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SOURCES OF MAGNETIC SENSORY INPUT TO IDENTIFIED NEURONS ACTIVE DURING CRAWLING IN THE MARINE MOLLUSC *TRITONIA DIOMEDEA*

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Summary

Although the nudibranch mollusc *Tritonia diomedea* orients to the geomagnetic field, the anatomical site and the mechanism of the geomagnetic transducer are not known. Previous work on semi-intact preparations of *Tritonia diomedea* in which the brain is intact and nerve connections to the periphery are maintained showed that identifiable pedal ganglion neurons Pd5 fired an increased number of action potentials when the horizontal component of the ambient magnetic field was rotated. This response disappeared when all nerves emerging from the brain were cut, suggesting a peripheral locus for the geomagnetic transducer.

In the present work, we recorded intracellularly from Pd5 in preparations in which all peripheral nerves were cut except those containing the axons of neurons Pd5 (pedal nerves 2 and 3). These uncut, mixed, sensory-motor trunks innervate the locomotory epithelium of the foot upon which the animal crawls. In this further-reduced preparation, Pd5 again responded to magnetic field rotations with action potentials. To determine the direction of this action potential transmission in response to magnetic field rotations, we analyzed extracellular recordings from nerves containing the Pd5 axons and found that action potentials elicited in Pd5 by magnetic stimuli originate centrally and are transmitted peripherally.

In addition, we have explored the behavioral function of Pd5 neurons by simultaneously recording intracellular electrical activity and crawling rate of the semi-intact animal. A significant correlation was found between crawling rate and Pd5 action potential rate. We also found that action potentials in dorsal swim interneurons depolarized both Pd5 and the established locomotion motoneuron Pd21.

Key words: *Tritonia diomedea*, geomagnetic field, magnetoreceptor, mollusc, pedal neurone, treadmill, pedal peptide.

Introduction

Behavioral evidence for the use of the geomagnetic field in orientation and navigation exists for organisms ranging from bacteria to mammals (for a review, see Wiltschko and Wiltschko, 1995). Moreover, effects of magnetic fields on physiological processes have been reported at levels of organization such as biochemical reactions (Hendee et al., 1996), ionic channels (Smith et al., 1987), gene expression (Goodman et al., 1987; Phillips, 1993), single cells (Blakemore, 1975; Phillips, 1987) and brain nuclei (Demaine and Semm, 1985; Walker et al., 1997). Sensory transduction mechanisms based on ferromagnetic materials (Lowenstam, 1962; Kirschvink and Gould, 1981; Schiff, 1991; Hsu and Li, 1994), electromagnetic induction (Kalmijn, 1978), the modulation of biochemical reactions (Leask, 1977; Schulten and Windemuth, 1986) and ion channel cyclotron resonance (Liboff et al., 1987) have been proposed. In contrast with the magnetic orientation of bacteria, however, the ‘elusive quantity called mechanism’ has not yet been defined for the transduction of magnetic field information to neuronal activity in any species (Phillips, 1993).

The nudibranch mollusc *Tritonia diomedea* is a model for the investigation of behavioral and neural responses to magnetic fields. Open-field body axis orientation and Y-maze navigation in *Tritonia diomedea* are modulated by the natural magnetic field (Lohmann and Willows, 1987). Furthermore, the nervous system of the animal is accessible to neurophysiological investigation (Willows, 1967). Intracellular recordings from semi-intact preparations (all major nerves intact) indicate that identifiable neurons (Pd5) from the pedal ganglia fire action potentials with increased frequency when the horizontal component of the ambient magnetic field is rotated. Interestingly, this response is observed in semi-intact preparations, where the brain is *in situ*, but cannot be elicited when the brain is isolated (Lohmann et al., 1991; I. R. Popescu and A. O. D. Willows, unpublished observations). These two electrophysiological observations suggest a peripheral location for magnetosensory neurons.

To investigate the localization of these receptor cells, we cut all nerves emerging from the brain except pedal nerves 2 and 3 (the further-reduced preparation) and exposed the animals to

the magnetic field stimulus used by Lohmann et al. (1991). In the further-reduced preparation, Pd5 neurons responded to magnetic field rotations as they did in the semi-intact preparation, suggesting that pedal nerves 2 and 3 innervate or contain peripheral magnetosensory structures, among other possibilities.

