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Mechanism for food avoidance learning in the central pattern generator of feeding behavior of *Pleurobranchaea californica*

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ABSTRACT **Food-avoidance conditioning in the mollusk *Pleurobranchaea* results in suppression of the feeding response to food stimuli. In conditioned animals, identified interneurons of the central pattern generator (CPG) for feeding behavior, the Int-2s, respond to a food stimulus with greater and more long-lasting excitation than controls. Enhanced Int-2 responsiveness to food stimuli is associated with markedly heightened Int-2 excitability. Sustained activity in the Int-2s arrests motor output of the oscillatory CPG in the protraction/retraction movement cycle of feeding through tonic excitation of a population of retractor interneurons and inhibition of protractors. The CPG locus of the learning mechanism is permissive of sensory excitation of alternative behavior and leaves the possibility open for release of the suppressed behavior in a fully aroused state.**

Identification and characterization of learning mechanisms is an important goal in understanding the organization of animal behavior. One animal in which the neurophysiology of learning has been usefully studied is the marine slug *Pleurobranchaea*, a predatory and voracious feeder on other invertebrates and carrion. *Pleurobranchaea* readily learns to avoid specific food stimuli and suppress its feeding behavior when food is presented in association with a noxious electric shock (1–3). Learning occurs efficiently over 1–10 training trials and is influenced by motivational state (4); memory persists over many days (2). This type of learning has clear adaptive significance for the foraging strategies and food selection of omnivores and predators (5).

Neural correlates of learning were previously reported (6, 7). In the hungry untrained animal, food applied to the chemosensory oral veil normally causes strong synaptic excitation and spiking of the paracerebral neurons (PCNs) of the brain (cerebropleural ganglion). PCN activity is important in the initiation and maintenance of feeding behavior (8). In contrast, in food-avoidance-trained animals food stimuli cause profound synaptic inhibition of the PCNs coincident with suppression of feeding behavior (6, 7). When the inhibitory pathway was described, it was found to have widespread synaptic effects throughout the motor network for feeding behavior (9–12). Moreover, activity in this pathway suppressed feeding behavior (11, 12).

The following experiments were undertaken to elucidate the roles of the inhibitory neurons in learning. The neurons of the inhibitory pathway were found to be more active in conditioned animals, a result largely due to enhanced excitability in one of the neuron groups: the interneuron 2 population (Int-2s). Evidence that they substantially contribute to learned suppression of feeding establishes these neurons as loci where feeding behavior is regulated by learning experience. The mode of action whereby the Int-2s inhibit behavior provides an interesting mechanism with wider implications for the organization of behavior.

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MATERIALS AND METHODS

Conditioning Procedures. *Pleurobranchaea californica* (150–900 g) were obtained from R. Fay (Pacific BioMarine, Venice, CA) and from M. Morris (Sea-Life Supply, Sand City, CA) and were maintained in Instant Ocean at 13°C. Behavioral testing was performed in accord with previously established procedures (1–3, 6, 7). Serial dilutions (1:10) of a homogenate of squid (SH; squid/seawater, 1:1, wt/wt) were prepared for behavioral measurement of the thresholds of easily observed components of feeding behavior, proboscis extension, and the succeeding bite/strike (7). Thresholds for eliciting proboscis extension and the bite/strike were measured by applying 1.5 ml of SH solutions over 10 sec to the oral veil, in order of increasing strength, while observing the lowest effective SH concentration for eliciting the behavior. Latencies for responses were measured with full-strength SH applied until the animal either responded or 90 sec elapsed.

Animals were conditioned for food avoidance as described (1–3, 6, 7), except that a random schedule was used. Animals were paired on the basis of size, thresholds, and latencies, and they were assigned to experimental or control groups by coin toss. Animals in the experimental group were trained in trials in which full-strength SH was applied to an animal's oral veil until proboscis extension began or until 90 sec elapsed with successful total suppression of the feeding response. Animals failing to attain the 90-sec criterion received 1 min of shock (70 V square pulses of 20 msec duration at 20 Hz) delivered to the head region with bipolar electrodes (6, 7). Control animals received food stimuli and shocks separated by intervals of 1–59 min drawn from a table of random numbers. Control animals did not show significant changes in behavioral thresholds or latencies after random food/shock trials. More than 80% of experimental animals showed threshold elevations >100-fold and response latencies prolonged >90 sec. Detailed results of the random conditioning are in preparation for separate publication. Only animals attaining the 90-sec criterion were used in neurophysiological analyses.

