imately 0.5 °C. This decrease was readily reversible upon re-exposure of the rat to 100% O_2.

Similar effects of hypoxia were observed when the chamber temperature had been lowered to 12 °C. Under conditions of 100% O_2 and 30 min of cooling the chamber, there occurred an average rise in temperature of the BAT of 0.7 ± 0.15 °C and of the core, 0.3 ± 0.24 °C. These cold-induced thermogenic responses were abolished when the atmosphere was changed from 100% to 12% O_2. In such an environment, the temperature of both the brown fat and core fell precipitously (after 1 h, BAT temperature was down 3.0 °C and core, 2.3 °C). Furthermore, after 15 min of exposure to hypoxia, the temperature of the BAT no longer exceeded that of the core. Return to a 100% O_2 atmosphere (still at 12 °C) resulted in a rapid increase in both the BAT and core temperatures and after 5 min, the temperature of the BAT again exceeded that of the core in all rats. At the end of the experiment, 4 of the 6 rats were returned to warm conditions (while breathing 100% O_2) and within 15 min the temperature of the brown fat had decreased to approximately that of the core.

These rapid and reversible hypoxic-induced changes in the temperatures of the brown fat and core are similar to those previously reported for rats under anesthesia[2]. Additionally, in the present study, exposure to hypoxia depressed or abolished the response of brown fat to injected NE (Table). That is, although a positive response to NE was observed in all 6 animals breathing 100% O_2 at 26 °C, only 3 of these rats exhibited such a response when breathing 12% O_2 at 26 °C, and none when exposed to 12% O_2 at 12 °C.

These data thus indicate that the hypoxic-induced depression of brown fat thermogenesis does not reflect lack of sympathetic activation at the level of the tissue. Rather the absence of a thermogenic effect of injected NE on the brown fat of cold exposed rats breathing low O_2 supports the view that the metabolism of the tissue is affected directly by the hypoxic environment. That this effect may be mediated via oxygen-limitation of cellular respiration is a conclusion similar to that reached by HEIM and HULL[3] for their observations in the newborn rabbit. Accordingly, the hypoxic inactivation of cold-induced brown fat thermogenesis is most likely a direct reflection of the concomitantly lowered arterial pO_2, although other indirect effects of low oxygen breathing (e.g. altered pCO_2) are not excluded by the data presented. Decreased regional blood flow to BAT would further limit oxygen delivery to the tissue. Such an effect for 8% O_2 inhalation has been observed in the unanesthetized euthermic woodchuck[4] but not in the anesthetized rat[5].

Résumé. Chez des rats adaptés au froid, non anesthésiés, placés brièvement dans une atmosphère de 12% d'oxygène, on observe un arrêt immédiat de la thermogénèse de la graisse brune. Cet effet semble s'expliquer par le manque d'oxygène au niveau cellulaire plutôt que par l'absence d'activation par le système sympathique. L'absence d'une réaction de thermogénèse en réponse à une injection de noradrénaline chez des rats soumis au froid et dans des conditions hypoxiques plaide en faveur de cette explication.

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The Presence of Octopamine in the Brain of *Helix aspersa* and its Action on Specific Snail Neurones

Octopamine, *p*-hydroxy-*β*-hydroxyphenylethylamine, was first identified in the salivary glands of *Octopus vulgaris*[1]. The distribution of octopamine in the octopus nervous system has recently been determined[2]. Octopamine has been found to be a normal constituent of rat sympathetic nervous system[3] and brain[4] and to be present in a wide range of species[4]. Octopamine would appear to be largely confined to nervous tissue since following denervation the octopamine content of an organ falls drastically[4]. Both dopamine and noradrenaline have been shown to be present in the *Helix* nervous system[5,6], but the present of octopamine has not been reported.

Materials and methods. All experiments in this study were made on the garden snail, *Helix aspersa*. A sensitive enzymatic assay for octopamine, developed by MOLINOFF, LANDSBERG and AXELROD[7], was used in the present study. The levels of noradrenaline were assayed using the method of SAELENS, SCHOEN and KOVACSICS[8]. The isolated snail brain was prepared for electrophysiological recording by a method previously described[9]. Intracellular recordings were made from identifiable neurones using glass microelectrodes filled with molar potassium acetate. Bio-electric potentials were amplified and displayed on a Tektronix 502A oscilloscope. Permanent traces were made on an AEI pen oscillograph. Drugs were dissolved in snail Ringer and added close to the preparation in a volume of 0.2 ml. The isolated snail brain was bathed in 10 ml Ringer. The Ringer was changed following each application of drug. The composition of the snail Ringer has previously been described[9].

Results and discussion. The levels of noradrenaline and octopamine were determined for brain and heart. The levels of noradrenaline in the brain were 0.82 ± 0.18 μg/g protein and for heart were 2.04 ± 0.35 μg/g protein. The results are the mean of 12 determinations. The levels of octopamine in the brain were 0.75 ± 0.15 μg/g protein and for heart were 1.32 ± 0.45 μg/g protein. The results are the mean of 6 determinations. The standard deviation is indicated in each case.

