Effects of Octopamine, Dopamine, and Serotonin on Production of Flight Motor Output by Thoracic Ganglia of *Manduca sexta*

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SUMMARY

Effects of biogenic amines on a centrally generated motor pattern in *Manduca sexta* were examined by pressure injecting nanomole to micromole amounts of octopamine, dopamine or serotonin into thoracic ganglia. Motor output was recorded extracellularly from a pair of antagonistic flight muscles and their motor neurons. The monoamines were found to alter production of a motor pattern that produces rhythmic wing flapping (10 Hz) and exhibits phase relationships similar to those in the flight pattern of intact moths.

In mesothoracic ganglia with sensory nerves intact, octopamine (4×10^{-9} mol) injected into lateral regions evoked regular firing of a single motor neuron, whereas a higher dose (4×10^{-8} mol) often elicited the flight motor pattern. In the absence of sensory input, these doses of octopamine had little effect. Low doses (10^{-10} mol) greatly enhanced motor responses to electrical stimulation of a wing sensory nerve.

Dopamine (2×10^{-10} mol) injected into the medial region of the mesothoracic ganglion elicited the flight motor pattern in the presence or absence of sensory input. Rhythmic output induced by dopamine (5×10^{-10} mol) was suppressed by injecting serotonin (5×10^{-10} mol) into the same region.

These findings demonstrate that dopamine, octopamine, and serotonin have different effects on motor output in *Manduca* and suggest that these amines are involved in initiating, maintaining and terminating flight behavior, respectively. Octopamine may elicit flight production by enhancing the efficacy of sensory transmission thereby increasing excitability or arousal. Dopamine may act on interneurons involved in generating the flight motor pattern.

INTRODUCTION

Various biogenic amines influence the neural mechanisms that generate motor programs for behavior. Serotonin intensifies feeding motor output in *Aplysia* (Kupfermann and Weiss, 1981) and *Limax* (Gelperin, 1981) and initiates locomotion in *Aplysia* (Mackey and Carew, 1983) and leeches (Willard, 1981). Dopamine evokes the feeding motor pattern in *Limax* (Wieland and Gelperin, 1983) and enhances pyloric motor output in lobsters

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(Anderson and Barker, 1977, 1981). In locusts, octopamine elicits motor patterns associated with flight or walking, or it suppresses the oviposition digging pattern, depending on the site of iontophoresis in metathoracic or abdominal ganglia (Sombati and Hoyle, 1984b). In same cases, two biogenic amines have similar effects on a motor program. Both L-DOPA, a precursor of dopamine and norepinephrine, and 5-HTP, a precursor of serotonin, initiate walking motor patterns in spinal cats (Grillner, 1969, 1976; Ahlman et al., 1971) and spinal rabbits (Viala and Buser, 1969) and both dopamine and serotonin activate motor patterns associated with feeding in *Helisoma* (Trimble and Barker, 1984; Gränzow and Kater, 1977). In other cases, two amines have opposite effects on a motor program. Octopamine evokes tonic extension of the extremities in lobsters and suppresses tonic flexion, an opposing postural movement enhanced by serotonin (Livingston et al., 1980). In crayfish, octopamine intensifies walking and optokinetic responses, whereas serotonin suppresses both activities (Arnesen and Olivo, 1983). Octopamine also intensifies phasic flexion of the abdomen, a movement that contributes to the crayfish escape response; flexion of the abdomen is inhibited by serotonin (Glanzman and Krasne, 1983).

In the present study, the central effects of dopamine, octopamine and serotonin on production of rhythmic motor output were investigated in the moth *Maduca sexta*. These biogenic amines are synthesized by thoracic and abdominal ganglia of *Manduca* (Maxwell et al., 1978). Thoracic ganglia contain 4.2 pmol of octopamine (Klaassen, 1983); quantities of the two other compounds have not been determined. Two questions were addressed in this study: (1) What are the effects of the three amines on production of the flight motor pattern? (2) Do the amines that have similar effects on patterned output have similar mechanisms of action, or do they differ in their site and mode of action? To answer these questions, it was necessary to develop a dissected preparation in which compounds could be injected centrally and a response could be identified and monitored easily. During the study, it became apparent that the number of intact sensory nerves altered the effects of octopamine on motor output but not the effects of dopamine. Additional experiments were then designed to analyze the importance of sensory input in the action of these biogenic amines.