Neurophysiological Analyses. Animals were prepared for electrical recordings as "hemi-animal" preparations (10, 12–14), consisting of the head region of the animal, including the sensory apparatus (oral veil, tentacles, and rhinophores), the feeding apparatus (proboscis, mouth, buccal mass, and esophagus), and the nervous system (brain, buccal, stomatogastric, rhinophore and tentacle ganglia, and peripheral network). The preparation was placed in a water-cooled Lucite chamber (12°C–14°C) and immersed in filtered Instant Ocean adjusted to pH 7.5.

Application of squid homogenate to the oral veil in every case elicited feeding responses in naive animal preparations

Abbreviations: Int-1 and -2, interneurons 1 and 2; PCN, paracerebral neuron (phasic); CPG, central pattern generator; SH, squid homogenate.

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(13, 14); sensory papillae and rhinophores extended and the buccal mass frequently began cyclic protraction/retraction movements characteristic of feeding. During buccal mass movements pipetted SH could be seen to enter the buccal cavity and exit the cut end of the esophagus. Such motor responses waned after 3–4 hr, although neural responses were more robust; experiments were generally terminated within 2–2.5 hr. Avoidance-trained animals did not exhibit feeding behavior; motor responses to food were variously local withdrawal, backwardly directed movements of the lateral oral veil tentacles, or were absent.

The brain was elevated and pinned to a wax-coated platform and the area of the brain with the neuron somata of interest was desheathed. Standard electrophysiological recording methods were used; microelectrodes filled with 3 M KCl (resistance, 15–30 MΩ) were used for intracellular recording, and the signal was led to high impedance amplifiers with bridge circuits for current injection. All signals were displayed on an oscilloscope and permanent records were made on a chart recorder. In the tests of neural responses to SH, the person applying the stimuli did not know to which group the animal was assigned.

Measurements of neural responses were done double-blind. Analysts, unaware of experimental conditions, estimated recorded membrane potential (inclusive of background postsynaptic potential activity) preceding a test condition and outlined the test response. A second "blind" analyst digitized the relative depolarizations or hyperpolarizations for numerical analysis by using a digitizing pad (Houston Instruments "Hipad").

RESULTS

All neurons under study are elements of the rhythmic central pattern generator of feeding behavior. The PCNs are one of a few populations with the ability to drive the cyclic motor output of feeding (8). The inhibitory pathway to the PCNs consists of at least two serially connected neuron groups, with identified synaptic connections shown in Fig. 1 (9, 11, 12). A bilateral population of four to six neurons, the Int-1s, lie near the PCNs and make inhibitory monosynaptic connection on them. Int-1s in turn are monosynaptically excited and driven by a nearby bilateral population of six to eight cells, the Int-2s. The Int-2s receive direct chemosensory inputs from the animal's oral veil, in contrast to the PCNs, which do not (12). The Int-2s are electrically coupled ipsilaterally, and contralateral populations are connected by an excitatory polysynaptic pathway. The Int-2s are potent

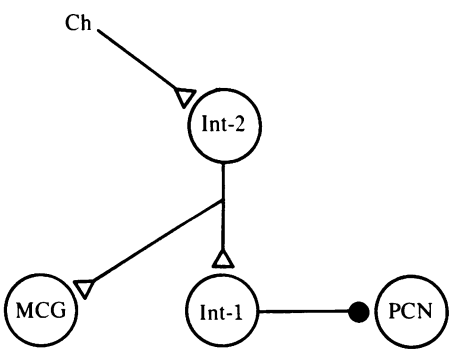

FIG. 1. Monosynaptic relations of the inhibitory pathway with identified neurons discussed (14). Ch, food chemosensory inputs; MCG, metacerebral giant neuron; PCN, phasic type PCN (see ref. 15). Not shown is a weakly inhibitory connection from Int-1 onto Int-2, the significance of which is unknown and not addressed here. △, Excitatory connections; ●, inhibitory connections. Additional detail is to be found in the text.

interneurons that affect the activity of many identified and unidentified neurons of the brain; sustained activity in a single Int-2 can suppress the ongoing activity of the feeding oscillator (12). Int-2 activity elicits an inhibitory postsynaptic potential barrage in the PCNs qualitatively similar to that caused by food stimuli in conditioned animals (9, 11, 12). The Int-2s also excite a variety of other feeding neurons in the brain, including the serotonergic metacerebral giant neurons, which in turn drive many neurons of the buccal ganglion (16).

