Serotonin-Induced Spike Narrowing in a Locomotor Pattern Generator Permits Increases in Cycle Frequency During Accelerations

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Satterlie, Richard A., Tigran P. Norekian, and Thomas J. Pirtle. Serotonin-induced spike narrowing in a locomotor pattern generator permits increases in cycle frequency during accelerations. J. Neurophysiol. 83: 2163–2170, 2000. During serotonin-induced swim acceleration in the pteropod mollusk Clione limacina, interneurons of the central pattern generator (CPG) exhibit significant action potential narrowing. Spike narrowing is apparently necessary for increases in cycle frequency during swim acceleration because, in the absence of narrowing, the combined duration of the spike and the inhibitory postsynaptic potential (IPSP) of a single cycle is greater than the available cycle duration. Spike narrowing could negatively influence synaptic efficacy in all interneuron connections, including reciprocal inhibitory connections between the two groups of antagonistic CPG interneurons as well as the interneuron-to-motoneuron connections. Thus compensatory mechanisms must exist to produce the overall excitatory behavioral change of swim acceleration. Such mechanisms include 1) a baseline depolarization of interneurons, which brings them closer to spike threshold, 2) enhancement of their postinhibitory rebound, and 3) direct modulation of swim motoneurons and muscles, all through inputs from serotonergic modulatory neurons.

INTRODUCTION

Transient changes in action potential shape have been documented as a primary means of altering synaptic efficacy in circuits in which the altered activity leads to behavioral changes. As notable examples, spike broadening has been found to produce synaptic changes that are involved in simple forms of learning (Baxter and Byrne 1989, 1990; Byrne and Kandel 1996; Ghirardi et al. 1992; Goldsmith and Abrams 1992; Hochner and Kandel 1992; Klein and Kandel 1978; Stark and Carew 1999; Sugita et al. 1997; Walters et al. 1983), in the initiation of feeding behavior (Gillette et al. 1980), and in peptide secretion (Whim and Kaczmarek 1998). In some instances, spike duration was shown to be directly related to the frequency of firing within a burst (Aldrich et al. 1979a,b; Coates and Bulloch 1985; Gillette et al. 1980; Ma and Koester 1995, 1996; Quattrocchi et al. 1994; Whim and Kaczmarek 1998) whereas in others, spike shape was altered by a variety of neuroactive peptides and amines (Abrams et al. 1984; Byrne and Kandel 1996; Critz et al. 1991; Ghirardi et al. 1992; Hochner et al. 1986; Rosen et al. 1989; Sugita et al. 1997). In general, increases in spike duration (broadening) appear to be associated with an enhanced synaptic efficacy and/or an elevated physiological or behavioral response (Baxter and Byrne 1990; Byrne and Kandel 1996; Goldsmith and Abrams 1992; Hochner and Kandel 1992; Hochner et al. 1986; Klein and Kandel 1978; Rosen et al. 1989; Sugita et al. 1992; Walters et al. 1983; for an exception see Spencer et al. 1989). We found a counterintuitive situation in which a clearly excitatory event (change from slow to fast swimming speed in the pteropod mollusk Clione limacina) is associated with significant spike narrowing in interneurons that comprise the primary pattern generator circuit for swimming. This decrease in spike duration occurs in parallel with an increase in pattern generator cycle frequency during the change to fast swimming. Both are specifically controlled by serotonergic inputs that serve as the main trigger for the speed change. This anomaly raises two important questions: what is the function of spike narrowing in a behavioral activity that represents a significantly elevated level of activity and, if spike narrowing is associated with a decrease in synaptic efficacy, how does the system achieve an overall increase in excitation?

Spike narrowing is a prerequisite for swim acceleration in Clione because maximum swim interneuron spikes and inhibitory postsynaptic potential (IPSP) durations are each in excess of 100 ms and the maximum wing-beat frequency is 5 Hz. Our data suggest that spike narrowing in Clione interneurons may be permissive, allowing up to a five-fold increase in cycle frequency. Any decrease in excitation that may be associated with narrowing is offset by other modifications both within and outside of the central pattern generator (CPG).

