*J. exp. Biol.* (1982), **98**, 415–428
*With 9 figures*
*Printed in Great Britain*



# EFFECTS OF TEMPERATURE ON THE ENDOGENOUS ACTIVITY AND SYNAPTIC INTERACTIONS OF THE SALIVARY BURSTER NEURONES IN THE TERRESTRIAL SLUG *LIMAX MAXIMUS*

BY DAVID J. PRIOR AND DEBRA S. GREGA

*Physiology Group, School of Biological Sciences, University of Kentucky, Lexington, Kentucky* 40506, *U.S.A.*

(*Received* 5 *November* 1981)

SUMMARY

(1) The activity of the endogenously active salivary burster neurones (SBs) shows temperature acclimation and has characteristic cold and warm blockade temperatures.

(2) Temperature acclimation affects the upper and lower limits of the temperature range over which SBs are active. The absolute range, in centigrade degrees, during warming, is unaffected by acclimation.

(3) Acclimatization of burster activity is a slow response to the mean ambient temperature.

(4) There is increased synchrony of activity between the right and left salivary bursters at low temperature which is correlated with an increased electrical coupling between the SBs and protractor motoneurones (B7s). There is a corresponding increase in the input resistance of B7 at low temperatures.

## INTRODUCTION

Temperature is one of the most significant environmental conditions, especially for poikilothermic animals. Since neuronal activity is particularly temperature sensitive, maintenance of appropriate nervous function and the resultant behavioural activities is a crucial problem for all poikilothermic animals.

Different neurone functions are known to have different temperature sensitivities (see Lagerspetz, 1974). It is known for example, that specific components of an animal's behaviour may function over quite different temperature ranges (e.g. Kivivuori, 1980; Moffett & Wachtel, 1976; Roots & Prosser, 1962; Staszak & Mutchmor, 1973). In a crayfish, for example, the righting reflex is blocked by raising the temperature to 19 °C, while the tail-flip reflex is stable up to 32 °C (Kivivuori, 1980). In some vertebrates and invertebrates, the hierarchy of temperature sensitivity of behavioural responses has been correlated with the thermal sensitivity of specific neural elements (Moffett & Wachtel, 1976; Prosser & Nagai, 1968; Friedlander, Kotchabhakdi & Prosser, 1976). In general, behavioural responses involving central

nervous system control appear to be more temperature labile than peripheral reflexes (Lagerspetz, 1974).

Long-term exposure to certain temperature regimes can result in compensatory responses (i.e. acclimation of the nervous system) among which are changes in the range of temperature over which neurones are active (e.g. Lagerspetz & Talo, 1967; Langley, 1979*a*, *b*; Figs. 2–4 in the present results), changes in the resting membrane potential (e.g. Marchiafava, 1970; Carpenter, 1967) and changes in membrane permeability, and the properties of ion pumps (e.g. Merickel & Kater, 1974; Zecevic & Levitan, 1978).

To study the effect of temperature on the interactions between neurones involved in the control of specific motor patterns, we have examined the effects of both long-term and short-term changes in temperature on the activity of the salivary burster neurones (SBs) in the terrestrial slug, *Limax maximus*. The paired SBs (Fig. 1) are autoactive motoneurones, each of which innervates the ipsilateral salivary duct (Prior & Gelperin, 1977; Beltz & Gelperin, 1980*a*, *b*). The basic pattern of endogenous SB activity is composed of cyclical bursts of impulses (Fig. 1), which during expression of the feeding motor programme (FMP) become synchronized with the protraction phase of the FMP (Prior & Gelperin, 1977; Gelperin, Chang & Reingold, 1978). This synchronization is due to electrotonic coupling between the SBs and protractor neurones such as RB7 and LB7 (see Fig. 1) which results in SB activity being modified by the synaptic drive of the FMP.

In the present study we have examined the effects of temperature acclimation on the endogenous activity of the salivary bursters and the effects of short-term temperature changes on their interaction with other neuronal elements in the salivary-feeding system.

## METHODS AND MATERIALS

The specimens of *Limax maximus* used in this study were collected locally or were reared in the laboratory from eggs. No differences were noted between collected or laboratory-reared animals. The slugs were kept in plastic containers lined with wet paper towelling and fed with rat chow and vegetables. All groups of slugs were acclimated for 2–3 weeks in controlled light and temperature conditions. Initially an L:D cycle of 12/12 h was used for both 5 and 25 °C groups. 'Spring animals' were generated from 25 °C animals by exposing them to an L:D cycle of 15/9 h and a temperature cycle of 27 °C, day/21 °C night.

Brains were dissected from anaesthetized animals in cold saline (1–4 °C) and placed in the recording chamber which had been pre-cooled to 1–4 °C. With the brains at this temperature, suction electrodes were applied to the salivary nerves and buccal ganglion neurones were penetrated with single- or double-barrelled glass microelectrodes for standard intracellular recording and current injection procedures. Brain temperature was controlled by a Peltier device and monitored with a YSI recording telethermometer. The thermistor probe, which had about the same dimensions as the brain, was fixed to the bottom of the recording chamber next to the brain.

