could generate the *trans*-4-keto-β-ionone found by Albone in the supracaudal gland. Isoe, Hyeon, and Sakan (*22, 23*) have shown that β-ionone and dihydroactinidiolide can be formed in a photochemically induced reaction of β-carotene with oxygen (*24*).

An interesting hypothesis could be made of the possible biogenesis of Δ^3-isopentenyl methyl sulfide (**1**) from isopentenyl pyrophosphate and methionine via a *S*-methylsulfonium intermediate. Thus, Δ^3-isopentenyl pyrophosphate (*25*), the first essential reactive isoprenoid intermediate in the long chain of terpene biogenesis leading to sterols and steroid hormones, could be the source of a powerful and fairly persistent olfactory marker, **1**, present in concentrations that could signal an increase of steroid hormone synthesis in preparation for the mating season in the fox. Alternatively, it could be that the steroid requirements have been met and that precursors are available to be diverted to other substances to advertise that fact.

It remains to be determined whether the concentrations of these volatile components (**1** to **4**) of the red fox urines undergo individual and seasonal changes that correlate with the already reported endocrine and behavioral changes characteristic of the mating period. The availability of synthetic mixtures simulating the natural ratios of these known volatiles will also make possible controlled behavioral studies with foxes in their natural habitat.

There is a close chemical relation between the thio ether **1** and mustelan (2,2-dimethylthietane), the malodorous substance from the anal gland of the mink (*Mustela vison*) and the polecat (*Mustela putorius*), and its companion compounds, 3,3-dimethyl-1,2-dithiolane and diisopentyl disulfide (*26*), all of apparent isoprenoid origin. A component (3-methyl-1-butanethiol) of the scent of the striped skunk (*Mephitis mephitis*) contains also (*27*) a putative isoprene unit, although in this case the presence of unbranched thiols and disulfides might suggest other than terpene origin.

The presence of 2-methylquinoline (quinaldine) in male red fox urine, but not detectable in female urine, is an interesting sex-related difference. Albone (*7*) refers without citation to an old report of the occurrence of 2-methylquinoline in skunk scent. This quinoline derivative could have originated from tryptophan or other indole derivative by way of a ring-enlargement, a reaction with well-known biochemical precedent.

The red fox, considered the least social of the wild canids (*1*), depends more than other canid species on olfactory means of communication. Whether any of the compounds reported above may have pheromonal activities is not yet known.

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Command Neurons in *Pleurobranchaea* Receive Synaptic Feedback from the Motor Network They Excite

Abstract. *Command neurons that cause rhythmic feeding behavior in the marine mollusc* Pleurobranchaea californica *have been identified in the cerebropleural ganglion (brain). Intracellular stimulation of single command neurons in isolated nervous systems, semi-intact preparations, and restrained whole animals causes the same rhythmic motor output pattern as occurs during feeding. During this motor output pattern, action potentials recorded intracellularly from the command neurons occur in cyclic bursts that are phase-locked with the feeding rhythm. This modulation results from repetitive, alternating bursts of excitatory and inhibitory postsynaptic potentials, which are caused at least in part by synaptic feedback to the command neurons from identified classes of neurons in the feeding network. Central feedback to command neurons from the motor network they excite provides a possible general physiological mechanism for the sustained oscillation of neural networks controlling cyclic behavior.*

Command neurons are single nerve cells that elicit a recognizable motor component of behavior (*1*). After they were described by Wiersma (*2*), command neurons were studied in several animal groups, including crustaceans (*3*), insects (*4*), molluscs (*5*), and mammals (*6*). On the basis of motor responses to electrical stimulation, these cells have been presumed to serve as simple, one-way relay pathways that convey instructions for specific movements from higher to lower motor "centers" (*6, 7*). This hypothesis has not been tested, however, owing to the paucity of studies involving recordings from command

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neurons. Here we describe a population of command neurons that causes cyclic feeding behavior in the mollusc *Pleurobranchaea californica*. By means of intracellular stimulation and recording we have demonstrated that these command neurons receive synaptic feedback from neurons in the motor network they excite. Our study shows that in this motor system, at least, command neurons not only activate the motor output pattern, but are also incorporated into the neuronal network that generates the pattern.

