi-direct-current) component consisting of a baseline-shift during the interruption (Metzner and Heiligenberg, 1991). The base level maintained during the interruption, however, depends on the geometry of the electric field of a chirping fish. The sender's head voltage, for instance, remains negative during an interruption on either side of its body. For a neighboring fish, which is exposed to these chirps, the base level is negative for the side of the body wall opposite the chirping fish, whereas it is positive for the body side facing the chirping fish (Heiligenberg et al., 1991; Metzner and Heiligenberg, 1993). This difference is encoded in the low-frequency ampullary system and results in a different temporal pattern of activity in neurons in the dorsal torus and nE that respond selectively to chirps by combining ampullary and tuberous information (Metzner and Heiligenberg, 1991; Heiligenberg et al., 1991). Moreover, in virtually all these combination-sensitive neurons, which represent at least 25 % of all neurons in the dorsal torus (J. Juranek and W. Metzner, unpublished observation), only input originating from the same receptive field type of lower-order ampullary and tuberous neurons converges, i.e. from ampullary and tuberous on-center cells ('E-type') or ampullary and tuberous off-center cells ('I-type') (Metzner and Heiligenberg, 1991; Heiligenberg et al., 1991; Rose and Call, 1992). Since these E–E- and I–I-type combination-sensitive neurons should be much less excited by the fish's own chirps than by the chirps of a neighbor, they could also enable the fish to perform this discrimination task (Metzner and Heiligenberg, 1993).

The use of an efference copy of its own chirp, which could, for instance, originate from the chirp-controlling portion of the CP/PPn, represents another possibility. In *Eigenmannia*, dendrites of chirp-sensitive neurons in a particular portion of the diencephalic nucleus electrosensorius complex (nEAR; Fig. 2) appear to extend into the neuropil that also receives projections from the PPnC (Heiligenberg et al., 1991; Wong, 1997a). Moreover, in the *Apteronotus* CP/PPn, some neurons controlling chirp production through their projection to the pacemaker nucleus could simultaneously relay this information *via* collaterals to the preglomerular nucleus (PG; see Fig. 2; Zupanc and Horschke, 1997a), which appears also to receive electrosensory input from the nE complex (Striedter, 1992; the connection between the nE and the PG was, however, not observed in *Eigenmannia* by Keller et al., 1990).

Finally, inhibitory commissural cells, so-called ovoid cells, in the first-order electrosensory nucleus, the electrosensory lateral line lobe (ELL), have been implicated in 'common-mode rejection' (Rose, 1989; Bastian et al., 1993; Maler and Mugnaini, 1994; Berman and Maler, 1998). This mechanism would reject bilaterally symmetrical stimuli, such as the sensory feedback caused by the fish's own chirps, but would facilitate the detection and identification of asymmetrical signals, such as a neighbor's chirps. None of these mechanisms, however, has so far been verified experimentally.

A sensory brain map for each behavior?

All electroreceptor afferents terminate in a somatotopic manner in the first-order nucleus of the electrosensory system of gymnotiform fish, the electrosensory lateral line lobe (ELL), where the rostral ELL represents the fish's head region. The ELL consists of four mediolaterally adjacent segments: the medial segment (MS) receives input from ampullary afferent axons, whereas tuberous primary afferents trifurcate and each collateral innervates the three remaining segments, the centromedial (CMS), centrolateral (CLS) and lateral (LS) segments (for reviews, see Carr and Maler, 1986; Heiligenberg, 1991). No intermap connections have been found (Shumway, 1989b). The existence of these multiple sensory maps with identical receptor inputs and mirror-image boundaries has always been a mystery. Despite the lack of qualitative differences between the three tuberous maps, previous investigations have yielded some quantitative differences in their physiological and immunohistochemical properties (Shumway, 1989a,b; Johnston et al., 1990; Metzner and Heiligenberg, 1991; Turner et al., 1996; Metzner et al., 1998). Their behavioral significance, however, remained unclear.

Results from recent lesion experiments in the electrosensory system of *Eigenmannia* and *Apteronotus* might shed some light on this problem (Metzner and Juranek, 1997a).