To distinguish between Pd5 peripheral sensory and motor roles, we determined the direction of action potential transmission in its major processes. Extracellular nerve recordings revealed that Pd5 action potentials were conducted away from the brain and towards the periphery in all cases.

We also investigated the locomotory functions of the Pd5 neurons. These neurons are excited, either directly or indirectly, by magnetic field stimuli which are used for orientation and navigation, and so we made intracellular measurements of their activity during crawling on a treadmill. There was a significant correlation between action potential firing frequency and the rate of treadmill turning. Support for the relevance of these results was provided by a pilot experiment in which it was found that Pd5 action potential frequency correlated with the rate of particle transport by the locomotory cilia of the foot. A correlation was also observed between trains of spikes elicited by intracellular stimulation in single Pd5 neurons and increased crawling rate on the treadmill.

It has been previously reported that the serotonergic pedal cell Pd21 of *Tritonia diomedea* is a crawling motoneuron (Audesirk, 1978). Here, we have shown that action potentials in the dorsal swim interneurons (DSIs) cause depolarization of both Pd21 and the peptidergic pedal neuron Pd5 (Lloyd et al., 1996). DSIs fire action potentials with increased frequency after the animal has concluded a swimming episode, at a time when fast crawling occurs (Getting et al., 1980; Katz et al., 1994).

Materials and methods

The semi-intact preparation

Tritonia diomedea (Bergh) were obtained by trawling in Bellingham Bay, Washington State. They remained at the University of Washington, Friday Harbor Laboratories, in running seawater aquaria for at least 48 h prior to experiments. The semi-intact preparation has been described in detail by Willows et al. (1973). For experiments involving magnetic fields, the brain was pinned to the support platform using non-magnetic cactus needles instead of minuten pins. At the end of the dissection, the nerves emerging from the central ganglia were cut as required by the experimental protocols.

Animals used for measurement of glass bead transport on the foot were dissected similarly, but from the ventral side and after cooling for 1 h in a small seawater bath over ice. The animals were then pinned on a submerged wax platform, foot upwards. After the initial incision, the buccal ganglia were removed, and the buccal mass was cut at the esophagus and pulled anteriorly. The cerebral nerves were cut, and the brain was rolled to expose its dorsal side (the Pd5 side). Care was taken to avoid cutting the pedal nerves and commissures or twisting them excessively. Intracellular recordings were made using glass microelectrodes (resistance 10–40 MΩ) filled with 3 mol l^{-1} KCl.

Magnetic field measurements and manipulations

The magnetic field intensity in the laboratory was 16 000–18 000 nT in the horizontal plane and 43 000–46 000 nT in the vertical plane when measured with a single-axis magnetometer (Schonstedt Instruments, Virginia, USA). The field vectors were altered using 2 sets of square Rubens coils (Rubens, 1945) 182 cm long built by Dr K. J. Lohmann (University of North Carolina). The animal preparation was placed at the center of the cube formed by the coils. During a magnetic field trial, the animal was exposed for 1 min to a magnetic field whose horizontal vector was rotated 60 ° clockwise, then for 1 min to the natural field. This was repeated for 30 min. The rotation of the field was accomplished in 1 s. When rotated, the magnitude of the horizontal magnetic vector was ±10 % of the natural field, while the vertical magnetic vector remained unchanged. During control trials, the ambient field was unaltered. According to the recording protocol, impalement with the microelectrode was followed by a 1 h rest period before monitoring the activity of the neuron for a suitable baseline. A baseline was defined as 45 spikes or fewer over a 15 min period. Baseline measurements were followed either by a magnetic field trial or a control trial, as determined by flipping a coin. After another 1 h rest period, a new baseline was sought. This second baseline measurement was followed by a magnetic field trial if the first baseline measurement had been followed by a control trial, and *vice versa*. For each animal, we obtained both a control and a magnetic field trial using the protocol outlined during a single, continuous intracellular recording.

