OCTOPAMINE MODULATES THE RESPONSES AND PRESYNAPTIC INHIBITION OF PROPRIOCEPTIVE SENSORY NEURONES IN THE LOCUST SCHISTOCERCA GREGARIA

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Summary
A multineuronal proprioceptor, the femoral chordotonal organ (feCO), monitors the position and movements of the tibia of an insect leg. Superfusing the locust metathoracic feCO with the neuromodulator octopamine, or the octopamine agonist synephrine, affects the position (tonic) component of the organ’s response, but not the movement (phasic) component. Both octopamine and synephrine act with the same threshold (10⁻⁶ mol l⁻¹).

Individual sensory neurones that respond tonically at flexed tibial angles show increased tonic spike activity following application of octopamine, but those that respond at extended angles do not. Tonic spiking of phaso-tonic flexion-sensitive neurones is enhanced but their phasic spiking is unaffected.

Bath application of octopamine to the feCO increases the tonic component of presynaptic inhibition recorded in the sensory terminals, but not the phasic component. This inhibition should at least partially counteract the increased sensory spiking and reduce its effect on postsynaptic targets such as motor neurones. Furthermore, some phasic sensory neurones whose spiking is not affected by octopamine nevertheless show enhanced tonic synaptic inputs.

The chordotonal organ is not known to be under direct efferent control, but its output is modified by octopamine acting on its sensory neurones to alter their responsiveness to mechanical stimuli and by presynaptic inhibition acting on their central branches. The effects of this neuromodulator acting peripherally on sensory neurones are therefore further complicated by indirect interactions between the sensory neurones within the central nervous system. Increases of sensory neurone spiking caused by neuromodulators may not necessarily lead to parallel increases in the responses of postsynaptic target neurones.

Key words: octopamine, neuromodulation, chordotonal organ, locust, Schistocerca gregaria, presynaptic inhibition, gain control.

Introduction
Functioning of the nervous systems of both vertebrates and invertebrates depends critically on the levels of circulating neurohormones and on more specifically released neuromodulators (Kaczmarek and Levitan, 1987; Harris-Warrick and Marder, 1991). In vertebrates, for example, the enkephalin analogue (D-Ala²)enkephalin inhibits the release of substance P from cultured sensory neurones of chick (Mudge et al., 1979). In Crustacea, the biogenic amine octopamine modulates responses of primary sensory neurones from several proprioceptors (Pasztor and Bush, 1989; Pasztor and Macmillan, 1990), although the sign of the effect on a particular receptor can be opposite in different species. In insects, levels of circulating octopamine are elevated during stress (Orchard et al., 1981) and during particular types of behaviour such as flight (Goosey and Candy, 1980); moreover, particular octopaminergic neurones that innervate skeletal muscles are activated during specific movements involving those muscles (Burrows and Pflüger, 1995). Octopamine induces plateau potentials in some interneurones of the flight and ventilatory systems of Locusta migratoria (Ramirez and Pearson, 1991a,b), alters the contractile properties of insect muscle (O’Shea and Evans, 1979) and modulates the firing of sensory neurones. Octopamine alters the spike pattern of the locust wing hinge stretch receptor (Ramirez and Orchard, 1990), a proprioceptor that responds with a burst of spikes near the top of the upstroke of each wingbeat. Stimulation of octopaminergic dorsal unpaired median (DUM) neurones similarly modulates the stretch receptor, so these neurones may provide a behaviourally relevant pathway for the proposed action of octopamine.

Octopamine also acts on sensory neurones of another proprioceptor, the femoral chordotonal organ (feCO), which monitors movements of the tibia relative to the femur (see Field and Matheson, 1997). The feCOs of locusts and stick