Octopamine, with a threshold of 50 pmol, was found to mimic the action of dopamine and noradrenaline on

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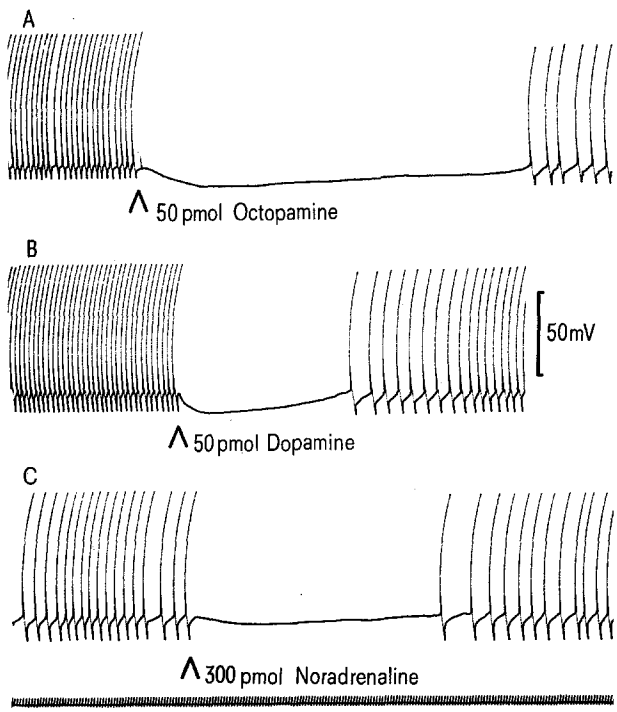
certain dopamine-sensitive cells, Figure. The activity of these cells was inhibited by all 3 compounds and the membrane potential hyperpolarized. The potency of octopamine compared with dopamine varied from equipotent to 100 times less potent. Cells desensitized to repeated applications of octopamine while still responding to a standard dose of dopamine. Cells which responded to octopamine also responded to tyramine but were about 1000 times less sensitive to tyramine. Low doses of ergometrine which blocked dopamine and noradrenaline failed to block octopamine but higher doses of ergometrine did block octopamine. Dibenyline blocked dopamine, noradrenaline and octopamine but propranolol failed to antagonize the 3 compounds. All the cells which responded to octopamine had a dopamine synaptic input. Dopamine-sensitive cells without a dopamine input were unaffected by doses of octopamine up to 500 nmol. Cocaine at a concentration of 10 µg/ml did block the action of 50 pmol octopamine but failed to block doses of octopamine in the range of 5–50 nmol.



The effects of octopamine, dopamine and noradrenaline on the spontaneous activity of a neurone from the brain of *Helix aspersa*. A) 14 mV hyperpolarization following the application of 50 pmol octopamine; B) 14 mV hyperpolarization following the application of 50 pmol dopamine; C) 5 mV hyperpolarization following the application of 300 pmol noradrenaline. The time scale is in intervals of 1 sec and the voltage scale represents 50 mV.

In the mammal octopamine is considerably less potent than noradrenaline[10,11]. In contrast, in *Helix* it is sometimes equipotent with dopamine and at least 10 times more potent than noradrenaline. In the mammal octopamine may exert a dual action, acting in part directly on catecholamine receptors and partly to release endogenous noradrenaline from presynaptic nerve terminals[11,12]. It seems unlikely that octopamine is acting directly on dopamine receptors of *Helix* neurones since in an earlier study[13], octopamine was inactive on certain dopamine-sensitive neurones. These cells do not have a dopamine synaptic input. This finding together with the observation that cocaine will block the action of low doses of octopamine provides evidence for an indirect action of the compound. Studies are in progress to determine whether this action of octopamine is a direct one on the post-synaptic membrane, an indirect one to release endogenous catecholamine or a combination of the two mechanisms.

Résumé. L'octopamine est présente dans le tissue nerveus de l'escargot, *Helix aspersa*. Elle a un effet fortement inhibitoire sur certains neurones. Ces neurones sont aussi sensibles à la dopamine et à la noradrénaline. Le mécanisme possible de l'action de l'octopamine est discuté.

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Délimitation des voies ascendantes responsables de l'activité ponto-géniculo-occipitale chez le chat

Les pointes ponto-géniculo-occipitale (PGO) apparaissent de façon synchrone au niveau des noyaux géniculés latéraux (NGL) au cours du sommeil paradoxal[1-3] ou après injection de Réserpine[4-7] (PGO_R). Nos expériences, réalisées sur 40 chats immobilisés au Flaxedil artificiellement ventilés et ayant reçu une injection de 0,5 mg/kg de Réserpine, ont permis de préciser la topographie du générateur des PGO_R, leurs voies ascendantes et d'expliquer leur synchronisme bilatéral.

Résultats. 1. Suppression du synchronisme bilatéral des PGO_R. 90 min. après injection de Réserpine, des potentiels phasiques d'une durée de 130–140 msec, d'une amplitude de 100–300 µv et à deux composantes séparées par 80 msec sont enregistrés au niveau des NGL. Ils se produisent de façon synchrone au niveau de ces deux structures (Figure 1, A). Cependant le tracé peut présenter des potentiels phasiques à une composante soit isolés, soit dans un arrangement répétitif complexe. Une section sagittale médiane de 10 mm au niveau du tegmentum pontique et une section au niveau de la décussation supra-optique sont nécessaires et suffisantes pour obtenir un asynchronisme des PGO_R qui sont alors constituées par une seule composante (Figure 1, D).

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