Treadmills

Treadmills (Fig. 1) were made from plastic cylinders (3 cm diameter) wrapped in 210 μm Nitex mesh. The discs at the ends of the cylinders were divided radially into 90 (4 °) alternating black or white sectors. The image of these disks was transferred by video camera to a monitor. A light sensor (either Fotonic Sensor KD-38 from MTI Instruments, MA, USA, or a similar model built in the laboratory) was trained at the image of the disc on the monitor. Direct current signals were generated by the motion detector as black or white sectors swept past. This signal was penned on one channel of the chart recorder, and the Pd5 membrane voltage on another. Subsequently, the number of action potentials and treadmill sectors turned during each consecutive minute were determined from the chart record.

Identification and impalement of dorsal swim interneurons and Pd21 neurons

In semi-intact preparations, dorsal swim interneurons (DSIs) were identified by the their location in the immediate rostral vicinity of the orange-pigmented cell field at the

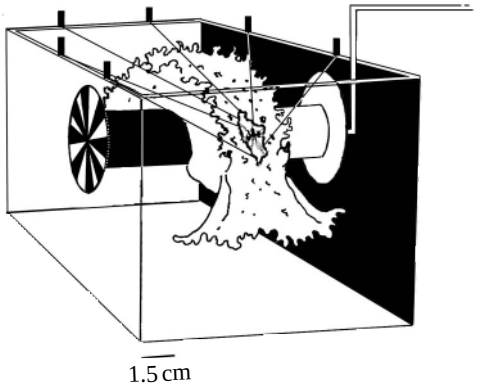

Fig. 1. A semi-intact preparation of *Tritonia diomedea* on the treadmill. The rotation of the cylinder is recorded with the help of a motion detector (not shown). The slug is held in place by hooks set in the margins of the incision exposing the brain and connected by threads to pegs on the rim of the aquarium.

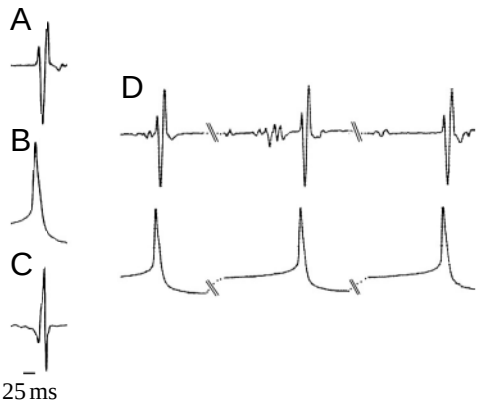

Fig. 2. The direction of Pd5 spike propagation as determined by extracellular nerve recordings. Spikes recorded and elicited with an intracellular electrode in the soma (B) were also recorded by two extracellular wire electrodes placed under pedal nerve 3 (A,C). In A, the negative extracellular electrode was closer to the soma and, therefore, recorded the action potential before the positive electrode. In C, the positive extracellular electrode was closer to the soma. Knowing that these action potentials originated in the soma, we were able to determine the shape of the extracellular recording when either the positive or negative electrode recorded the action potential first. Subsequently, by knowing the placement order of the extracellular electrodes, we could determine the direction of action potential propagation. (D) Three consecutive Pd5 spikes fired during magnetic field stimulation recorded both intracellularly (lower recording) and extracellularly (upper recording). A comparison of the polarity of the extracellular spikes with the polarity of the calibration examples in A and C shows that they were recorded by the proximal (negative) electrode first (as in A).

cerebral/pleural ganglion junction and near the visible white axon tract leading to cerebral nerve 1. The DSIs were approximately 50 μm in diameter, translucent and showed intermittent, spontaneous, non-bursting action potential electrical activity when the slugs were not swimming. In isolated brain preparations, DSIs were identified using these criteria and by the phase of their action potential bursts during fictive swimming elicited by a 2 s (10 Hz, 2 ms, 2 V) stimulus applied to pedal nerve 3 through a suction electrode (Getting et al., 1980).

Simultaneous impalement of ipsilateral Pd5 and Pd21 required the pedal ganglion to be supported with minuten pins so that its caudal edge faced upwards, making the caudal–ventral and caudal–dorsal areas accessible to microelectrodes.