METHODS

Larvae of *Manduca sexta* were reared on a carrageen-based artificial diet and maintained on a long-day (16 h), short-night (8 h) cycle. Adult moths, 1–2 days following eclosion, were dissected ventrally to expose the thoracic ganglia. In experiments studying the role of sensory input, sensory nerves and connectives to the head and abdomen were severed, thus isolating thoracic ganglia from peripheral and central inputs. In some experiments, fine wire recording electrodes were placed in two antagonistic flight muscles of the mesothorax, the dorsal longitudinal depressor (dl_1) and the dorsal oblique elevator (dl_2) (nomenclature according Nüesch, 1953). In all experiments, either a suction recording electrode was placed on IIN1b, a nerve that branches to dl_1 and dl_2 (Nüesch, 1957; Eaton, 1974), or a pair of electrodes was positioned on the IIN1b branches innervating dl_1 and dl_2, designated here as Ndl1 and Ndl2, respectively.

Dopamine, DL-octopamine or serotonin (Sigma Chemical Company) dissolved in saline ($1\text{-}10 \times 10^{-2}$ *M*) was pressure injected into medial or lateral regions of thoracic ganglia. Controls were injected with equimolar concentrations of glucose in saline or with saline alone. Saline

composition was 53 mM NaCl, 9.3 mM KCl, 6.1 mM $CaCl_2$, 7.9 mM $MgCl_2$, 114 mM Na-methanesulfonate, and 27.8 mM Tris-methanesulfonate, pH 7.0. Micropipets used to inject the solutions were 1.2 mm O.D. capillary tubing pulled to a fine tip with a Brown-Flaming P-77 puller. The tip was broken under 250 × magnification to allow its insertion through the sheath; in some experiments the sheath was treated with 3% pronase for 1–1.5 min to facilitate penetration. Injection pipets were calibrated using an ocular micrometer to measure the diameter of the droplet ejected during a pressure pulse, which was supplied by a picospritzer (General Valve Corporation). The volume of injections ranged from 5–100 nL. In some experiments, larger volumes (0.1–1 μL) were delivered using a 12 mL syringe to supply the pressure (Kinnamon et al., 1984). The injection pipet was inserted through the ventral surface of thoracic ganglia until the pipet tip had just penetrated the sheath. All experiments were performed at 20–23°C.

The influence of sensory input on the effect of octopamine was examined by stimulating electrically a wing sensory nerve (IIN1c) before and after injection. A 200 ms stimulus train consisting of 30 pulses, each of 2 ms duration, was given every 2.5 s at a voltage (1–4 V) that elicited short bursts of motor activity in untreated preparations.

Motor neurons innervating flight muscles dl_1 or dl_2 were visualized by backfilling their respective branches, Ndl1 and Ndl2, with 0.25 M $CoCl_2$ followed by ammonium sulfide development (Pitman et al., 1973) and silver intensification (Bacon and Altman, 1977). Although sensory afferents and neurosecretory axons are present in the nerve (Wasserman, 1982), only the axons of flight motor neurons were large enough to be backfilled successfully.

RESULTS

Flight Motor Pattern (FMP).

Rhythmic motor output can be recorded from intact moths during fixed flight and from dissected preparations during large amplitude wing flapping. In *Manduca sexta* and other hawkmoth species, the recruitment, patterning and phase relationships of direct and indirect flight muscles during tethered flight have been well characterized (Kammer, 1967, 1968, 1971). Indirect wing depressor and wing elevator muscles provide power for the downstroke and upstroke, respectively, and exhibit an easily identifiable antiphasic motor pattern during nonturning flight [Fig. 1(A)]. Flight in tethered moths can be elicited by delivering air puffs or tactile stimuli to head or wings.

In dissected preparations of *Manduca sexta*, wind or tactile stimuli evoke from depressor and elevator flight muscles rhythmic activity that is similar to the flight pattern of intact moths [Fig. 1(B)]. As observed in nonturning fixed flight, antagonistic flight muscles of the dissected moth are active in antiphase and generate vigorous large-amplitude wing flapping.

To characterize further the motor output in ventrally dissected preparations, the activity of two antagonistic flight muscles was correlated with activity in a thoracic motor nerve, IIN1b. The two muscles chosen for study, the mesothoracic depressor dl_1 and elevator dl_2, are active in antiphase during straight flight (Kammer, 1971) and are innervated by branches Ndl1 and Ndl2 of nerve IIN1b (Nüesch, 1957; Eaton, 1974). Extracellular recordings from nerve IIN1b revealed a motor pattern in phase with the alternating rhythmic excitation of dl_1 and dl_2 that is observed during large-amplitude wing flapping [Fig. 2(A)]. Simultaneous recordings of activity in Ndl1 and Ndl2 demonstrated that the centrally-generated motor output has two antiphasic components, one in each branch of IIN1b [Fig. 2(B)]. Combined, the activity from both branches forms a motor pattern that excites the dl_1 and dl_2 antagonistic flight muscles to power rhythmic up and down movements of the wing.

