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# Identifiable neurons inhibited by Earth-strength magnetic stimuli in the mollusc *Tritonia diomedea*

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**Summary**

**Diverse animals use the Earth's magnetic field as an orientation cue, but little is known about the sensory, processing and motor elements of the neural circuitry underlying magnetic orientation behavior. The marine mollusc *Tritonia diomedea* has both a magnetic compass sense and a simple nervous system accessible to electrophysiological analysis. Previous studies have revealed that four identifiable neurons, known as LPd5, RPd5, LPd6 and RPd6, respond with enhanced electrical activity to changes in Earth-strength magnetic fields. Here we report that two additional neurons, LPd7 and RPd7, are inhibited by magnetic stimuli.**

**Cobalt fills of the Pd7 neurons indicated that two prominent neurites emerge from the soma and project to the periphery through the ipsilateral cerebral nerves CeN6 and CeN3; in some cases, a third neurite was visible in CeN2. The nerves extend to the anterior region of the animal where they innervate the lateral body walls, oral veil and mouth region. Action potentials in the Pd7 neurons propagate from the central ganglia toward the periphery. Thus, the Pd7 cells have characteristics of efferent neurons. The precise function of these cells during magnetic orientation behavior, however, remains to be determined.**

Key words: *Tritonia diomedea*, magnetic, magnetoreception, orientation, navigation, mollusc.

## Introduction

Numerous behavioral experiments have demonstrated that animals use the Earth's magnetic field as a directional cue during orientation and navigation (reviewed by Wiltschko and Wiltschko, 1995). In contrast, comparatively few studies have examined the neural mechanisms that underlie magnetic orientation behavior (see reviews by Lohmann and Johnsen, 2000; Ritz et al., 2002). In some animals, regions in the central nervous system (CNS) have been identified that may play a role in magnetic orientation (Semm et al., 1984; Beason and Semm, 1987; Semm and Beason, 1990; Walker et al., 1997; Nemec et al., 2001). However, the neural mechanisms used to detect the Earth's magnetic field, process magnetic information and ultimately produce magnetic orientation behavior remain poorly understood.

The marine mollusc *Tritonia diomedea* is a promising model system for investigating the neural mechanisms underlying magnetic orientation behavior. Behavioral studies have demonstrated that *Tritonia* has a magnetic compass sense, which the animals may use during onshore and offshore movements (Lohmann and Willows, 1987; Willows, 1999). In addition, this animal has large, identifiable brain cells and a central nervous system readily accessible to electrophysiology (Chase, 2002). Intracellular recordings have shown that two bilaterally symmetric pairs of neurons in the brain of *Tritonia*, known as left and right pedal 5 (LPd5 and RPd5) and left and right pedal 6 (LPd6 and RPd6) (Fig. 1), respond with increased spiking to changes in Earth-strength magnetic fields (Lohmann et al., 1991; Popescu and Willows, 1999; Cain, 2001; Wang et al., 2003).

In this study, we present evidence that another pair of neurons also responds to changes in the ambient magnetic field. These cells, known as the pedal 7 (Pd7) neurons, are adjacent to the Pd5 and Pd6 cells (Fig. 1) and have similar features. All three pairs of neurons have similar coloration, produce neuropeptides known as TPeps (Willows et al., 1997) and have electrophysiological and anatomical characteristics of efferent neurons. In contrast to the Pd5 and Pd6 neurons, however, the Pd7 neurons respond with decreased spiking to magnetic stimuli.

## Materials and methods

*Tritonia diomedea* Bergh were trawled from Bellingham Bay, Washington, USA at depths of 20 to 30 m. The animals were taken to the University of Washington Friday Harbor Laboratories in Friday Harbor, Washington and kept in tanks

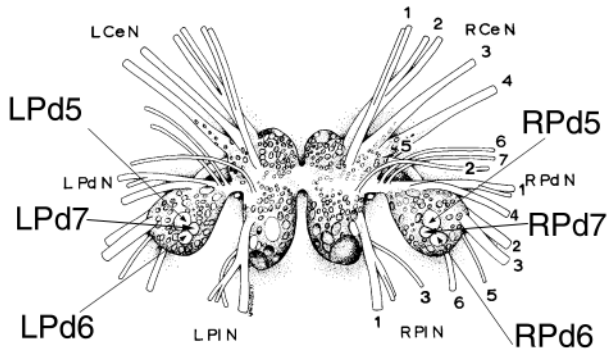


Fig. 1. Diagram of the central ganglia of *Tritonia diomedea* indicating the location of the identifiable neurons LPd7, RPd7, LPd6, RPd6, LPd5 and RPd5. L/R, left/right; Ce N, cerebral nerve; Pd N, pedal nerve; Pl N, pleural nerve.

with filtered flow-through seawater at 11°C. Animals were fed sea pens (*Ptilosarcus gurneyi*) *ad libitum*. For dissections and electrophysiological experiments, animals were transferred to a dissection chamber that contained flow-through 11°C seawater.