Experiments were performed on *P. californica*. Isolated nervous system preparations consisted of the brain (cerebropleural ganglion) and buccal ganglion, connected by the two cerebrobuccal connectives. Nervous systems were removed from medium sized specimens (volume, 100 to 200 ml) as described (*8*) and pinned on Sylgard in filtered seawater or filtered *Pleurobranchaea* blood. Semi-intact preparations consisted of the intact central nervous system connected to the oral veil and buccal mass. Whole-animal preparations were made by exposing the brain and suspending the animal in seawater by hooks in the margins of the incision (*9*). The brain was lifted and stabilized with small pins on a micromanipulated wax platform. Extracellular recordings from nerves that mediate feeding (*8*) were made with glass capillary suction electrodes. Intracellular stimulation and recording was accomplished by means of glass capillary microelectrodes filled with 3*M* KCl or 2*M* potassium acetate (tip resistance, ~10 megohms).

The somata of putative command neurons in the brain were identified previously by the retrograde transport of cobaltous chloride taken up by the descending axons in the cerebrobuccal connectives (*10*). Three discrete populations of somata were identified by this method (Fig. 1, top): the paired metacerebral giant neurons (*11*), the paracerebral neurons, and the opisthocerebral neurons. In this report we describe the paracerebral neurons.

The data in Fig. 1A are typical of those obtained from 25 paracerebral neurons in 12 isolated nervous system preparations. Prior to stimulation of the impaled soma, the neural network that generates the feeding rhythm was inactive, as signified by the absence of cyclic bursts of efferent action potentials in feeding nerves (trace 1 of Fig. 1A). Injection of steady (d-c) current into the soma through a bridge circuit caused action potentials in the impaled paracerebral neuron that were followed in 1.5 seconds by the onset of the same rhythm as occurs during feeding in nerves of the brain (*sovn*) and buccal ganglion (*r3*) (trace 2 of Fig. 1A). Rhythmic, coordinated output of this type occurred for as long as the paracerebral cell was depolarized (3 minutes) and terminated immediately on cessation of stimulation (trace 4 of Fig. 1A). In several hungry, untrained whole-animal preparations, every paracerebral neuron studied was depolarized by application of food stimuli to the oral veil, and most such neurons discharged action potentials that were accompanied by visible feeding movements (Fig. 1C). Intra-