Bead transport

To determine the direction of local ciliary beating, a micropipette was used to position glass beads (50–100 μm diameter) from a seawater suspension on a region between the brain and the left margin of the foot. A piece of metric ruler mounted on a micromanipulator was positioned along the path of bead flow. Beads were observed through the microscope and timed by stopwatch as they passed the 0 and 10 mm marks on the ruler. These events were also marked on a chart recorder together with a record of the ipsilateral Pd5 membrane voltage. Transport measurements were taken during 26 sequential intervals over the course of 1 h, each interval starting when the beads used in the previous interval had been cleared by the cilia from the measurement path. The action potential frequency was determined from the number of action potentials in Pd5 during the 24 s prior to the end of each observed transport time.

Determining the direction of Pd5 spikes in pedal nerve 3

The direction of Pd5 action potential propagation in pedal nerves 2 and 3 was determined from differential extracellular nerve recordings. The observed spike polarity was determined by the order in which the active (+) and inactive (−) silver wire electrodes recorded the passage of the action potential. This polarity was reversed when the order in which the electrodes were placed on the nerve was reversed (Fig. 2A–C). We used the information inherent in the polarity to determine the direction of spike transmission (Fig. 2D). A Pd5 action potential evoked intracellularly in the soma was used to calibrate the signal polarity generated by a spike traveling towards the periphery. Simultaneous intracellular recordings confirmed that all extracellular spikes analyzed were propagating in Pd5.

Results

The responses of Pd5 to magnetic field stimuli

We measured the effect of magnetic field rotations on 11 Pd5 neurons in 11 further-reduced preparations in which all nerves leaving the brain, except pedal nerves 2 and 3, were cut. For this purpose, the change in the number of spikes fired during the 30 min imposed magnetic field trials (change over the preceding 15 min baseline value) was compared with the change in the number of spikes fired during the 30 min control trials (change over the preceding baseline control trial). The increase in the number of action potentials during the imposed field trials was significantly greater than that during control

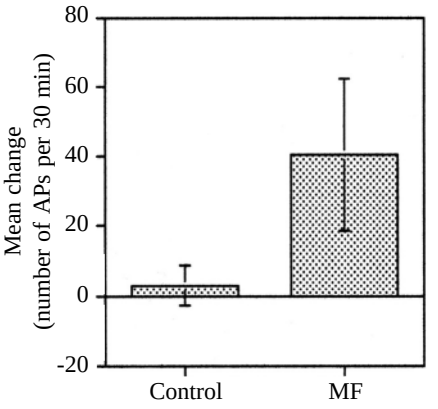

Fig. 3. Summary of changes over baseline levels in Pd5 action potential (AP) numbers in further-reduced preparations when the horizontal component of the ambient magnetic field (MF) was rotated 60° once per minute for 30 min. During control trials, the ambient magnetic field was unaltered. *N*=11. Values are means ± S.E.M.

trials (P<0.02, Wilcoxon signed-ranks test). On average, Pd5 firing increased by 40.3±22.0 action potentials during imposed field trials and by 3.1±5.7 action potentials during control trials (means ± S.E.M., *N*=11, Fig. 3).

The direction of Pd5 spike propagation in pedal nerve 3

We determined the direction of Pd5 action potential transmission in pedal nerve 3 during three imposed magnetic field trials in three further-reduced preparations. For this purpose, we first noted the polarity of the two-electrode, extracellular nerve recordings of outgoing Pd5 spikes in pretrials using spikes evoked intracellularly in the soma (see Fig. 2A–C). We then compared the polarity of the evoked spike recording with the polarity of spike recordings elicited during imposed field trials. The polarity of all 52 action potentials analyzed from Pd5 axons recorded from pedal 3 nerves demonstrated propagation from the neuron soma in the brain towards the periphery.