The standard saline had a final composition of: $Na^+$ = 55·6 mM, $K^+$ = 4·2 mM, $Ca^{2+}$ = 7·0 mM, $Mg^{2+}$ = 4·6 mM, $Cl^-$ = 80·3 mM, $H_2PO_4^-$ = 0·2 mM, $HCO_3^-$ =

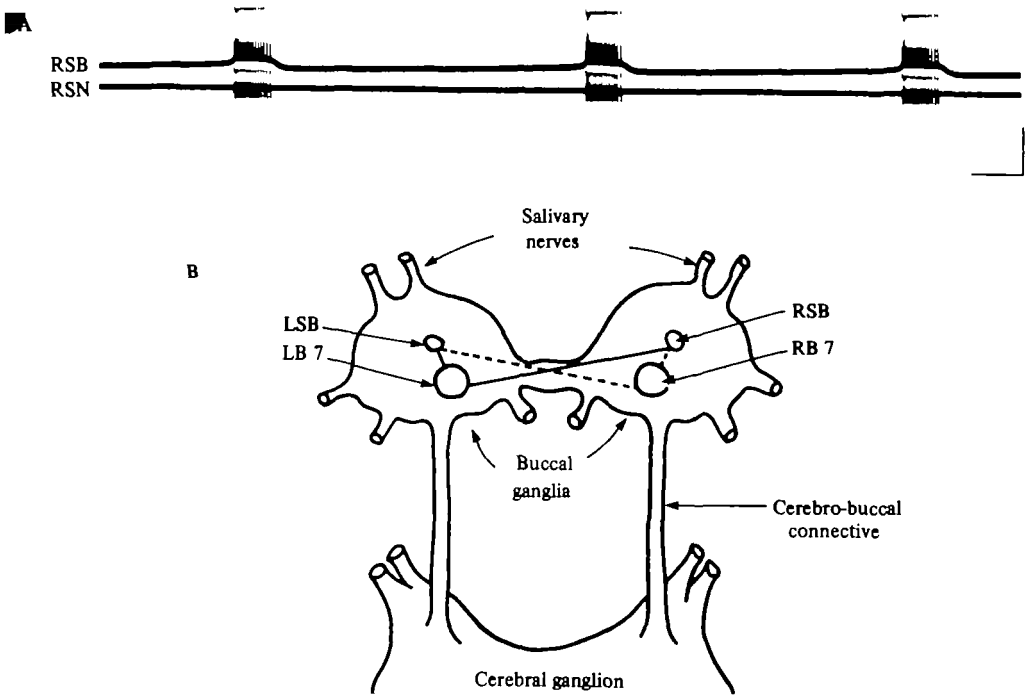


Fig. 1. (A) is an example of a simultaneous recording from the soma of the right salivary burster neurone (RSB; intracellular) and the corresponding RSB axonal activity (recorded extracellularly) in the right salivary nerve (RSN). This is the typical endogenous burst pattern recorded from the burster at 20 °C. (B) illustrates the brain–buccal ganglia preparation with the brain (cerebral ganglion shown) and buccal ganglia attached by the cerebral–buccal connectives. The salivary nerves (RSN, LSN), the somata of the salivary bursters (RSB, LSB) and the somata of protractor motoneurones (RB7, LB7) are illustrated. The calibration for the RSB recordings in A are 60 mV, 1·0 s.

5·0 mM and glucose = 5·0 mM. In increased $Mg^{2+}$ saline ($Mg^{2+}$ = 27·6 mM) the concentration of $Ca^{2+}$ was lowered to 0·5 mM.

In the experiments illustrated in Figs. 3–5, the temperature of the preparation was changed in a cyclical manner from near zero to 25–28 °C, and back to zero. This full cycle was repeated three times on each preparation. A complete temperature cycle (zero to 28 °C to zero) took approximately 15–20 min.

## RESULTS

Each salivary burster neurone innervates the ipsilateral salivary duct musculature by way of a single axon in the salivary nerve. Simultaneous recordings from the soma of the right SB and the right salivary nerve illustrate the presence of the SB axon (Fig. 1). Fig. 1 also shows the known electrotonic connexions between certain protractor motoneurones (RB7, LB7) and the SBs (Prior & Gelperin, 1977).

During our initial studies of temperature effects we found the endogenous activity of the bursters to be particularly temperature-sensitive. Variations in temperature altered the duration and frequency of bursts and, at certain high and low temperatures,

