culating opioid pentapeptide, [Met]enkephalin, in one recent investigation (*9*). Plasma β-endorphin concentrations were apparently not measured in that study. Another group reported a similar reduction in β-lipotropin to undetectable levels in three patients treated with dexamethasone (*10*). The suppression of plasma β-lipotropin by dexamethasone (*9, 10*) suggests that, in our study, the β-endorphin antibody with a 50 percent cross-reactivity to β-lipotropin was not measuring β-lipotropin but was probably more specific for β-endorphin in the plasma samples taken after dexamethasone administration.

These findings of suppressed ACTH and β-lipotropin after treatment with dexamethasone and our evidence for the lack of β-endorphin suppression by dexamethasone suggest another pathway for β-endorphin release not directly linked to the ACTH secreting system. Along this line, it has recently been shown with immunohistochemical techniques that β-endorphin immunoreactivity and ACTH are not necessarily present in the same human anterior pituitary cells simultaneously (*11*). In addition to its location in the pituitary gland, β-endorphin has been found in other parts of the brain and in peripheral organs such as the pancreas (*12*), although no direct evidence is available yet to indicate that ACTH-independent pituitary cells or tissues of the brain or of peripheral organs contribute to the production of plasma β-endorphin. Our data indicate a dissociation between the effects of a low dose of dexamethasone on plasma β-endorphin and plasma cortisol concentrations. We conclude that in humans and rhesus monkeys the feedback mechanisms regulating the hypothalamic-pituitary-adrenal system and the β-endorphin system are not identical.

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Phase-Sensitive Midbrain Neurons in *Eigenmannia*: Neural Correlates of the Jamming Avoidance Response

Abstract. *Neurons in the torus semicircularis of the weakly electric fish* Eigenmannia *encode phase differences between sinusoidal electrical stimuli received in different body regions. These fish normally experience time-varying phase differences when the electric organ discharge fields of two or more individuals overlap. These phase differences supply information necessary for the animal's jamming avoidance behavior.*

South American weakly electric fish [gymnotoid fish (*1*)] generate electric fields by rhythmically discharging electric organs located in their elongate tails. This field results in a voltage generated across the skin (transepidermal voltage), which is monitored by electroreceptors distributed over the animal's surface. Objects having an electrical impedance different from that of the surrounding water distort this electric field, thereby altering the transepidermal voltage patterns associated with the electric organ discharge (EOD). The animals can "electrolocate"—detect and identify objects in their environment—by analyzing the patterns of transepidermal voltage encoded by their electroreceptors (*1a*).

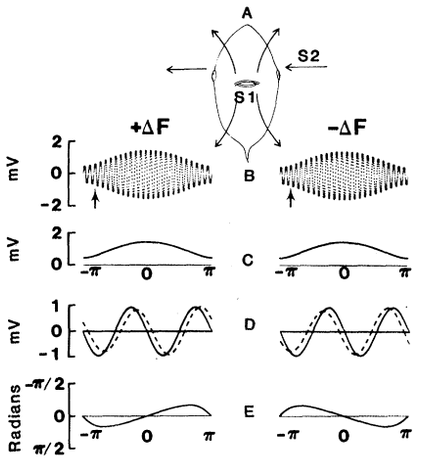

Fig. 1. (A) Head-on view of *Eigenmannia* illustrating the patterns of current flow resulting from the radially presented EOD replacement, *S1*, and the transversely presented jamming signal, *S2*. (B) Beat waveform produced by the addition of *S1* and *S2*; ΔF = 4 Hz. The *S1* frequency was set at 100 Hz for illustrative purposes. (C) Envelopes of the signals of (B). These are identical for opposite ΔF's as long as the two components of the beat, *S1* and *S2*, are sinusoidal. (D) Individual cycles of the beat waveform (dashed lines) from the region indicated by the arrows in (B) on an expanded time scale. The solid lines show the *S1* component of these waveforms. The phase relationships between these two signals are opposite for the two signs of ΔF. (E) Plots of the phase of the beat waveform relative to the *S1* over the time of one beat cycle. The depths of the amplitude modulation (C) and of the phase modulation (D) approach zero as the intensity of *S2* approaches zero.