Enhanced Response to Food in the Inhibitory Pathway of Conditioned Animals. For the naive hungry animal, stimulation of the oral veil and rhinophores with SH normally causes some excitation of both Int-1s and Int-2s; this parallels a general excitation of the feeding network that precedes and culminates in rhythmic motor output (8, 12, 13, 16). In 9 control animals, 11 Int-2s responded in 42 trials (two to five each) of SH stimulation with an overall average of 10.2 ± 2.0 (SEM) action potentials. Fourteen Int-2s tested similarly; in 35 (two to three each) trials on 11 conditioned animals responded with 23.1 ± 3.5 spikes, significantly greater than controls ($P < 0.005$; one-tailed t test).

The activity of the Int-1 followers of the Int-2s was also increased by training. In two Int-1s of two control animals, SH stimulation caused only 2.5 ± 2.3 spikes (averaged from three trials per cell), while in two conditioned animals, the responses averaged at 36 ± 2.7 spikes. These results are statistically significant ($P < 0.025$; one-tailed t test), suggesting that although the number of observations is low, the Int-1s participate in suppressing PCN activity in the food-avoidance conditioned animal.

Enhanced Depolarization in the Int-2s. The preceding results led us to examine the Int-2s for electrical manifestations that might underlie the intensified and prolonged spike activity in conditioned animals. Our studies focused on the Int-2s because of their demonstrated wide range of potent synaptic outputs within the feeding motor network (12).

For the Int-2s of conditioned animals, depolarization by food stimuli was more intense and enduring than in controls. This increase in depolarization was associated with learning-contingent enhancement of Int-2 excitability. Intracellular recordings from Int-2s of conditioned and control animals responding to food stimuli showed a major difference in the form of the depolarization underlying the excitatory response (Fig. 2): in the conditioned animal, depolarization decayed at a much slower rate. Data from 17 experiments indicate that the duration of the food-stimulated depolarization and its intensity (area between the excitatory depolarization and voltage baseline) were much enhanced in experimental animals relative to controls (Table 1). Possible differences in the amplitude of the excitatory responses were not detectable in these experiments.

Enhanced Excitability in the Int-2s. The capacity to sustain a prolonged depolarization that outlasts the excitatory stimulus is exhibited in Int-2s only of conditioned animals; furthermore, it acts in a voltage-dependent fashion. Depolarization of the Int-2s of conditioned animals with injected current for 1 to several sec is followed by a depolarizing afterpotential whose duration and amplitude vary positively with the duration and amplitude of the depolarizing stimulus (Fig. 3). In 11 experiments on conditioned animals in which 14 Int-2s were examined in repeated trials, all Int-2s of the conditioned animals always exhibited the ability to sustain prolonged depolarizations (Fig. 3 *Upper*). Conversely, all of 11 Int-2s examined in nine control animals showed only hyperpolarizing afterpotentials following stimulated trains of spikes (Fig. 3). The conclusiveness of these results points to a learning-induced change in Int-2 function.

Three Int-1s examined in three conditioned animals showed no such depolarizing afterpotentials as seen in the Int-2s. While more data are necessary for a clear conclusion,