### *Semi-intact animal preparations*

Intracellular recordings from neurons were carried out in semi-intact preparations (Willows, 1967; Willows et al., 1973). A small incision was made directly above the brain on the animal's dorsal surface. Non-magnetic tungsten hooks were used to retract the body wall, support the animal in the seawater bath, and expose the central nervous system (CNS). A wax-covered platform was placed beneath the brain and tungsten pins were used to immobilize the central ganglia. Animals were allowed to recover for at least 1 h after the brain had been immobilized. Neurons were then identified by their size, coloration and location within the CNS and specific cells were impaled with glass microelectrodes filled with 3 mol $l^{-1}$ KCl (15–30 MΩ). Electrical signals were amplified, monitored on an oscilloscope, digitized using a CED 1401 A-D board, and analyzed using CED Spike 2 software (Cambridge Electronics Design, Cambridge, UK).

### *Magnetic field measurements and magnetic field manipulations*

The dissection chamber was located in the center of a Merritt 4-coil system (Merritt et al., 1983). Each of the four square coils measured 1.02 m on a side. The coil system was powered by a BK Precision 1760 Triple output DC power supply, and a Radio Shack TRS-80 portable computer was used to switch the coil on and off. Animals were placed in the dissection chamber so that they faced magnetic 300° (with 0° indicating magnetic north). When the coil was turned on, the magnetic field was rotated 60° clockwise so that the animals were oriented toward 240°. When the coil was off, the intensity of the ambient magnetic field at the recording site was 53.0 μT and the inclination was 76.9°. When the coil was turned on, alterations to the total magnetic intensity and inclination angles were minimal (the total intensity of the magnetic field was changed by +0.2% and the inclination angle by +0.4°).

### *Magnetic experiments with left pedal 7*

Because the Pd7 neurons share many characteristics with the magnetically sensitive Pd5 and Pd6 neurons, we investigated whether the Pd7 neurons also respond to changes in the ambient magnetic field. We arbitrarily focused our experiments on left pedal 7 (LPd7). Each LPd7 neuron was tested with two treatments: a magnetic stimulus treatment and a control treatment. The magnetic stimulus treatment (Popescu and Willows, 1999; Cain, 2001) consisted of a 15 min baseline period followed by a 30 min magnetic stimulus period. During the stimulus period, the magnetic field was first rotated 60° clockwise in a single step; then, after 1 min, it was rotated 60° counterclockwise back to its original position (Lohmann et al., 1991; Popescu and Willows, 1999; Cain, 2001; Wang et al., 2003). This was repeated so that the magnetic field was rotated once every minute (Popescu and Willows, 1999; Cain, 2001). The control treatment consisted of a 15 min baseline and a subsequent 30 min period during which the magnetic field was not changed (Popescu and Willows, 1999; Cain, 2001).

For each animal, the order of the two treatments was randomly determined. After the first treatment, a recovery interval of at least 1 h elapsed before the other treatment was applied. After the second trial had been completed, spike frequencies (spikes $min^{-1}$) were calculated for each baseline period and for the subsequent magnetic stimulus or control period (Popescu and Willows, 1999; Cain, 2001). The LPd7 neurons of nine different animals were tested using these procedures.

In addition, we tested the responses of several Pd7 neurons to a single 60° clockwise rotation of the magnetic field. In these trials, a 15 min baseline was first recorded from the Pd7 neuron before the magnetic field was rotated. Recordings continued for 15 min after the single rotation.

While recording from the Pd7 neurons, we simultaneously recorded from one or more of the following when possible: the RPd7 cell, the Pd5 neurons and the Pd6 cells. These simultaneous recordings allowed us to monitor the responses of these neurons to magnetic stimuli and also to determine whether LPd7 neurons had common synaptic inputs with LPd5 and LPd6.