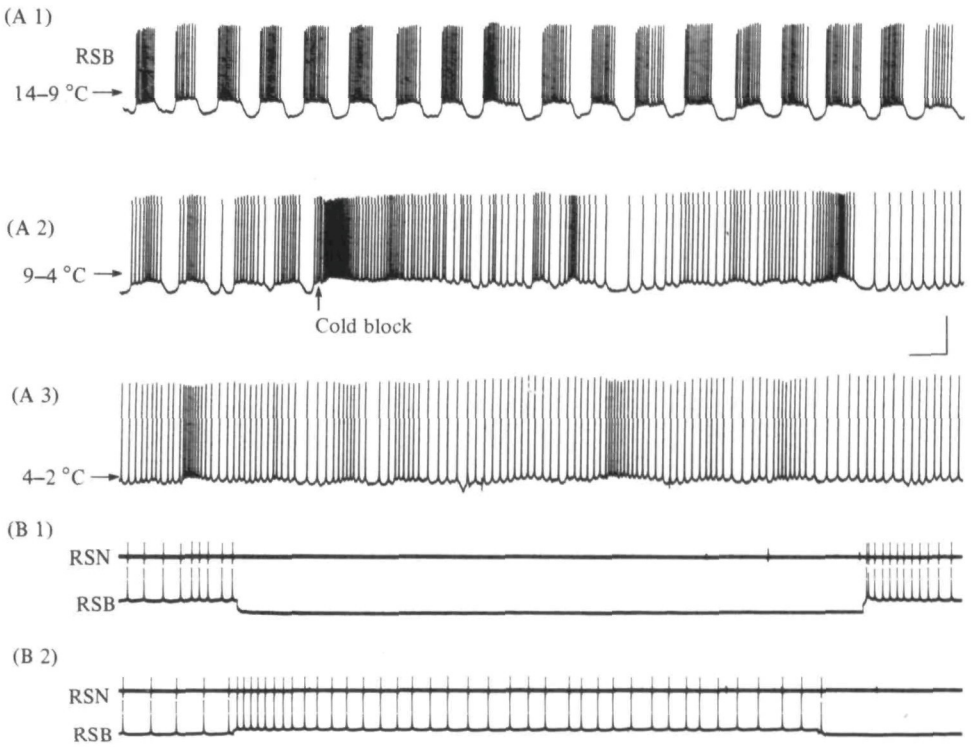


Fig. 2. (A) Continuous intracellular recording from a salivary burster during progressive reduction in temperature. A reference potential (arrows) is indicated in each trace to illustrate the depolarization that occurs at reduced temperature. The frequency of impulses within a burst declines and cold block of burst activity occurs at about 5 °C. This burster was from a winter slug and continued to be tonically active following cessation of burst activity. Upper calibration for (A) is 30 mV, 10 s. (B) Simultaneous recordings from the right salivary nerve (RSN) and the some of the right burster (RSB; intracellular). This was a 'cold-blocked' winter burster. Neither hyperpolarization (B1) nor depolarization (B2) resulted in reinitiation of burst generation. Calibration for RSB in B is 87 mV, 4 s.

burst activity was blocked (Figs 2, 3). The decline in intensity of burst activity at reduced temperatures was accompanied by progressive depolarization of the SBs (Fig. 2) similar to that which has been reported for several other types of neurones (e.g. Konishi & Kravitz, 1978; Merickel & Kater, 1974; Carpenter, 1967).

*Endogenous SB activity*

The burst frequency and the frequency of impulses within a burst declined with reduced temperature and at the cold block temperature (3–5 °C), the SBs became either silent or tonically active (depending upon the season; Figs 2, 3). To determine if the change in resting potential alone was responsible for cessation of burst generation, cold, tonically active SBs were depolarized and hyperpolarized (Fig. 2B). These alterations of membrane potential failed to reinitiate bursting in the SBs. This was probably due to the independence of membrane potential and burst generation in their responses to temperature (see Carpenter, 1967).

The sensitivity of SB activity to long-term changes in temperature was examined

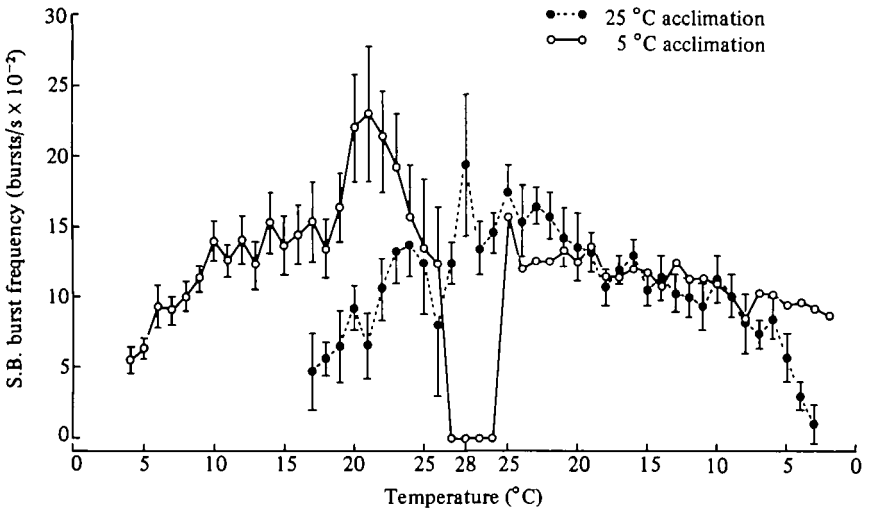


Fig. 3. This graph shows the relationship between temperature and burst frequency of the salivary burster during a temperature change from near 0 to 28 °C and back again, a full cycle taking approximately 15–20 min. The responses of bursters from 25 and 5 °C acclimated slugs are illustrated. Each point represents the mean (±S.E.M.) of 7–16 measurements from eight 25 °C acclimated slugs and six 5 °C acclimated slugs.

using slugs acclimated to 25 or 5 °C. After 2–3 weeks of acclimation, isolated brain–buccal ganglia preparations were made from animals in each temperature group. The responses of the SBs to cyclical changes in temperature were examined by recording from the salivary nerves (see Fig. 2). Fig. 3 illustrates the effect of temperature cycles on the activity of SBs from 25 and 5 °C acclimated slugs. As a preparation was warmed from near zero to 28 °C, 25 °C SBs were silent until the temperature reached 17–20 °C whereupon bursting activity was initiated. In 5 °C SBs however, burst activity began when the preparation had been warmed to 4–5 °C. Thus the 'on-point' of burst activity was about the same as the acclimation temperature of the slug from which the preparation had been made.

25 °C SBs were active from about 17–20 °C to about 35 °C, whereas the 5 °C SBs were active from 4–5 °C to about 25 °C. The absolute magnitude of the temperature range in both cases was about 20 deg. C. Therefore temperature acclimation affects the limits, but not the magnitude of the temperature range over which the SBs were active.

