conclude that the eyestalk of the stomatopod *Squilla mantis* contains the neurodepressing hormone, that its NDH has the same general characteristics as the hormone obtained from decapods (a small, hydrophilic and neutral peptide), and that it cross-reacts with the *Procambarus bouvieri* bioassay. The negative results obtained with the total and crude extracts could be due to the presence of interfering substances present in the eyestalk and which are efficiently eliminated by paper electrophoresis at pH 1.8 and pH 10.0. It can be stated now that the NDH is present not only in decapods but also in the more primitive stomatopods. This speaks in favour of an evolutionary stability with general conservation of its structure, commensurate with its physiological importance as a modulator of crustacean nervous activity. In view of the above, we suggest that for analytical purposes our accelerated procedure should be used directly to detect the presence of the neurodepressing hormone in hitherto untested species.

1 This investigation was supported by grant No. 192 from the Consejo Nacional de Ciencia y Tecnología, and by a grant from Fondo de Fomento Educativo. We are very thankful to Dr Victor Anaya for performing the bioassays.
2 T.W. Roberts, Anat. Rec., suppl. *81*, 46 (1941).
3 E. Schallek, J. exp. Zool. *91*, 155 (1942).
4 E. Naylor and B.G. Williams, J. exp. Biol. *49*, 107 (1968).
5 H. Aréchiga, A. Huberman and E. Naylor, Proc. R. Soc. Lond. *187B*, 299 (1974).
6 H. Aréchiga, A. Huberman and A. Martínez-Palomo, Brain Res. *128*, 93 (1977).
7 H. Aréchiga, C. Cabrera-Peralta and A. Huberman, J. Neurobiol. *10*, 409 (1979).
8 A. Huberman, H. Aréchiga, A. Cimet, J. de la Rosa and C. Arámburo, Eur. J. Biochem. *99*, 203 (1979).
9 A. Huberman, C. Arámburo and H. Aréchiga, Proc. 6th Am. Pept. Symp. Ed. E. Gross and J. Meienhofer. Pierce Chemical Co., Rockford, Ill., p. 853 (1979).
10 A. Huberman, H. Aréchiga, C. Arámburo and I. González, Comp. Biochem. Physiol., in press (1981).

Reserpine prevents goldthioglucose hypothalamic lesions in mice

E. Briese and E. Murzi

Behavior Physiology Laboratory, Universidad de Los Andes Apartado 109, Merida (Venezuela), 17 March 1980

Summary. In reserpinized mice the occurrence of goldthioglucose hypothalamic lesions was significantly lower than in control mice. Some protection was also conferred by serotonin-receptor blockers and by treatment with nialamide + DL-α-methyldopa, but the protective effect of reserpine was not reversed by serotonergic and dopaminergic agonists, alone or in combination, nor by insulin.

When injected into mice, goldthioglucose (GTG) accumulates in the ventromedial hypothalamus (VMH) and destroys it[1]. This makes the mice eat more, and they become obese[2]. Diabetic mice are protected from GTG lesions[3], but insulin restores their vulnerability[4]. This is one of the main arguments in favour of the glucostatic theory of feeding which holds that feeding is inhibited by the VMH when enough glucose enters it[5-7]. It has been shown, however, that treatments other than with insulin, including certain mildly stressful procedures[8], may restore vulnerability to GTG in diabetic mice[9,10]. Further investigations in this laboratory have suggested a possible role for monoamine release in this phenomenon since the immunity of the diabetic mouse appeared to be enhanced by pretreatment with reserpine, a drug known to deplete brain monoamines[11].

In the present study we have further investigated the interaction of GTG and reserpine by examining their effects in normal, non-diabetic mice. In addition we examined the effect of other agents, affecting monoamine function in a more specific manner than reserpine, on GTG lesions.