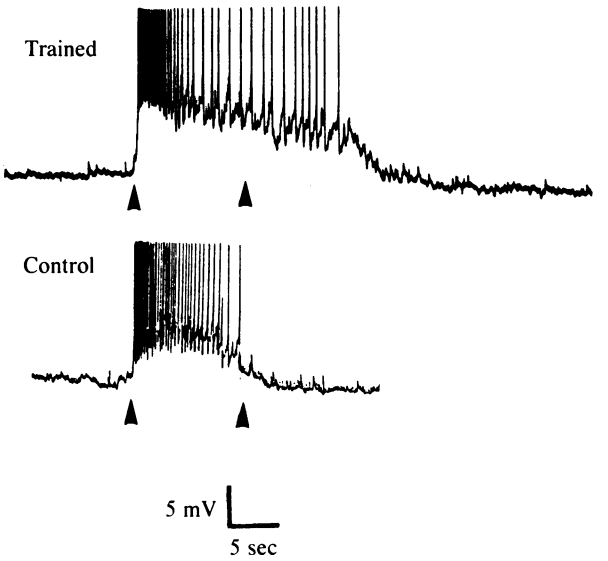

FIG. 2. Int-2s of conditioned animals are more excited by food stimulation of the oral veil and by intracellular current injection than those of control animals. The records show representative Int-2 responses to food stimulation of the oral veil in trained and control animals. SH (1.5 ml) was pipetted onto the animal's oral veil over a 10-sec interval (arrows). The Int-2s of trained animals responded with more spiking activity and more enduring depolarization (Table 1).

the enhanced food-stimulated activity of the Int-1s in conditioned animals could arise largely from the presynaptic activity of the Int-2s.

Roles of the Int-1s and Int-2s in Learned Suppression of Feeding. One specific neural correlate of food-avoidance conditioning in *Pleurobranchaea* is food-stimulated inhibition of PCN activity (6, 7). To ascertain the role of the Int-2/Int-1 pathway in food-stimulated inhibition of PCNs, we measured the effect of hyperpolarizing single interneurons of conditioned animals. In these trials, a PCN and one of the interneurons were simultaneously recorded; the interneuron was alternately allowed to fire normally or was hyperpolarized by current injection to prevent spiking activity. PCN responses were measured from prestimulation voltage baseline for the first 10 sec from start of stimulation. Fig. 4*A* shows one of three pairs of such trials in which the suppression of a single Int-1 reduced inhibitory postsynaptic potential frequency, resulting in significant disinhibition of the PCN.

Similarly, sustained hyperpolarization of Int-2s reduced both the amplitude and the duration of PCN inhibition. For the experiment shown in Fig. 4*B*, in 14 alternating trials

Table 1. Food-stimulated depolarization in Int-2s of control and conditioned animals

Condition	Maximum amplitude, mV	Duration, sec	Area, mV·sec
Control (n = 10)	8.5 ± 3.0	10.9 ± 1.3	281.9 ± 17.8
Trained (n = 7)	9.9 ± 2.4	19.4 ± 1.7*	516.8 ± 21.3†

Animals were tested with 1.5 ml of SH pipetted onto the oral veil; response measurements were averaged from 22 trials on 16 cells in 10 control animals, and from 17 trials on 10 cells in 7 experimental animals. Both the duration and magnitude (duration × amplitude) of the averaged depolarization (±SEM) were significantly greater in conditioned animals (one-way analysis of variance). Significant difference in maximum amplitudes was undetected. *, $P < 0.01$; †, $P < 0.001$ (Scheffe test).

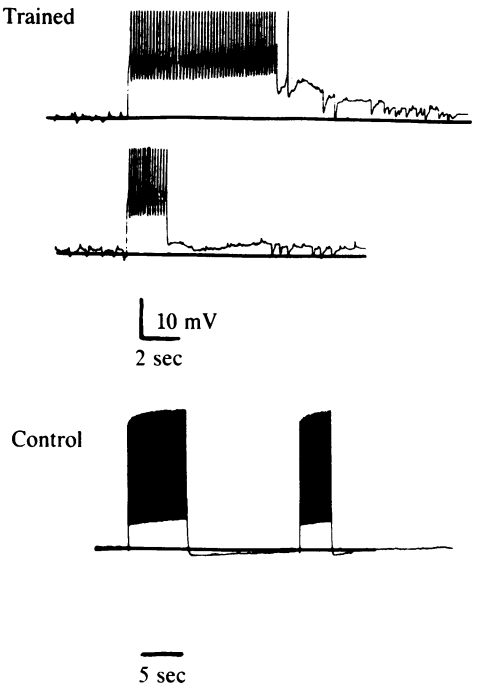

FIG. 3. Int-2 responses to depolarizing current injection (bars). Depolarization and spiking of Int-2s in a trained animal are followed by a prolonged and slowly decaying depolarizing afterpotential; the upper two records show graded responses to depolarizing current injection of long and short duration. Such prolonged afterpotentials are entirely absent after stimulation of the Int-2s of untrained control animals, which instead show small hyperpolarizations (bottom record). The duration of depolarizing current injection is indicated by the periods of rapid spiking. Reference lines are superimposed on voltage baselines.

