Effects of temperature on a central synapse between identified motor neurons in the locust

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Summary. Changing the temperature from 10–40 °C modifies the transmission at an established monosynaptic connection between the fast extensor tibiae (FETi) and flexor tibiae motor neurons in the metathoracic ganglion of the locust Schistocerca gregaria (Forskål). Striking changes occur to the shape of the spikes, to membrane resistance, to the synaptic delay, and to the evoked synaptic potentials.

In the presynaptic FETi motor neuron, raising the temperature reduces the amplitude of an antidromic spike recorded in the soma by a factor of 10 (40 mV to 4 mV), reduces the time taken to reach peak amplitude by 5 (3.5 to 0.7 ms) and decreases the duration at half maximum amplitude by 0.5. The conduction velocity of the spike in the axon is increased by 50% from 10 °C to 40 °C. Orthodromic spikes are affected by temperature in a similar way to the antidromic spikes.

The membrane resistance of both pre- and postsynaptic motor neurons falls as the temperature is raised. The membrane resistance of FETi falls by a factor of 4 (about 4 MΩ at 10 °C to 1 MΩ at 40 °C). A contributory component to this fall could be the increase in the frequency of synaptic potentials generated as a result of inputs from other neurons. No temperature dependence could be demonstrated on the voltage threshold relative to resting potential for evoking orthodromic spikes, but because the resistance changes, the current needed to achieve this voltage must be increased at higher temperatures.

The latency measured from the peak of the spike in the soma of FETi to the start of the EPSP in the soma of a flexor motor neuron decreases by a factor of 20 (10 ms at 10 °C to 0.5 ms at 40 °C).

In a postsynaptic flexor tibiae motor neuron, the amplitude of the evoked synaptic potential increases by a factor of 3.4 (5 mV to 17 mV), its duration at half maximum amplitude decreases by 3 (7 ms at 12 °C to 2.3 ms at 32 °C) and its rate of rise increases by 3. An increased likelihood that spikes will occur in the flexor contributes to the enhanced amplitude of the compound EPSP at temperatures above 20 °C.

Introduction
Among the many specialisations that enable the hind legs of a locust to produce the considerable power for jumping and kicking are direct connections between some of the leg motor neurons in the central nervous system. A spike in the single fast motor neuron that innervates the main power producing muscle, the extensor tibiae, causes depolarizations that are sufficient to generate spikes in some of the motor neurons that innervate the antagonistic flexor tibiae muscle (Hoyle and Burrows 1973). The connections should increase the excitability of the flexor motor neurons during the preparatory phase for a jump during which the flexor and extensor muscles co-contract. They are, however, only one of a number of factors that contribute to the co-contraction, because if all fast extensor spikes are suppressed flexor motor neurons will still show a pattern of spikes comparable to that seen in a normal kick (Heitler and Bräunig 1988). Despite the compelling anatomical and physiological evidence which suggests that these connections are monosynaptic, the measured synaptic delay of 1.6–2.0 ms is long (Burrows et al. 1989). The physiological experiments were, however, performed at about 20 °C whereas the typical body temperature for a locust in the wild during the daytime (see Uvarov 1977, for review), or when offered a behavioural choice in the laboratory (Miles 1985) is in
the range 32–44 °C. The effects of altering the temperature on the synaptic transmission between these motor neurons have therefore been examined.