METHODS

Clione limacina were collected from the breakwater at Friday Harbor Laboratories, Friday Harbor, WA. Specimens were held in natural seawater in one-gallon jars in a refrigerator or were partially submerged in continuous-flow seawater tables. Animals were dissected in Sylgard-coated petri dishes containing seawater. Reduced preparations consisted of the central ganglia and wings, which were immobilized with cactus spines (Opuntia sp.). The preparations were treated for 3 to 5 min in 1 mg/ml protease (type XIV, Sigma, St. Louis, MO) to soften the ganglionic sheaths. Intracellular recordings were conducted with 2 M potassium acetate-filled microelectrodes with resistances of 10–40 MΩ. Electrophysiological signals were amplified, displayed, and recorded using conventional techniques. Intracellular stimulation was provided via amplifier bridge circuits.
For experiments on postinhibitory rebound, interneurons were isolated from the ganglion according to the techniques of Arshavsky et al. (1986). In short, once a stable penetration was achieved, slow pulling pressure was exerted on the micromanipulator so that the electrode pulled the penetrated cell from the ganglion. The process, which resulted in a totally isolated soma with a short segment of axon, typically took 30–90 min for complete isolation. If the membrane potential changed by 5–10 mV during the isolation technique and/or did not settle back to the original value, the preparation was discarded. Isolated cells maintained stable resting potentials for up to 3 h. Postinhibitory rebound (PIR) was induced in current-clamp recordings with hyperpolarizing currents that were adjusted to give PIR depolarizations of 5–20 mV. In two isolated interneurons, a range of hyperpolarizations was used to produce PIR of varying amplitudes, both in the absence and presence of 10^{-4} M serotonin. The latency from hyperpolarization release to PIR peak was measured from the termination of the injected hyperpolarization to the peak of the PIR depolarization. In two additional isolated interneurons, several pre- and post-serotonin PIRs were induced; however, the stimulus parameters used in the trial immediately before serotonin application were left unchanged for the trial immediately after serotonin addition.

Serotonin was applied to the recording dish with a graduated pipette into a known volume of saline to achieve the desired final concentration. Similar methods were used for application of tetraethylammonium bromide (TEA) and mianserin.

Action potential and IPSP duration measurements were made in the following ways. For statistical comparisons and graphic data, action potential durations were measured from the vertical inflection point indicated on Fig. 3C. For individual action potentials in which the inflection point was not evident, obvious inflection points on other action potentials from the same recording were used to estimate nonobvious inflections. Care was taken to make sure that no frequency changes occurred in these records. The resultant duration values eliminated initiating inputs such as synaptic inputs and postinhibitory rebound depolarizations, but also resulted in an underestimation of the total duration. For this reason, total duration measurements, taken from baseline takeoff to baseline return, were used when estimating the percentage of total interspike interval (ISI) occupied by action potentials.

**FIG. 1.**  
A: stimulation of a cerebral serotonergic neuron (top trace) resulted in acceleration of swim frequency as noted in a recording from a central pattern generator (CPG) swim interneuron (si). Note the baseline depolarization that accompanies the acceleration.  
B: higher speed trace of a swim interneuron (si) shows the difference between control action potentials and those that come after stimulation of a Cr-S neuron.
potentials. For these measurements, the baseline was estimated by running hard copy records at slow chart speed and estimating the most horizontal phase of the post action potential period in a record that included at least 15 cycles. For statistical comparisons, IPSP durations were measured at half amplitude in an attempt to control for amplitude-dependent differences. Again, for estimates of total IPSP duration as a percentage of ISI, total baseline initiation-to-baseline return measurements were recorded using the same baseline determination described for action potentials.

RESULTS

The swim CPG in the pedal ganglia of Clione consists of two groups of antagonistic interneurons coupled by monosynaptic reciprocal inhibitory synapses (Arshavsky et al. 1985c; Satterlie 1985). The interneurons exhibit strong postinhibitory rebound, which permits the pattern generator to cycle without tonic input from other parts of the nervous system (Satterlie 1985). Two groups of serotonergic neurons are involved in the slow-to-fast swimming speed change and are capable of initiating swimming in quiescent animals through modulatory inputs to CPG interneurons and swim motoneurons (Satterlie 1995; Satterlie and Norekian 1995). Serotonergic modulation of the swim CPG includes three significant changes that are intrinsic to CPG interneurons: baseline depolarization, enhancement of postinhibitory rebound, and spike narrowing.

These intrinsic changes contribute to the increase in cycle frequency that is characteristic of the change from slow to fast swimming.