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insects contain neurones that respond to tibial position, velocity or acceleration, or to combinations of these signals (Hofmann et al. 1985; Hofmann and Koch, 1985; Matheson, 1990), and which make direct excitatory output synapses with motor neurones and interneurones of the segmental ganglion (locust; Burrows, 1987). These proprioceptors participate in the reflex control of movements of the tibia (Field and Burrows, 1982) and other leg joints (Field and Rind, 1981), and their signals are carried by intersegmental interneurones to other ganglia (Laurent, 1986; Laurent and Burrows, 1988) where they can influence the reflex movements of adjacent legs (Laurent and Burrows, 1989). In the stick insect Cuniculina impigra, octopamine acts in the periphery to increase the tonic but not the phasic spike response of sensory neurones of the feCO, with a threshold at 5×10⁻⁷ mol⁻¹ l⁻¹ (Ramirez et al. 1993). Despite these alterations in sensory responsiveness, octopamine does not alter a resistance reflex mediated by this sense organ (Büschges et al. 1993). Either the sensory neurones affected by octopamine do not participate in the reflex, or a mechanism exists to compensate for their altered responses.

There is no known direct neural efferent control of the locust feCO, but the sensory neurones receive presynaptic inhibitory inputs on their central terminals (Burrows and Laurent, 1993; Burrows and Matheson, 1994). These depolarising inhibitory inputs shunt the action potentials and reduce transmitter release onto postsynaptic motor neurones. The presynaptic inputs are mediated by unidentified interneurones and are known to have at least three sources (Wolf and Burrows, 1995). First, they may be initiated by central neurones that become active in particular types of behaviour. Second, they may be initiated by sensory pathways from other joints or other legs. Third, and most relevant to the present study, they may be initiated by other sensory neurones of the same chordotonal organ (Burrows and Laurent, 1993; Burrows and Matheson, 1994). A particular movement of the tibia of a hind leg stimulates many of the feCO sensory neurones, whose spikes propagate in parallel to the central nervous system (CNS). Some of these sensory neurones activate interneurones which, in turn, presynaptically inhibit other sensory neurones that may be responding to the same movement. Each sensory neurone has a characteristic pattern of inhibitory inputs which indicates that it is inhibited by many but not all of the other neurones from the same receptor. This represents an automatic gain control mechanism that regulates the flow of information from feCO sensory neurones to postsynaptic targets depending on the pooled and weighted outputs of the other active sensory neurones. This mechanism may explain the observation made in the stick insect that octopamine alters sensory responsiveness without changing a resistance reflex mediated by the same sensory neurones: the increased sensory firing is counteracted by increased presynaptic inhibition. This explanation is only credible if the particular sensory neurones that mediate the inhibition are themselves affected by octopamine.

The main objective of this study was to determine whether changes in feCO spiking activity caused by octopamine are accompanied by alterations in presynaptic inhibition mediated by the gain control pathway. Although octopamine is known to alter the responsiveness of feCO neurones in the stick insect Cuniculina impigra, it was not known whether comparable effects occur in the locust Schistocerca gregaria, in which the presynaptic gain control mechanism was characterised. The first step was therefore to characterise the responses of locust feCO neurones to octopamine. These experiments also permitted the effects of octopamine on feCO sensory neurones of the locust to be compared with those previously published for the stick insect.

### Materials and methods

Adult male and female locusts, Schistocerca gregaria (Forskål), from our crowded colony were restrained ventral side upwards using Plasticine so that the femur of the left hind leg was horizontal, with its anterior surface uppermost. The femoro-tibial joint was fixed at an angle of 60°, and the apodeme of the metathoracic feCO was exposed in the distal femur. Great care was taken to minimise disruption of the tracheal supply to the organ. The apodeme was grasped with forceps distal to the point where fine strands insert upon it (Field, 1991; Shelton et al. 1992) and then cut distally. The forceps were attached to a vibrator (Ling Dynamic, type 101), the movements of which were controlled by an analogue ramp generator (Matheson and Ditz, 1991). The flexor strand and ventral ligament of the chordotonal organ remained attached to the apodeme of the flexor tibiae muscle. Control experiments, in which the chordotonal organ was completely isolated from all muscles, showed that the effects reported in this paper could not be attributed to alterations of muscle properties.

Linear movements of the chordotonal apodeme mimicked angular extension or flexion movements of the tibia, within the range normally used by the animal [0–120°, where 0° is fully flexed; see Field and Burrows (1982) for the relationship between movements of the apodeme and the femoro-tibial angle]. Movements of the apodeme are referred to as if the entire tibia had been moved; i.e. a distal stretch of the apodeme is referred to as a flexion movement, whereas relaxation of the apodeme is referred to as an extension movement.