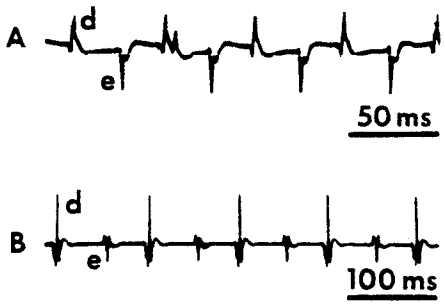

Fig. 1. Extracellular recordings from antagonistic flight muscles (dl_1 and dl_2) during flight activity. (A) Flight pattern from a tethered moth. Muscle potentials were recorded during fixed flight by inserting fine copper wires through the cuticle into depressor (d) and elevator (e) muscles. Flight frequency: 20 Hz. (B) Flight pattern from a dissected preparation. Muscle potentials were recorded as in A with wire electrodes placed into exposed depressor (d) and elevator (e) muscles. Flight frequency: 10 Hz.

Although the flight-like rhythms expressed in dissected preparations are elicited by relevant stimuli and show correct phase relationship between antagonistic muscles, the patterned activity is produced at a lower frequency (10 Hz) than in intact moths (20 Hz). This reduction in frequency is likely due to a decrease and/or distortion in sensory input resulting from surgery and from the restricted wing and body movements of ventrally-dissected preparations. Similar reductions in flight frequency have been observed in intact moths with restricted wing movements (Kammer and Rheuben, 1976) and in dissected preparations of locust (Robertson and Pearson, 1982; Sombati and Hoyle, 1984b).

To determine the number and distribution of motor neurons innervating dl_1 and dl_2, the branches of IIN1b were backfilled with cobalt chloride.

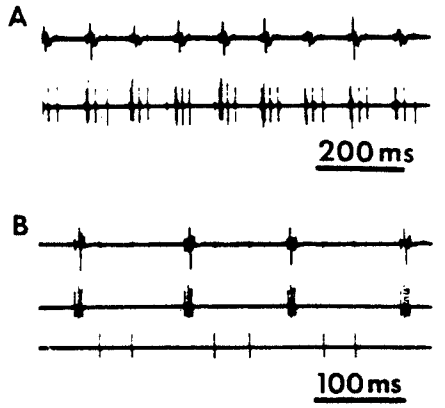

Fig. 2. The flight motor pattern (FMP) recorded from nerve IIN1b. (A) Top trace, muscle potentials recorded from a wing depressor muscle, dl_1, during flight activity. Bottom trace, simultaneous recording from nerve IIN1b showing the activity of motor neurons that innervate dl_1 and a wing elevator muscle, dl_2. (B) Top trace, dl_1 muscle potentials as in A. Middle trace, a simultaneous recording of depressor motor neuron activity from Ndl1, the branch of IIN1b that innervates dl_1. Bottom trace, a simultaneous recording of elevator motor neuron activity from Nd12, the branch innervating dl_2. The combined output of both branches forms the FMP. A and B are different preparations that were treated with 5×10^{-9} mol dopamine.

Seven cell bodies in the thoracic ganglia were stained consistently (Fig. 3). Five of the seven were stained by backfilling Ndl1, the IIN1b branch that innervates the depressor dl_1. Four of these neurons have cell bodies in the prothoracic ganglion ipsilateral to the filled nerve with the fifth having a contralateral soma in the mesothoracic ganglion. Cobalt backfills of Ndl2, the branch that innervates the elevator dl_2, stained two cells with somata ipsilateral to the filled nerve and adjacent to the dl_1 prothoracic motor neurons.

Extracellularly recorded motor neuron activity that showed appropriate phase relationships (Fig. 2) provided a means by which the production of flight motor output could be identified. This neural activity is designated the flight motor pattern (FMP). Motor activity not patterned or not exhibiting the antiphasic flight patterning is described as unpatterned or nonflight activity.