During slow swimming, each CPG interneuron produced a single broad spike per swim cycle followed by a single inhibitory synaptic potential representing spike activity in antagonistic interneurons (Fig. 1). The mean action potential duration measured during periods of slow swimming was $94.93 \pm 8.26$ ms (SE), with a maximum spike duration of 120 ms ($n = 70$) (10 consecutive cycles were measured before an initiating stimulus in each of 7 preparations). In induced bouts of fast swimming, mean action potential duration was $76.34 \pm 7.79$ ms ($n = 70$) (10 consecutive cycles after the initiating stimulus in each of 7 preparations; $t$-test; $P < 0.0001$). After stimulation of a cerebral modulatory neuron (either Cr-SA or Cr-SP) (Satterlie and Norekian 1995), CPG interneurons exhibited a decrease in cycle period that outlasted the duration of modulatory neuron firing (Fig. 1 and dashed trace in Fig. 2). Cycle period decreased rapidly, down to nearly 50% of control in the first two cycles after the initiation of the modulatory input. During this period, spike duration showed a slower transient decrease and reached a minimum between the fourth and sixth poststimulus cycles (solid trace in Fig. 2; $n = 7$). A comparison of control with maximally narrowed action potentials showed that both had similar rising phase kinetics and a similarity in
the initial part of the falling phase (Fig. 3). In control action potentials, the latter portion of the falling phase included a distinct shoulder that was missing in the narrowed action potential.

In recordings in which a triggered burst of spikes in a cerebral modulatory neuron induced a wing-beat acceleration of 2–3 Hz, mean total spike duration in the prestimulus period accounted for 20% of the ISI whereas mean total IPSP duration was 32% of the ISI. In these measurements, total durations of spikes and IPSPs were used. Thus a combination of total spike and IPSP durations comprised 52% of the ISI. As shown in Table 1, spike, IPSP, and spike-IPSP combination percentages increased after the cerebral modulatory neuron was stimulated. The fastest swim frequency encountered in *Clione* was 5 Hz whereas the shortest spikes and IPSPs were ~50 and ~75 ms in duration. If these numbers are used to represent maximal acceleration, Table 1 shows that as swimming speed increases, spikes and IPSPs account for larger percentages of the total ISI. If no spike narrowing were to occur, we could extrapolate percentages for 3, 4, and 5 Hz swimming. For these hypothetical estimates, 2 Hz spike and IPSP duration data were held

<table>
<thead>
<tr>
<th>Cycle Frequency</th>
<th>Spike Duration</th>
<th>IPSP Duration</th>
<th>Spike-IPSP Duration</th>
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<tr>
<td>Normal—spike narrowing</td>
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<tr>
<td>2 Hz*</td>
<td>20%</td>
<td>32%</td>
<td>52%</td>
</tr>
<tr>
<td>3 Hz†</td>
<td>24%</td>
<td>36%</td>
<td>60%</td>
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<tr>
<td>5 Hz‡</td>
<td>25%</td>
<td>38%</td>
<td>63%</td>
</tr>
<tr>
<td>Hypothetical—no spike narrowing§</td>
<td></td>
<td></td>
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<tr>
<td>3 Hz</td>
<td>30%</td>
<td>48%</td>
<td>78%</td>
</tr>
<tr>
<td>4 Hz</td>
<td>40%</td>
<td>64%</td>
<td>104%</td>
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<tr>
<td>5 Hz</td>
<td>50%</td>
<td>80%</td>
<td>130%</td>
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CPG, central pattern generator; IPSP, inhibitory postsynaptic potential. * Data collected before stimulation of cerebral modulatory neuron. † Data representing maximal cycle frequency and maximal shortening of spikes and IPSPs. § Hypothetical percentages representing lack of spike or IPSP narrowing (durations taken from 2 Hz experimental data).