The distal part of the femur containing the exposed chordotonal organ was surrounded by Plasticine to form a well into which was passed a continuous flow of locust saline. This flow could be switched to a separate supply of saline containing DL-octopamine (Sigma) or synephrine (Sigma) at concentrations of 10⁻⁸ to 10⁻⁴ mol⁻¹ l⁻¹. The volume of the well was approximately 0.15 ml, and the flow rate was adjusted so that this volume was replaced approximately every 20 s. Extracellular recordings of the spikes of sensory neurones from the feCO were made with hook electrodes on nerve 5B1 in the femur just proximal to the organ.

For intracellular recordings of individual chordotonal organ sensory neurones, the thorax was opened and the mesothoracic and metathoracic ganglia were stabilised on a wax-coated silver platform. The ganglionic sheath was treated with a 0.1%
Modulation of sensory neurones in the locust

(w/v) solution of protease (Sigma type XIV) for 40 s before recording began. Intracellular recordings were made using electrodes filled with 2 mol l\(^{-1}\) potassium acetate (resistance 50–80 MΩ). The axons of sensory neurones from the feCO were impaled in nerve 5 where it meets the metathoracic ganglion or in the neuropile close to the entry point of nerve 5. The sensory neurones were identified by their specific responses to controlled movements of the chordotonal apodeme and by correlating the spike recorded intracellularly with one recorded extracellularly in nerve 5B1 close to the organ. For measurement of synaptic inputs, all neurones were held hyperpolarised by a constant current of approximately 2 nA throughout the experiment. Recordings were stored on magnetic tape (Racal Store 7 DS) for later analysis, and displayed on a Gould TA 240 thermal array recorder. The timing of spikes was measured using a window discriminator and stored using a Cambridge Electronic Design (C.E.D.) computer interface and software. In Figs 6, 7, the sensory spikes have been truncated so that a high gain could be used to display synaptic inputs. The results presented here are based on recordings made from chordotonal organ sensory neurones in 159 animals.

Results

The metathoracic femoral chordotonal organ of the locust contains approximately 90 sensory neurones that respond to the position and movements of the tibia relative to the femur (Zill, 1985; Matheson and Field, 1990; Matheson, 1990, 1992a,b). A series of ramp-and-hold movements of the main apodeme was used to activate the sensory neurones, whose spikes were recorded in nerve 5B1 (Fig. 1A). The overall tonic spike rate increased as the main apodeme was pulled distally (tibial flexion) and decreased during extension (Fig. 1A,B). Phasic bursts of larger spikes were evident during movements in either direction (Fig. 1A,C).

Octopamine enhances tonic spike activity

Bath application of octopamine (10\(^{-4}\) mol l\(^{-1}\)) to the femoral chordotonal organ caused a significant increase in tonic spike frequency when the tibia was fully flexed (0°, Fig. 2A; paired t-test, \(P<0.05, N=10, \text{range } 0–72\%\)). It had no significant effect at extended angles. The elevated tonic spike frequency returned to control levels during a subsequent wash in normal saline. The spike frequencies measured during the movement phases of the ramp-and-hold sequence in the same preparations were not affected by the application of octopamine at any tibial angle, for either flexion or extension movements (Fig. 2B).

When the apodeme was held fully stretched (tibial flexion), application of 10\(^{-4}\)mol l\(^{-1}\) octopamine caused an increase in tonic spiking within 30 s. The increase reached a peak within 3 min and declined to baseline levels within a further 10–20 min (Fig. 3A), even when octopamine was still present in the bath. There was a further decline in tonic spiking over the next 30 min. In control experiments (no octopamine applied), tonic spiking showed only a gradual decline (Fig. 3B), perhaps reflecting a gradual wash-out of endogenous modulatory substances. When this decline was subtracted from the corresponding experimental values, the true time course of the octopamine effect was revealed to be a transient increase and subsequent decrease to baseline spiking rate (Fig. 3C).