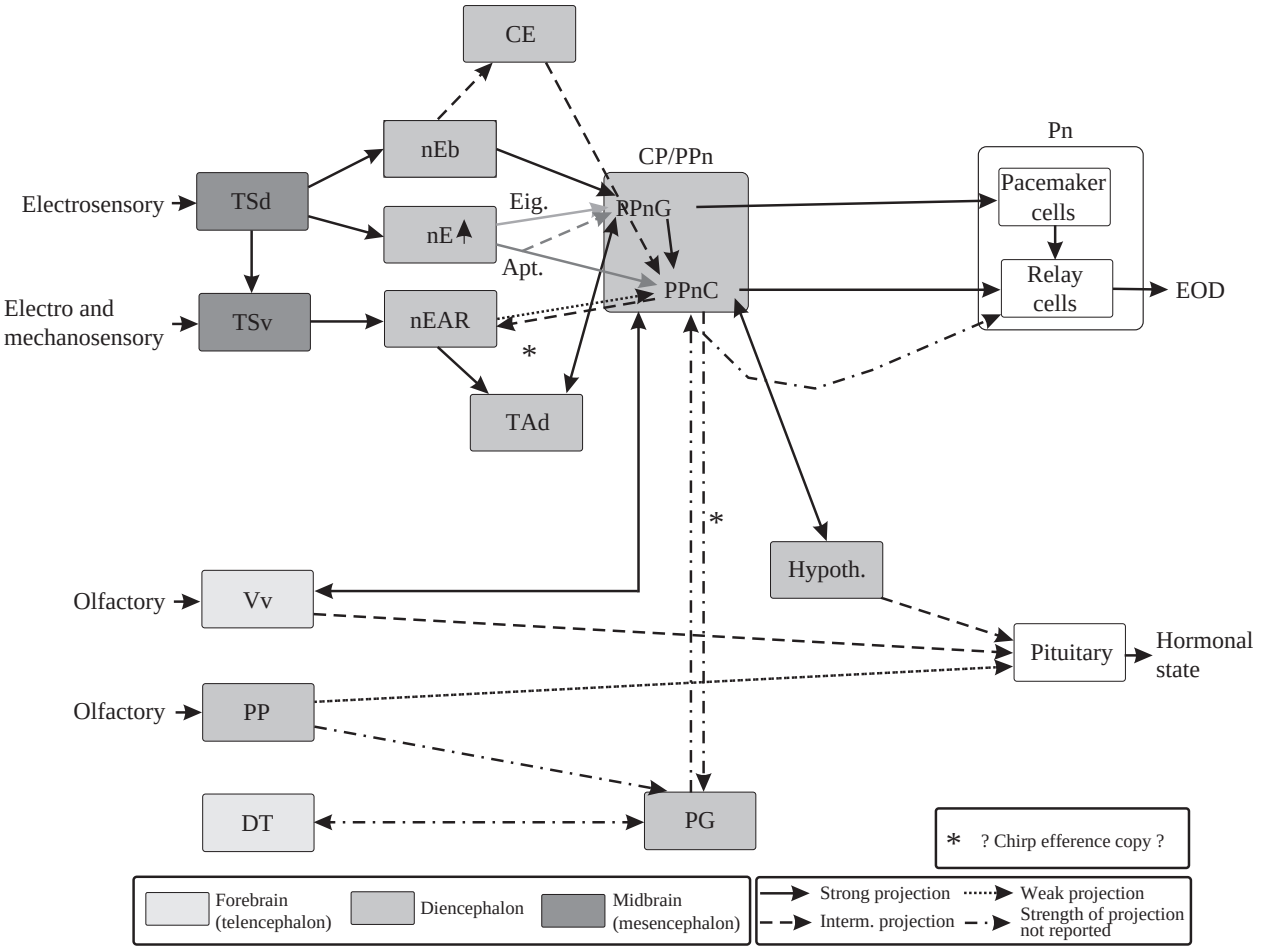

Fig. 2. The neural network potentially involved in sensory processing and premotor control of communicatory signals in *Eigenmannia* (*Eig*.) and *Apteronotus* (*Apt*.) is rather complicated and might also involve multimodal integration of electrosensory, mechanosensory and olfactory information. Moreover, some of these connections are also under endocrine control and change with the fish's reproductive status. The output of this network can affect the animal's electric organ discharge (EOD) as well as its hormonal state. For details, see text. (After Kawasaki et al., 1988; Dye et al., 1989; Keller et al. 1990; Keller and Heiligenberg, 1989; Zupanc and Heiligenberg, 1989; Heiligenberg et al., 1991, 1996; Zupanc, 1991b; Johnston and Maler, 1992; Sas et al., 1993; Metzner and Viete, 1996a; Wong, 1997a,b; Zupanc and Maler, 1997.) CE, central nucleus of the inferior lobe; CP/PPn, diencephalic prepacemaker nucleus complex; DT, dorsal telencephalon; Hypoth, hypothalamus; nEAR: nucleus electrosensorius, acusticolateralis region; nEb, nucleus electrosensorius, beat region; nE↑, dorsal portion of the diencephalic nucleus electrosensorius complex whose stimulation causes gradual rises in EOD frequency; PG, preglomerular nucleus; Pn, pacemaker nucleus; PP, periventricular preoptic nucleus; PPnG, dorsomedial portion of the diencephalic prepacemaker nucleus in which stimulation causes a gradual rise in EOD rate; PPnC, ventrolateral portion of the diencephalic prepacemaker nucleus in which stimulation causes chirp production; TAd, dorsal portion of the anterior tuberal nucleus; TSd, dorsal torus semicircularis; TSv, ventral torus semicircularis.; Vv, ventral nucleus of the ventral telencephalon; interm, intermediate strength.

Pharmacological inactivations of different ELL maps resulted in markedly different behavioral deficits (Fig. 3). The centromedial map is both necessary and sufficient for the JAR, whereas it does not affect the communicative response to external electric signals. Conversely, the lateral map does not affect the JAR but is necessary and sufficient to evoke communication behavior. The behavioral role of the CLS is still unclear. It does not seem to be involved, however, in the encoding of signals yielding a JAR or evoking chirping.

These findings are consistent with several earlier behavioral, physiological and histochemical studies. For instance, pyramidal cells in each map respond differently to the frequency of sinusoidal amplitude modulations: most pyramidal cells in the CMS respond best to sinusoidal amplitude modulations of 1–3 Hz, whereas those in the LS prefer rates above 8 Hz (Shumway, 1989a). In contrast, pyramidal cells in the CMS have a smaller receptive field size, thus showing higher spatial resolution but lower temporal resolution than those in the LS and *vice versa* (Shumway, 1989a). High spatial resolution is a prerequisite for the computational mechanisms controlling the JAR (Heiligenberg, 1991), and high temporal resolution is presumably required to encode the beat pattern that evokes chirps (Metzner and Heiligenberg, 1991). Indeed, pyramidal cells in the CMS extract features relevant for the JAR better than

those in the LS (Metzner et al., 1998), whereas pyramidal cells in the LS encode simulations of brief chirps better than those in the CMS or CLS (Metzner and Heiligenberg, 1991). Correspondingly, in behavioral experiments, the JAR is elicited most strongly by frequency differences of 1–6 Hz between the fish's EOD (or its mimic) and the external signal, whereas frequency differences between 8 and 16 Hz evoke chirp responses best (Bullock et al., 1972; Dye, 1987; Maler and Ellis, 1987; Shumway, 1989a; Heiligenberg, 1991; Zupanc and Maler, 1993; Heiligenberg et al., 1996). Finally, serotonin, which seems to represent a neuromodulatory agent for the processing of conspecific communication signals in gymnotiform fish (see above), is found at higher densities in the LS than in the CMS or CLS (Johnston et al., 1990).