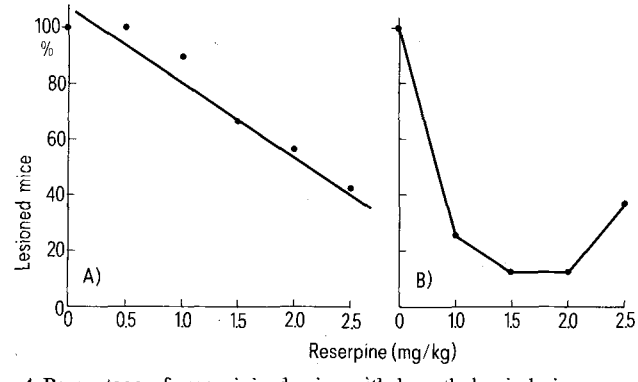
One of 2 doses (250 or 450 mg/kg, i.p.) of GTG was administrated to each mouse after treatment with reserpine or after administration of a specific synthesis - or receptor-blocker of monoamine transmitter. Hypothalamic lesions were assessed histologically in the brains of mice that had survived 3 days, as described elsewhere[10].

The protective effect of reserpine from GTG lesions can be seen in the figure A, which shows a linear dose-effect relationship. The protection was effective with 450 mg/kg of GTG, a much higher dose than ED_{100} which, in this batch of GTG and strain of mice, was of 250 mg/kg (see

Protective effect of $SQ_{10,631}$ against lesions produced by 250 mg/kg of GTG in the VMH of mice. $SQ_{10,631}$ was injected 2 h before GTG. Values are the number of mice

$SQ_{10,631}$ (mg/kg)	Total surviving mice	Lesions Yes	No
0	22	22	0
100	10	6	4
130	13	10	3
160*	5	3	2

$X^2 = 10.0$ ($p < 0.05$); $r = 0.46$ ($p < 0.01$). * LD_{50} (E.R. Squibb & Sons, Inc.; personal communication). $SQ_{10,631}$ was a gift from S.J. Lucania, The Squibb Institute for Medical Research, Princeton, N.J.



A Percentage of reserpinized mice with hypothalamic lesions produced by 450 mg/kg of GTG plotted against the dose of reserpine given 24 h prior to GTG. Regression line is $Y = -25X + 107$; $r = -.97$; $p < .001$. *B* Same results after only 250 mg/kg (ED_{100}) of GTG. Hypothalamic lesions were determined as reported in detail elsewhere[10] by photography of histological sections of the brains of the mice surviving 3 days.

control group in the table). With smaller doses of GTG the dose-response relationship was not linear, as shown in the figure, B. We were able partially to reproduce the protective effect of reserpine by methiothepin (1 mg/kg) and 2-chlorocinanserin ($SQ_{10,631}$), drugs which are serotonin-receptor blockers[12] and with a combination of nialamide (500 mg/kg) + DL-α-methyldopa (500 mg/kg) that, according to Corrodi[13], blocks the accumulation of serotonin (5HT) and norepinephrine but does not affect dopamine (DA) content in mouse brain. The protective effect of methiothepin was increased when it was given in combination with pimozide (0.5 mg/kg). In the replication of the reserpine effect, the best results were obtained with $SQ_{10,631}$, as given in the table. The reserpine effect was not reproduced with monoamine synthesis blockers and catecholamines receptor antagonists. We were not able to render reserpinized mice vulnerable to GTG either by serotoninergic and dopaminergic agonist, alone or in combination, nor by insulin.

One of the interpretations of this finding would be that we might be dealing here with a permissive factor which, in addition to insulin, is necessary for GTG to accumulate in and destroy the VMH. This factor could possibly be 5-HT and DA intervening with insulin in a chain of biochemical processes permitting the capture of GTG and of glucose by VMH. To a certain extent, the stimulation of appetite by 5-HT receptor blocking agents[14] and the anorexia produced by serotonin-like drugs[15,16] supports this view.