An individual's electrosensory system is sensitive to electrical signals of any origin as long as those signals meet the amplitude and frequency requirements of the electroreceptors (*2*). Therefore, certain extraneous signal sources can interfere with the animal's analysis of its personal electric field; that is, the system is sensitive to jamming (*3*). Extraneous signals similar in amplitude and frequency to the animal's own EOD significantly impair electrolocation (*4*). Conspecifics, of course, form a large population of sources of jamming signals in the animal's environment, and the fish have evolved a jamming avoidance response (JAR) that reduces the deleterious effects of jamming signals (*3-5*).

We studied the gymnotoid fish *Eigenmannia virescens*, which has nearly sinusoidal EOD's of stable, though individually different, fundamental frequency (*F1*) between 250 and 600 Hz. Electrolocation is most severely impaired if the animal is exposed to a second EOD-like signal having a slightly different frequency (*F2*). Signals separated by difference frequencies (ΔF, where $\Delta F = F2 - F1$) ranging between ± 2 to ± 8 Hz are most detrimental; fish ex-

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posed to this mixture of signals alter their EOD frequency in the direction that will enlarge the frequency difference (*3*). Enlarging ΔF reduces the deleterious effects of the mixed signal on the electrolocation performance (*4, 5*). This JAR has been the object of many recent behavioral and neurophysiological studies (*6*), including recent determinations (*7*) of the following minimal stimulus conditions under which the behavior will occur. (i) Actual EOD's are not needed. An animal's EOD can be abolished by a neuromuscular blockade (*8*); a pure sinusoidal signal, *S1*, of appropriate amplitude, frequency, and field geometry can be used as a substitute. A second sinusoidal signal, *S2*, whose frequency differs by ΔF from that of *S1*, can be used for the interfering signal. The would-be EOD frequency of the curarized animal can be determined by recording from the spinal motorneurons that drive the electric organ (*7*). This signal changes its frequency according to the sign and the magnitude of the ΔF. As in the natural situation, the pacemaker frequency is raised or lowered in response to negative or positive ΔF's, respectively. (ii) Neither *S1* nor *S2* need be equal to or harmonically related to the animal's would-be EOD frequency. This fact strongly supports the hypothesis that the JAR can be controlled solely by electroreceptive afferences with no reference to signals from the central nervous system related to the would-be EOD frequency. (iii) The sinusoidal EOD substitute *S1* and the sinusoidal jamming signal *S2* must be presented with different geometries if the behavior is to occur. If *S1* and *S2* are added electronically and the resulting beat signal is presented through a single electrode pair, no JAR is elicited. A necessary condition for the JAR, not met when both are presented by a single electrode pair (*7*), is that different regions of the fish receive different relative intensities of *S1* and *S2*.

The EOD replacement, *S1*, was presented between an internal electrode and electrodes lateral to the fish so that current attributable to the *S1* flowed radially. The jamming stimulus, *S2*, was given transversely between electrodes lateral to the fish so that current crossed the skin in opposite directions on either side of the body (Fig. 1A). This stimulus geometry ensures that different regions of the body receive different relative magnitudes of the *S1* and *S2* signals. Since the *S1* is radial, all body regions will receive nearly the same magnitude of current through the skin from this signal. The transverse *S2* causes different regions of the body to receive different amounts of *S2* current. For example, current through the skin in the dorsal and ventral regions of the body will be minimal since the transverse *S2* current is tangential to the skin at these points.

Figure 1B shows the voltage waveform recorded across the skin of the animal when ΔF is $\pm$ 4 Hz. The envelopes of the signals are shown in Fig. 1C. The time course of the amplitude modulation of the signal is identical for either sign of ΔF. The magnitude of this amplitude modulation will vary around the perimeter of the animal's cross section, reaching a maximum at lateral positions and a minimum at dorsal and ventral positions.