We tested the possibility that the acclimation effect was due to a change in input resistance of the SBs. Using double-barrelled microelectrodes we measured the input resistance of 5 and 25 °C SBs. Measurements were made during the longer interburst interval of preparations cooled to 10 °C. The input resistances of 5 and 25 °C SBs were not significantly different (5 °C SBs: 34·4, 27·4, 40·0, 38·6 mΩ, $\bar{x} = 35{\cdot}1 \pm 5{\cdot}6$ mΩ S.D.); (25 °C SBs: 26·0, 50·0, 31·5, 21·0 mΩ, $\bar{x} = 32{\cdot}1 \pm 12{\cdot}6$ mΩ S.D.).

Acclimation of the temperature range for activity was not observed in all *Limax* neurones. For example, in buccal neurone B1 (not involved in the feeding-salivary system) there was no effect of the acclimation temperature on the 'on-point' or

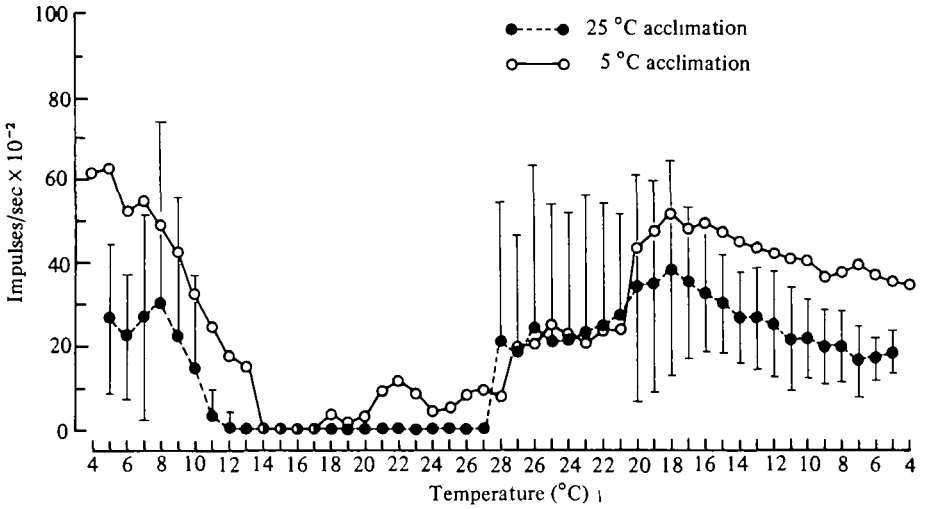


Fig. 4. This graph illustrates the responses of buccal neurone B1 to temperature cycles (as in Fig. 3). The impulse frequency of this tonically active neurone is plotted against temperature. The responses of neurones from 25 °C and 5 °C acclimated slugs are shown. Each point represents the mean (±S.E.M.) of measurements from six 25 °C acclimated slugs and four 5 °C acclimated slugs.

'off-point' for activity. Although the mean impulse rate was generally higher in the 5 °C neurones, the large variance around the means suggested that the differences between the 5° and 25 °C neurones were not significant (Fig. 4).

*Temperature acclimatization of SB activity*

There was a variation in the temperature responses of SBs from slugs collected in the winter (or laboratory animals in L:D, 14:10 or 12:12) and those collected in the summer. SBs from winter slugs were tonically active below the cold block temperature for bursting activity while SBs from summer animals were silent at the same low temperature (Fig. 2). This seasonal effect could be abolished by maintaining winter slugs in an L:D cycle of 15:9 and a day–night temperature cycle of 27 °C: 21 °C. SBs from slugs kept in these conditions responded to temperature cycles the same as SBs from summer slugs.

To determine how rapidly temperature acclimatization can occur in the field, we collected three slugs at 8 °C (3·00 a.m. one November day) and removed them directly to the laboratory where SB responses to temperature cycles were immediately measured. The 'on-points' of the SBs were between those of 5 and 25 °C SBs (14, 14, 15 °C). Later the same day (1.30 p.m.) three more slugs were collected from the same site which then had an air temperature of 24 °C. The 'on-points' of the SBs in this group (13, 13, 16 °C) were not significantly different from those of the earlier group. Rather than being correlated with the temperature at the time of collection, the 'on-points' of the SBs were close to the mean of the daily temperature extremes.

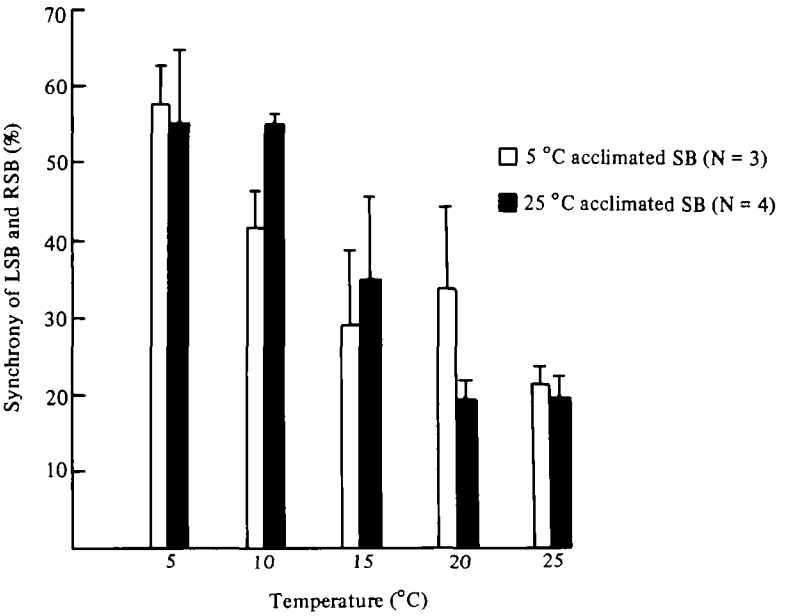


Fig. 5. This graph illustrates the relationship between LSB–RSB synchrony and temperature. Each bar represents the mean number of LSB bursts occurring during an RSB burst (i.e. synchrony). From 13–57 RSB bursts were measured in each preparation (three 5 °C slugs; four 25 °C slugs) at each temperature. The bars represent S.E.M.