While these data suggest that tuberous information is processed in strictly segregated pathways in the electrosensory system, there is also some evidence that a certain degree of 'crosstalk' occurs between the two electrosensory submodalities, i.e. tuberous and ampullary information processing.

Let us first consider chirping behavior. In earlier studies, chirp-selective neurons were found in higher-order brain centers (TSd and nEAR). When tested for their ampullary and tuberous responses, it turned out that both modalities had to be presented simultaneously to evoke a response. Either one of them alone was insufficient to cause them to spike (Metzner and Heiligenberg, 1991; Heiligenberg et al., 1991). Why would it make sense for a fish to not rely on only one of the two modalities to detect chirps?

Chirps contain not only stimulus parameters that affect the tuberous system but also low-frequency ampullary information

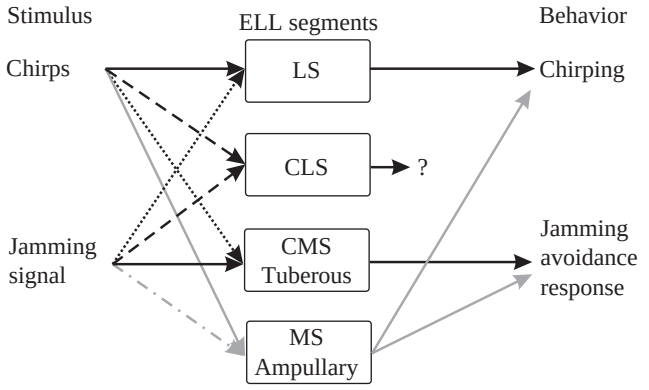

Fig. 3. Tuberous information yielding chirping and jamming avoidance response (JAR) behavior, respectively, appears to be processed by the various electrosensory lateral line lobe (ELL) segments in a strictly segregated manner (Metzner and Juranek, 1997a). The centromedial segment (CMS) is necessary and sufficient to process electrosensory signals resulting in a JAR, whereas the lateral segment (LS) is necessary and sufficient for chirping behavior. The role of the intermediate, centrolateral ELL segment (CLS) is still unclear (?). Ampullary information, however, also affects both chirping behavior and the JAR. This might reflect a first evolutionary step from a strictly segregated towards the 'distributed hierarchical' organization (van Essen and Gallant, 1994) seen in most other vertebrate sensory systems. MS, medial segment.

(see above). A high-frequency component before and after each chirp stimulates the tuberous system, and a low-frequency component, which is caused by the shift from the mean alternating current to the base level of the head–tail voltage, stimulates the ampullary system. If a fish used only its tuberous system to detect chirps, it would use a detection system that is very susceptible to high-frequency 'noise'. This noise originates from transient signals caused by lightning, which occurs abundantly in the tropics and travels over hundreds of kilometers (Hopkins, 1973). It turns out that every 2–3 s, on average, lightning causes pulses to occur with an electric field strength that is high enough to be detected by an electric fish (e.g. *Gymnarchus niloticus*). To avoid jamming by this high-frequency background noise, electric fish could use information provided by the ampullary system, which is tuned to the low-frequency component of chirps. The exclusive use of ampullary information alone, however, is insufficient, because the low-frequency component is slightly modulated by the movement of the fish relative to the low-frequency voltage source. In other words, any movement of either receiver or sender while an interruption occurs will alter the level of the voltage maintained during the chirp. This generates 'noise' in the ampullary system. Therefore, natural selection should favor the convergence of tuberous and ampullary information in order to increase the overall signal-to-noise ratio: ampullary information about chirps could secure the neuronal detection system against the background noise caused by lightning, and tuberous information could correct for fluctuations in the direct current level caused by the relative movement of the fish (Metzner and Heiligenberg, 1991; Heiligenberg et al., 1991).

Preliminary evidence indicates that ampullary information most surprisingly also affects the JAR (Fig. 4). Inactivations of the medial ELL map can in some cases dramatically reduce the JAR by more than half (Fig. 4A–C) or, in other cases, increase the JAR, almost doubling it (Fig. 4D,E). Whether the JAR is reduced or increased appears to be independent of the sex of the animal, but its social status seems to play a role. Fish that we considered dominant (e.g. they occupied the most attractive hiding places in the tank, the EOD frequency of the males was among the lowest and that of the females among the highest measured in the tank) usually showed an increased JAR; in subdominant fish, the JAR was reduced after lesions of the medial ELL segment. These are quite puzzling results since, according to our current understanding, tuberous information alone carries all the cues about the distortions in the electric field caused by jamming, i.e. beat patterns that strongly modulate the phase and amplitude of the electric image (Heiligenberg, 1991; see also Takizawa et al., 1999; Kramer, 1999). It is therefore a mystery why the ampullary system would be of any use for the JAR.

Discussion

Evolutionary considerations on segregated sensory processing and premotor control

What is the reason for the distinctly distributed organization of the tuberous electrosensory system in these fish?