Fig. 1. (A) The overall tonic activity of the femoral chordotonal organ increased as the apodeme was pulled distally (tibial flexion) (A,B). In contrast, phasic activity during 20° movements at 1000 °s\(^{-1}\) was approximately equal at all but the most extended angles (C). B and C are mean ± s.d. for three repeats of these movements in a single animal. Tonic spike frequencies were measured during a 1 s period following the end of each movement.
maximal spike frequency was 60% above baseline levels \((N=8)\). In some experiments, the octopamine applied to the preparation was washed out with normal saline for 10–60 min following the peak of its effect, but this did not affect the time course of the decline in response. Further applications of octopamine, even of higher concentration, failed to produce subsequent increases in spiking. In two preparations, the feCO was isolated completely from all muscles before the application of octopamine. The overall spike rates in these preparations showed the same response to octopamine as described above, indicating that it is not caused by alterations in muscle properties.

With the femoro-tibial angle set to fully flexed, the threshold for the effect of octopamine was determined to be at least \(10^{-6}\) mol l\(^{-1}\) (e.g. Fig. 4A). In one preparation, \(10^{-7}\) mol l\(^{-1}\) octopamine caused a small (3%) increase in tonic spiking. Higher concentrations of octopamine caused larger increases...
in peak spike frequency, with the effect beginning to saturate at $10^{-4}\text{mol}\text{l}^{-1}$ (Fig. 4B).

The octopamine agonist synephrine was bath-applied to the feCO in the same way as for octopamine ($N=23$). At a concentration of $10^{-4}\text{mol}\text{l}^{-1}$, synephrine increased the tonic spiking of sensory neurones by 36\% (range 19–51\%, $N=4$) with a similar time course to that described above (e.g. Fig. 4C). The threshold for an effect was $10^{-6}\text{mol}\text{l}^{-1}$.

**Octopamine enhances the tonic spiking of individual sensory neurones**

Extracellular recordings of the summed discharge of the entire feCO do not distinguish between enhanced spiking of individual neurones and recruitment of additional units. To investigate this, intracellular recordings were made from the axons of feCO neurones in nerve 5 ($N=36$).

Tonic spiking of individual sensory neurones that signal tibial flexion was increased by bath application of octopamine to the feCO in 16 of 17 recordings (e.g. Fig. 5A,B). Tonic neurones that respond at fully extended tibial angles are less commonly encountered in intracellular recordings from axons, but in two such neurones, $10^{-4}\text{mol}\text{l}^{-1}$ octopamine had no effect on their tonic spike rate. This supports the conclusion drawn from extracellular recordings that octopamine acts predominantly on tonic neurones that signal tibial flexion.

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**Fig. 4.** (A) An increase in tonic spiking of femoral chordotonal organ sensory neurones was apparent at an octopamine concentration of $10^{-6}\text{mol}\text{l}^{-1}$. (B) The effectiveness of octopamine on the overall tonic spike frequency was dependent on concentration. Data are expressed as a mean ± S.E.M. percentage increase in spike frequency for the $N$ values indicated. (C) $10^{-4}\text{mol}\text{l}^{-1}$ synephrine also caused an increase in tonic spiking. Extracellular recordings were made from nerve 5; bin width 30 s in A and C.

**Fig. 5.** Intracellular recordings from the axons of individual femoral chordotonal organ sensory neurones showed that octopamine caused an increase in tonic but not phasic spiking. The shaded area indicates the duration of the phasic stimulus. The record in A was obtained under control conditions, that in B after octopamine ($10^{-4}\text{mol}\text{l}^{-1}$) had been perfusing for 5.5 min and that in C after a further 7 min, by which time the response had returned to control levels (see Fig. 3). The bottom trace indicates the imposed movement.
Sensory neurones that did not spike at a particular tibial angle under control conditions often began to spike tonically following application of octopamine (e.g. Fig. 7). For flexed angles, therefore, application of octopamine both caused an increase in spiking of active sensory neurones and recruited previously silent sensory neurones.