*Temperature effects on SB–SB interaction*

The effects of temperature on the interaction between right and left salivary bursters was examined by means of simultaneous extracellular recordings from the salivary nerves (SNs) during cyclical temperature changes. As the temperature was increased there was a decrease in the occurrence of simultaneous SB–SB bursts (Fig. 5). Acclimation temperature seemed to have no consistent effect on this temperature-dependent variation in SB–SB synchrony. Although the temperature dependence of SB–SB synchrony was usually obvious, there was considerable variation in the burst patterns in different preparations and at different temperatures. In some cases there was considerable SB–SB synchrony at both high and low temperatures while in some there was no apparent synchrony at any temperature (20% of the preparations). For this reason many measurements from several preparations were pooled to illustrate temperature-dependent synchrony of the SBs (Fig. 5).

The majority of synchronous SB–SB bursting occurred following spontaneous multi-unit bursts that occurred simultaneously in both SNs (Fig. 6). These bursts could be blocked with high $Mg^+$ saline thus suggesting the involvement of chemically mediated synaptic input. Simultaneous generation of prolonged bursts in each SB constituted a second type of synchronizing activity (Fig. 7). These SN bursts involved simultaneous activation of both SBs and resulted in synchronization of subsequent SB activity. Both the burst frequency and the duration of subsequent SB bursts were increased (Figs. 6, 7A, B). In addition, the phase relationship of the burst patterns was altered so that the SBs generated synchronous bursts (Fig. 6).

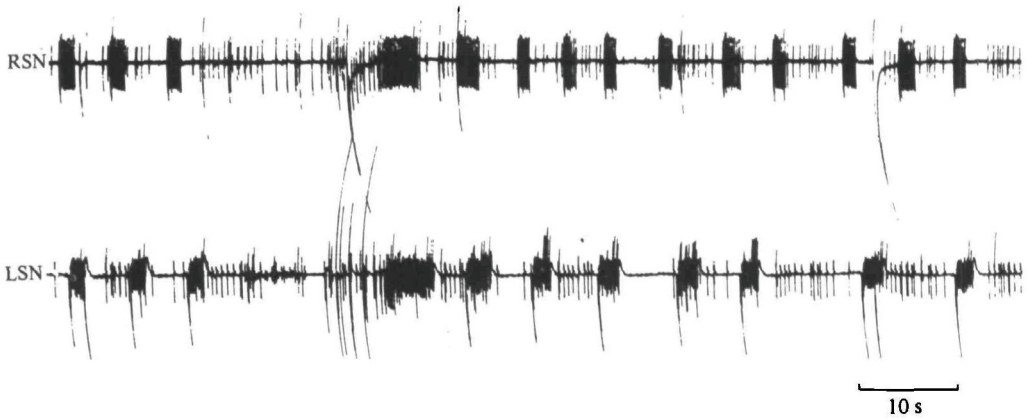


Fig. 6. This simultaneous recording from both right and left salivary nerves (RSN, LSN) illustrates the effect of a multi-unit synchronizing burst on the activity of the right and left salivary bursters. Following the prolonged synchronizing burst, the first subsequent bursts of the bursters are longer and occur synchronously. Calibration is 10 s.

Temperature dependence of the synchrony between SBs was illustrated by comparing the responses of the SBs to synchronizing bursts at three temperatures in a single isolated brain preparation (Fig. 7). At 18 °C there was one simultaneous SB–SB burst following a prolonged synchronization burst (Fig. 7A), at 13 °C both burst duration and frequency were increased (Fig. 7B), and at 8 °C, a single synchronizing burst resulted in four overlapping SB–SB bursts (Fig. 7C). This alteration of the phase relationships of the SBs was especially well illustrated by preparations in which the SBs had different burst frequencies. In Fig. 8A, the phase shift caused by the synchronizing burst resulted in four simultaneous SB–SB bursts.

In addition to the transient synchronization of the SBs, in many preparations, the activity of one SB seemed to activate the other. This was especially noticeable at low temperatures in some preparations where the endogenous activity of the SBs was highly synchronized (Fig. 8B). The timing and frequency of SB bursts and even the number of impulses composing bursts were essentially identical. This suggested that synchronization might also involve mutually excitatory interactions between the SBs and connected neurones. It was possible that chemically mediated synaptic input shared by the SBs could have been involved. It was also possible that conductance changes resulting from chemical synaptic input had altered the efficacy of electrotonic coupling involving the SBs (e.g. Carew & Kandel, 1976; B7-SB in Fig. 1).