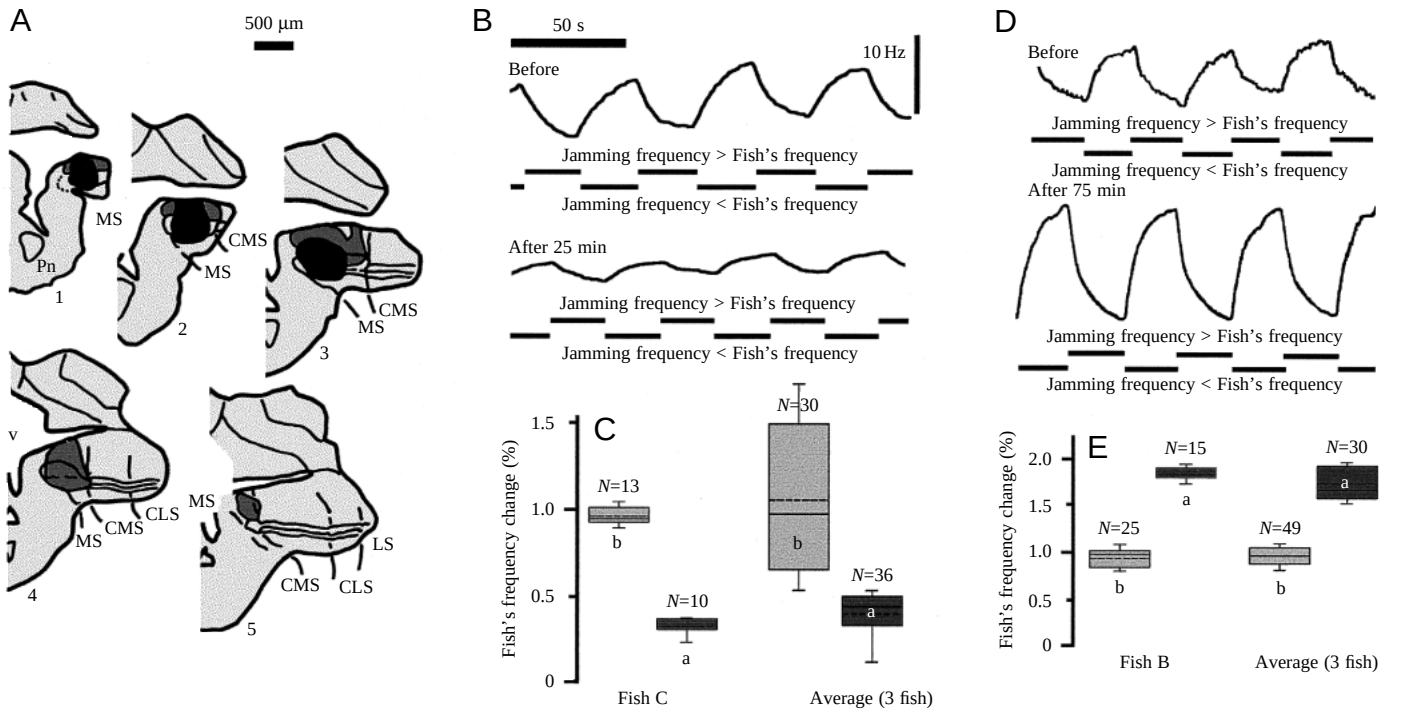

Fig. 4. Lesions in the medial segment of the first-order nucleus of the electrosensory pathway, the electrosensory lateral line lobe (ELL), unexpectedly also affect the jamming avoidance response (JAR). (A) Frontal sections (right half) through the ELL, arranged from caudal (1) to rostral (5). Black, center of injection; dark gray, periphery of injection (containing somata of pyramidal cells and dendrites reaching into the center of injection); small black circles, labeled somata of pyramidal cells (see Metzner and Juranek, 1997a,b, for details). Sections are approximately 200 µm apart. MS, CMS, CLS, LS, medial, centromedial, centrolateral and lateral segment; Cer, cerebellum; Pn, pacemaker nucleus; V, fourth ventricle. (B) In some fish, the JAR was reduced after lesions of the MS. The JAR before (upper trace) and after (lower trace) injection of biotinylated ibotenic acid as shown in A. Each trace represents frequency/time plots of the electric organ discharge (EOD). Rows of horizontal bars indicate when the jamming frequency was higher (upper bars) or lower (lower bars) than the frequency of the fish's EOD mimic. (C) The JAR decreases after lesions of the MS. Quantitative analysis of JAR before ('b', light gray) and after ('a', dark gray) the lesion. Boxes on the right indicate medians of EOD frequency changes before and after lesion for the case depicted in A and B and are average values for three fish. The lower and upper ends of the boxes define the twenty-fifth and seventy-fifth percentile, respectively, with a solid horizontal line at the median and a hatched line at the mean. Error bars indicate the tenth (lower) and ninetieth (upper) percentiles. *N*, number of JAR cycles analyzed. (D,E) In another fish, the JAR increases after MS lesion. The display follows the conventions used in B and C.

Electrosensory systems might share a common evolutionary lineage with the mechanosensory (lateral line) system. It has been suggested repeatedly that electrosensory brain structures could have evolved by duplication from mechanosensory areas (McCormick and Braford, 1988; New and Singh, 1994). Hence, it is tempting to speculate that duplication of existing brain maps could efficiently accommodate the increased information flow associated with a growth in the behavioral repertoire, as originally proposed for the mirror-image organization of sensory maps in the mammalian cortex (Allman and Kaas, 1971; Kaas, 1982). A further evolutionary increase in the complexity of sensory scenes and motor actions, such as in visually guided mammalian behavior, might eventually have required a greater flexibility in information processing. This might have yielded the shared use of circuit elements originally anchored in separate information streams by bridging between maps, which resulted in the present distributed hierarchical organization of most vertebrate sensory systems. It is conceivable that the relatively simple nature of electric signals controlling a limited behavioral repertoire in electric fish caused the tuberous electrosensory system to retain this 'primitive' character of a distinct modularity (Metzner and Juranek, 1997a).

On the premotor side, accurate electrolocation requires an extremely low jitter in EOD discharges (Bullock, 1972; Heiligenberg, 1991; Moortgat et al., 1998). This demand for highly synchronous and regular discharge of the pacemaker nucleus was probably the leading selective pressure yielding the strict functional separation of its cellular components into pacemaking units, i.e. pacemaker cells, and output units, i.e. relay cells (Bennett, 1971). This rigid design of the premotor command nucleus may, instead, have required its synaptic inputs to be modified in order to produce the different motor patterns underlying different behaviors. For instance, in the *Apteronotus* pacemaker nucleus, an excitatory, tonic input as in *Eigenmannia*, could introduce a high risk of jitter stemming from spontaneous fluctuations in this tonic drive (Heiligenberg et al., 1996). This would be particularly detrimental to the performance of this system since the especially high discharge rates in *Apteronotus* dictate an extremely high firing accuracy (Bullock, 1972; Moortgat et al., 1998).