### *Cobalt fills of the Pd7 neurons*

To visualize the morphology of the Pd7 neurons, 500 mmol $l^{-1}$ $CoCl_2$ was pressure injected into the somata ($N$=5 for LPd7; $N$=5 for RPd7) using a PV820 Picopump (World Precision Instruments, Sarasota, FL, USA). After the $CoCl_2$ diffused throughout the neuron, the CNS was removed from the animal. The brain was incubated for 15 min in 11°C filtered seawater with several drops of concentrated ammonium sulfide (Croll, 1986). The CNS was then washed with seawater, fixed with 10% formalin in filtered seawater for 24 h, dehydrated with an ascending ethanol series, cleared with methyl salicylate, and mounted

on a glass slide. The Pd7 somata and neurites were visualized using light microscopy.

*Determining the direction of LPd7 action potentials*

The direction of action potential propagation in LPd7 was determined by recording intracellularly from this cell while simultaneously recording extracellularly from left cerebral nerve 6 (LCeN6) and left cerebral nerve 3 (LCeN3), the two nerves found to contain Pd7 neurites (see Results). Extracellular signals were recorded using *en passant* suction electrodes in differential recording mode and amplified using a differential AC amplifier (A-M Systems, Carlsborg, Washington, USA). LPd7 units in the nerves were identified by determining which units corresponded one-to-one with evoked potentials in the soma during stimulation (*via* current injection through the intracellular electrode). Spontaneous LPd7 spikes were then used to determine the direction of action potential propagation within the left cerebral nerves.

## Results

*Magnetic experiments with the LPd7 neurons*

To determine whether the spike frequency of LPd7 changed during periods of magnetic stimulation, each preparation was subjected to a control treatment and a magnetic stimulus treatment (Fig. 2). In the control treatment, no difference was observed between the spike frequency during the baseline period (mean spike frequency=6.5±1.4 spikes $min^{-1}$, $N$=9, mean ± S.E.M.) and the spike frequency during the subsequent control period (mean spike frequency=6.7±1.3 spikes $min^{-1}$, $N$=9) (Wilcoxon Signed Ranks Test, $P$>0.30, $N$=9) (Fig. 3A). In the magnetic stimulus treatment, however, a significantly lower spike frequency occurred during the magnetic stimulus period (mean spike frequency=4.1±1.1 spikes $min^{-1}$, $N$=9) than during the baseline (mean spike frequency= 7.5±1.7 spikes $min^{-1}$, $N$=9) (Wilcoxon Signed Ranks Test, $P$<0.01, $N$=9) (Fig. 3B).

Although the statistical analysis demonstrated that decreased spiking occurred during the magnetic stimulus periods, responses in individual trials were variable. Decreases in spike frequency occurred in eight of the nine individual trials. When decreases occurred, they ranged in magnitude from –0.3 to –8.7 spikes $min^{-1}$. Decreases in spike frequency occurred after a latency of about 3 to 10 min after the first field change (Figs 2, 4).

For each animal, the change in spike frequency between the baseline and the subsequent magnetic stimulus period (mean change in spike frequency=–3.4±1.0 spikes $min^{-1}$, $N$=9) was compared to the change between the baseline and the subsequent control period (mean change in spike frequency=0.2±0.2 spikes $min^{-1}$, $N$=9). A significantly larger change in spike frequency occurred during the magnetic stimulus treatment than during the control treatment (Wilcoxon Signed Ranks Test, $P$<0.01, $N$=9) (Fig. 3C).

Although the focus of this study was on LPd7, we were able to record simultaneously from RPd7 (the bilaterally symmetric mate of LPd7) in two animals during a magnetic stimulus treatment. In both cases, a decrease in spike frequency occurred during the magnetic stimulus period (Fig. 4A). We also recorded the responses of the Pd7 neurons to a single 60° clockwise rotation of the field in three animals, twice testing LPd7 and once testing RPd7 (Fig. 4B). In all three trials, spike frequency decreased.