The activity of Pd5 during crawling

We recorded Pd5 firing intracellularly from seven semi-intact *Tritonia diomedea* preparations crawling on treadmills. The firing rate was correlated with the rate of treadmill turning (Fig. 4A,B). The coefficients of correlation for these slugs

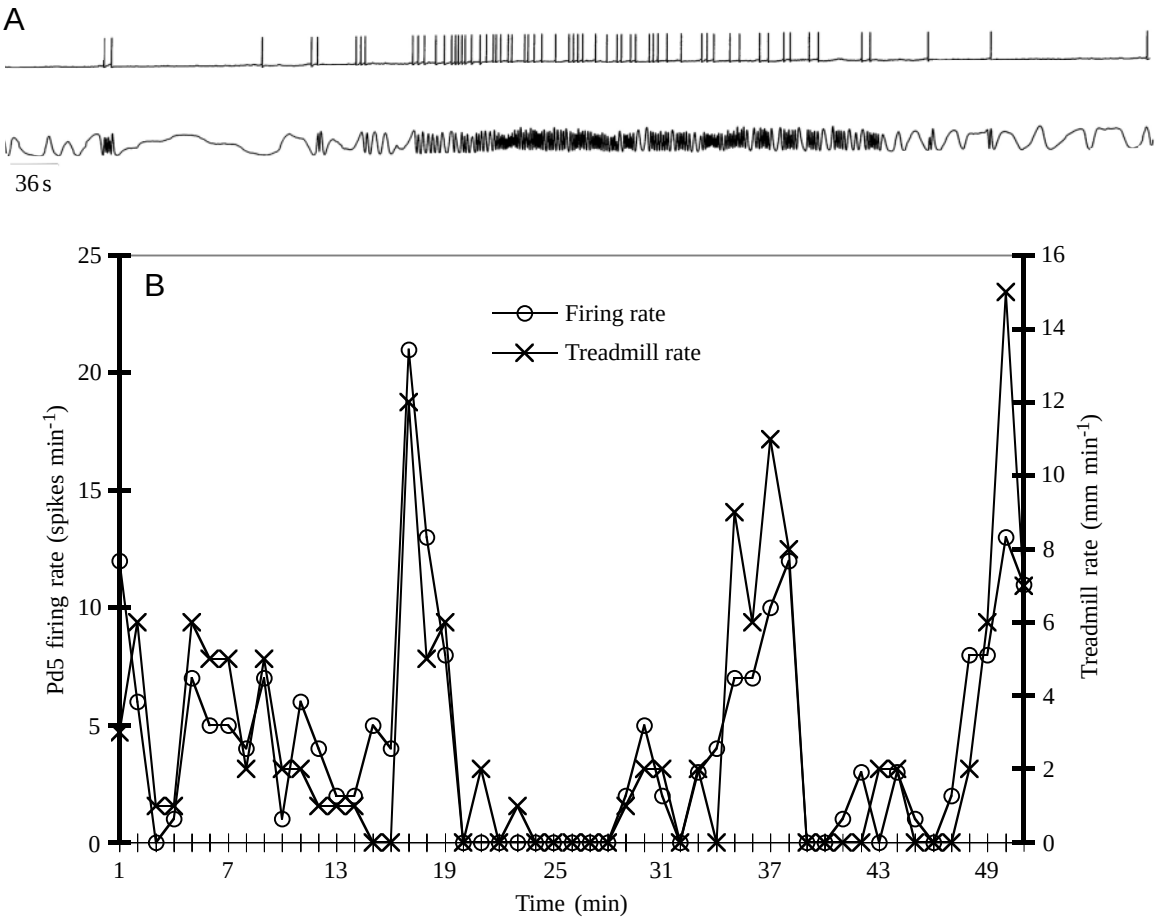

Fig. 4. Monitoring crawling and Pd5 membrane potential simultaneously. (A) Each full up or down deflection in the treadmill recording (shown in the lower trace) represents a 1.05 mm (4°) turn of the cylindrical treadmill. The action potentials shown in the upper trace measure between 85 and 100 mV. (B) Data from a longer recording of crawling and Pd5 action potentials have been been binned at 1 min intervals and plotted together.