Similar to such changes in tonic activity levels underlying the very different roles of the nE↓/SPPn pathway during the JAR in *Eigenmannia* and *Apteronotus*, other rather subtle changes, in activity levels, connectivity or receptor expression for instance, may provide additional possible substrates upon which Nature may act to form new behaviors (for a review, see Heiligenberg et al., 1996). Many *Apteronotus*, for example, produce chirps in response to jamming stimuli and do so preferentially for either higher or lower jamming frequencies (Dye, 1987; Zupanc and Maler, 1993). Such 'sign-selective' chirping appears to be mediated by the nE↑/PPnC/Pn pathway in *Apteronotus*. *Eigenmannia* only rarely chirps in response to jamming stimuli and L-glutamate stimulation of the nE↑ does not cause chirping, suggesting that the neural substrate is indeed present, but may normally be only very weakly expressed or may be actively suppressed. Likewise, an 'inverse sign-selectivity' to the nE↑/PPnG/Pn pathway based upon a non-selective response may reflect circuitry underlying behaviors that are only transiently expressed in developing *Eigenmannia* (Hagedorn et al., 1988; Viete and Heiligenberg, 1991). Some young fish at first show only a frequency rise when presented with jamming stimuli, and the JAR develops soon thereafter. A few fish transiently express an 'inverse' JAR, and in some the JAR and frequency rise combined appear remarkably similar in form to the JAR of *Apteronotus*.

There may be many more weak connections, too weak perhaps to reveal an obvious physiological significance. During further evolution, they could, however, strengthen and eventually become behaviorally significant. Conversely, such weak connections could also be remnants of connections that were stronger in the past and fulfilled a function now lost or taken over by other structures. In addition, variations in the relative expression or location of receptor types in the pacemaker nucleus over the course of evolution, for instance, could readily alter the temporal dynamics of specific pacemaker modulations (Heiligenberg et al., 1996; Juranek and Metzner, 1997, 1998). So far, these findings indicate that relatively small changes in the strength of connections between (sub)nuclei and in the anatomical, physiological and pharmacological properties of individual neurons can lead to an extensive diversification of behaviors even in closely related genera.

Portions of the work described in this paper were supported by grants from NIDCD, NSF and the Academic Senate of UCR to the author. Many thanks to S. Viete, J. Juranek, L. Maler and R. Turner for comments on earlier versions of the manuscript.

References

Allman, J. M. and Kaas, J. H. (1971). A representation of the visual field in the caudal third of the middle temporal gyrus of the owl monkey (*Aotus trivirgatus*). *Brain Res.* **31**, 84–105.

Alves-Gomes, J. A., Orti, G., Haygood, M., Heiligenberg, W. and Meyer, A. (1995). Phylogenetic analysis of the South American electric fishes (order Gymnotiformes) and the evolution of their electrogenic system: a synthesis based on morphology, electrophysiology and mitochondrial sequence data. *J. Mol. Biol. Evol.* **12**, 298–318.

Bass, A. H. (1986). Electric organs revisited. In *Electroreception* (ed. T. H. Bullock and W. Heiligenberg), pp. 13–70. New York: Wiley.

Bastian, J., Courtwright, J. and Cawford, J. (1993). Commissural neurons of the electrosensory lateral line lobe of *Apteronotus leptorhynchus*: morphological and physiological characteristics. *J. Comp. Physiol.* A **173**, 257–274.

Bastian, J. and Yuthas, J. (1984). The jamming avoidance response of *Eigenmannia*: properties of a diencephalic link between sensory processing and motor output. *J. Comp. Physiol.* A **154**, 895–908.

Bennett, M. V. L. (1971). Electric organs. In *Fish Physiology* (ed. W.S. Hoar and D.J. Randall), pp. 493–574. New York: Academic Press.

Berman, N. J. and Maler, L. (1998). Inhibition evoked from primary afferents in the electrosensory lateral line lobe of the weakly electric fish, *Apteronotus leptorhynchus. J. Neurophysiol*. (in press).

Bullock, T. H. (1969). Species differences in effect of electroreceptor input on electric organ pacemakers and other aspects of behavior in electric fish. *Brain Behav. Evol.* **2**, 85–118.

Bullock, T. H. (1970). Reliability of neurons. *J. Gen. Physiol.* **55**, 565–584.

Bullock, T. H., Hamstra, R. H. and Scheich, H. (1972). The jamming avoidance response of high frequency electric fish. I. General features. *J. Comp. Physiol.* **77**, 1–22.

Carr, C. E. and Maler, L. (1986). Electroreception in gymnotiform fish. In *Electroreception* (ed. T. H. Bullock and W. Heiligenberg), pp. 319–373. New York: Wiley.

Dulka, J. G. and Maler, L. (1994). Testosterone modulates female chirping behavior in the weakly electric fish, *Apteronotus leptorhynchus*. *J. Comp. Physiol.* A **174**, 331–343.

Dulka, J. G., Maler, L. and Ellis, W. (1995). Androgen-induced changes in electrocommunicatory behavior are correlated with changes in substance P-like immunoreactivity in the brain of the electric fish, *Apteronotus leptorhynchus*. *J. Neurosci.* **15**, 1879–1890.

Dye, J. (1987). Dynamics and stimulus-dependence of a vertebrate communicatory behavior: chirping in the weakly electric fish, *Apteronotus*. *J. Comp. Physiol.* A **161**, 175–185.

Dye, J. (1988). An *in vitro* physiological preparation of a vertebrate communicatory behavior: chirping in the weakly electric fish, *Apteronotus*. *J. Comp. Physiol.* A **163**, 445–458.