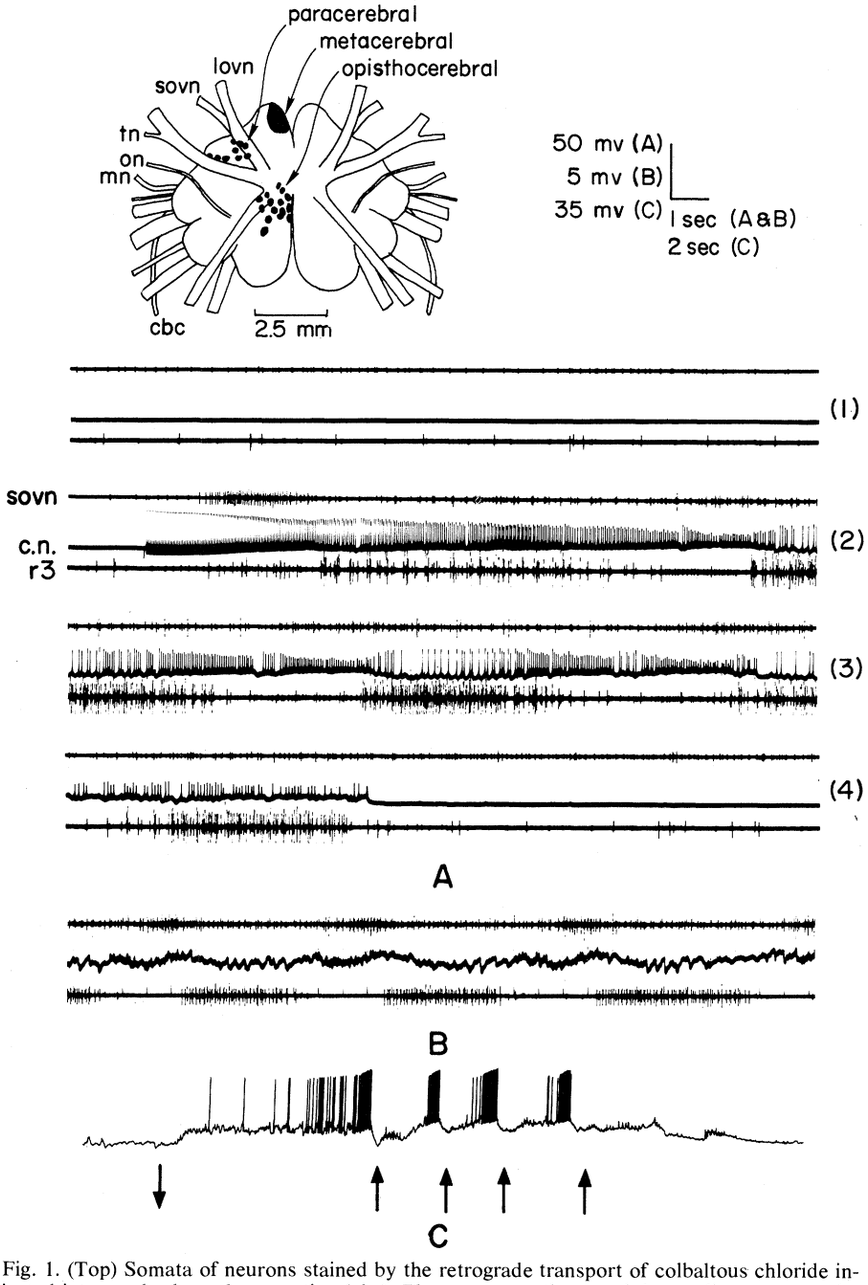

Fig. 1. (Top) Somata of neurons stained by the retrograde transport of colbaltous chloride injected into cerebrobuccal connective (*cbc*). These neurons thus send descending axons to the buccal ganglion (*10*). Stimulation (A) and recording (B and C) from a paracerebral feeding command neuron of *Pleurobranchaea*. (A) The effect of intracellular stimulation of one paracerebral command neuron (*c.n.*) in an isolated nervous system. The feeding rhythm is monitored by recording extracellularly from the small oral veil nerve (*sovn*) of the brain (see inset) and the third nerve root of the buccal ganglion (*r3*). The sovn normally discharges during proboscis eversion, while r3 fires during proboscis withdrawal (*8*). Traces 1 to 3 are continuous, whereas 2.5 minutes of continual stimulation of the paracerebral neuron separate traces 3 and 4. (B) Intracellular recording from the same paracerebral neuron that was stimulated in (A) during feeding output generated by tonic extracellular stimulation of both stomatogastric nerves. Note the EPSP's during sovn activity and IPSP's during r3 bursts. Traces arranged as in (A). (C) Intracellular recording from a single paracerebral neuron in a whole-animal preparation during application of food stimuli (squid extract) to the oral veil, beginning at the first downward arrow. Subsequent upward arrows designate occurrence of vigorous bites. Additional abbreviations: *lovn*, large oral veil nerve; *tn*, tentacle nerve; *on*, optic nerve; *mn*, mouth nerve.

cellular stimulation of more than 50 individual paracerebral neurons in 24 whole-animal preparations at physiological discharge frequencies caused visible, rhythmic feeding movements in most cases. Thus, the paracerebral neurons exhibit every major property expected of command neurons for feeding behavior. Neither dye injection nor physiological mapping revealed axonal branches of the paracerebral neurons in anterior nerves of the brain or in nerves of the buccal ganglion (*12*). Thus the paracerebral neurons may be true interneurons.

Once the feeding rhythm commenced, spike activity in a paracerebral neuron became cyclic and phase-locked with the feeding rhythm, both in isolated nervous systems (Fig. 1A) and in behaving, whole-animal preparations (Fig. 1C). Paracerebral neurons discharge normally during eversion; their burst typically is followed immediately by a visible bite (Fig. 1C) and proboscis withdrawal. When a paracerebral neuron was hyperpolarized to prevent spiking during the feeding rhythm, cyclic oscillation of the membrane potential continued, caused by alternating barrages of excitatory postsynaptic potentials (EPSP's) during the proboscis eversion phase, and inhibitory postsynaptic potentials (IPSP's) during the proboscis withdrawal phase (Fig. 1B).

To determine the possible sources of these EPSP's and IPSP's, we analyzed synaptic interactions between the paracerebral neurons and other classes of neurons that mediate feeding by direct intracellular stimulation and recording from somata in quiescent preparations. The paracerebral neurons interact with at least two classes of such neurons in the buccal ganglion: the corollary discharge (CD) and anterior ventral (AV) neurons (*13*). The AV and CD neurons are organized into two functionally antagonistic populations: those that are active during eversion, and those active during withdrawal. Paracerebral neurons connect with eversion AV and CD neurons in an excitatory loop; that is, paracerebral neurons excite AV neurons (Fig. 2A, part 1), which in turn excite ascending CD neurons that cause EPSP's in the paracerebral neuron (Fig. 2A, part 2). Thus the feeding command neurons are functionally connected to other members of the motor network by means of a positive feedback loop (Fig. 2A, part 3) that can, in principle, account for the observed EPSP's in paracerebral neurons during proboscis eversion (Fig. 1B).