FIG. 4. A: spike narrowing in a CPG interneuron induced by bath application of $10^{-5}$ M serotonin (5-HT). B: depolarization of a CPG interneuron sufficient to produce an acceleration in swimming frequency did not produce spike narrowing.
FIG. 5. A: relationship between postinhibitory rebound (PIR) amplitude and hyperpolarization release-to-PIR peak latency is linear for both control (small squares) and serotonin-bathed (large circles) trials in acutely isolated CPG interneurons. B: bath-applied serotonin enhances the amplitude of the PIR in response to identical pre- and postserotonin current injections. Hyperpolarization-release-to-PIR peak latency is shorter for the larger PIR depolarization. The two traces were superimposed to better show PIR differences and thus do not show the serotonin-induced baseline depolarization in the serotonin trace.

constant as cycle frequency was increased. Table 1 shows that fast swimming above 3 Hz in the absence of spike/IPSP narrowing would not be feasible because the combination of spike and IPSP durations would be longer than the interspike interval.

Bath-applied serotonin (10 μM) produced spike narrowing of a magnitude comparable to that observed after stimulation of cerebral modulatory neurons (Fig. 4A). Similar narrowing was observed in “spontaneous” accelerations. Narrowing triggered by both exogenous serotonin and stimulation of cerebral modulatory neurons was reversibly blocked by the serotonin antagonist mianserin (10 μM).

In some preparations, ongoing swimming activity showed a slightly elevated swim frequency and decreased spike durations (40–50 ms) for long periods. In these instances, neither exogenous serotonin nor stimulation of cerebral modulatory neurons produced further shortening. In preparations in which interneurons showed narrow spikes for relatively long periods of time, 10 mM TEA was able to increase spike duration to values similar to those seen during normal slow swimming (80–120 ms). Once broadened by TEA, interneron spikes could not be shortened by subsequent application of serotonin or stimulation of cerebral modulatory neurons. Similarly, in slow swimming preparations (long-duration action potentials), TEA did not cause further broadening and subsequent serotonin application failed to produce spike narrowing. Thus TEA appears to block serotonin-induced spike narrowing in CPG interneurons.

Aside from neuromodulation, additional mechanisms have been shown to trigger changes in spike duration in other preparations. For example, frequency-dependent spike broadening has been shown in several molluscan cells (Gillette et al. 1980; Ma and Koester 1996; Whim and Kaczmarek 1998). Also, spike narrowing in jellyfish was found to be associated with an increase in firing synchrony in electrically coupled neurons, as well as with the level of depolarization of these neurons (Spencer 1981; Spencer et al. 1989). In Clione, spike duration is not dependent on interspike interval because spike narrowing occurs with a different time course than do changes in interspike interval (Fig. 2). Furthermore, in measurements taken from control and swim-accelerated preparations, spike duration showed no significant statistical correlation with interspike interval (linear regression — control: r = 0.2526, P = 0.06; swim-accelerated: r = 0.1383, P = 0.20). Direct depolarization of recorded CPG interneurons, both of the magnitude observed during serotonergic modulation and at higher values sufficient to produce higher frequency cycling, did not alter spike duration (Fig. 4B).

Although it is difficult to draw concrete conclusions concerning synaptic efficacy changes in current-clamp recordings of the type used in this study, an analysis of reciprocal inhibitory connections between CPG interneurons did suggest that spike narrowing was accompanied by a similar decrease in IPSP duration. Mean IPSP duration at half amplitude in control preparations was 108.26 ± 19.92 ms (n = 60 from six preparations; see Fig. 1B) whereas in measurements taken in the 10 cycles after stimulation of a cerebral modulatory neuron, the mean IPSP duration was 90.83 ± 13.96 ms (n = 60 from six preparations; t-test, P = 0.01).

In addition to spike narrowing, exogenous serotonin and cerebral modulatory inputs produced a 1–5 mV baseline depolarization in CPG interneurons that peaked 4 or 5 cycles after initiation of the modulatory input and that slightly outlasted the period of serotonin application/modulatory input (Fig. 1A). As with spike narrowing, the baseline depolarization was blocked by mianserin application.

Cerebral modulatory neuron inputs, as well as exogenous serotonin, produced one other notable change in CPG interneurons in addition to the increase in cycle frequency, spike narrowing, and baseline depolarization. PIR is a major property of CPG interneurons and it is large enough that an IPSP from one group of CPG interneurons is sufficient to produce a rebound spike in the antagonistic interneurons (Satterlie 1985). Under the influence of exogenous serotonin, the amplitude of
the PIR depolarization was enhanced by an average of 26.8% (n = 12; t-test, P < 0.001) (Fig. 5B). In the subthreshold range for spike production, the latency from hyperpolarization-release to PIR peak decreased with increasing size of the PIR depolarization (Fig. 5A) (linear regression; r = -0.8361, P < 0.0001).