Dye, J., Heiligenberg, W., Keller, C. H. and Kawasaki, M. (1989). Different classes of glutamate receptors mediate distinct behaviors in a single brainstem nucleus. *Proc. Natl. Acad. Sci. USA* **86**, 8993–8997.

Dye, J. C. and Meyer, J. H. (1986). Central control of the electric organ discharge in weakly electric fish. In *Electroreception* (ed. T. H. Bullock and W. Heiligenberg), pp. 71–102. New York: Wiley.

Elekes, K. and Szabo, T. (1982). Synaptic organization in the pacemaker nucleus of a medium-frequency weakly electric fish, *Eigenmannia* sp. *Brain Res.* **237**, 267–281.

Elekes, K. and Szabo, T. (1985). Synaptology of the medullary command (pacemaker) nucleus of the weakly electric fish (*Apteronotus leptorhynchus*) with particular reference to comparative aspects. *Exp. Brain Res.* **60**, 509–520.

Ellis, D. B. and Szabo, T. (1980). Identification of different cell types in the command (pacemaker) nucleus of several gymnotiform species by retrograde transport of horseradish peroxidase. *Neurosci.* **5**, 1917–1929.

Hagedorn, M. and Heiligenberg, W. (1985). Court and spark:

signals in the courting and mating of gymnotid fish. *Anim. Behav.* **33**, 254–265.

Hagedorn, M., Heiligenberg, W. and Carr, C. E. (1988). The development of the jamming avoidance response in the weakly electric fish, *Eigenmannia*. *Brain Behav. Evol.* **31**, 161–169.

Heiligenberg, W. (1973). Electrolocation of objets in the electric fish, *Eigenmannia*. *J. Comp. Physiol.* **87**, 137–164.

Heiligenberg, W. (1977). *Principles of Electrolocation*. Berlin, Heidelberg, New York: Springer-Verlag.

Heiligenberg, W. (1986). Jamming avoidance responses, model systems for neuroethology. In *Electroreception* (ed. T. H. Bullock and W. Heiligenberg), pp. 613–649. New York: Wiley.

Heiligenberg, W. (1991). *Neural Nets in Electric Fish*. Cambridge: MIT Press.

Heiligenberg, W., Finger, T., Matsubara, J. and Carr, C. E. (1981). Input to the medullary pacemaker nucleus in the weakly electric fish, *Eigenmannia*. *Brain Res.* **211**, 418–423.

Heiligenberg, W., Metzner, W., Wong, C. J. H. and Keller, C. H. (1996). Motor control of the jamming avoidance response in *Apteronotus leptorhynchus*: evolutionary changes of a behavior and its neuronal substrates. *J. Comp. Physiol.* A **179**, 653–674.

Heiligenberg, W., Keller, C. H., Metzner, W. and Kawasaki, M. (1991). Structure and function of neurons in the complex of the nucleus electro-sensorins of the gymnotiform fish Eigenmannia. *J. Comp. Physiol.* A **169**, 151–164.

Hopkins, C. D. (1973). Lightning as a background noise for communication among electric fish. *Nature* **242**, 270–286.

Hopkins, C. D. (1974). Electric communication: functions in the social behavior of *Eigenmannia virescens*. *Behaviour* **50**, 270–305.

Johnston, S. A. and Maler, L. (1992). Organization of the hypophysiotrophic system in the electric fish, *Apteronotus leptorhynchus*. *J. Comp. Neurol.* **317**, 421–437.

Johnston, S. A., Maler, L. and Tinner, B. (1990). The distribution of serotonin in the brain of *Apteronotus leptorhynchus*: an immunohistochemical study. *J. Chem. Neuroanat.* **3**, 429–465.

Juranek, J. and Metzner, W. (1997). Cellular characterization of synaptic modulations of a neuronal oscillator in electric fish. *J. Comp. Physiol.* A **181**, 393–414.

Juranek, J. and Metzner, W. (1998). Segregation of behavior-specific synaptic inputs to a vertebrate neuronal oscillator. *J. Neurosci.* **18**, 9010–9019.

Kaas, J. H. (1982). The segregation of function in the nervous system: why do sensory systems have so many subdivisions? *Contrib. Sens. Physiol.* **7**, 201–240.

Kawasaki, M. and Heiligenberg, W. (1989). Distinct mechanisms of modulation in a neuronal oscillator generate different social signals in the electric fish, *Hypopomus*. *J. Comp. Physiol.* A **165**, 731–741.

Kawasaki, M. and Heiligenberg, W. (1990). Different classes of glutamate receptors and GABA mediate distinct modulations of a neuronal oscillator, the medullary pacemaker in gymnotiform electric fish. *J. Neurosci.* **10**, 3896–3904.

Kawasaki, M., Maler, L., Rose, G. J. and Heiligenberg, W. (1988). Anatomical and functional organization of the prepacemaker nucleus in gymnotiform electric fish: the accommodation of two behaviors in one nucleus. *J. Comp. Neurol.* **276**, 113–131.

Keller, C. H. (1988). Stimulus discrimination in the diencephalon of *Eigenmannia*: the emergence and sharpening of a sensory filter. *J. Comp. Physiol.* A **162**, 747–757.

Keller, C. H. and Heiligenberg, W. (1989). From distributed sensory processing to discrete motor representations in the diencephalon of the electric fish, *Eigenmannia*. *J. Comp. Physiol.* A **164**, 565–576.

Keller, C. H., Kawasaki, M. and Heiligenberg, W. (1991). The control of pacemaker modulations for social communication in the weakly electric fish, *Sternopygus*. *J. Comp. Physiol.* A **169**, 441–450.

Keller, C. H., Maler, L. and Heiligenberg, W. (1990). Structural and functional organization of a diencephalic sensory–motor interface in the gymnotiform fish, *Eigenmannia*. *J. Comp. Neurol.* **293**, 347–376.