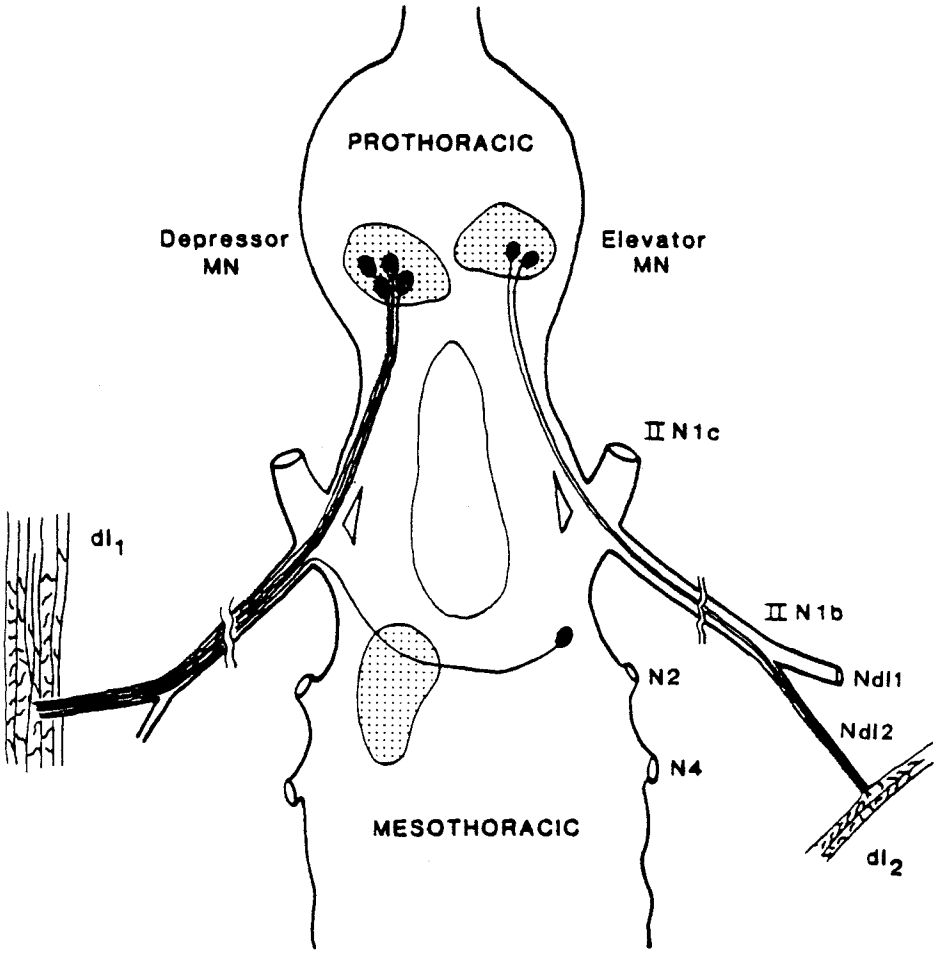

Fig. 3. A diagrammatic representation of the prothoracic and mesothoracic ganglia and IIN1b flight motor neurons. The flight motor nerve IIN1b innervates both dl_1 and dl_2. Cobalt backfills of the dl_1 branch (Ndl1) stained five motor neurons (left). Backfills of the dl_2 branch (Ndl2) stained two motor neurons (right). Shaded areas represent dendritic arborizations of the filled neurons. All motor neurons are located bilaterally in the thoracic ganglia. Nerve IINlc is a sensory nerve from the wing.

Effects of Biogenic Amines on the FMP

In the first set of experiments, dopamine, octopamine and serotonin were injected into prothoracic and mesothoracic ganglia of preparations with intact sensory nerves. The output of flight motor neurons was monitored in nerve IIN1b. In all preparations, no activity was present in IIN1b for at least 10 min before an injection.

Effects of Dopamine

Injection of dopamine (3×10^{-8} mol) into the prothoracic ganglion evoked single, large-amplitude spikes within 2 min in 4 of 5 trials [Fig. 4(A)]. These action potentials in IIN1b were constant in amplitude and were produced at a cycle time of 20–35 ms. The single spikes were the only activity elicited by prothoracic injection; at no time was bursting activity or the FMP observed.

Injection of dopamine into the mesothoracic ganglion had different effects on production of the FMP depending on the region injected. Dopamine ($2-4 \times 10^{-8}$ mol) injected into lateral regions elicited no activity in IIN1b in 5 of 6 trials [Fig. 4(B)], although leg and abdomen movements were detected. However, smaller doses ($1\text{-}5 \times 10^{-9}$ mol), when injected into the medial region of the mesothoracic ganglion, triggered the FMP [N = 5; Fig. 4(C)]. Flight motor output occurred within seconds after injection with little or no non-flight activity preceding or following the FMP. Duration of the dopamine-induced FMP was dose-dependent for injections greater than 5×10^{-11} mol (Fig. 5). At the highest dose tested (10^{-8} mol), dopamine maintained the FMP 20 min, at which time the experiment was terminated.