Since the patterns of amplitude modulation are identical for either sign of ΔF, this parameter cannot supply the information needed by the animal to decide whether to increase or decrease its EOD

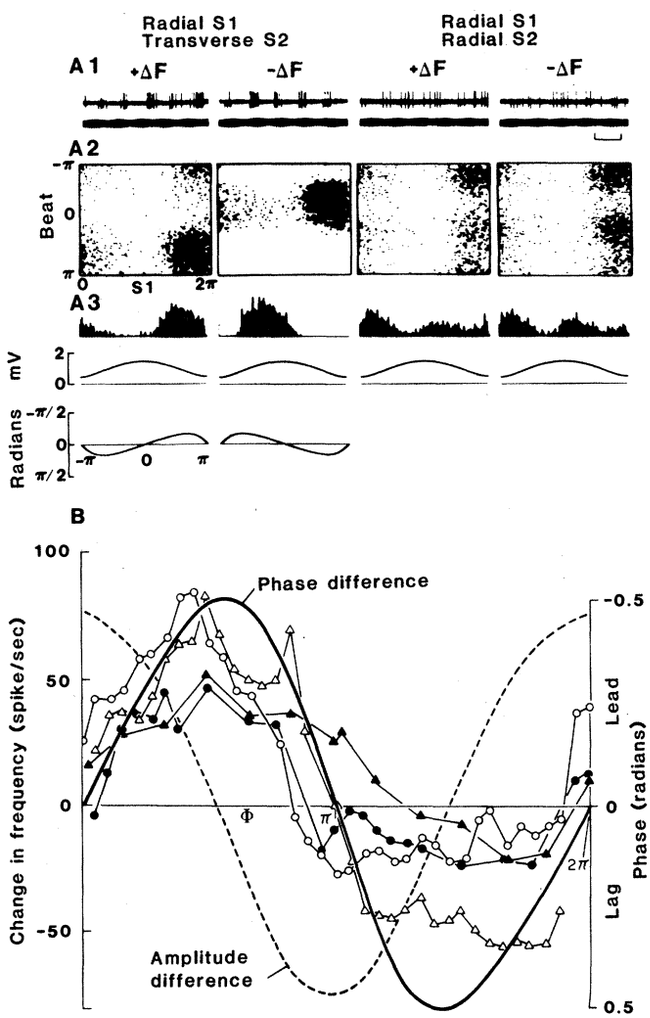

Fig. 2. (A1) Responses of torus sign-sensitive cells (upper trace) and the stimulus waveform (lower trace) recorded with the indicated patterns of stimulation. Time mark, 250 msec; *S1* frequency, 400 Hz; *S1* amplitude, 1.0 mV measured across the skin; *S2* amplitude, 0.5 mV. The stimulus was monitored between an electrode implanted in the dorsal musculature and one just touching the lateral body surface. (A2) Raster displays of the responses of (A1). The length of the vertical axis equals the duration of one beat cycle (250 msec) beginning at the top, the time of the minimum beat amplitude ($-\pi$). The length of the horizontal axis equals the duration of one *S1* cycle (2.5 msec) beginning with the negative zero-crossing. Each consecutive *S1* cycle within the beat is displaced downward an equal amount and the cathode-ray tube was brightened on the Z axis when each action potential occurred, thereby generating the pattern of dots seen in the display. (A3) Upper row: poststimulus time histograms computed over the time of one beat cycle. Middle row: time course of the amplitude modulation of the beat waveform. Third row: phase modulation of the beat relative to *S1*. No phase modulation is indicated beneath the histograms of data from radial *S1*, radial *S2* stimulation since, under these conditions, the animal was unable to make a phase measurement. (B) Responses of sign-sensitive cells to 100-msec tone bursts of the phase-shifted signal Del *S1* plotted against the degree of phase shift (ϕ) between *S1* and Del *S1*. The solid line shows the maximum possible phase change in radians between the composite signal (*S1* + Del *S1*) and *S1* plotted as a function of ϕ. The dashed line shows the maximum changes in amplitude of the composite signal relative to *S1* as a function of ϕ. Maximum and minimum values for these amplitude changes are $\pm$ 0.5 mV. Different symbols indicate data from different cells.