Simultaneous recordings of the Pd5, Pd6 and Pd7 neurons indicated that the same magnetic stimuli that elicited decreased spiking in the Pd7 neurons elicited increased spiking in the Pd5 and Pd6 cells. In all four instances in which the activities of LPd6 and LPd7 were simultaneously recorded, LPd6

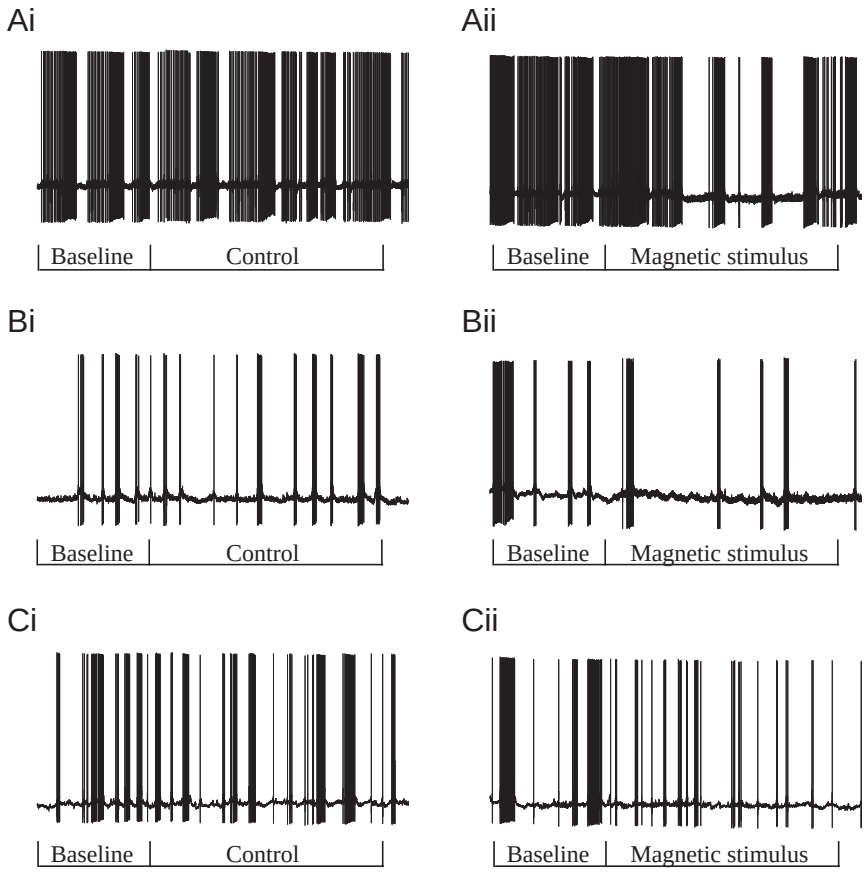


Fig. 2. Representative electrophysiological recordings of LPd7 from three different animals during control treatments (Ai–Ci) and magnetic treatments (Aii–Cii). Traces in the same row (A–C) are from the same animal. Action potentials are between 90 and 100 mV in amplitude. The brackets beneath each trace indicate the 15 min baseline period and the 30 min control or magnetic stimulus period.

increased its firing rate during the magnetic stimulus period. Similarly, increased spiking was observed in LPd5 in four of five animals. One example of these multiple recording experiments is shown in Fig. 5A. These multi-cell recordings also allowed us to observe the postsynaptic potentials (PSPs) in the neurons. The results indicated that the postsynaptic potentials of LPd5, LPd6 and LPd7 are sometimes synchronous. However, LPd5 and LPd6 share many more synchronous PSPs with each other than with LPd7 (Fig. 5B).