Likewise, the paracerebral neurons connect with withdrawal CD and AV neurons in a disinhibitory feedback loop involving two inhibitory synapses connected in series. That is, paracerebral neurons inhibit withdrawal AV neurons (Fig. 2B, part 1). These same AV neurons excite ascending CD neurons, which in turn induce a long-latency and, therefore, presumably polysynaptic inhibition of the paracerebral neurons (Fig. 2B, part 2). Such a feedback loop (Fig. 2B, part 3) would be expected to protect paracerebral neurons from inhibition during eversion, but to permit IPSP's to occur during withdrawal, as observed (Fig. 1B).

These findings have several possible implications for the neuronal control of movement (*7*). Perhaps most important in the context of cyclic behavior, recip-

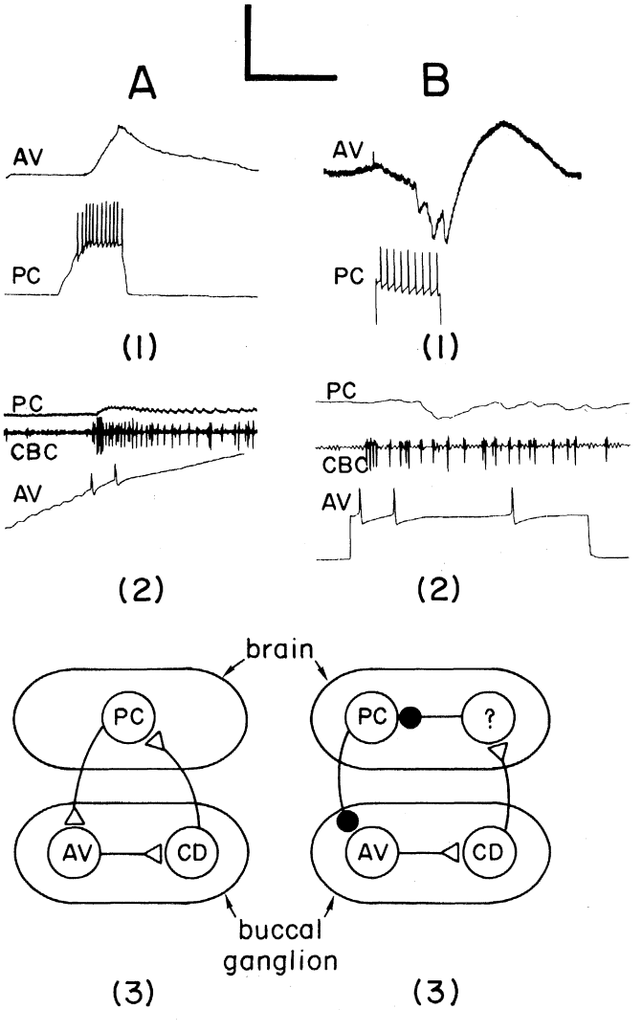

Fig. 2. Synaptic connections between single paracerebral command (*PC*) neurons and other feeding neurons. (A) Reciprocal excitation between a PC neuron and other feeding neurons that are active during the eversion phase of feeding. (B) Reciprocal (polysynaptic) inhibition between a PC neuron and other feeding neurons that are active during the withdrawal phase of feeding. In part 1 of (A), a single PC neuron is depolarized by intracellular current injection. The resulting action potentials cause smoothly summating EPSP's in a feeding neuron of the buccal ganglion [an "anterior ventral," or AV neuron; (*12*)]. Part 2 of (A) illustrates that intracellular stimulation of the same AV neuron excites several ascending neurons recorded extracellularly in the ipsilateral cerebrobuccal connective [the "corollary discharge," or CD, neurons (*10*)], one of which causes apparently unitary EPSP's in the PC neuron. AV neurons reliably exhibited the "triggering" effect in all preparations examined. Part 3 in (A) shows a hypothetical synaptic circuit capable of explaining the data in parts 1 and 2 of (A). In part 1 of (B), intracellular stimulation of a PC neuron causes IPSP's in a different AV neuron. Intracellular stimulation of the same AV neuron, shown in part 2 of (B), activates ascending CD neurons in the cerebrobuccal connective (*CBC*) which initiates long latency (presumably polysynaptic) inhibition of the same PC neuron as in part 1 of (B). The hypothetical circuit in part 3 of (B) could account for data in B(1) and B(2). Calibration mark (top): (A) part 1, vertical, 10 mV (top trace), 100 mV (bottom trace); horizontal, 1 second; (A) part 2, vertical, 10 mV (top trace), 200 mV (bottom trace); horizontal, 1 second; (B) part 1, vertical, 2 mV (top trace), 200 mV (bottom trace); horizontal, 1 second; (B) part 2, vertical, 20 mV (top trace), 200 mV (bottom trace); horizontal, 0.4 second. In parts 3 of (A) and (B), △ = excitation, • = inhibition.

