of sympathetic neurons. However, the reduced half-life of cardiac noradrenaline after chronic administration of guanethidine indicates that synthesis of NA still occurred and that the organism tried to compensate for the depleted stores of the neurotransmitter, noradrenaline. These biochemical results are supported by histochemical findings. A complete depletion of NA in the sympathetic nerves of rat iris was observed after 3 weeks treatment with guanethidine (25 mg/kg/day). However, after a prolonged treatment with the same dose up to 5 weeks, a refillment in some of the sympathetic nerves in rat iris was observed 15 min after i.v. injection of 0.2 µg/kg α-methyl-noradrenaline[12].

Zusammenfassung. Chronische Behandlung mit Guanethidin führte bei Ratten neben einer starken Verminderung des endogenen Noradrenalingehaltes im Herzen zu einer ausgeprägten Verminderung der Aufnahme von ^{3}H-(−)-Noradrenalin und zu einem stark beschleunigten Noradrenalinumsatz.

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Aphrodisiac Effect of L-DOPA and Apomorphine in Male Sexually Sluggish Rats

In our first report on the stimulatory effect of parachlorophenylalanine (PCPA) on male sexual activity in rats, we observed that male to male mounting behavior produced by this drug was greatly potentiated by pargyline, a monoamine-oxidase inhibitor[1]. Since the administration of pargyline to animals treated with PCPA, a specific inhibitor of serotonin synthesis[2], produces a selective accumulation of brain catecholamines in the absence of serotonin, we suggested that this monoamine might inhibit and catecholamines stimulate sexual behavior in male animals[1].

The present report shows that either apomorphine, or a combination of Ro 4-4602 with L-DOPA, increases the copulatory behavior of sexually sluggish male rats, and that this effect is prevented by haloperidol. In addition, haloperidol suppresses the spontaneous copulatory behaviour of rats with high level of sexual activity. These results suggest that brain dopamine stimulates copulatory behaviour in male rats.

Male wistar rats, weighing 300–350 g, were housed individually, starting at least 1 week before the beginning of the experimental period, under a reversed light-dark cycle (with light from 21.00 h to 09.00 h) and fed ad libitum. Each rat underwent 4 mating tests with a female in oestrus, at weekly intervals, as described below. At the end of this training period, 50 of these rats which did not reach ejaculation in 3 out of 4 of the mating tests, were classified as sexually sluggish and selected for this study.

Female wistar rats were ovariectomized 3 weeks before use and brought into heat by s.c. injections of oestradiol and progesterone in olive oil[3]. Mating tests were carried out during the dark phase of the cycle, from 10.00 h to 12.00 h, in a red light.

A female was introduced into the male's own cage and the test was terminated if the male rat failed to ejaculate within 30 min. Patterns of copulatory behaviour were scored according to Beach[4]. Every animal received one mating test under each of the 6 treatments tested, according to a latin square design.

The Table shows the effect of apomorphine and a combination of L-DOPA with Ro 4-4602, a peripherally

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Stimulation by apomorphine or L-DOPA of the copulatory behaviour of male sluggish rats and inhibition of this effect by haloperidol

Treatment	Haloperidol (mg/kg i.p.)	% of animals exhibiting at least one		
		Mounting[a]	Intromission[a]	Ejaculation[a]
Saline	—	58	54	14
Apomorphine	—	80	80	62
Ro 4-4602 + L-DOPA	—	90	90	64
Saline	1	0	0	0
Apomorphine	1	20	20	12
Ro 4-4602 + L-DOPA	1	10	10	0

Each values was obtained from 50 rats. Each rat underwent different mating tests, at weekly interval, with and without treatment. Two doses of L-DOPA (100 mg/kg each) were injected i.p. 20 and 50 min after Ro 4-4602, respectively. The experiment was performed $^1/_2$ h after the last treatment. [a] Occurring within 30 min after male and female rats were paired. Apomorphine (0.5 mg/kg) and haloperidol were injected 15 min and 2 h prior to the mating test, respectively.

acting decarboxylase inhibitor[5,6], on the copulatory behaviour of male sluggish rats. Both treatments significantly increased the percentage of animals showing mounts, intromissions and ejaculations.

The aphrodisiac effect of L-DOPA and apomorphine was prevented by the administration of haloperidol, a specific inhibitor of dopamine receptors in brain[7]. In addition, haloperidol completely suppressed the copulatory behaviour spontaneously present in male rats.

The present investigation has shown that the copulatory behaviour of sexually sluggish male animals is stimulated by a combination of L-DOPA with Ro 4-4602 and by apomorphine. Since the administration of L-DOPA to rats treated with Ro 4-4602 has been shown not only to increase the content of brain catecholamines[5], but also to decrease that of serotonin[8], the stimulatory effect on male copulatory behaviour can be ascribed to either mechanism. On the other hand, since apomorphine is considered to act as a direct stimulant of the dopamine receptors in brain[9], the finding that this compound also stimulates the copulatory behaviour in male rats supports the hypothesis that dopamine plays a stimulatory role on male sexual behaviour.