To test these possibilities we examined the temperature sensitivity of SB–SB synchrony in high $Mg^{2+}$ saline. This saline blocked junction potentials in the salivary duct musculature, and the compound e.p.s.p.s in the protractor neurone, B7, resulting from electrical stimulation of buccal root 1. In high $Mg^{2+}$ saline there was usually an increase in the duration of SB bursts (probably due to reduction in the $Ca^{2+}$ dependent slow $K^+$ current responsible for the interburst hyperpolarization in 'bursters'; Meech, 1979). There was, however, no measurable change in the temperature-sensitive SB–SB synchrony, which suggested that chemically mediated synaptic interactions were not necessary for the observed synchrony.

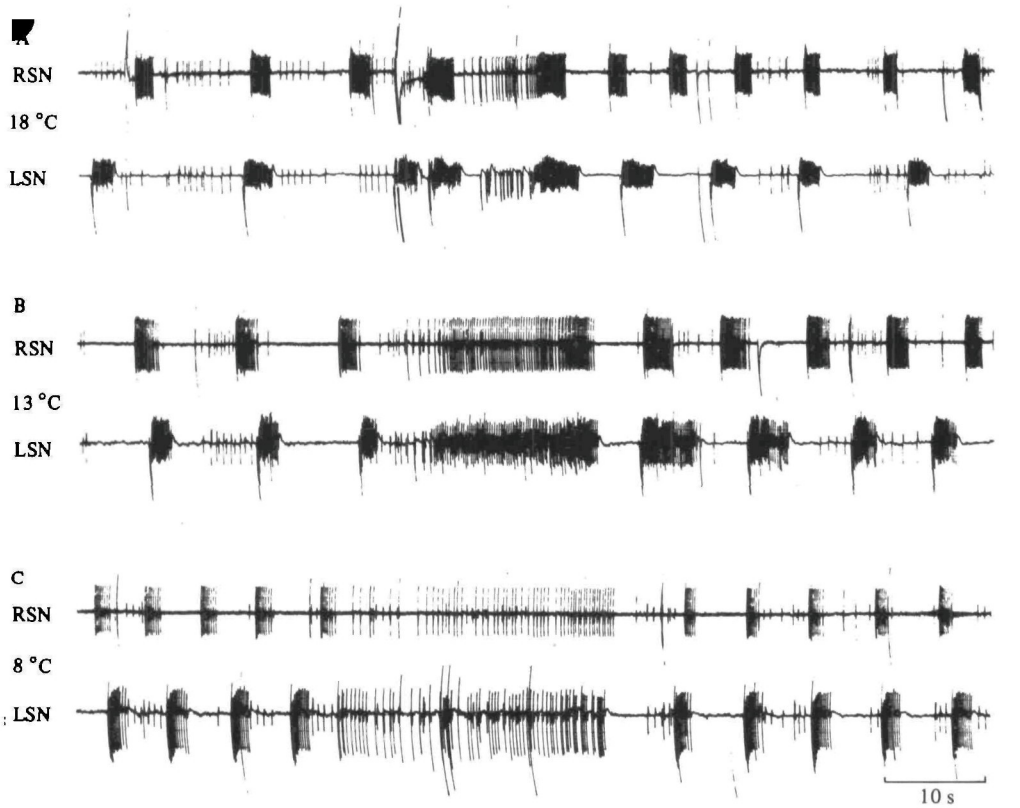


Fig. 7. Each set of records (A–C) is from the same preparation in which simultaneous extracellular recordings were made from both salivary nerves (RSN, LSN) at different temperatures In (A) (18 °C) a prolonged burst in each burster resulted in one subsequent synchronous burst and phase shifting of the activity of both bursters. In (B) (13 °C), a prolonged simultaneous burst in the bursters resulted in synchronization of the first subsequent burst and a slight overlap of the next. In (C) (8 °C), the prolonged simultaneous burst results in synchronization of four subsequent SB-SB bursts. Calibration is 10 s.

It was possible that temperature-dependent variation in SB–SB synchrony was due to variation in the electrotonic coupling between the SBs and the motoneurones, RB7 and LB7 (see Fig. 1). Activation of either B7 (synaptically or by current injection) can result in activation of both SBs (see Prior & Gelperin, 1977). Likewise, intense activity in one SB could activate the other SB via the B7s. To test this hypothesis we examined the temperature-sensitivity of electrical coupling between protractor neurone, RB7, and the ipsilateral SB. While recording the activity of the SBs, we measured the amplitude of the current pulses injected into B7 that just blocked, or initiated, a burst in the ipsilateral SB (Fig. 9). The magnitude of current pulses required to alter SB activity changed as a function of temperature, which indicated that the efficacy of the electrotonic coupling between RB7 and RSB was temperature-dependent. This could have been due to decreased resistance of electrotonic junctions or increased resistance of non-junctional membrane. Because we could not measure the junctional resistance, we were unable to properly assess this alternative. However,