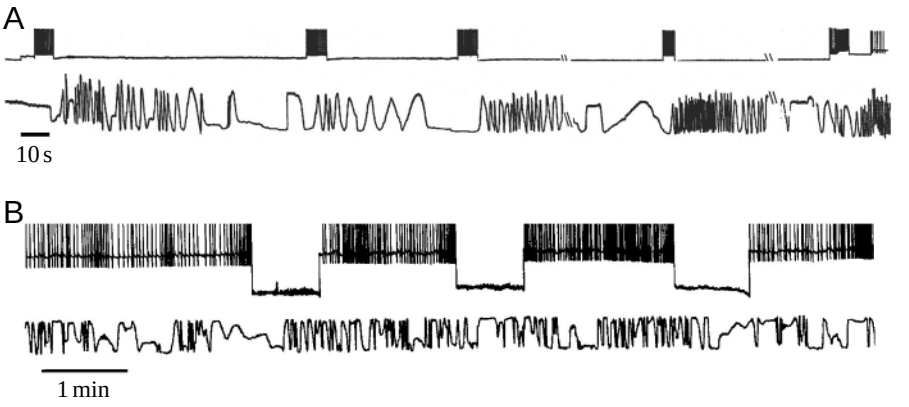

Fig. 5. Effect of depolarization or hyperpolarization of Pd5 on locomotion detectable by monitoring treadmill movement. (A) In one example, five consecutive trains of spikes elicited in Pd5 by depolarization (shown in the upper trace) caused acceleration of the treadmill. (B) During spontaneous firing, while the treadmill was turning, hyperpolarizing Pd5 appeared to have no effect on slug crawling.

were 0.826 (P<0.001), 0.638 (P<0.01), 0.882 (P<0.001), 0.559 (P<0.001), 0.781 (P<0.001), 0.888 (P<0.001) and 0.765 (P<0.001). The maximal treadmill turning rate observed was 46 mm min^{-1}.

To investigate whether Pd5 plays a motor role in locomotion, it was depolarized intracellularly (action potential frequency <3.2 Hz) when the treadmill was stationary or turning slowly (indicating slow crawling). In 14 out of 20 attempts (five animals), depolarization of Pd5 was followed by a sharp increase in the turning rate of the treadmill. In the case of one animal, treadmill turning rate increased sharply after the beginning of driven Pd5 action potential bursts in five out of five sequential trials (Fig. 5A). Hyperpolarizing Pd5 below threshold during a spontaneous spike train produced no obvious effect on the slug's crawling activity (Fig. 5B).

The Pd5 spiking rate also correlated with bead transport rates in our pilot experiment (Fig. 6). The fastest bead moved at 0.62 mm s^{-1}. Increases in spiking frequency usually preceded faster bead transport.

Synaptic connections from dorsal swim interneurons to Pd21 and Pd5

Depolarizations leading to action potentials in DSIs elicited depolarizations of contralateral Pd5 and Pd21 (Fig. 7). Each action potential in the DSI caused one excitatory postsynaptic potential (EPSP) in Pd5. The depolarization of Pd5 caused by DSI bursts was sometimes followed by a hyperpolarization response similar in peak latency (approximately 2.5 s, Fig. 7) to the slow inhibitory postsynaptic potential elicited by neuron C2 (Snow, 1982). Small bursts of DSI action potentials caused depolarization of Pd21 without one-to-one EPSPs, sometimes eliciting several closely grouped spikes. Spikes elicited in Pd5 by passing depolarizing current through the intracellular electrode had no effect on DSIs or Pd21 membrane potentials, and driving Pd21 had no effect on DSIs or Pd5.

Discussion

In the magnetic stimulation experiments with the further-reduced preparation (only pedal nerves 2 and 3 remaining uncut), the activity of Pd5 increased by an average of 40.3 action potentials during the 30 min imposed field trials. In an earlier study on the semi-intact preparation (all nerves intact) by Lohmann et al. (1991), the mean increase in the number of action potentials fired during the first magnetic field trial was 27.9. The larger response seen here may have resulted from the fact that we deliberately avoided using hyperpolarizing current to lower the background firing rate. Accordingly, our baseline period criterion was the firing of 0–45 action potentials by Pd5 during 15 min, in contrast with 0–19 action potentials during 20 min used by Lohmann et al. (1991). This difference resulted in recordings with a wider range of action potential rates and may underlie the higher mean increase as well as the higher variability of the action potential frequency response here. Furthermore, neural and behavioral responses to identical stimuli may differ with, and depend on, the physiological and behavioral state of the animal. For example, it has been reported that some animals respond to magnetic fields only when moving (Kreithen and Keeton, 1974). Sensory afferents are also gated by the locomotory status in locusts (Reichert et al., 1985). It has been suggested that the activity of peripheral sensory cells in *Tritonia diomedea* may be modulated by serotonin released from the peripheral terminals of neurons with centrally located cell bodies (Moroz et al., 1997). To search for a less variable response, our future