**DISCUSSION**

The acceleration from slow to fast swimming in *Clione* involves alterations in CPG interneuron activity through a combination of synaptic and modulatory inputs. Changes to CPG interneurons include shortening of the cycle period through synaptic inputs from type 12 and delayed V-phase interneurons (Arshavsky et al. 1985d, 1989). In the present study, we show that swim acceleration is also accompanied by spike narrowing, enhancement of postinhibitory rebound, and a 1–5 mV baseline depolarization of CPG interneurons. With the exception of spike narrowing, all of these changes serve to either increase the level of excitation in the CPG and/or increase the frequency of CPG cycling. Spike narrowing is problematic because it has the potential of decreasing synaptic efficacy in connections that are vital for CPG and motoneuron function. Serotonin application or activation of the serotonin-immunoreactive cerebral modulatory neurons Cr-SA and Cr-SP (Satterlie and Norekian 1995) induces a transient decrease in spike duration of 20–30% in most preparations, but was recorded at nearly 50% in a few preparations. Spike narrowing can be blocked by the serotonin antagonist mianserin regardless of whether serotonin is added to the preparation or its release is triggered by activation of the cerebral modulatory neurons. Other potential contributors to spike narrowing, including frequency dependency (Aldrich et al. 1979a,b; Coates and Bulloch 1985; Gillette et al. 1980; Jackson et al. 1991; Ma and Koester 1995, 1996; Quattrocki et al. 1994; Whim and Kaczmarek 1998), increase in firing synchrony in electrically coupled interneurons (Spencer 1981), and depolarization-induced narrowing (Spencer et al. 1989), have been ruled out as major factors.

Our data suggest that spike narrowing may involve a serotonin-induced increase in potassium conductance because TEA not only blocked spike narrowing in slow-swimming preparations, but also because TEA increased spike duration to normal slow-swimming levels in preparations in which the interneuron spikes were already short (fast swimming). A decrease in potassium currents has been implicated in spike broadening in several cells (Aldrich et al. 1979a,b; Critz et al. 1991; Jackson et al. 1991; Ma and Koester 1995, 1996; Quattrocki et al. 1994) and includes the closure of a serotonin-sensitive potassium channel (Baxter and Byrne 1990; Goldsmith and Abrams 1992) or multiple potassium channels (Byrne and Kandel 1996; Critz et al. 1991; Ma and Koester 1996). Although the cellular mechanisms of serotonin-sensitive spike narrowing in *Clione* require more detailed investigation, the present experiments lend corroborative support for the role of serotonin in spike narrowing in *Clione* CPG interneurons and suggest that the spike narrowing is likely to involve alteration of potassium currents.

Spike broadening has been shown to be involved in synaptic facilitation (Abrams et al. 1984; Baxter and Byrne 1989, 1990; Byrne and Kandel 1996; Critz et al. 1991; Ghirardi et al. 1992; Goldsmith and Abrams 1992; Hochner and Kandel 1992; Hochner et al. 1986; Klein and Kandel 1978; Rosen et al. 1989; Stark and Carew 1999; Sugita et al. 1992, 1997; Walters et al. 1983) whereas spike narrowing produces the opposite effect—synaptic depression (Rosen et al. 1989). The potential impact of spike narrowing on synaptic efficacy in *Clione* CPG interneurons is extremely important because outputs of these cells include reciprocal inhibitory connections to antagonistic CPG interneurons as well as excitatory synaptic connections to synergistic motoneurons and inhibitory connections to antagonistic motoneurons (Arshavsky et al. 1985a–c; Satterlie 1993; Satterlie and Spencer 1985). Changes in synaptic efficacy are difficult to demonstrate in the reciprocal inhibitory connections of the CPG because excitatory modulation not only results in spike narrowing but also involves baseline depolarization, enhancement of postinhibitory rebound, and recruitment of synaptic inputs from type 12 and delayed V-phase interneurons (Arshavsky et al. 1985d, 1989). Although we have not yet conducted a detailed mechanistic investigation of IPSP changes, we have shown that IPSP durations decrease after serotonin application or stimulation of cerebral modulatory neurons, and that these decreases are of similar magnitude to spike duration decreases.