Effects of Octopamine

Injection of octopamine (2×10^{-8} mol) into the prothoracic ganglion of preparations with intact sensory nerves elicited multiple large-amplitude

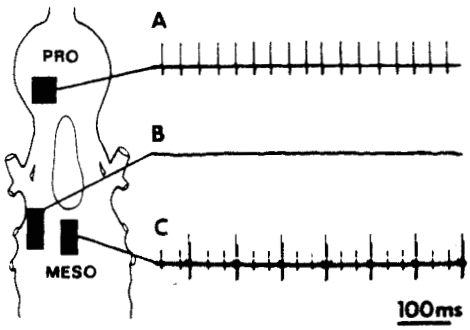

Fig. 4. Effects of dopamine in the lateral and medial regions of thoracic ganglia. Shaded areas outline regions of injections. Traces are extracellular records of activity in nerve IIN1b. (A) Prothoracic (PRO) injection of dopamine (3×10^{-8} mol) elicited single, large amplitude spikes; the FMP was not produced. (B) Mesothoracic (MESO) injections ($2–4 \times 10^{-8}$ mol) into lateral regions had no effect. (C) Mesothoracic injection ($1\text{-}5 \times 10^{-9}$ mol) into the medial region triggered the FMP.

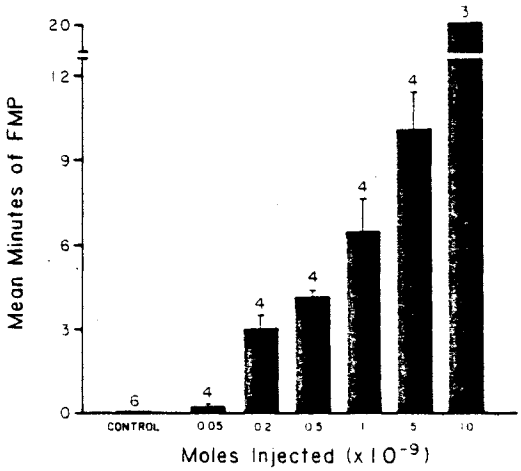

Fig. 5. Duration of the FMP induced by injecting dopamine into the medial region of the mesothoracic ganglion. Maximum observation time was 20 min. Controls were injected with saline or glucose dissolved in saline. *N* for each dose is shown above bar. Error bar indicates one standard deviation.

action potentials in IIN1b within 2 min of injection in 4 of 5 trials [Fig. 6(A)]. The unpatterned activity appeared to involve only prothoracic motor neurons as demonstrated by severing the connective containing the axon of the mesothoracic contralateral motor neuron and observing no change in IIN1b activity. Unpatterned motor output was the only response elicited by prothoracic injection of octopamine.

The effect of octopamine in the mesothoracic ganglion varied with the region of injection. Octopamine (4-6 $\times$ 10^{-8} mol), when injected into the medial region, elicited no activity in IIN1b within 2 min in 7 of 9 preparations [Fig. 6(C)]; large-amplitude, single spikes were produced in the other two cases. Injections of a lower dose (4 $\times$ 10^{-9} mol) into lateral regions of the

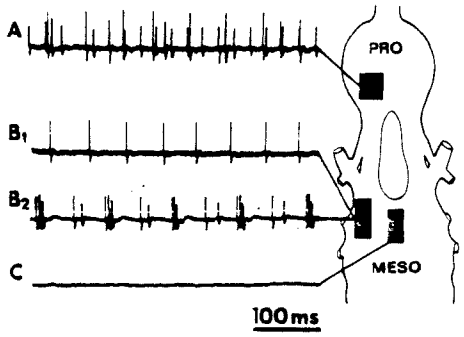

Fig. 6. Effects of octopamine in thoracic ganglia. Shaded areas outline regions of injections. Traces are recordings from nerve IIN1b. (A) Prothoracic (PRO) injection of octopamine (2 $\times$ 10^{-8} mol) activated multiple spikes from prothoracic motor neurons; the FMP was not produced. (B_1)Mesothoracic (MESO) injections (4 $\times$ 10^{-9} mol) at lateral regions elicited single, large-amplitude spikes. (B_2) At doses exceeding 10^{-8} mol, the spiking activity in B_1 was often followed by the FMP. (C) Mesothoracic injection (4-6 $\times$ 10^{-8} mol) at the medial region had no effect.