frequency. In addition to the obvious amplitude modulations, the beat signal also undergoes cyclic phase modulations relative to either of the individual sinusoidal components. In Fig. 1D, two cycles from the region of the beat indicated by the arrows in Fig. 1B are plotted along with *S1*, on an expanded time scale. With positive ΔF, the beat waveform lags behind the *S1* waveform during the first half of the beat cycle, and it leads *S1* during the second half of the beat cycle. The opposite phase relationship is observed with negative ΔF beats. The value of this phase difference changes in a continuous fashion over the time course of one beat cycle (Fig. 1E) (*9*). At any given time within the beat cycle, the phase relationship between the beat waveform and *S1* will be opposite for opposite signs of ΔF. This phase information must be evaluated with reference to the concurrent amplitude modulation to distinguish the sign of ΔF and thereby supply the animal with the cue needed to make a correct decision as to whether to increase or decrease the EOD frequency. Behavioral studies show that both the amplitude and the phase information are necessary; if either is missing, no JAR will occur (*7*). This leads to the question "How can the animals extract this information from their electroreceptor afferences?"

Two types of electroreceptors, T and P receptors (*10*), can encode the two crucial stimulus parameters, phase and amplitude. The T receptors fire one spike per stimulus cycle, phase-locked near the positive zero-crossing of the stimulus waveform. The P receptors fire less regularly, but as a function of the amplitude of the stimulus. Information about amplitude modulation is thereby transformed into changes in the rate of firing of the P receptors. The T receptors seem well suited to measuring the phase because of their precise timing relative to the stimulus waveform. However, in order to evaluate phase, the animals must compare the firing times of T receptors relative to some phase reference. A phase reference of absolute zero could be supplied by T receptors situated in a body region receiving no *S2* stimulation, and a relative phase reference could be supplied by T receptors receiving a smaller *S2* stimulation. In either case, a phase measurement could be made by comparing the timing of T receptor firings from areas of the body receiving different amounts of *S2* stimulation (*7*). Presenting both *S1* and *S2* with identical stimulus geometries, through the same electrode pair, ensures that all areas of the body receive the same ratio of *S2* to *S1* stimulation. This procedure eliminates beat-related phase differences among T receptor outputs, and no JAR can be elicited.

We have monitored the activity of electroreceptors and single cells in various regions of the CNS in order to identify those cell types whose response properties are correlated with the presence and the sign of the JAR. We have discovered a class of cells in the torus semicircularis that are sensitive to the sign of ΔF, provided that the stimulus geometry allows phase modulations to be detected. The torus semicircularis is a major mesencephalic structure in lower vertebrates, which is probably homologous to the inferior colliculus of higher forms. Figure 2A shows the responses of such a cell to beats of $\pm$ 4 Hz ΔF presented with different (left) and identical (right) *S1* and *S2* geometries. Only in the first case do we see a sign-specific response pattern. In a study of 22 of these cells, the time of peak activity within the beat cycle shifted an average of 154° (standard deviation, 20.8) when the sign of ΔF was changed. When the same stimuli were presented with identical *S1* and *S2* geometries, all regions of the body received the same relative intensities of *S1* and *S2*, and, therefore, the signal received by all electroreceptors showed the same degree of phase modulation. This situation precludes the possibility that a phase measurement can be made by the animal, since measurements between any groups of T receptors will show no ΔF sign-dependent phase difference.