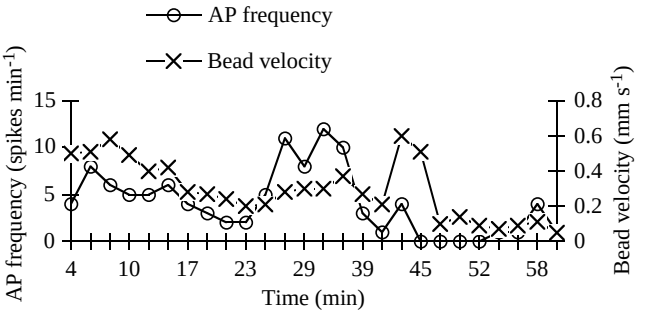

Fig. 6. Pd5 action potential (AP) frequency plotted against the velocity at which glass beads were transported on the foot of the slug. The time taken by beads to be transported 10 mm was measured 26 times during 1 h. The number of Pd5 action potentials fired during the 24 s prior to the end of each bead transport time measurement was used to determine the action potential frequency for that data point.

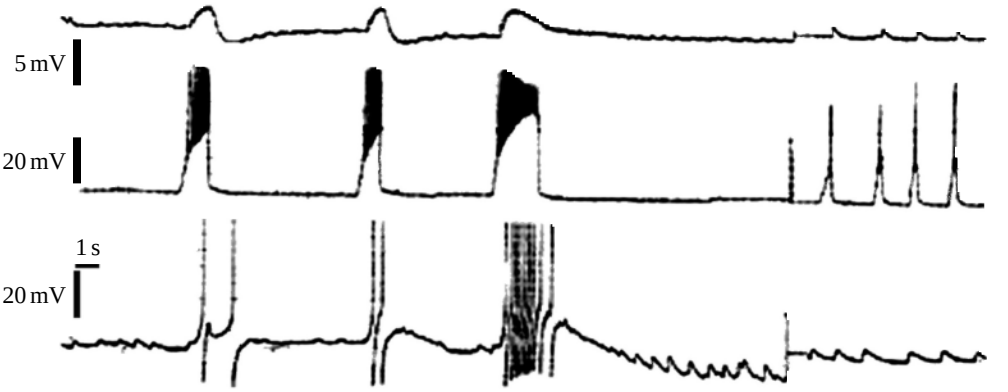

Fig. 7. Dorsal swim interneuron (DSI) bursts due to microelectrode current injection (shown in middle trace) cause depolarization of both contralateral Pd5 (shown in upper trace) and contralateral Pd21 (shown in lower trace). Single spikes elicited in a DSI correspond one-to-one with excitatory postsynaptic potentials in Pd5, but not in Pd21.

electrophysiological experiments involving magnetic stimuli will be conducted while monitoring crawling rate. Finally, although Pd5 may receive EPSPs in response to magnetic field stimuli, the resulting depolarization may be insufficient to fire the neuron in the absence of other active inputs. The magnetic field response seen in Pd5 may be an increase in spike frequency within trains triggered by other, coincidental stimuli. It is possible that this issue could have been resolved by recording postsynaptic currents in voltage-clamp mode during magnetic field stimulation if it had been possible to space-clamp this giant neuron.

The Pd5 neurons responded to magnetic stimuli in the semi-intact preparations (all nerves intact), and the response persisted in the present study in the further-reduced preparations. This contrasts with an earlier observation (Lohmann et al., 1991) that, if all nerves are cut, the rate of Pd5 spiking is no longer affected by magnetic stimuli. It is possible that the activity of central magnetoreceptors impinges upon Pd5 *via* a peripheral route no longer in place when the brain is isolated or that Pd5 becomes less excitable after axotomy (Stinnakre and Tauc, 1969). This is supported by the observation that the depolarization of Pd5 by other pedal neurons ceases when pedal nerves 2 and 3 are cut (Murray et al., 1992).