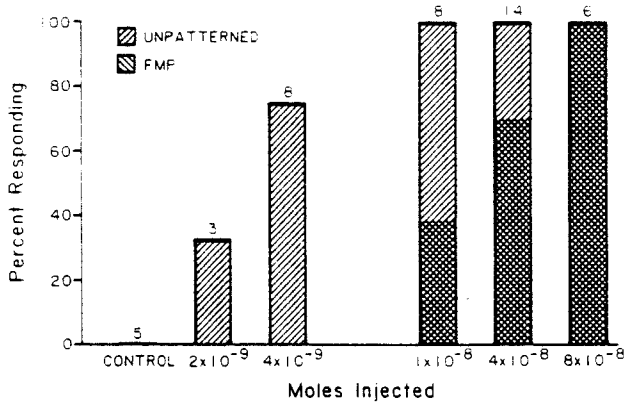

Fig. 7. Effect of injected octopamine on the production of nonflight and flight motor activity in IIN1b. Increasing doses of octopamine injected into lateral regions of the mesothoracic ganglion increased the occurrence of unpatterned, large-amplitude spiking (right diagonal lines). At higher doses, octopamine increased the number of preparations that also produced the FMP (left diagonal lines). Controls were injected with saline or glucose dissolved in saline. N for each dose is shown above bar.

mesothoracic ganglia elicited single, large-amplitude action potentials in Ndl1 within 2 min in 6 of 8 trials [Fig. 6(B_1)]. When the connective containing the axon from the mesothoracic motor neuron was severed in 4 animals producing large amplitude spikes, activity continued unabated indicating that the lateral mesothoracic injection of octopamine activates a depressor motor neuron in the prothoracic ganglion.

At doses exceeding 10^{-8} mol, injections of octopamine into lateral regions of the mesothoracic ganglion elicited one and then several large-amplitude spikes that often became organized as the FMP [Fig. 6(B_2)]. The effect of octopamine on the production of non-flight and flight motor output was dose-dependent (Fig. 7). At 8×10^{-8} mol, the FMP was elicited in 6 of 6 preparations. Duration of the octopamine-induced FMP was variable (2–20 min) and appeared related to the number of intact peripheral nerves. Upon termination of the FMP, preparations produced unpatterned activity. Tactile stimulation of the wing or head increased motor output and often reinitiated the FMP.

Effects of Serotonin

Injecting serotonin into thoracic ganglia did not elicit motor activity in nerve IIN1b. To determine whether serotonin inhibited flight output, serotonin was injected after the FMP had been initiated by dopamine or octopamine. Medial injection of serotonin (5×10^{-10} mol) into the mesothoracic ganglion terminated the dopamine-induced (5×10^{-10} mol) FMP within 60 s in 8 of 8 trials. In contrast, lateral mesothoracic injection of serotonin did not inhibit the FMP. In control experiments, medial injection of 0.1 μL saline had no effect on the dopamine-induced FMP in 5 of 6 trials.

Serotonin had little effect on motor activity elicited by 10^{-8} mol octopamine. Although serotonin (10^{-8} mol) decreased the number of spikes per burst or stopped activity in 8 of 13 trials, the effect was only temporary with

nonflight motor output returning within 4–6 min in 50% of those cases. Injection of an equal volume (1 μL) of saline also temporarily suppressed activity in 3 of 3 trials.

Influence of Sensory Input on the Amine-Induced FMP

To determine whether the effect of octopamine or dopamine on the FMP required sensory input, all sensory nerves and connectives to the head and abdomen were cut, thereby isolating thoracic ganglia from peripheral and central inputs. In these preparations, octopamine (2-4 $\times$ 10^{-8} mol) injected into lateral mesothoracic regions elicited no IIN1b activity within 10 min (N = 5; Fig. 8). This is in contrast to the response of sensory-intact thoracic ganglia, where octopamine elicited motor output within 2 min following lateral mesothoracic injection.

Low doses of dopamine (2-10 $\times$ 10^{-10} mol) injected into ganglia isolated from sensory input elicited activity similar to that induced in intact ganglia; injection at the medial mesothoracic region triggered the FMP (N = 6; Fig. 8). Thus, unlike octopamine, the effect of dopamine on the FMP appears to be independent of sensory input.

Effect of Octopamine on the Response to Electrical Stimulation

To determine whether octopamine increases the response of thoracic ganglia to sensory input, the wing sensory nerve IIN1c was stimulated electrically before and after injecting low doses of octopamine (1-4 $\times$ 10^{-10} mol) into