Kennedy, G. and Heiligenberg, W. (1994). Ultrastructural evidence of GABA-ergic inhibition and glutamatergic excitation in the pacemaker nucleus of the gymnotiform electric fish, *Hypopomus*. *J. Comp. Physiol.* A **174**, 267–280.

Kramer, B. (1987). The sexually dimorphic jamming avoidance response in the electric fish, *Eigenmannia*. *J. Exp. Biol.* **130**, 39–62.

Kramer, B. (1990). *Electrocommunication in Teleost Fish*. Berlin: Springer-Verlag.

Kramer, B. (1999). Waveform discrimination, phase sensitivity and jamming avoidance in a wave-type electric fish. *J. Exp. Biol.* **202**, 1387–1398.

Larimer, J. L. and MacDonald, J. A. (1968). Sensory feedback from electroreceptors to electromotor pacemaker centers in gymnotids. *Am. J. Physiol.* **214**, 1253–1261.

Maler, L. and Ellis, W. G. (1987). Inter-male aggressive signals in weakly electric fish are modulated by monoamines. *Behav. Brain Res.* **25**, 75–81.

Maler, L. and Mugnaini, E. (1994). Correlating gamma-aminobutyric acidergic circuits and sensory function in the electrosensory lateral line lobe of a gymnotiform fish. *J. Comp. Neurol.* **345**, 224–252.

McCormick, C. A. and Braford, M. R. (1988). Central connections of the octavolateralis system: evolutionary considerations. In *Sensory Biology of Aquatic Animals* (ed. J. Atema, R. R. Fay, A. N. Popper and W. N. Tavolga), pp. 733–756. New York: Springer-Verlag.

Metzner, W. (1993). The jamming avoidance response in *Eigenmannia* is controlled by two separate pathways. *J. Neurosci.* **13**, 1862–1878.

Metzner, W. and Heiligenberg, W. (1991). The coding of signals in the electric communication of the gymnotiform fish, *Eigenmannia*: From electroreceptors to neurons in the torus semicircularis dorsalis of the midbrain. *J. Comp. Physiol.* A **169**, 135–150.

Metzner, W. and Heiligenberg, W. (1993). The neuronal processing of communicatory signals in *Eigenmannia*. *J. Comp. Physiol.* A **173**, 722–726.

Metzner, W. and Juranek, J. (1997a). A sensory brain map for each behavior? *Proc. Natl. Acad. Sci. USA* **94**, 14798–14803.

Metzner, W. and Juranek, J. (1997b). A method to biotinylate and histochemically visualize ibotenic acid for pharmacological inactivation studies. *J. Neurosci. Meth.* **76**, 143–150.

Metzner, W., Koch, C., Wessel, R. and Gabbiani, F. (1998). Feature extraction by burst-like spike patterns in multiple sensory maps. *J. Neurosci.* **15**, 2283–2300.

Metzner, W. and Viete, S. (1996a). The neuronal basis of communication and orientation in the weakly electric fish, *Eigenmannia*. I. Communication behavior or: seeking a conspecific's response. *Naturwissenschaften* **83**, 6–14.

Metzner, W. and Viete, S. (1996b). The neuronal basis of communication and orientation in the weakly electric fish, *Eigenmannia*. II. Electrolocation and avoidance of jamming by neighboring conspecifics. *Naturwissenschaften* **83**, 71–77.

Moller, P. (1995). *Electric Fishes*. London, New York: Chapman & Hall.

Moortgat, K. T., Keller, C. H., Bullock, T. H. and Sejnowski, T. J. (1998). Submicrosecond pacemaker precision is behaviorally modulated: the gymnotiform electromotor pathway. *Proc. Natl. Acad. Sci. USA* **95**, 4684–4689.

New, J. G. and Singh, S. (1994). Central topography of anterior lateral line nerve projections in the channel catfish, *Ictalurus punctatus*. *Brain Behav. Evol.* **43**, 34–50.

Richards, S. and Maler, L. (1996). The distribution of met-enkephalin in the brain of *Apteronotus leptorhynchus*, with emphasis on the electrosensory system. *J. Chem. Neuroanat.* **11**, 173–190.

Rose, G. J. (1989). Rejection of common mode signals in the central electrosensory system. *Soc. Neurosci. Abstr.* **15**, 348.

Rose, G. J. and Call, S. J. (1992). Differential distribution of ampullary and tuberous processing in the torus semicircularis of *Eigenmannia*. *J. Comp. Physiol.* A **170**, 253–261.

Rose, G. J., Kawasaki, M. and Heiligenberg, W. (1988). 'Recognition units' at the top of a neuronal hierarchy? Prepacemaker neurons in *Eigenmannia* code the sign of frequency differences unambiguously. *J. Comp. Physiol.* A **162**, 759–772.

Sas, E. and Maler, L. (1991). Somatostatin-like immunoreactivity in the brain of an electric fish (*Apteronotus leptorhynchus*) identified with monoclonal antibodies. *J. Chem. Neuroanat.* **4**, 155–186.

Sas, E., Maler, L. and Tinner, B. (1990). Catecholaminergic systems in the brain of a gymnotiform teleost fish: an immunohistochemical study. *J. Comp. Neurol.* **292**, 127–162.

Sas, E. L., Maler, L. and Weld, M. (1993). Connections of the olfactory bulb in the gymnotiform fish, *Apteronotus leptorhynchus*. *J. Comp. Neurol.* **335**, 486–507.

Shumway, C. A. (1989a). Multiple electrosensory maps in the medulla of weakly electric gymnotiform fish. I. Physiological differences. *J. Neurosci.* **9**, 4388–4399.

Shumway, C. A. (1989b). Multiple electrosensory maps in the medulla of weakly electric gymnotiform fish. II. Anatomical differences. *J. Neurosci.* **9**, 4400–4415.

