stimulation indicated that there are several types of excitatory and inhibitory synaptic responses. Some of these synaptic responses decremented to repeated stimulation of the siphon but were not definitely related to habituation of the siphon withdrawal. Figure 2B shows a central neuron that responded to tactile stimulation of the siphon. Action potentials in these cells were not preceded by synaptic potentials, which suggests that the spike was initiated in a peripheral process of the cell and conducted to the soma. This also suggests that the neuron is a central sensory cell with processes in the periphery. However, this cell could also have been activated by a synapse with a peripheral sensory neuron. The arrangement of central sensory cells has been noted before in *Aplysia, Spisula,* and in the leech (*9, 19*). The latency of siphon contraction in Fig. 2B was approximately 100 msec, and the neuronal action potentials were concurrent with the onset of contraction. Since contraction was initiated at the same time that information reached the central neurons, it seems likely that the contribution of the central neurons to the siphon response in the intact animal must occur no sooner than the contraction initiated by the peripheral system.

At this time we have evidence that there are neurons in the isolated siphon preparation, as there are in other peripheral structures of *Aplysia* (*7, 20*). The number of neurons in the peripheral and central systems that mediate the siphon responses is unknown as is the nature of the receptors for light and tactile stimuli. Our results show that the nervous system which supports the adaptive behavior of siphon withdrawal is composed of a central and a peripheral system. These two systems influence each other and are, therefore, parts of an integrated system. Kupfermann *et al.* (*11*) proposed that the peripheral and central systems which control the gill withdrawal response of *Aplysia* are independent systems, yet state in the same paper that the systems may interact. Our results show that the peripheral system alone is capable of mediating a considerable amount of adaptive response and that the interaction between the central and peripheral systems may be quite complex.

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13. The jet of seawater (2-second duration) was produced by a commercially available Water Pik. This has been used previously (*16*).
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15. The sucrose block was arranged by positioning a glass pipette over the nerve to direct a stream of isotonic sucrose over the nerve. The block was complete in 4 minutes as verified by monitoring the electrical activity. Shutting off the sucrose flow unblocked the nerve in 30 seconds. A piece of tubing under the nerve siphoned off the used sucrose.
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Rat Origin of CHB Cells

Growing interest in neurobiology has created a great demand for established cell strains of neurological origin. Several established cell strains of both neuronal (*1*) and glial (*2*) origin have been provided. One glial cell strain, CHB, which was first reported by Lightbody *et al.* (*3*), has been derived from a human astrocytoma. We wish to warn the scientific community that existing cultures of CHB cells and lines derived from them, although possessing some differentiated characteristics of glial cells, are of rat origin and not human origin.

Cytological examination of the chromosome numbers and karyotypes of CHB cultures in several laboratories indicated that these cells are of rat origin. Furthermore, T. B. Shows (Roswell Park Memorial Institute) compared the starch gel electrophoretic patterns of three soluble enzymes (glucose-phosphate isomerase, aspartate aminotransferase, and malate dehydrogenase) present in extracts of CHB cells, WI-38 human cells, and rat kidney. The isozyme profiles confirmed that CHB cells were of rat origin. Cultures of CHB cells from several laboratories have been examined in hopes of discovering the human glial cell strain; however, no human strain has been found. The relation of CHB cells to other known rat glial cell strains has not been established, but cytological and biochemical differences indicate that CHB is not identical to the widely studied C-6 strain of rat glial cells (*2*).

Publications in which some or all of the results reported were obtained with CHB cells or derived cells are cited (*4*).

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