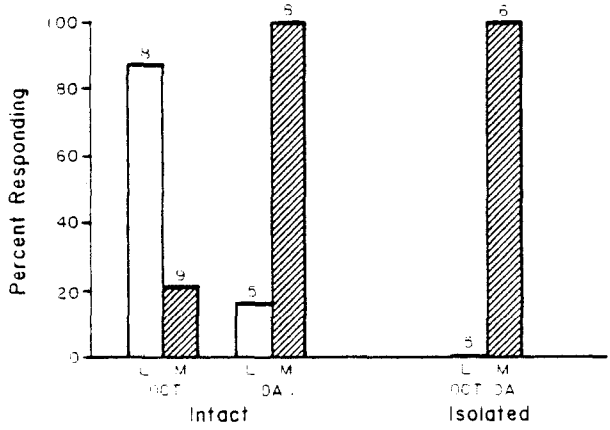

Fig. 8. Effects of octopamine (OCT) and dopamine (DA) injections in lateral (L) and medial (M) regions of mesothoracic ganglia in the presence and absence of sensory input. In intact thoracic ganglia, octopamine injected in lateral regions (4 $\times$ 10^{-9} mol) elicited a response more often than did injections into the medial region (4–6 $\times$ 10^{-8} mol). In contrast, injections of dopamine evoked more responses in the medial region (1–5 $\times$ 10^{-9} mol) than in lateral regions (2–4 $\times$ 10^{-8} mol). In isolated thoracic ganglia, lateral injections of octopamine elicited no response (2–4 $\times$ 10^{-8} mol), whereas dopamine remained effective in the medial region (2–10 $\times$ 10^{-10} mol). Response to injection was defined as appearance of motor output in nerve IIN1b within 2 min following injection. N for each set of experiments is shown above bar.

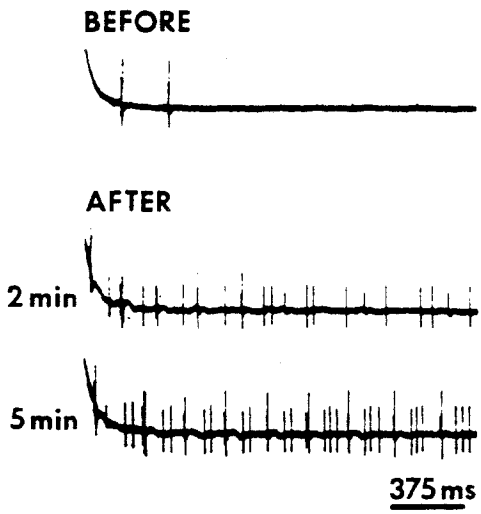

Fig. 9. Recordings of IIN1b activity generated by electrical stimulation of a wing sensory nerve before and after injecting octopamine. Each trace begins immediately following stimulation. Top trace, before octopamine injection; few spikes were produced in response to stimulation. Middle trace, 2 min after injection; irregular spiking activity followed electrical stimulation. Bottom trace, 5 min after injection; the FMP was produced following stimulation.

lateral regions of thoracic ganglia. Such doses are below the threshold for eliciting spontaneous activity.

In the untreated preparation, a high frequency stimulus train delivered every 2.5 s elicited low frequency bursting in nerve IIN1b (Figs. 9,10). After injecting octopamine, stimulation of the wing sensory nerve increased bursting activity within 2 min in all preparations and evoked low-frequency FMP within 10 min in 5 of 6 preparations (Figs. 9,10).

DISCUSSION

Results from this study provide evidence that dopamine, octopamine and serotonin act on the central nervous system to initiate, modulate, or terminate the production of flight motor output in *Manduca sexta*. Dopamine and octopamine excite production of the FMP whereas serotonin inhibits the activity. Serotonin acts within the medial region of the mesothoracic ganglion, the same location at which dopamine exerts excitatory actions.

Although both dopamine and octopamine elicit the FMP from sensory-intact preparations, they act at different locations in mesothoracic ganglia. Dopamine triggers the FMP when injected into the medial region, whereas octopamine is effective when injected into lateral regions (Fig. 8). Localization of amine action to either lateral or medial regions applies to motor activity in nerve IIN1b and does not exclude actions of these compounds in other regions of the CNS.

Results from injections into isolated thoracic ganglia show that dopamine and octopamine also differ in their mechanisms of action. When sensory nerves are cut, dopamine can activate the FMP whereas octopamine cannot.