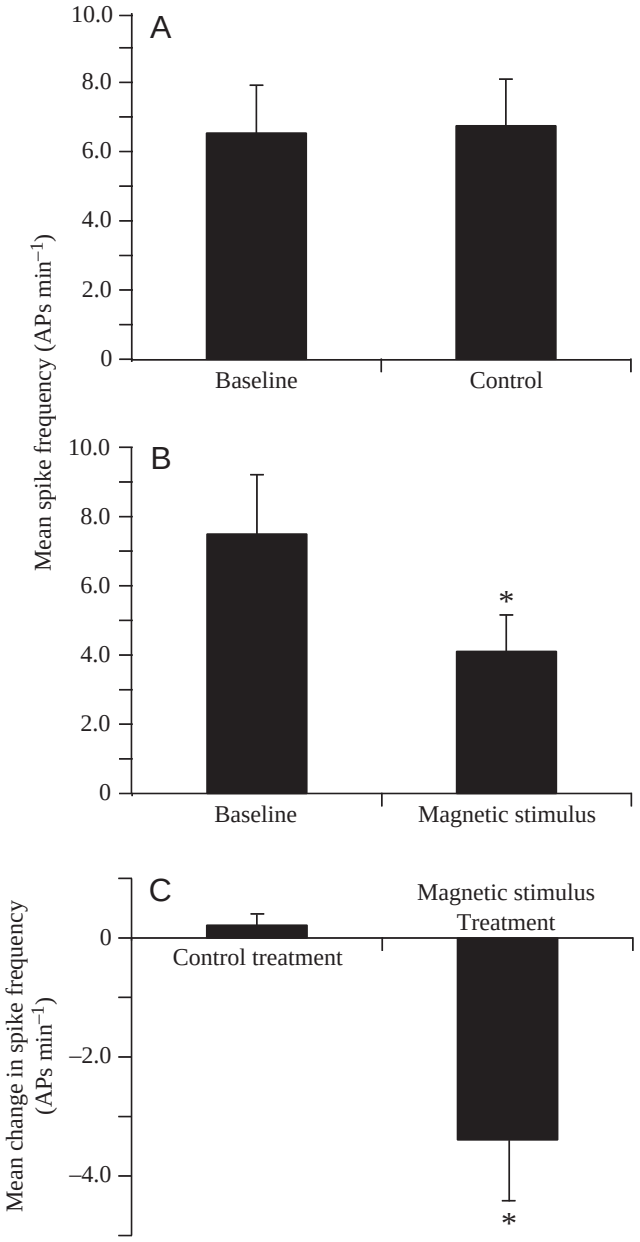


Fig. 3. Summary of LPd7 responses during control treatments (in which the magnetic field was not changed) and magnetic stimulus treatments (see text). (A) Results from control treatments. Bar graphs indicate the mean spike frequencies during the baseline and control periods (no significant difference; Wilcoxon Signed Ranks Test, $P>0.30$, $N=9$). (B) Results from the magnetic stimulus treatments. Bar graphs indicate the mean spike frequencies during the baseline and magnetic stimulus periods (*significant difference, Wilcoxon Signed Ranks Test, $P<0.01$, $N=9$). (C) Bar graphs indicate the mean changes in action potentials between the baseline period and the control period or magnetic stimulus period (*significant difference, Wilcoxon Signed Ranks Test, $P<0.01$, $N=9$). Values are means ± S.E.M. AP, action potential.

*Cobalt fills of LPd7*

The somata of the Pd7 neurons are located in the dorsal, posterior region of the pedal ganglia and often measure 200–250 μm in diameter (Fig. 6). Cobalt fills revealed that each Pd7 cell possesses a large, primary neurite, which emerges from the soma and enters the cerebral ganglia. At the cerebral–pedal connective, a small neurite branches and enters