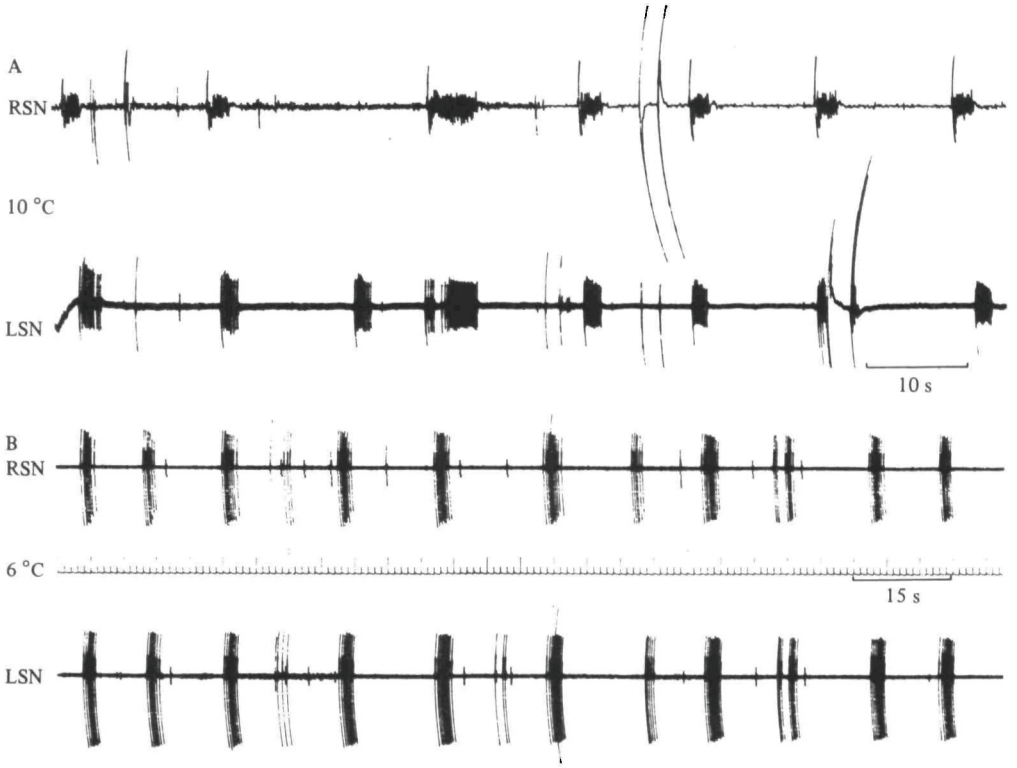


Fig. 8. (A) Simultaneous recording from both salivary nerves (RSN, LSN) in a preparation in which a prolonged burst synchronizes two salivary bursters whose endogenous activities were dissimilar. (B) illustrates an extreme example of SB–SB synchrony at low temperature (6 °C) in which the activity of the bursters is almost completely synchronous even with irregular burst timing, impulse frequency and number. The upper calibration (A) is 10 s and the lower calibration (B) is 15 s.

using double-barrelled microelectrodes, we have made qualitative measurements of the input resistance of B7 as a function of temperature. As in various other neurones (Carpenter, 1967; Zecevic & Levitan, 1978; Langley, 1979), the input resistance of B7 was reversibly increased by reduced temperature (18 to 8 °C; 30–60% increases in four neurones). Although preliminary, these results are consistent with the observed increase in effective B7–SB coupling.

## DISCUSSION

The results presented here describe both long- and short-term effects of temperature on the activity of the salivary burster neurones and their interaction with protractor motoneurones in the feeding motor system. As with the other gastropod 'burster neurones' (e.g. Carpenter, 1967; Moffett & Wachtel, 1976; Zecevic & Levitan, 1979), certain properties of the SBs are temperature-sensitive, among which are the frequency of impulses within a burst, the interburst interval, the burst duration and the temperature range over which the SBs are active (Fig. 2).

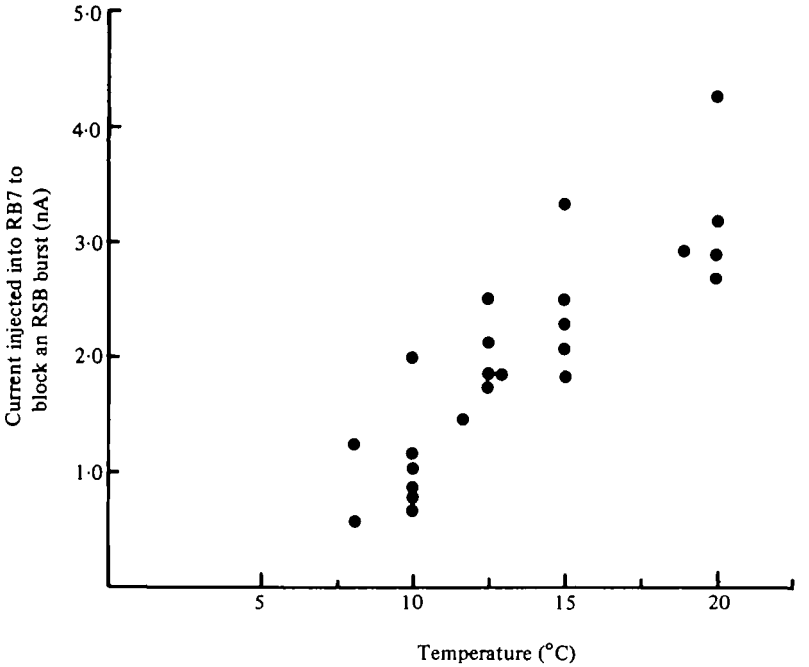


Fig. 9. This graph illustrates the relationship between temperature and the current that had to be injected into RB7 to block an RSB burst. Each point respresents one measurement from one of five preparations.

*Temperature acclimation*

Temperature range over which the SBs are active is a function of the temperature to which the slug had been acclimated. Both the 'on-point' of SB activity during an increase in temperature and the heat block temperature are significantly affected (Fig. 2). The effect of acclimation on 'on-point' can also be seen in the data of Langley (1979, Fig. 2) on neurone F1 in *Helix*. Thus, this transition point in the mechanism underlying burst generation seems to be particularly sensitive to acclimation temperature. It has been shown that neurone F1 in *Helix* takes 2–3 weeks for acclimation (Langley, 1979). Langley's experiment did not, however, address the question of *acclimatization* to the daily temperature cycle. We observed no difference in the activity of SBs from slugs collected during cold or hot periods of a single day. This finding argues against the existence of a short-term response to each high and low of daily temperature cycles. Instead, our results suggest the existence of a slow acclimatization in response to the mean ambient temperature.