unrestrained Int-2 activity caused nearly 1.5 times greater hyperpolarization of the PCN than when the Int-2 activity was suppressed ($P < 0.005$, one-tailed t test). It may be noted that hyperpolarization of a single Int-2 will have inhibitory effects within the entire Int-2 population. Ipsilateral Int-2s are closely coupled by nonrectifying electrical connections, while contralateral Int-2s are mutually excitatory via chemical synapses (Fig. 1) (9, 11, 12). Thus, negative current injected into a single Int-2 will hyperpolarize the ipsilateral population and reduce positive feedback between the bilateral populations.

From the results of the experiments on hyperpolarization of single interneurons, it may be presumed that the full populations are proportionately more effective. These results indicate that activity in both the Int-1 and Int-2 populations is a major source of the learning-contingent and food-induced synaptic inhibition of the PCNs. Since the Int-2s have many potent synaptic outputs within the feeding motor network of the brain ganglion, the effects of their heightened activity must be similarly widespread.

DISCUSSION

It was previously shown that the Int-2 neuron population has the ability to suppress feeding behavior, and it was suggested that they might act in this capacity in food-avoidance-conditioned and satiated animals (12). The present evidence supports the interpretation that the Int-2s are indeed loci where the initiation of feeding is regulated. The data have shown the following: (*i*) Int-2 activity in food-avoidance-conditioned animals is altered toward that known to suppress the cyclic motor output of the network; (*ii*) the intensified and prolonged activity of the Int-2s is explainable in whole or in part by an observed enhancement of excitability, which is

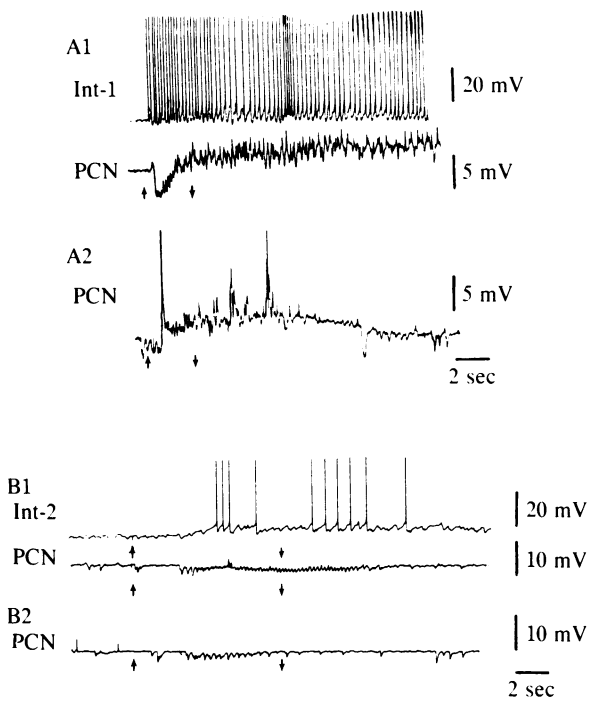

FIG. 4. Individual Int-1s and Int-2s contribute significantly to food-induced inhibition in PCNs of conditioned animals. Simultaneous intracellular records. (*A1*) Food stimulation of the animal's oral veil with squid homogenate (arrows) stimulates spiking in an Int-1 and concurrent inhibitory postsynaptic potentials in a PCN. (*A2*) Tonic hyperpolarization of the Int-1 by >20 mV to prevent spiking resulted in significantly less PCN inhibition, particularly evident in the initial portion of the record. The hyperpolarized trace is off the record. The response of the PCN in the 10 sec after the beginning of food stimulation, represented as the area between the voltage trace and the voltage baseline of pre-food stimulation, was averaged over three trials at 115.5 ± 2.8 mV·sec when the Int-1 was active, versus 214.6 ± 9.6 mV·sec when the Int-1 was hyperpolarized ($P < 0.001$; one-tailed t test). (*B*) Greater inhibition occurred when an Int-2 was allowed to fire normally (*B1*) in 14 trials (−280.2 ± 5.4 mV·sec) than when the Int-2 neuron was hyperpolarized (*B2*; −150.5 ± 3.8 mV·sec; $P < 0.005$).

either intrinsic to the Int-2s or to local positive feedback loops; and (*iii*) the Int-2s contribute significantly to neural activity accompanying suppression of feeding behavior (inhibition of the PCNs).