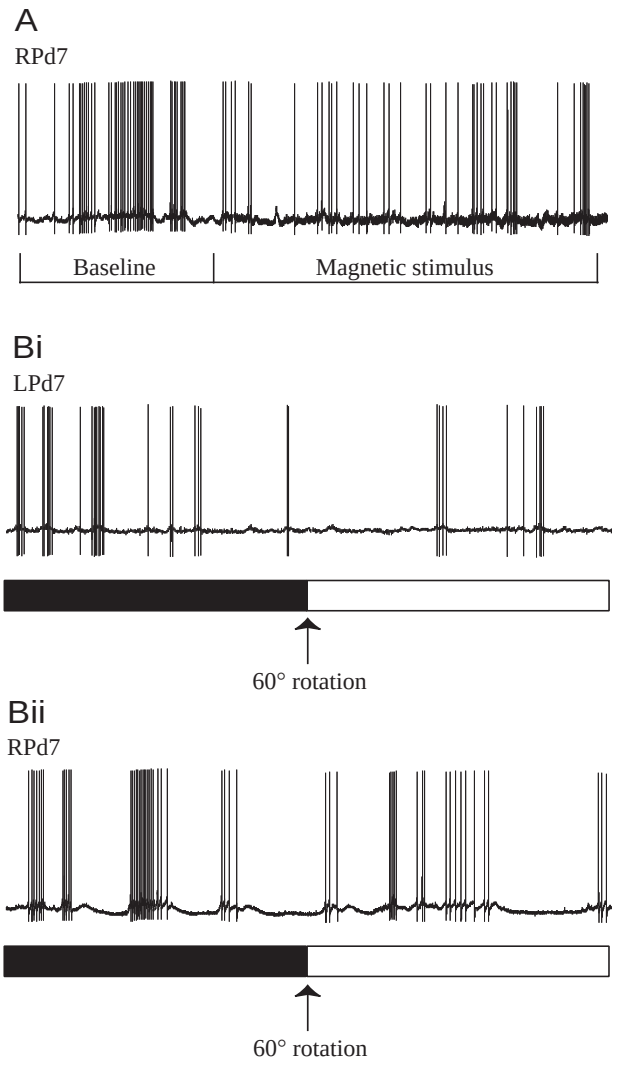


Fig. 4. (A) Electrophysiological recording of RPd7 during a magnetic stimulus treatment. Conventions as in Fig. 2. (B) Recordings of LPd7 (Bi) and RPd7 (Bii) before and after a single 60° clockwise rotation of the magnetic field. The white and black bars indicate a period of 900 s. In both traces, the number of spikes decreased after the 60° rotation. Action potentials are between 90 and 100 mV in amplitude.

CeN6. The main neurite continues into the cerebral ganglion before entering CeN3. In two of the five preparations, the main neurite bifurcated within the cerebral ganglion with one branch entering CeN3 while the other entered cerebral nerve 2 (CeN2).

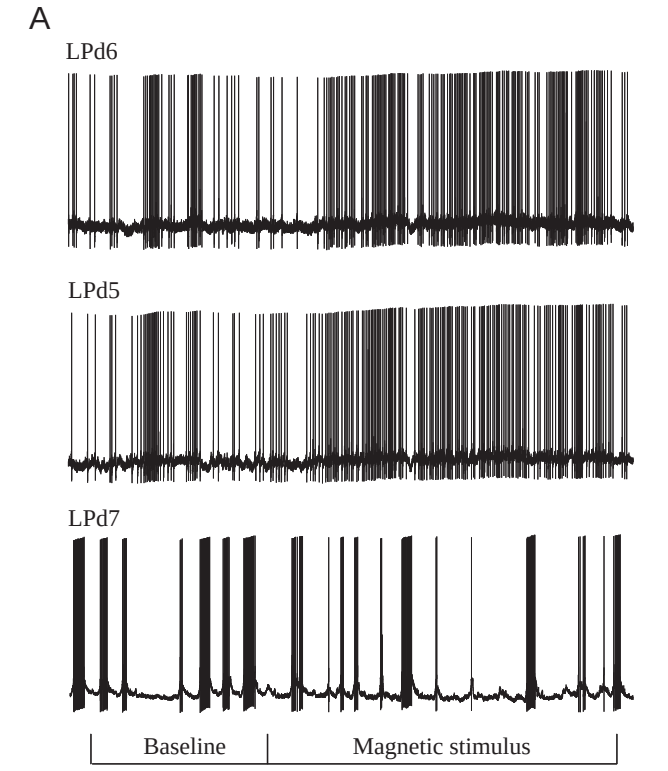


Fig. 5. Simultaneous intracellular recordings of LPd5, LPd6 and LPd7 neurons during a magnetic stimulus treatment. Action potentials are between 90 and 100 mV in amplitude. Conventions as in Fig. 2. Spiking in LPd5 and LPd6 increased in response to the magnetic stimulus while spiking in LPd7 decreased. (B) Representative simultaneous intracellular recordings of LPd5, LPd6 and LPd7 showing common postsynaptic potentials (PSPs). The first two vertical lines indicate times at which common synaptic input occurred in all three neurons. Subsequent vertical lines indicate times at which synchronous PSPs occurred in LPd5 and LPd6 but not in LPd7. In this and many other recordings, LPd5 and LPd6 shared more synchronous PSPs with each other than with LPd7.

*Direction of LPd7 action potential propagation*

Simultaneous extracellular and intracellular recordings of LPd7 were performed to determine the direction of action potential propagation. In all preparations ($N$=3), spontaneous action potentials were observed in the LPd7 soma before the corresponding extracellular units were recorded in LCeN6 or LCeN3 (Fig. 7). This demonstrated that action potentials in LPd7 neurons propagate from the central ganglia toward the periphery.

## Discussion

The results indicate that the electrical activity of the Pd7 neurons decreased when *Tritonia* was subjected to changes in Earth-strength magnetic fields. Decreased spiking occurred in response to magnetic stimuli consisting either of a series of magnetic field rotations (Figs 2, 3, 4A, 5A) or a single 60° clockwise rotation of the magnetic field (Fig. 4B). In contrast, spiking in the Pd5 and Pd6 neurons increased in response to these same magnetic stimuli (Fig. 5A; Lohmann et al., 1991; Popescu and Willows, 1999; Cain, 2001; Wang et al., 2003). No other neurons in *Tritonia* tested so far appear to be affected by changes in Earth-strength magnetic fields (Lohmann et al., 1991; Wang et al., 2003).