If it is assumed that this narrowing of IPSPs represents a negative, or at least a neutral, influence on synaptic efficacy within the CPG during swim acceleration, then action potential narrowing requires comment in terms of its significance to CPG function during periods of acceleration. One possible explanation emerges when the percentages of total ISI occupied by action potentials and IPSPs are considered (Table 1). As noted in RESULTS, swim acceleration is associated with an increase in the total percentage of the cycle period occupied by the combined action potential/IPSP of CPG interneurons. Without significant spike (and IPSP) narrowing, wing-beat frequencies >3 Hz would be impossible (spike and IPSP would occupy more than 100% of the cycle period). Thus one functional aspect of spike narrowing simply may be to allow higher wing-beat frequencies during fast swimming.

If spike narrowing merely permits higher wing-beat frequencies and, as a consequence, presents either a detrimental or neutral influence on overall excitability in the swimming system, then compensatory mechanisms must exist to maintain or raise the overall level of excitation in the CPG and effectors. Three types of such serotonin-induced modifications occur within the CPG. 1) The baseline depolarization of CPG interneurons brings the electrically coupled network of neurons closer to the firing threshold. 2) The enhancement of postinhibitory rebound by serotonin could, on its own, explain an increase in cycle frequency caused by the faster and larger depolarization toward spike threshold. However, absolute PIR amplitude would depend on a complex interplay between several conflicting factors. Preliminary data suggest that PIR amplitude is positively correlated with both the duration and amplitude of the “conditioning” hyperpolarization and negatively correlated with baseline depolarization above the resting membrane potential. Two serotonin effects would thus tend to counteract PIR enhancement: decrease in interneuron IPSP duration and baseline depolarization. Data from isolated interneurons indicated that serotonin increases PIR amplitude despite baseline depolarization; however, the influence of decreased IPSP duration is yet to be tested. 3) Reconfiguration of the CPG during fast swimming involves the addition of direct
synaptic inputs to the CPG from recruited interneurons (such as type 12 interneurons). The main function of CPG reconfiguration is to shorten the cycle period through “early” inhibitory and excitatory synaptic inputs to CPG interneurons. Together, these changes promote and allow an increase in cycle frequency. In addition, cerebral modulatory neuron inputs induce firing activity in general excitor motoneurons, increase activity in small motoneurons, and excite pedal modulatory neurons (Pd-SW). These latter inputs serve to increase wing contractility. The combination of increased cycle frequency and increased wing contractility results in acceleration from slow to fast swimming.

Parallel modulatory inputs serve to excite the swimming system through alterations extrinsic to the CPG. These include serotonin-induced baseline depolarization and increase in firing frequency in the subset of wing motoneurons that are active during both slow and fast swimming (Satterlie 1993; Satterlie and Norekian 1995, 1996). Similarly, a second subset of swim motoneurons is depolarized and recruited into firing activity during the serotonin-induced speed change. Finally, pedal serotonergic (Pd-SW) neurons (Satterlie 1995) provide modulatory inputs to the swim musculature and increase the force of ongoing wing contractions without altering wing-beat frequency. Under the influence of the cerebral modulatory neurons, the Pd-SW neurons show an increase in firing frequency that, in turn, results in an increase in wing contractility (Satterlie 1995; Satterlie and Norekian 1995). Thus modulatory output of the cerebral modulatory neurons is designed to increase excitation to the swimming system by selectively increasing wing contractility in three ways. They contribute to the recruitment of a subset of swim motoneurons that, in turn, recruit fast-twitch wing musculature (Satterlie 1991); and they increase the activity of peripheral modulatory neurons (Fig. 6).

Accelerating from slow to fast swimming in Clione involves a variety of modulatory changes to the CPG interneurons, swim motoneurons, and swim musculature. All of these changes except one appear to increase the level of excitation in the swimming system. The one possible exception is spike narrowing in CPG interneurons, which appears to play a permissive role in allowing significant increases in wing-beat frequency. Any deleterious effects of spike narrowing on CPG and swim system function are counteracted by compensatory mechanisms that occur at all levels of the swimming system. The need for a combination of shortened cycle period and increased effector activity represents a common problem for rhythmic motor systems that are capable of significant cycle frequency accelerations. It will be of interest to see if other motor systems solve this problem by including both permissive and compensatory mechanisms similar to those found in Clione.

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