A Model for Regulation of Feeding Behavior by the Int-2s. Present knowledge of the Int-2s allows formulation of a simple model of their action in regulating behavior in *Pleurobranchaea*. Simply, in conditioned animals, the enhanced excitability and sustained activity of the Int-2s acts to halt the rhythmic central pattern generator (CPG) of feeding behavior in the retraction phase of its cycle.

In the model pictured in Fig. 5, the oscillatory CPG is depicted as a simple half-cell (17) comprising two major classes of neurons active in antiphase during the feeding cycle, labeled P (protractor) and R (retractor) (18). The capacity of the Int-2s for experience-dependent change in their excitability is depicted as a positive feedback loop with variable gain.

The Int-2s themselves are part of the central pattern generator (12). During normal feeding behavior, the Int-2s fire cyclically; they are driven in phase with the retraction movement of the buccal mass by alternating synaptic excitation and inhibition from (as yet unidentified) retractor and protractor neurons, respectively. The Int-2s have extensive and potent excitatory outputs among retractor neurons and they cause inhibition of protractors; tonic Int-2 activity drives sustained activity in retractor units of the brain (12). The

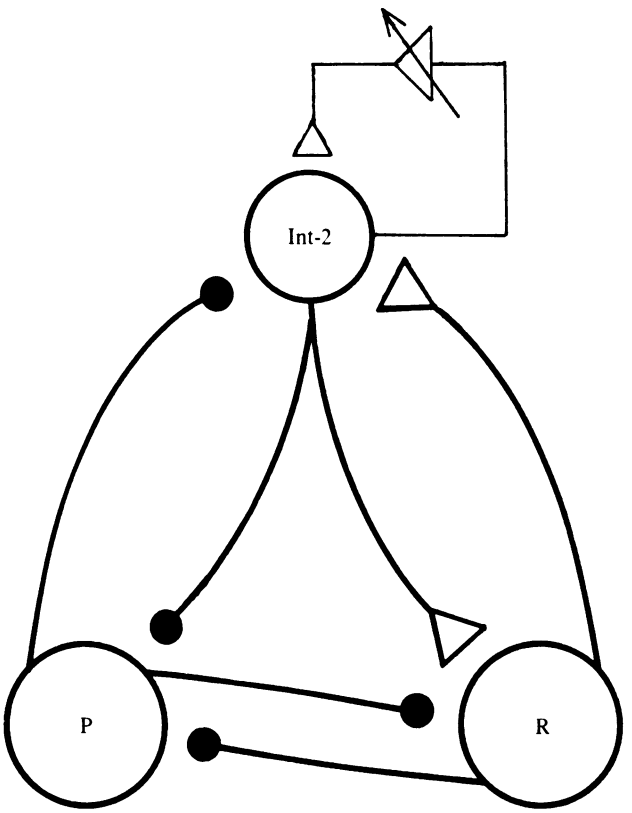

FIG. 5. A circuitry model of the role of the Int-2s in regulating feeding behavior, based on variable gain in a positive feedback loop. Synaptic relations are not necessarily monosynaptic. △, Excitatory connections; ●, inhibitory connections. At low gain (as in a hungry naive animal), the Int-2s fire cyclically during feeding, entrained by alternating excitatory inputs (△) from the retractor neuron population (R) of the oscillator network and by inhibitory inputs (●) from protractors (P). At high gain (food-avoidance-conditioned animal) the Int-2s fire tonically, causing tonic excitation of retractor neurons and like inhibition of protractors. Thus, in learned suppression of feeding, the oscillator is biased in some phase of retraction.

result is that when the Int-2s fire tonically, they arrest the oscillator and thus suppress the expression of feeding behavior (12). Tonic activity of the more excitable Int-2s of conditioned animals must be driven in part by direct chemosensory excitation from the food-sensing oral veil (12).