The function of the Pd7 neurons is not known. Given that *Tritonia* uses the Earth's magnetic field as an orientation cue (Lohmann and Willows, 1987), the Pd7 neurons appear likely

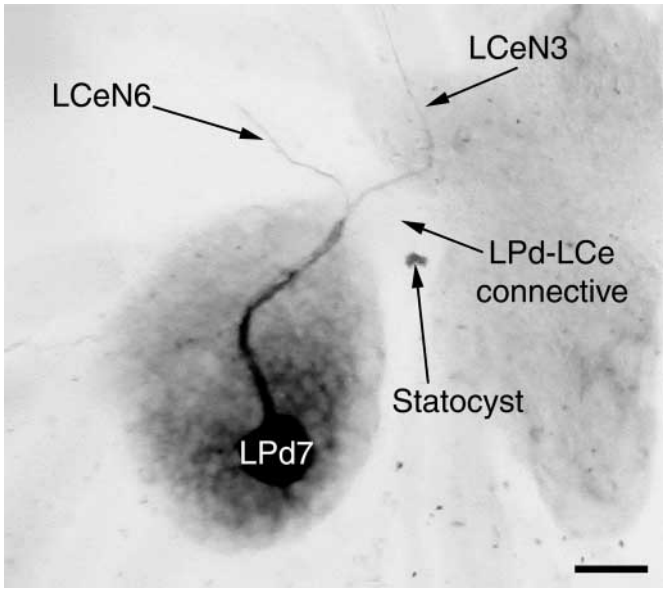


Fig. 6. Cobalt fill of LPd7 showing the large soma and primary neurite within the left pedal ganglion. The primary neurite extends into the left cerebral ganglion before entering left cerebral nerve 3 (LCeN3). At the left pedal–cerebral connective, a small neurite branches off and enters left cerebral nerve 6 (LCeN6). Scale bar, 250 μm.

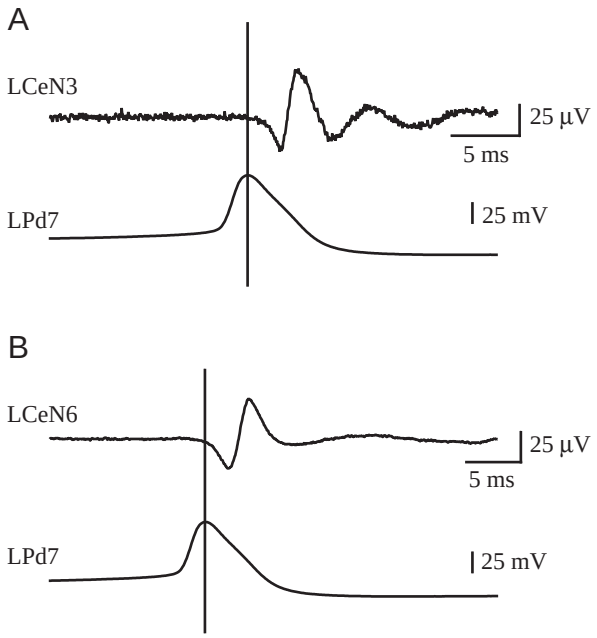


Fig. 7. Action potential propagation in LPd7 through LCeN3 and LCeN6. Single units corresponding to LPd7 were identified in LCeN3 and LCeN6 (see text for details). Simultaneous intracellular (LPd7) and extracellular (LCeN3 or LCeN6) recordings indicate that spontaneous action potentials were detected in the cell soma before they were detected in the cerebral nerves. Thus, action potentials propagate from the cell body toward the periphery of the animal.

to be components of the neural circuitry that underlies magnetic orientation behavior. Among several possibilities, the cells might play a role in detecting magnetic fields, in processing magnetic field information, in generating a motor response that involves crawling along a specific magnetic heading, or in suppressing behavior that might otherwise impede orientation.