Although octopamine increased the tonic spiking of phasotonic sensory neurones, their phasic component remained unchanged (Fig. 5). Octopamine had no effect on the spiking of purely phasic (velocity- or acceleration-sensitive) neurones in 14 of 16 recordings.

Octopamine enhances synaptic inputs to sensory neurones

Intracellular recordings made from the axons of feCO sensory neurones near the metathoracic ganglion revealed depolarising inhibitory synaptic inputs, upon which were superimposed the sensory spikes arriving from the periphery (A). The amplitude of the inputs was emphasised in these recordings by hyperpolarising the sensory neurone with a constant current of approximately 2 nA. Perfusion of the feCO with octopamine (10^{-4} mol l^{-1}) for 5 min increased the tonic spiking rate of a sensory neurone (B) and simultaneously increased the amplitude of the inputs (C). The recordings shown in A and B are superimposed in C. An almost purely phasic feCO sensory neurone also received tonic synaptic inputs (D). Perfusion of the feCO with octopamine (10^{-4} mol l^{-1}) for 3 min increased the amplitude of these inputs, in the absence of an increase in tonic spiking and in the absence of any shift in membrane potential (D). The effect was reduced after a 9 min wash in normal saline (E). The spikes have been truncated to permit display of the inputs at high gain.
Tonic inputs

Octopamine applied to the feCO increased the amplitude of tonic synaptic inputs in all 11 sensory neurones tested that had maximal inputs at flexed tibial angles (mean 28\%, range 14–42\%). For example, stretching of the feCO apodeme (tibial flexion) was signalled by a phaso-tonic feCO sensory neurone with a rapid burst of spikes during flexion and a lower frequency of tonic spikes throughout the sustained 0.5 s period of static flexion (Fig. 6A). The neurone did not spike during tibial extension. The neurone was held hyperpolarised by approximately 2 nA of constant current throughout the experiment to reveal the synaptic potentials. At the same time as the neurone spiked, it received a continuous barrage of synaptic inputs upon which the spikes were superimposed (Fig. 6A). The amplitude of this depolarisation increased almost linearly as the joint was flexed, remained at a constant level throughout the period of sustained flexion, and returned to the baseline level during joint extension. When octopamine (10^{-4} \text{mol}\cdot\text{l}^{-1}) was applied to the feCO, the tonic spike rate of the sensory neurone recorded at a tibial angle of 0° increased by approximately 20\% and the first spike during the flexion movement occurred after a shorter delay. At the same time, the amplitude of the synaptic input increased by 36\% (Fig. 6B,C). Increases in the amplitude of synaptic inputs occurred in the absence of any change in the resting membrane potential of the sensory neurones. It is possible that the increased spiking of the sensory neurone contributed to its apparent depolarisation, either through alterations in its membrane properties caused by conductances associated with the spikes or through synaptic feedback onto its own terminals. Recordings from three phasic neurones that received tonic synaptic inputs rule out this possibility. In all, octopamine caused an increase in the tonic synaptic inputs (mean 31\%, range 25–42\%), even though the spike response was unchanged. For example, the neurone represented in Fig. 6D,E did not spike tonically at fully flexed tibial angles, but nevertheless received a sustained synaptic input. Application of octopamine (10^{-4} \text{mol}\cdot\text{l}^{-1}) did not affect the spiking of the neurone but did increase the amplitude of its synaptic input by 25\% (Fig. 6D), indicating that the increase in synaptic inputs was not caused by an increase in spiking of the sensory neurone itself. The amplitude of the input decreased during a subsequent wash in normal saline (Fig. 6E).