We performed another experiment to test the hypothesis that cells of the torus sensitive to the sign of ΔF actually evaluate modulations in phase by comparing the information received over T receptors from areas of the body receiving different intensities of *S2* relative to *S1*. A continuous radial *S1* was presented to the animal, and tone bursts of the same signal (Del *S1*) phase-shifted by various amounts, ϕ, were periodically applied transversely. The phase-shifted version of *S1* assumed the role of *S2* in the previous experiments. The addition of *S1* and Del *S1* results in a sinewave whose amplitude and phase relative to *S1* alone depend on the size of the phase shift between *S1* and Del *S1* (Fig. 2B). Depending upon the value of ϕ, cells were either excited or inhibited. No such effect was observed when both *S1* and Del *S1* were presented with identical field geometries.

If *S1* and its phase-shifted counterpart, Del *S1*, are presented with radial and transverse geometries, respectively, different intensity ratios between these two signals will exist at various places on the animal's body surface. The lateral parts of the body will receive the strongest Del *S1* components, whereas the dorsal and ventral surfaces will be nearly free of this component. For any given ϕ, the resulting phase difference between the mixed signal (*S1* + Del *S1*) and the pure *S1* will increase with the intensity ratio of Del *S1* to *S1*. The maximum difference will occur at the lateral regions of the body (solid line in Fig. 2B). The amplitude difference between the composite signal and S1 alone also increases with the intensity ratio of Del *S1* to *S1* (dashed line in Fig. 2B).

The close agreement between the phase difference of the resultant signal relative to the pure *S1* as a function of ϕ and the responses of the sign-sensitive torus cells suggest that these cells are driven by, or encode, the phase difference between signals arriving from different regions of the body surface.

We conclude that there is a population of cells in the torus semicircularis capable of resolving the differences in the phases of signals arriving at different regions of the body (*11*). A conservative estimate of the threshold phase difference for these cells is 0.1 rad, which corresponds to a time delay of 40 μsec when *S1* is 400 Hz. The output of these cells taken in conjunction with the output of cells sensitive only to the amplitude modulation of a beating signal could provide the animal with an unambiguous measure of the sign of the difference in frequencies between its own EOD and that of a neighbor (*12*). This information is necessary for correct jamming avoidance behavior.

The evaluation of phase differences in electric signals received by different regions of the animal's body resembles the evaluation of phase differences in auditory signals arriving at the two ears in mammals (*13*). In both instances, differences in arrival times of signals from neurons phase-locked to a sinusoidal input signal are compared; the comparison is made in homologous brain areas, the torus semicircularis of fish and the inferior colliculus of mammals.

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8. Since the electric organs of most weakly electric fish, excluding the apteronotids, are modified muscles, the normal discharge can be eliminated with curarelike drugs such as Flaxedil or Alloferin.
9. An *S2/S1* ratio of 0.5 was chosen for this figure; with this large *S2* intensity, the steepness of the slopes of the phase function is significantly different in the regions of 0 and π. Since the direction of the phase shift in the regions of 0 and π is also opposite for opposite signs of ΔF, the fish could theoretically decode the sign of the ΔF from the phase function alone. Behavioral experiments (*7*) show that the phase information alone is insufficient for evoking the JAR.
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11. It is likely that these torus sign-sensitive cells are the same as the torus cells previously shown to respond to beats of opposite sign with large (160°) phase shifts of peak activity within the beat cycle when quasi-natural (slant-clipped sine-wave) stimulation is used (*6*).
12. At present, the site of this integration is unknown. The previously described ΔF decoder cells (*6*) could be the cells accomplishing this task.
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Recall (Versus Recognition) of Taste and Immunization Against Aversive Taste Anticipations Based on Illness

Abstract. *Two experiments show that, after taste-aversion conditioning, rats can use external retrieval cues to recall or anticipate the aversive taste solution and avoid its location without making contact with the flavor. They also show that the rat's avoidance of a conditioned aversive taste and its consumption of the aversive flavored solution can be attenuated by giving it prior runway training in which taste reward is given inconsistently on a partial reinforcement schedule.*