### *Characteristics of Pd7 neurons*

The Pd7 neurons have neurites in nerves that innervate the anterior body wall, oral veil and mouth. Each Pd7 neuron has neurites located in cerebral nerve 6 (CeN6) and in cerebral nerve 3 (CeN3) (Fig. 6). In two of five preparations, neurites were also detected in cerebral nerve 2 (CeN2). Although the cobalt fills did not allow us to identify the precise targets that the Pd7 neurons innervate, CeN6 innervates the ipsilateral body wall near the base of the rhinophores (Willows et al., 1973) as well as the eye. CeN3 and CeN2 innervate ipsilateral areas of the oral veil and mouth (Willows et al., 1973). Simultaneous intracellular and extracellular recordings of LPd7 show that spontaneous action potentials propagate away from the central ganglia and toward the periphery through LCeN6 and LCeN3 (Fig. 7). Such an action potential propagation pattern, from CNS to periphery, is typical of efferent neurons (Bullock and Horridge, 1965; Willows et al., 1973).

The Pd7 neurons produce a class of neuropeptides known as TPeps (Lloyd et al., 1996; Willows et al., 1997; Beck et al., 2000). TPeps increase the ciliary beating frequency of isolated foot epithelial cells (Willows et al., 1997), as well as the ciliary transport rates of foot patches (Willows et al., 1997), ciliated salivary ducts (Gaston, 1998) and esophageal ciliated epithelial patches (Pavlova et al., 1999). TPep-like immunoreactivity has been detected near ciliated epithelial cells in the statocysts, oviduct, foot, salivary ducts, foregut and esophagus (Willows et al., 1997; Gaston, 1998; Beck et al., 2000). Possible peripheral targets of the Pd7 neurons may therefore include ciliated epithelial cells located on the anterior body wall, oral veil and the mouth. TPeps have also been hypothesized to function as neurotransmitters or neuromodulators within the CNS (Beck et al., 2000). TPep immunoreactivity has been localized in cell bodies and neural processes throughout the CNS and in structures identified as axosomatic synapses in the buccal ganglia (Beck et al., 2000). Thus, the Pd7 neurons may interact with other neurons in the central ganglia.

An enigmatic characteristic of the Pd7 neurons, as well as the other magnetically responsive neurons in *Tritonia*, is that a long latency (about 1–15 min) occurs between the onset of the magnetic stimulus and the onset of the neural response (Figs 2, 4A, 5; Lohmann et al., 1991; Popescu and Willows, 1999; Wang et al., 2003). Similar or longer latencies have been observed in electrophysiological responses of honeybees (Korall and Martin, 1987) and guinea pigs (Semm et al., 1980; Semm, 1983) and in behavioral responses of spiny lobsters (Lohmann et al., 1995) and bobolinks (Beason, 1989). Some possible reasons for these latencies are that the receptor mechanism or neural processing of the magnetic information may require a significant period of averaging, or that the nervous system may only periodically update magnetic field information (Beason, 1989; Walcott, 1996; Wiltschko et al., 1998)

### *Comparison of Pd7 neurons with Pd5 and Pd6 neurons*

To date, six neurons in *Tritonia* (LPd7, RPd7, LPd6, RPd6, LPd5 and RPd5) have been shown to respond to changes in Earth-strength magnetic fields. These cells share several similarities including the production of TPeps (Lloyd et al., 1996; Willows et al., 1997), characteristics of efferent neurons (Popescu and Willows, 1999; Cain, 2001; Wang et al., 2003), responsiveness to other sensory stimuli such as rheotactic cues (Murray et al., 1992), and some common synaptic inputs as indicated by synchronous postsynaptic potentials (Fig. 5B).

The Pd7 neurons, however, differ from the Pd5 and Pd6 neurons in two ways. Firstly, the Pd7 neurons are inhibited by magnetic stimuli whereas the Pd5 and Pd6 neurons are excited. Secondly, the Pd7 neurons have neurites in nerves that project to regions of the anterior body wall, oral veil and mouth. In contrast, the Pd5 and Pd6 neurons have axons in nerves projecting to the foot (Cain, 2001; Wang et al., 2003).

Given that *Tritonia* crawls using cilia on the ventral surface of its foot, the Pd5 and Pd6 cells have been hypothesized to modulate cilia involved in crawling (Willows et al., 1997; Popescu and Willows, 1999; Wang et al., 2003). Anatomical

and electrophysiological characteristics of the Pd7 neurons suggest that they too have efferent functions, but the role that these neurons play during magnetic orientation remains poorly understood. Although further studies will be needed to clarify the function of the Pd7 neurons, the identification of these magnetically responsive cells represents another step toward understanding the sensory, processing, and motor components that underlie magnetic orientation behavior in *Tritonia* and other animals.

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