Phasic inputs

Peripheral application of octopamine increased phasic synaptic inputs in only 2 of 16 neurones. In the others, there was no effect or a general decrease in phasic inputs. For example, a phasic feCO sensory neurone responded to tibial extension movements with a burst of spikes and sometimes responded with a single spike during flexion movements (Fig. 7A). It did not have a tonic discharge, although it spiked occasionally when the tibia was held fully flexed. Both extension and flexion movements caused large barrages of synaptic inputs, and there was a small sustained input during a 1 s period of tibial flexion. Application of octopamine to the feCO (10^{-4} \text{mol}\cdot\text{l}^{-1}) caused the neurone to spike tonically at flexed tibial angles (Fig. 7B) and increased the amplitude of the sustained synaptic input by 33\% (Fig. 7B). At the same time, the amplitude of the phasic inputs was reduced. During a subsequent wash in normal saline, the spiking response of the sensory neurone declined, as did the phasic and tonic synaptic inputs (Fig. 7C). The gradual decline in phasic inputs probably reflects a gradual decrease in sensory responsiveness (similar to that shown in Fig. 3B) rather than a specific effect of octopamine.

Discussion

Sensory neurones of the locust femoro-tibial chordotonal organ respond phasically to imposed movements of the tibia and tonically to maintained changes in tibial position. Peripheral application of the biogenic amine octopamine or its agonist synephrine increase the overall tonic spike rate at flexed tibial angles, with threshold concentrations between 10^{-7} and 10^{-6} \text{mol}\cdot\text{l}^{-1}. There is no effect on tonic spiking at extended angles or on the phasic spike rate during imposed movements. Not only is there increased spiking of individual receptors, but octopamine also recruits previously silent neurones. The tonic spiking rate of an individual phaso-tonic neurone can be enhanced by octopamine, while the phasic component of its response remains unchanged. In parallel with the increased spiking of sensory neurones, there is an increase in tonic inhibitory synaptic inputs recorded near their output branches within the CNS, while phasic inputs are unaffected. It is not known whether the increase in tonic synaptic inputs occurs at all tibial angles; it could only be analysed in neurones whose maximal inputs occurred at flexed angles.

Octopamine levels in locusts

Levels of circulating octopamine in insects are elevated during stress (Orchard et al. 1981) and during particular types of behaviour such as flight (Goosey and Candy, 1980). The basal haemolymph concentration of octopamine measured from the metathoracic legs of *Schistocerca gregaria* is 4.9×10^{-8} \text{mol}\cdot\text{l}^{-1} (Davenport and Evans, 1984). This rises during stress induced by noxious mechanical or chemical stimuli to 7.9×10^{-8} to 4×10^{-7} \text{mol}\cdot\text{l}^{-1} (Davenport and Evans, 1984) or, during flight, to at least 2×10^{-7} \text{mol}\cdot\text{l}^{-1} (Goosey and Candy, 1980). These elevated levels are similar to the threshold for an effect on the feCO measured in the present study (no consistent response at 10^{-7} \text{mol}\cdot\text{l}^{-1}, but a clear response at 10^{-6} \text{mol}\cdot\text{l}^{-1}, see Fig. 4B), suggesting that the effects observed here will occur when haemolymph octopamine levels increase under natural conditions. The threshold was determined from extracellular nerve recordings so it is probable that individual sensory neurones are more sensitive than suggested by this value, which is effectively the mean of a range of thresholds of all the neurones represented in the recording.

Octopamine levels differ in different haemolymph compartments of an individual animal (Davenport and Evans, 1984), presumably because octopamine is released locally by specific modulatory neurones. Some octopaminergic dorsal
unpaired median (DUM) neurones that innervate the metathoracic leg of the locust are activated during specific types of behaviour, such as kicking (Burrows and Pflüger, 1995), raising the possibility that these neurones elevate octopamine levels locally within the leg during behaviour patterns in which the feCO plays an important role. Such effects, if present, would be superimposed on those caused by alterations in the level of octopamine in circulating haemolymph. The only known outputs of metathoracic DUM neurones in the legs are the extensor and flexor tibiae muscles, but other possible targets have not been investigated in detail. Recent evidence indicates that DUM neurones may innervate a wider range of peripheral targets than has previously been recognised (Bräunig et al., 1994), so it would be valuable to search for output branches on the femoral chordotonal organ and other sensory structures known to be affected by octopamine.

