Coactivation of electrically coupled sensory neurons alters the active propagation of peripherally generated spikes

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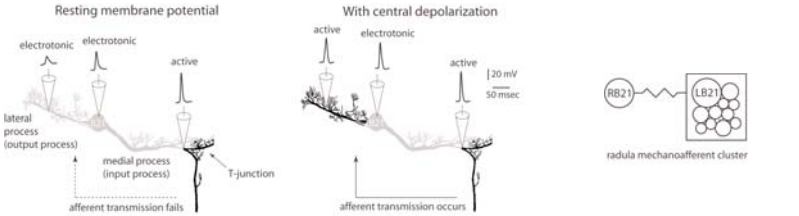
1 BACKGROUND

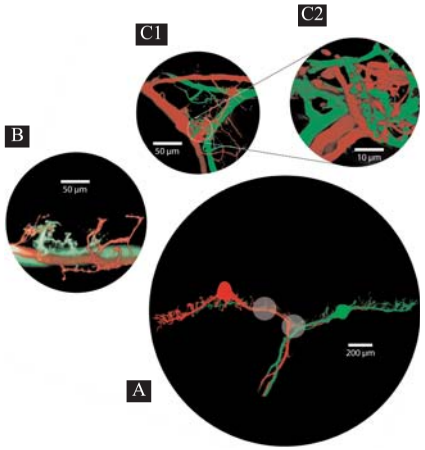


We study an Aplysia sensory neuron, a bipolar radula mechanoafferent B21. When B21 is peripherally activated at its resting membrane potential, a spike propagation failure occurs. Afferent activity is not transmitted to follower neurons contacted by an output region, the lateral process. When B21 is centrally depolarized, the propagation failure is relieved, and afferent transmission is restored. Under physiological conditions B21 is centrally depolarized via electrical coupling with other neurons.

One central source of depolarization in B21 is the electrical coupling that exists between B21 and other cells of the radula mechanoafferent cluster. Particularly strong is the electrical coupling between each B21 and the bilaterally symmetrical 'sister' cell. In this study we sought to determine whether this type of input could alter spike propagation in B21.

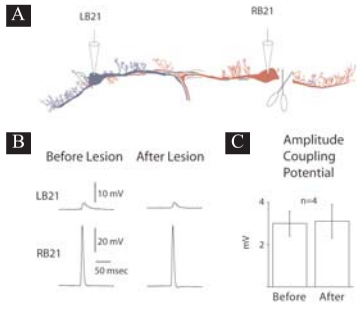
2 REGION OF CONTACT BETWEEN THE TWO B21 NEURONS

Confocal and multiphoton imaging of the two B21 neurons suggests that contact between the two B21 neurons occurs via the medial process



(A) Low power (10X) confocal imaging of two B21 neurons. One neuron was injected with Alexa Fluor 568, and the other neuon was injected with Alexa Fluor 488. Approximate regions indicated by the gray circles are shown at a higher magnification in (B) and (C). (B) and (C1) High power (60X) multiphoton imaging of the regions labeled (B) and (C). (C2) is the region indicated in (C1) with 5x digital zoom. Note the apparent proximity between the medial processes of the two B21 neurons.

Electrophysiological data confirming contact between medial parts of B21

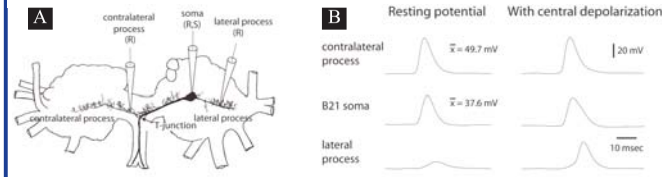


Coupling potentials were generated by triggering a spike in one B21 neuron (B, left). Cells were then lesioned as indicated in (A) and coupling potentials again measured (B, right). Note that removing the lateral process did not produce a significant effect on coupling potential amplitude (C).

Conclusion: Contact between the two B21 neurons is mediated via the medial process.