In the laboratory, temperature acclimation resulted in a shift of the specific temperature range over which the SBs were active (Fig. 2). Both 5 °C and 25 °C SBs were active over a range of about 20 centigrade degrees (5° SBs; 4–25 °C; 25° SBs: 17 to 35–37 °C). Thus temperature acclimation in this case resulted in maintaining the correspondence between the functional range of the SBs and the ambient temperature.

Temperature acclimation is known to result in significant alterations in enzyme

and transport function (Somero & Hochachka, 1976) and the fluidity of synaptosomal membranes (Cossins *et al.* 1977; Cossins and Prosser, 1978). It is possible therefore, that changes in membrane resistance or the currents involved in burst-generation could be the basis of the observed acclimation effects. If membrane resistance were a function of acclimation temperature such that cold acclimation increased resistance, a given current could result in quite different changes in membrane potential. For example, a given membrane current which in a warm-acclimated slug would result in a subthreshold change in potential, might, in a cold-acclimated slug, result in initiation of burst activity. Our results indicate that temperature acclimation did not have a significant effect on either the input resistance of the SB soma or the electrotonic coupling between SB and B7 (Fig. 5). Similar analysis of membrane currents will require voltage-clamp techniques.

*Seasonal effects*

Although there seemed to be no seasonal variation in the sensitivity of SBs to temperature cycles, SBs from winter animals did remain tonically active at temperatures below cold block (Fig. 2). Our inability to initiate bursting activity in cold-blocked SBs (by current injection) indicated that changes in resting potential alone were not sufficient to explain the temperature-dependent cessation and initiation of activity. This is likely due to the independence of temperature effects on the resting potential and the burst-generation mechanism (Carpenter, 1967). Thus, the effects of temperature on SB activity were not mediated by the temperature-dependent changes in resting potential that have been seen in this and other neurones (e.g. Merickel & Kater, 1974). This may likewise apply to other preparations in which increased temperature results in membrane hyperpolarization and initiation of burst activity (e.g. Carpenter, 1967, in *Aplysia*; Konishi & Kravitz, 1978, in lobster). In *Aplysia*, seasonal effects have been described for the responses of several central neurones (Moffett & Wachtel, 1976). Interestingly, the effect was on the 'on-point' of neuronal activity following cold block. If the animals were kept in the laboratory for only a short time before use, it is possible that the effect was due to temperature 'acclimatization' in response to the ambient water temperatures at the collection site.

*Effects of temperature on SB–SB synchrony*

At low temperature, there was an increase in the occurrence of synchronous SB–SB bursts (Fig. 5). This involved alteration of the phase relationship between the SBs by prolonged 'synchronizing bursts' (Figs 6, 7, 8). The intense activity of the two SBs during a synchronizing burst resulted in a phase shift of SB activity similar to that which occurs following activation of SBs by depolarization of protractor motoneurone B7 (see Prior & Gelperin, 1977). This effect was particularly evident in preparations in which the endogenous SB activity was irregular (Fig. 8). The high degree of SB–SB synchrony in these preparations at low temperature, and the simultaneity of spontaneous, prolonged SB–SB burst can be explained by enhanced electrotonic coupling between the SBs and associated neurones (Fig. 9). Due to the increased coupling efficiency between B7s and SBs at low temperature, excitatory synaptic input (e.g. FMP or 'synchronizing burst') to these neurones, or others in the circuit, would be mutually excitatory.

The variation in effective B7–SB coupling could be explained by an increase in non-junctional resistance which would enhance electrotonic spread of current. Consistent with this possibility is our observation that the input resistance of B7 was increased at low temperature. Similar results have been obtained for the lateral giant cell in *Helisoma* (Merickel & Kater, 1974), cell F1 in *Helix* (Langley, 1979) and the septate axon of crayfish (Payton, Bennett & Pappas, 1969). However, this interpretation is complicated by the report that, in the crayfish septate axon, the junctional resistance is also increased by reduced temperature (Payton *et al.* 1969). We could not measure the actual junctional resistance between B7 and SB. We can, however, say that if B7–SB junctional resistance does increase, it does not prevent an increase in effective coupling between the neurones at reduced temperature.

Poikilothermic animals are often exposed to wide daily ranges of temperature. In the autumn the night lows are often close to the cold-block temperature for chemically mediated synapses. Reduction in the efficacy of such synaptic interactions can interrupt synchronized motor patterns. A temperature-dependent increase in electrotonic coupling could facilitate maintenance of synchrony in motor output as the efficacy of chemically mediated inputs declines. Temperature compensation of this sort could be involved in both the maintenance and initiation of behaviour during short term exposure to reduced ambient temperatures.

We thank Dr P. Schofield and a very helpful *J. exp. Biol.* reviewer for comments on this paper. This work was supported by N.S.F. grant BNS-74-15217-AO1 and an Alfred P. Sloan Foundation Fellowship to Dr Prior. Dr Prior is the recipient of Research Career Development Award NSI-EA-104 NS-490-02 from the National Institutes of Health. This is contribution 148 from the Tallahassee, Sopchoppy and Gulf Coast Marine Biological Association.

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