The physical basis of the positive feedback loop regulating Int-2 activity could be (*i*) a voltage-dependent property intrinsic to the Int-2s, such as the slow depolarizing current regulated by cyclic AMP in other *Pleurobranchaea* neurons (19); (*ii*) enhanced chemical or electrical coupling among the Int-2 neuron population; or (*iii*) enhanced excitatory coupling between the Int-2 neurons and other unidentified retractor neurons in the brain ganglion. The mechanism remains to be described, as does the nature of the presumed heterosynaptic pathway mediating the effects of noxious shock in the learned behavior.

The model shows division of the oscillator into only two phases of action, a simplification. The P and R populations divide the many and various components of the motor network, including the PCNs (elements of the P population) and the MCGs and Int-1s (R population). The P and R populations are shown coupled in a mutually inhibitory relation, incorporating prior observations on connectivity in the feeding network (8, 14, 16, 17). In fact, the retraction phase is probably further divisible (unpublished observations), rather like the feeding oscillator of the pulmonate snail *Lymnaea* (20). Thus, the Int-2s may drive a specific subpop-

ulation of retractors whose identities remain to be more fully described (ref. 12; unpublished observations).

Learning Mechanisms in CPGs: Significance. For virtually all rhythmic and complex behaviors, the initial output parameters of timing and intensity emerge from the properties of hard-wired CPGs (21). CPG output is subject to modulation on a moment-to-moment basis by sensory feedback. Our results constitute documentation of a learning mechanism localized within a CPG for a rhythmic behavior. Other loci of associative learning mechanisms have been previously documented at the level of primary sensory neurons in mollusks: in mechanoreceptors effecting reflex gill withdrawal in *Aplysia* (15, 22) and in photoreceptors controlling phototaxis in *Hermissenda* (23). In contrast, the Int-2s are purely interneurons: they form part of the CPG for feeding behavior, and they integrate inputs from both sensory and motor sources (12). The localization of a mechanism for learning in a CPG rather than in sensory or other pathways is likely to have special significance for the organization of behavior.

One important consequence is that the location of a learning mechanism within the CPG can permit retention of behavioral arousal while overt feeding is suppressed. While feeding behavior is suppressed by being locked in the retraction phase, excitatory sensory inputs to the oscillator are still active and the attendant neuromodulatory activity remains. For instance, the serotonergic metacerebral giant neurons have been shown to be tonically excited by food stimuli in conditioned animals during suppression of feeding (6), driven by tonic Int-2 activity (12). The metacerebral neurons act to augment the excitatory state of the network in *Pleurobranchaea* (16) and may act as important neuromodulators of the aroused feeding state as they do in *Aplysia* (24). Thus, a potential for releasing the behavior in a fully aroused state remains.

A second consequence of the location of a mechanism for food-avoidance learning within the CPG is that sensory pathways are left available for stimulation of alternative behaviors. An example of this is the active avoidance behavior often released during suppression of feeding in *Pleurobranchaea* (2, 6). Possibly, there are other sites for learning mechanisms within sensory or other pathways afferent to the feeding motor network; these also might act to confer other special qualities to learned behavior, such as selective avoidance to different food stimuli.

Finally, a testable prediction of the model is that learning-induced alteration of function in a CPG component ought to cause alteration of any CPG function involving that component. In addition to feeding, other rhythmic behaviors are produced by the same CPG in *Pleurobranchaea*, acting in a different state of coordination, including rejection of material from the buccal cavity (12, 25). The potential consequences to other behaviors from learning-induced changes in Int-2 function have yet to be measured.

Conclusion. The Int-2s lie within the central pattern generator for feeding behavior where their excitability is regulated by associative food-avoidance learning. The resulting change causes suppression of feeding by biasing the oscillatory CPG toward one phase of its normal cycle. The CPG site of learning has unique and adaptive consequences for the organization of behavior, among them the preservation of the physiological state of arousal during suppression of the feeding behavior and release of an alternative avoidance behavior driven by food stimuli.

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