**Effect of octopamine**

Application of octopamine to the feCO increases the spiking of its tonic neurones that signal tibial flexion and enhances tonic synaptic inputs recorded in their central terminals. The increase in tonic spike frequency only at flexed tibial angles means that octopamine extends the range of frequencies used to code position; this should increase the position-sensitivity of its femoro-tibial control system. The overall effect of octopamine on the transmission of sensory information to postsynaptic targets within the CNS is, however, more subtle than this. In parallel with the increased tonic spike activity in some sensory neurones, there is an increase in inhibitory synaptic inputs recorded in their central branches.

The most likely mechanism for the increase in synaptic inputs is through the pathway described by Burrows and Laurent (1993) and Burrows and Matheson (1994) in which particular feCO sensory neurones presynaptically inhibit others via interposed interneurones. The increased spiking of sensory neurones caused by octopamine would be reflected in increased activity of the inhibitory interneurones and thus in the presynaptic inputs recorded in the terminals of other sensory neurones. The increase in presynaptic inputs causes a reduction in the strength of output synapses made by the sensory neurones (Burrows and Matheson, 1994). An important feature of the mechanism described by Burrows and colleagues is that sensory neurones are presynaptically inhibited by others that respond best to the same tibial positions or movements. It is therefore interpreted as acting as an automatic gain control system that could prevent saturation of postsynaptic neurones during bursts of high-frequency sensory spiking. The same gain control system may act to negate the increased spiking of sensory neurones caused by octopamine.

Application of octopamine (5×10⁻³ mol l⁻¹) to the feCO of the stick insect does not induce noticeable changes in resistance reflexes (Büsches et al., 1993), despite the increase in tonic sensory neurone spiking (Ramírez et al., 1993). The finding made in the present paper that octopamine also increases presynaptic inhibition of sensory neurones provides one possible explanation for this observation. That is, increases in sensory spiking may automatically be counteracted by simultaneous enhancement of inhibitory inputs acting on the sensory neurone output terminals to reduce their synaptic gain (Burrows and Matheson, 1994).

The automatic gain control mechanism relies on the pooling of sensory inputs so that any particular sensory neurone is inhibited by a summed and presumably weighted input from a particular group of other sensory neurones that code similar tibial positions or movements. This specificity of input to each sensory neurone means that octopamine may have different effects on the synaptic inputs recorded in different sensory neurones. Increased presynaptic inhibition caused by elevated levels of octopamine may therefore not simply negate all increases in sensory spike activity, but may instead change the balance of information passing from the population of sensory neurones to the CNS. Until we understand better what rules underlie the connections made by specific sensory neurones to specific targets, this problem will remain difficult to solve. An initial step would be to assess the reflex responses of identified fast and slow tibial motor neurones during application of octopamine to the chordotonal organ.

**Comparison with the stick insect**

Locusts and stick insects avoid predation using different types of behaviour: locusts kick and jump to escape whereas stick insects remain motionless, using their cataleptic behaviour to mimic a twig. Catalepsy requires a high resistance reflex gain and high velocity-sensitivity of the femoro-tibial control loop (Ebner and Bässler, 1978; Kittmann, 1991); whereas kicking and jumping require a low or negative resistance reflex gain. Do increases in haemolymph octopamine concentration during attack by a predator cause alterations in sensory signalling that facilitate these different types of behaviour? In the stick insect, octopamine enhances tonic spiking of feCO neurones at all tibial angles (Ramírez et al., 1993), whereas in the locust the increase is seen only at flexed angles. This latter effect means that, when octopamine levels are elevated in a locust, tibial angles are encoded by a wider range of firing frequencies, presumably enhancing precision. Coding of flexed tibial angles should be particularly important during these types of behaviour, as the tibia must be completely flexed to enable the extensor muscle to develop the force needed to generate a kick or jump.

Elevation of haemolymph octopamine concentration does not affect the spiking responses of velocity-sensitive neurones in either locusts or stick insects. In the locust, however, the increase in tonic presynaptic inhibition of some velocity-sensitive neurones should reduce the flow of phasic information to postsynaptic targets and thus the velocity-sensitivity of the control loop. The challenge now is to determine whether octopamine causes a different pattern of changes in synaptic inputs to stick insect feCO sensory neurones, as predicted by the high gain and velocity-sensitivity of the system in cataleptic animals.

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