Consistently, the effect of apomorphine and L-DOPA was prevented by haloperidol, a specific inhibitor of dopaminergic receptors in brain[7]. Moreover, this drug also suppressed the spontaneous copulatory behaviour of male rats with high basal level of sexual activity.

Riassunto. L'apomorfina e l'associazione di L-DOPA con Ro 4-4602, un inibitore della decarbossilasi non cerebrale, stimolano il comportamento copulatorio nei ratti maschi con scarsa attività sessuale di base. L'effetto afrodisiaco della L-DOPA e dell'apomorfina è prevenuto dall'aloperidolo.

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Effects of GABA on Presynaptic Nerve Terminals in Bullfrog (*Rana catesbiana*) Sympathetic Ganglia

It is known that the synaptic transmission in mammalian sympathetic ganglia is inhibited by γ-amino-butyric acid (GABA)[1]. The synaptic transmission in bullfrog sympathetic ganglia is also inhibited by the action of this drug. The present communication deals with the presynaptic inhibitory mechanism underlying such an inhibitory action of GABA.

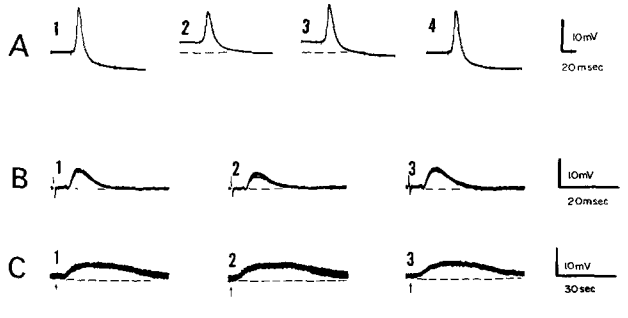

Fig. 1. Effects of 0.1 m*M* GABA on the nicotinic transmission in bullfrog sympathetic ganglia, which was partially blocked by D-tubocurarine (7×10^{-3} m*M*). A) Inhibition of the nicotinic response of ganglion cells. These potentials were recorded by the sucrose-gap method before (1), 1 min (2) and 7 min (3) after an application of GABA, and record 4 was taken 30 min after its withdrawal. Note the original potential level shown by broken lines. B) The fast EPSP recorded from a single ganglion cell before (1) and 1 min (2) after an application of GABA. Record 3 was taken 5 min after its withdrawal. Note the original potential level shown by broken lines. C) The nicotinic ACh depolarization produced by direct applications of ACh before (1) and 1 min (2) after an application of GABA. Record 3 was taken 3 min after its withdrawal. 0.2 cm^3 ACh solution (10 m*M* ACh-Cl in Ringer's solution) was injected into the perfusate at the moments shown by arrows. Note the original potential level shown by broken lines.

Methods. Paravertebral sympathetic ganglion chains of bullfrog (*Rana catesbiana*) were used. The synaptic (nicotinic) transmission mediated by the fast excitatory postsynaptic potential (fast EPSP) was observed by applying supramaximal electrical stimulations (0.5 msec pulses) to preganglionic B nerve fibres[2]. The membrane potentials of preganglionic nerve axons, preganglionic nerve terminals, and ganglion cells were recorded by the sucrose-gap method[3,4]. The intracellular potential of ganglion cells was also recorded[5]. Ionic compositions of the Ringer's solution are as follows: 112 m*M* NaCl, 2 m*M* KCl, 1.8 m*M* $CaCl_2$, and 2 m*M* $NaHCO_3$. Na ions in the Ringer's solution were totally replaced by equimolar *tris* (hydroxymethyl) aminomethane for preparing the Na-free *Tris* solution. Preparations were continuously perfused with a solution flowing through a chamber ($50 \times 5 \times 4$ mm) at the rate of 0.2 cm^3/sec.

Results. The nicotinic transmission in bullfrog sympathetic ganglia was inhibited in the presence of GABA in concentrations of $1\text{–}10^{-2}$ m*M*. Such an inhibition could be observed when GABA was applied to preparations of which nicotinic transmissions were partially blocked by D-tubocurarine (7×10^{-3} m*M*) (Figure 1-A). Sympathetic ganglion cells were depolarized under the effect of GABA[1]. As seen in Figure 1-A, the nicotinic transmission was partially restored, while the depolarization of ganglion cells remained unchanged, when GABA was applied for more than 5–10 min.

In order to clarify the mechanism underlying the inhibition of nicotinic transmission, changes in the fast

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