Spiro, J., Brose, N., Heinemann, S. F. and Heiligenberg, W. (1994). Immunolocalization of NMDA receptors in the central nervous system of weakly electric fish: functional implications for the modulation of a neuronal oscillator. *J. Neurosci.* **14**, 6289–6299.

Striedter, G. F. (1992). Phylogenetic changes in the connections of the lateral preglomerular nucleus in ostariophysan teleosts: a pluralistic view of brain evolution. *Brain Behav. Evol.* **39**, 329–357.

Stroh, T. and Zupanc, G. K. H. (1995). Somatostatin in the prepacemaker nucleus of weakly electric fish, *Apteronotus leptorhynchus*: evidence for a nonsynaptic function. *Brain Res.* **674**, 1–14.

Szabo, T. and Enger, P. S. (1964). Pacemaker activity of the medullary nucleus controlling electric organs in high-frequency gymnotid fish. *Z. Vergl. Physiol.* **49**, 285–300.

Takizawa, Y., Rose, G. J. and Kawasaki, M. (1999). Resolving competing theories for control of the jamming avoidance response: the role of amplitude modulations in electric organ discharge decelerations. *J. Exp. Biol.* **202**, 1377–1386.

Tokunaga, A., Akert, K., Sandri, C. and Bennett, M. V. L. (1980). Cell types and synaptic organization of the medullary electromotor nucleus in a constant frequency weakly electric fish, *Sternarchus albifrons*. *J. Comp. Neurol.* **192**, 407–426.

Turner, R. W., Maler, L., Deerinck, T., Levinson, S. R. and Ellisman, M. H. (1994). TTX-sensitive dendritic sodium channels underlie oscillatory discharge in a vertebrate sensory neuron. *J. Neurosci.* **14**, 6453–6471.

Turner, R. W., Plant, J. R. and Maler, L. (1996). Oscillatory and burst discharges across electrosensory topographic maps. *J. Neurophysiol.* **76**, 2364–2382.

van Essen, D. C. and Gallant, J. L. (1994). Neural mechanisms of form and motion processing in the primate visual system. *Neuron* **13**, 1–10.

Viete, S. and Heiligenberg, W. (1991). The development of the jamming avoidance response (JAR) in *Eigenmannia*: and innate behavior indeed. *J. Comp. Physiol.* A **169**, 15–23.

Watanabe, A. and Takeda, K. (1963). The change of discharge frequency by A.C. stimulus in a weak electric fish. *J. Exp. Biol.* **40**, 57–66.

Weld, M. M. and Maler, L. (1992). Substance P-like immunoreactivity in the brain of the gymnotiform fish, *Apteronotus leptorhynchus*: presence of sex differences. *J. Chem. Neuroanat.* **5**, 107–129.

Wong, C. J. H. (1997a). Afferent and efferent connections of the diencephalic prepacemaker nucleus in the weakly electric fish, *Eigenmannia virescens*: interactions between the electromotor system and the neuroendocrine axis. *J. Comp. Neurol.* **383**, 18–41.

Wong, C. J. H. (1997b). Connections of the basal forebrain of the weakly electric fish, *Eigenmannia virescens*. *J. Comp. Neurol.* **389**, 49–64.

Yamamoto, T., Maler, L. and Nagy, J. L. (1992). Organization of galanin-like immunoreactive neuronal systems in weakly electric fish (*Apteronotus leptorhynchus*). *J. Chem. Neuroanat.* **5**, 19–38.

Zupanc, G. K. H. (1991). The synaptic organization of the prepacemaker nucleus in the weakly electric knifefish, *Eigenmannia*: a quantitative ultrastructural study. *J. Neurocytol.* **20**, 818–833.

Zupanc, G. K. H. (1996). Peptidergic transmission: from morphological correlates to functional implications. *Micron* **27**, 35–91.

Zupanc, G. K. H. and Heiligenberg, W. (1989). Sexual maturity-dependent changes in neuronal morphology in the prepacemaker nucleus of adult weakly electric knifefish, *Eigenmannia*. *J. Neurosci.* **9**, 3816–3827.

Zupanc, G. K. H. and Heiligenberg, W. (1992). The structure of the diencephalic prepacemaker nucleus revisited: light microscopical and ultrastructural studies. *J. Comp. Neurol.* **323**, 558–569.

Zupanc, G. K. H. and Horschke, I. (1997a). Reciprocal connections between the periglomerular nucleus and the central posterior/prepacemaker nucleus in the diencephalon of weakly electric fish, *Apteronotus leptorhynchus*. *Neurosci.* **80**, 653–667.

Zupanc, G. K. H. and Horschke, I. (1997b). Neurons of the posterior subdivision of the nucleus preopticus periventricularis project to the preglomerular nucleus in the weakly electric fish, *Apteronotus leptorhynchus*. *Brain Res.* **774**, 106–115.

Zupanc, G. K. H. and Horschke, I. (1997c). A distinct population of neurons in the central posterior/prepacemaker nucleus project to the nucleus preopticus periventricularis in the weakly electric gymnotiform fish, *Apteronotus leptorhynchus*. *Brain Res.* **776**, 117–125.

Zupanc, G. K. H. and Maler, L. (1993). Evoked chirping in the weakly electric fish, *Apteronotus leptorhynchus*: a quantitative biophysical analysis. *Can. J. Zool.* **71**, 2301–2310.

Zupanc, G. K. H. and Maler, L. (1997). Neuronal control of behavioral plasticity: the prepacemaker nucleus of weakly electric gymnotiform fish. *J. Comp. Physiol.* A **180**, 99–111.

Zupanc, G. K. H., Maler, L. and Heiligenberg, W. (1991). Somatostatin-like immunoreactivity in the region of the prepacemaker nucleus in weakly electric knifefish, *Eigenmannia*: a quantitative analysis. *Brain Res.* **559**, 249–260.