rocal feedback connections between command neurons and other members of a motor network provide a possible means for self-supporting oscillation of the network (*14*). Such self-reinforcement by positive feedback can, in principle, help sustain a motor pattern beyond the initiating stimulus, providing one plausible neurophysiological explanation for the ethological concept of "triggering" (*15*), whereby a behavioral sequence long outlasts the initiating stimulus. Our data also imply that the activity of a central motor network can, in principle, be initiated at many loci. Direct excitation of the network mediating feeding in *Pleurobranchaea* can occur by stimulation of other neurons that are reciprocally connected to the command neurons (*16*). Owing to such reciprocal interconnections within a motor network, specialization of a given population of neurons for the command role may derive at least in part from privileged access to the central or sensory inputs that control the corresponding behavior. That is, command neurons may be differentiated from other members of a motor network not only on the basis of output effects, but also on the basis of input organization.

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Neural Correlate of Behavioral Plasticity in Command Neurons of *Pleurobranchaea*

Abstract. *Food stimuli normally excite the command neurons of* Pleurobranchaea *that cause feeding. In contrast, the same food stimuli selectively inhibit these neurons in specimens that have been trained to suppress feeding and withdraw from food by means of an avoidance conditioning paradigm consisting of paired food and conditional shock. Food stimuli excite the feeding command neurons of yoked control specimens exposed to unpaired food and shock, but inhibit the feeding command neurons of untrained specimens that have been satiated with food. These results suggest that the command neurons serve as a neural locus at which an animal's behavior is modulated by past experiences. These results also establish a neural correlate of behavioral plasticity, in the form of synaptic inhibition of the command neurons.*

Behavioral plasticity may be defined broadly as a modification of behavior that is acquired because of experience (*1*) and includes such phenomena as habituation, sensitization, and learning. One approach to understanding the neurophysiological mechanisms of behavioral plasticity is to analyze the neuronal circuitry that mediates a modifiable behavioral act, and then to examine this circuitry for physiological changes that accompany the behavioral modification (*2*). The feeding behavior of the mollusc *Pleurobranchaea californica* is amenable to this approach; the behavior (*3*) can be modified by classical (*4*) and avoidance (*5*) conditioning, as well as food satiation (*6*), and the neuronal circuitry controlling feeding is known in some detail (*7–9*). In particular, command neurons for feeding, termed paracerebral neurons (*8*), have been identified in two bilaterally symmetrical groups in the cerebropleural ganglion (brain). Intracellular stimulation of single command neurons in whole-animal preparations causes rhythmic, coordinated biting and swallowing that is indistinguishable from normal feeding (*8*).

As reported (*8*), paracerebral command neurons in hungry, untrained specimens are excited by application of food stimuli to the chemosensory oral veil, and such natural excitation of the command neurons is usually followed immediately by overt feeding. We report here that when *Pleurobranchaea* is trained in a conventional avoidance conditioning paradigm (*5*) to suppress feeding (passive avoidance conditioning) and withdraw from a normally palatable food substance (active avoidance conditioning), application of this food to the oral veil inhibits rather than excites the feeding command neurons. Likewise, when specimens are fed to satiety, the feeding command neurons are inhibited rather than excited by food stimuli. Our study therefore shows that in *Pleurobranchaea*, modulation of behavior by experience is accompanied and presumably caused by a corresponding modulation of the activity of command neurons that control the behavior.

Specimens of *P. californica* (volume, 50 to 350 ml) were purchased from Pacific Bio Marine (Venice, California), matched in pairs on the basis of size (± 10 percent), and divided into experimental and control groups by a random procedure (coin flip). Experimental animals were conditioned in seawater (12° to 15°C) in 10 to 18 hourly trials as described (*5*). In each trial, a liquid homog-

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