3 SPIKES ACTIVELY PROPAGATE THROUGHOUT MOST OF B21'S MEDIAL PROCESS WHEN IT IS PERIPHERALLY ACTIVATED AT ITS RESTING MEMBRANE POTENTIAL

Active propagation in the contralateral branch in the medial process when B21 is peripherally activated at its resting membrane potential



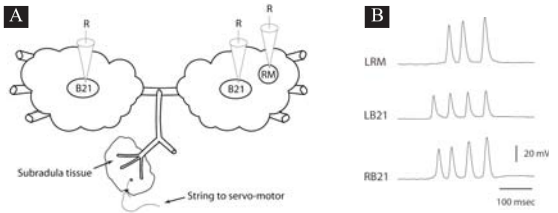
(A) Camera lucida drawing of B21 indicating electrode placement for the experiment shown in (B). R, indicates the use of an electrode for recording, S indicates current injection. (B) Left, simultaneous intracellular recordings from the lateral process, soma, and a branch of the medial process (the contralateral process) when B21 was peripherally activated at resting membrane potential. Note the full size spike in the contralateral process as compared to the attenuated depolarizing impulse in the lateral process. Right, simultaneous intracellular recordings from the lateral process, soma, and contralateral process when B21 was centrally depolarized prior to peripheral activation. Note that the lateral process was capable of active spike initiation, i.e., a full size action potential is now observed.

Consequences of contralateral spike propagation in B21 with peripheral activation at resting membrane potential

(C) Schematic based both on previous data and data obtained in the current study. Active spike propagation is indicated by dark red. The lighter red indicates electrotonic transmission, with the degree of attenuation indicated by the color. Note that active spike propagation tends to occur in medial regions of B21. Previously we have shown that central depolarization in these regions effects spike propagation to the lateral (output) process.

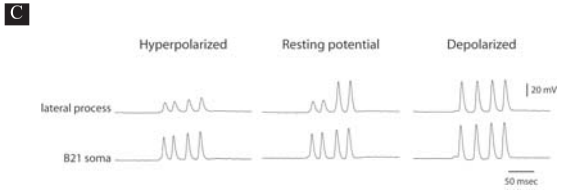
4 IF STIMULI CO-ACTIVATE RADULA MECHANOAFFERENTS, WILL SPIKE PROPAGATION TO THE LATERAL PROCESS BE OBSERVED WHEN B21 IS PERIPHERALLY ACTIVATED AT ITS RESTING MEMBRANE POTENTIAL?

Co-activation of radula mechanoafferents



(A) To co-activate radula mechanoafferents experiments were conducted in a semi-intact preparation with the subradula tissue attached. Intracellular recordings (R) were obtained from both left and right B21 neurons and an unidentified radula mechanoafferent (RM) in response to a brief pull. (B) Somatic intracellular recordings from the cells indicated in (A). Note all three neurons were activated by the stimulus. Four spikes were triggered in each B21 with each pull. Three spikes were triggered in the unidentified RM.

Active spike propagation to the lateral process of B21 is observed when radula mechanoafferent neurons are coactivated.



Peripheral RM activation with the stimulus described in (A) and simultaneous recording from the soma and lateral process of B21. Note that two of the peripherally triggered spikes did actively propagate to the lateral process when B21 was stimulated at its resting membrane potential (middle). As expected somatic hyperpolarization of B21 prevented lateral process spike propagation (left) and somatic depolarization resulted in active propagation of all triggered spikes (right).

SUMMARY AND CONCLUSIONS

When B21 is peripherally activated alone a spike propagation failure occurs at resting membrane potential. Spikes do not actively process to the lateral process (the output region of the neuron). In contrast, when a peripheral stimulus is applied that coactivates other radula mechanoafferent neurons, spike propagation to the lateral process can be observed. Thus, coactivation of radula mechanoafferent neurons can impact spike propagation in B21. This is likely to be a mechanism that couples afferent transmission to the strength of the applied peripheral stimulus.