It is well known that animals can learn to suppress the intake of flavored solutions associated with x-irradiation (*1*) and poisons (*2*). It has not been demonstrated that animals can avoid a flavor on the basis of the anticipation of its conditioned aversive taste. With few exceptions (*3*), the measurement of a taste aversion has been in terms of the suppression of fluid intake. The animals have first to make a direct contact with or "recognize" the taste or odor of the fluid; then they show suppression. We have found that rats could anticipate the upcoming aversive taste, or, in more cognitive terms, that rats could use alley cues to recall the memory of a taste. In a second experiment, we found that the learning or expression (or both) of a taste aversion could be attenuated by prior behavioral procedures. [Radiation-induced (*4*) and poison-induced (*5*) aversions can be blocked or attenuated by chemical agents.] The prior behavioral treatment in experiment 2 was partial reinforcement (PRF) training using as reward the particular flavored solution to which the aversion is conditioned.

The general experimental sequence of both experiments had three phases: (i) thirsty rats were trained to run in an alley for a flavored solution as reward; (ii) that taste was then made aversive through a conditioning procedure in the home cages; and (iii) the approach response to the flavored solution, learned in phase 1, was extinguished. The experiment was conducted during the light phase (0800 to 2200 hours) of a light-dark cycle. The training apparatus was a straight alley runway with a clear Plexiglas top and a black interior. It was 194 cm long, 7 cm wide, and 7.3 cm high. The first 35 cm comprised the start box and the last 35 cm comprised the goal box. Both compartments were separated from the run segment (alley) by guillotine doors. A round metal cup, 2.7 cm in diameter, 1 cm deep, and 1.5 cm above the floor, was attached to the end wall of the goal box. The time-scoring system began when the start door was mechanically lowered by pressing a button. Three light beams, positioned at 30 cm, 122 cm, and 152 cm from the start door, controlled three electric clocks that recorded start, run, and goal times to 0.01 second.

In experiment 1, 48 60- to 70-day-old female Sprague-Dawley rats bred in our laboratory were run in three squads of 16. Deprived of water for 24 hours, the rats were placed four times in the goal box with 1 ml of a 1.5 percent solution of vinegar (by volume in tap water) as a reward. (i) Phase 1 runway acquisition training, initiated 24 hours after goal box training, consisted of 30 continuous reinforcement (CRF) trials over 3 days, with 6, 12, and 12 trials per day. Reward was 1 ml of the vinegar solution placed in the goal box cup. The intertrial interval was 20 minutes. (ii) Phase 2, conducted 24 hours after the runway acquisition phase of training, was taste-aversion conditioning in the home cage. At this point, three of the four groups of rats were allowed to drink vinegar, a 0.2 percent saccharin solution (weight to volume in tap water), or water for 30 minutes in their home cages. The drinking was followed immediately by an intraperitoneal injection of 0.3*M* lithium chloride (LiCl) (3 percent of body weight). The fourth group was a control that drank vinegar solution but was injected with an equivalent amount of physiological saline. (iii) Phase 3 runway extinction (five trials) was conducted 24 hours after home-cage taste-aversion conditioning. On extinction trials the goal box cup was clean and empty. Thirty minutes after the runway extinction phase, all four groups of rats were allowed to drink vinegar solution in the home cage for 30 minutes as a test of the taste aversion conditioned in phase 2.

Figure 1A summarizes the runway acquisition and extinction data (*6*). All four groups of rats reached asymptotic running speeds within 30 trials. In extinction, the vinegar-LiCl group suppressed running speed on the first extinction trial after taste-aversion conditioning; and the response was extinguished below the operant level across trials. The saline control group was the slowest to extinguish. The other two poisoned groups—saccharin-LiCl and water-LiCl—ran faster than the vinegar-LiCl group, but, perhaps because they were still affected by the illness, these two poisoned groups were slower than the saline control group (*7*). These data (and particularly the first extinction point) are to our knowledge the first demonstration of the suppressive effects of the anticipation of an aversive taste. They also demonstrate a specific relationship between aversive taste conditioning (in the home cage) and the suppression of an instrumental response

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