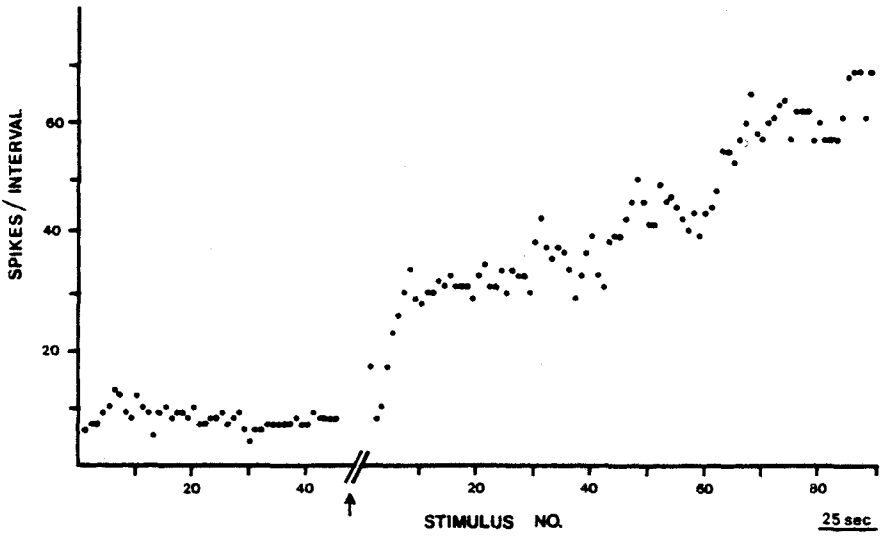

Fig. 10. Spiking activity in IIN1b in response to electrical stimulation of a wing sensory nerve before and after injecting octopamine. The number of spikes produced during the 2.5 s interval following each stimulus train was counted. Octopamine injected at arrow increased response to stimulation. Data, which are from one experiment, are representative of results from 4 of 5 similar experiments.

The action of octopamine requires sensory input; supplementing input via electrical stimulation increases the effectiveness of octopamine.

Although the effective doses of octopamine were relatively high compared to octopamine levels in thoracic ganglia, we believe these doses were required in the present study for two reasons: (1) It is not known what fraction of the octopamine injected is available for binding to the receptor. The effectiveness of applied amines may be reduced by monoamine oxidase inactivation and the presence of diffusion barriers in the CNS. (2) Since the effectiveness of octopamine depends on the level of sensory excitation, increased doses may be needed to compensate for reduced sensory input and/or for inhibitory inputs in dissected preparations.

Dopamine

Flight motor output in *Manduca* is activated more effectively by dopamine than by octopamine. Dopamine can elicit the FMP at lower concentrations than can octopamine, and in a shorter response time. The pattern induced by dopamine is produced abruptly following injection, is maintained at a relatively constant burst frequency for the duration, and is terminated abruptly. Little or no unpatterned motor activity precedes or follows the dopamine-induced flight pattern. These characteristic effects of dopamine on the generation of the FMP in either the presence or absence of sensory input suggest that dopamine acts on the flight generator, possibly as the transmitter for neurons that normally command the behavior.

Dopamine initiates motor patterns in other systems isolated from sensory input. In *Limax* (Wieland and Gelperin, 1983) and *Helisoma* (Trimble and Barker, 1984), isolated buccal ganglia are triggered by dopamine to produce

output similar to the centrally generated feeding motor pattern. In lobsters, dopamine induces a pattern, similar to the pyloric motor pattern, from isolated stomatogastric ganglia in which the spontaneous pyloric rhythm has been inactivated by TTX (Raper, 1979; Anderson and Barker, 1981).

Octopamine

In *Manduca*, octopamine increases the probability that the FMP will be produced, but only when injected into mesothoracic ganglia with intact sensory nerves. The effect of octopamine on these preparations is unlike that of dopamine because nonflight motor output often precedes and follows production of the FMP. A similar nonflight spiking pattern is observed when subthreshold doses of octopamine are applied to the mesothoracic ganglion. In addition, octopamine injected into the prothoracic ganglion elicits unpatterned activity from prothoracic motor neurons. It appears that octopamine can activate motor output via multiple pathways. Suprathreshold doses of octopamine injected into the mesothoracic ganglion may activate the flight pattern generator since the FMP is elicited. Moreover, since nonflight motor output is also observed, octopamine can activate IIN1b motor neurons by other pathways in both prothoracic and mesothoracic ganglia.

Sensory Input

That octopamine increases the efficacy of sensory input has recently been shown in locusts (Sombati and Hoyle, 1984a). In the present study, motor activity generated in response to a fixed electrical stimulus was increased up to 600% after injecting octopamine. Enhancement of the response to sensory input by octopamine could result from either increased release of transmitter from sensory afferents or increased responsiveness of postsynaptic premotor interneurons or motor neurons. In crayfish, octopamine increases the response of the lateral giant escape reaction to electrical stimulation of a sensory nerve by acting presynaptically to the lateral giant command neurons (Glanzman and Krasne, 1983). In lobsters, a presynaptic action of octopamine increases the excitability of abdominal extensor motor neurons by enhancing input to those neurons (Harris-Warrick and Kravitz, 1984).

The present study demonstrates the importance of considering sensory input in the action of neuroactive compounds. Dopamine and octopamine appear to have similar effects on flight production until the role of sensory input is examined. The results suggest that octopamine elicits flight activity by modulating sensory transmission, whereas dopamine appears to act directly on the central pattern generator.

Although the centrally generated motor pattern for flight can be produced without sensory input, it is likely that the central program is influenced by peripheral and central inputs, and that neuroactive compounds such as biogenic amines modulate that influence, as well as directly regulate production of the motor program.

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