Pedal nerves 2 and/or 3 may also contain magnetoreceptors or transmit action potentials from peripheral magnetoreceptors to the brain. Pedal nerves 2 and 3 innervate the ipsilateral anterior third and posterior two-thirds of the foot, respectively. Between the level of the mucous gland and the tip of the tail, pedal nerve 3 courses along a relatively straight line, branching occasionally into the foot and towards the animal's ciliated foot epithelium (MacFarland, 1966; I. R. Popescu and A. O. D. Willows, unpublished observation from dissections of Methylene-Blue-stained tissues). The large number of regularly arranged molecules necessary for some hypothetical magnetoreception mechanisms (e.g. magnetite-associated ion channels or cyclotron-resonant ion channels) could be embedded in a long cylindrical array in the Pd5 axonal plasma membrane. Pedal nerves 2 and 3 contain many axons, some transmitting action potentials peripherally and others centrally. Thus, although we do not know where action potentials transmitted towards the brain through these nerves originate, two possible sources are the ciliated epithelial cells of the foot and the neurons embedded in the nerve trunks. These sources are suggested by the knowledge that sensory neurons are generally derived from ciliated cells in evolutionary and developmental terms, and by their proximity to pedal nerves 2 and 3, respectively (Bullock and Horridge, 1965).

Intracellular depolarization of Pd5, causing high-frequency action potential firing, was followed by increased turning of the treadmill in 14 out of 20 trials. The variability of the response may be due to changing levels of axial friction in the treadmill resulting from mucus accumulation. Cleaning of the treadmill axle while recording from neurons is impossible. Alternatively, the motor (locomotory) function of Pd5 is probably accomplished through the release of *Tritonia diomedea* pedal ganglion peptides (TPeps). These neuromodulatory peptides also accelerate the beating of the ciliated, locomotory epithelial cells of the foot. Pd5 may act in concert with several other pedal neurons that contain these peptides or serotonin (Audesirk, 1978; Willows et al., 1997; Moroz et al., 1997; J. C. Beck, M. S. Cooper and A. O. D. Willows, in preparation). Simultaneous microelectrode access to Pd5 and the established serotonergic crawling motoneuron Pd21 is very difficult in the semi-intact preparation, because these cells are found on the dorsal and ventral faces of the pedal ganglion, respectively. Furthermore, we have shown here that both Pd5 and Pd21 receive excitatory, although probably differently routed, inputs from contralateral DSIs, suggesting that simultaneous activation of these pedal neurons often occurs. Suprathreshold depolarization of a DSI *via* the intracellular electrode causes immediate commencement of treadmill turning (W. Frost, personal communication). Indeed, in the fast crawling periods following a swimming episode, when the DSIs are active, peptidergic and serotonergic motoneuron firing may overlap. Interestingly, although water currents directed at the oral veil inhibit Pd21, they depolarize Pd5 in these highly rheotactic animals (Murray et al., 1992). This raises the question of whether just the peptidergic neurons or both these chemically distinct locomotory branches are activated by magnetic stimulation.

A motor role for Pd5 is suggested because (1) this neuron contains peptides (TPeps) that accelerate the beating of the locomotory cilia when applied directly to the surface of the

foot or to the isolated cells (Willows et al., 1997), (2) the Pd5 axons form part of pedal nerves 2 and 3, which innervate the foot (Lohmann et al., 1991), (3) TPeps-containing nerves are found in close proximity to the locomotory cilia (Willows et al., 1997), (4) Pd5 axons conduct action potentials away from the brain, (5) firing of Pd5 and the locomotion of the slugs are usually correlated, and (6) Pd5 receives excitatory input from DSIs, which are active during the fast crawling that follows escape swimming (Getting et al., 1980; Katz et al., 1994) and which also excite a known locomotion motoneuron (Pd21).

We conclude that Pd5 neurons from *Tritonia diomedea* are excited by earth-strength magnetic stimuli, for which the afferent pathway includes pedal nerves 2 or 3, and that Pd5s are likely to play a motor role in the orientation of the animal to magnetic fields.

Color images of *Tritonia diomedea* can be found on our website
(http://weber.u.washington.edu/ razvan/geomagnetic.html).

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