It is typical of heterotherm insects to be more active at higher body temperatures and for these temperatures to produce numerous effects on their neurons. For example, moths will not fly if their body temperature is below a critical level, whereas thoracic muscles are contracted in mechanically inefficient patterns that generate heat and not flight. The transition from this warm-up pattern to the real flight pattern can be effected by local warming of the pterothoracic ganglion (Hanegan and Heath 1970). Locusts show a general increase in behavioural activity when warm and a greater probability of kicking or jumping. Some of the neuronal mechanisms underlying this increased excitability at higher temperatures have been explored. Temperature receptors on the antennae of locusts give information about changes in temperature, with some transiently increasing their spike frequency to a reduction in temperature, but maintaining an increased frequency to a sustained elevation (Ameismeier and Loftus 1988). The sensitivity of transduction in insect mechanoreceptors (French and Kuster 1982; Miles 1985) and the frequency of their spikes is increased by elevating the temperature (Thurm 1963; French 1985; Miles 1985). Many neurons in the central nervous system of insects spike faster as the temperature is raised (Abrams and Pearson 1982; Murphy and Heath 1983; Janiszewski and Otto 1988). Specific changes in the properties of the fast extensor motor neuron of the locust have also been observed (Heitler et al. 1977; Abrams and Pearson 1982) that vary in different isogenic clones (Goodman and Heitler 1977). Heitler et al. (1977) found that the current needed to evoke a spike decreased with increasing temperature and thought this to be a major factor in explaining the increased excitability. Abrams and Pearson (1982), however, found the converse and instead attributed some of the increased excitability to an enhanced responsiveness of sensory neurons.

This paper shows that altering the temperature produces complex changes at the synapse between specific locust motor neurons, that include effects on the shape of the presynaptic spikes, on the duration of the synaptic delay and on the amplitude of the evoked postsynaptic potentials.

Materials and methods

Adult, male and female locusts, Schistocerca gregaria (Forskål) were taken from our crowded culture and fixed with their ventral surface uppermost. The metathoracic ganglion was exposed and supported on a wax-covered steel platform. Nerves 1 and 3a of the metathoracic ganglion and nerves 1, 3, 4 and 6 of the mesothoracic ganglion were cut to prevent contractions of the thoracic muscles and hence to improve stability of the ganglion. The thorax was perfused continuously with saline whose temperature was 10 °C in a reservoir bottle, but which could be raised in a controlled fashion to 40 °C by a small heating coil on the outlet pipe close to the locust. The temperature of the saline in the thoracic cavity was measured by a 1.5 mm O.D. thermistor placed next to the mesothoracic ganglion. Measurements from neurons were made only when a new temperature had been maintained for at least 5 min. The sheath of the metathoracic ganglion was treated with a 0.1% solution (wt/vol) of protease (Sigma Type XIV) before recording began. Microelectrodes, filled with 2 M potassium acetate, were driven across the sheath into the somata of the fast extensor (FETi) and fast flexor tibiae motor neurons. A particular flexor motor neuron could not be identified in each locust, but the effects of an FETi spike are the same in at least 5 flexors (Burrows et al. 1989). All recordings were made from somata as their large size provided a stable recording site throughout the approximately 2 h required to effect the changes of temperature. To measure the resistance of the membrane, two electrodes were inserted into the same cell body, one to pass current, the other to record voltage. The femur was cut half way along its length to eliminate sensory feedback from receptors associated with the femoro-tibial joint. Antidromic spikes were then evoked repetitively in FETi at intervals of 3 s by stimulating its axonal terminals with a pair of 50 μm steel wires implanted proximally in the extensor tibiae muscle. All recordings were stored on an FM tape recorder for later analysis and display on a digital oscilloscope and XY plotter. Signal averaging was performed with a CED 1401 interface (Cambridge Electronic Design) and microcomputer. The results are based on recordings from 16 locusts, in each of which measurements were made at a minimum of 5 different temperatures.

Results

A single spike in the fast extensor tibiae motor neuron evokes an EPSP in a flexor tibiae motor neuron at synapses in the metathoracic ganglion. If the presynaptic spikes are repeated at intervals of 3 s, the evoked EPSPs occur reliably at a constant delay, have a consistent amplitude, and each may give rise to spikes in the flexor motor neuron. At a given temperature the characteristic features of the antidromic spike as recorded in the soma of FETi, the postsynaptic potential recorded in the soma of a flexor, and the synaptic delay all remain stable. If the temperature is increased, however, the following changes occur (Fig. 1). The amplitude, duration and time to peak of the presynaptic spike decrease, while its conduction velocity in the axon increases. The synaptic delay decreases, and the amplitude and rate of rise of the EPSP in a flexor increase, while its duration decreases.

The presynaptic neuron

The soma membrane of FETi does not support an overshooting action potential. Both an ortho-