1 N.B. Marshall, R.J. Barrnett and J. Mayer, Proc. Soc. exp. Biol. Med. *90*, 240 (1955).
2 G. Brecher and S. Waxler, Proc. Soc. exp. Biol. Med. *70*, 498 (1949).
3 A.F. Debons, I. Krimsky, H.J. Likuski, A. From and R.J. Cloutier, Am. J. Physiol. *214*, 652 (1968).
4 A.F. Debons, I. Krimsky, A. From and R.J. Cloutier, Am. J. Physiol. *217*, 1114 (1969).
5 J. Mayer and M.W. Bates, Am. J. Physiol. *168*, 812 (1952).
6 J. Mayer, New Engl. J. Med. *249*, 13 (1953).
7 J. Mayer and N.B. Marshall, Nature *178*, 1399 (1956).
8 E. Murzi and E. Briese, unpublished observations.
9 C.A. Baile, C.I.L. McLaughlin, W. Zinn and J. Mayer, Am. J. Physiol. *221*, 150 (1971).
10 E. Briese, R. Rondon-Morales and L. Hernandez, Physiol. Behav. *7*, 807 (1971).
11 A.J. Azzaro, G.R. Wenger, C.R. Craig and R.E. Stitzel, J. Pharmac. exp. Ther. *180*, 558 (1972).
12 P.J. Langlais and S. Gabay, J. Neurosci. Res. *3*, 135 (1977).
13 H. Corrodi, J. Pharm. Pharmac. *18*, 197 (1966).
14 R.E. Noble, J. Am. med. Assoc. *209*, 2054 (1969).
15 R. Samanin, D. Ghezzi, L. Valzelli and S. Garattini, Eur. J. Pharmac. *19*, 318 (1972).
16 R. Samanin, C. Bendotti, F. Miranda and S. Garattini, J. Pharm. Pharmac. *29*, 53 (1977).

Spontaneous maze ambulation in two mouse strains

D. Sabolovic*, M. Robin and D. Oth

I.N.S.E.R.M., Unit of Experimental Cancerology and Radiobiology, Plateau de Brabois, F-54500 Vandœuvre-lès-Nancy (France), 8 April 1980

Summary. A simple device is described, which permits us to quantify several parameters of spontaneous behaviour of small animals. Using this device with mice we obtained statistically satisfactory results, showing a strong genetic influence on the behavioural characteristics tested.

An increasing number of psychobiological investigations on learning performances, memory processes and behavioural developments in mice require an adequate training apparatus. Various devices; behaviour equipments, 'torture chambers' such as the shuttle box[2-4], the running wheel[5], the Deutsch carousel[6], the photoswitch activity cage[7,8] etc. have been used, in which the animal is subjected to different escape and avoidance mechanisms, electric shocks, motion restriction, and deprivation of food and water[3,9,10].

The use of an aversive stimulus might be responsible for emotional components in subtle experiments on memory and learning in mice which can be variously interpreted. This paper describes a training apparatus which allows free exercise without any external stimulation, food and water deprivation or light or electric-shock conditioning.

Apparatus. As depicted in figure 1, the training apparatus (the apparatus is available from Medical Research Co., F-91160 Longjumeau) used for the study is composed of a 'departure' and an 'arrival' cage, both supplied with food and water, separated by a squared maze (40 × 40 cm) with multiple corridors. The latter can be modified as desired by rearrangement of walls and gates. There is a direct connection between the arrival cage and the departure cage by means of a 1-way tunnel. 1-way gates in the maze prevent the mouse from coming back.

From the departure cage, the mouse to be tested can enter the maze via a 1-way gate which will prevent it from returning to the departure cage. Entrance into the maze is detected by an opto-electronic unit which activates an electronic clock. When the mouse has found the exit and leaves the maze by a 1-way gate, it is detected by another opto-electronic unit which inactivates the electronic clock. At this moment, a printer prints the number of sec during which the mouse remained inside the maze, and the time of day in h and min. The mouse is then in the arrival cage and can return freely to the departure cage, by means of an external 1-way tunnel. A new round trip can be performed as often as the mouse decides.

Results and discussion. 2 identical mazes were used simultaneously, under the same external conditions (air-condi-

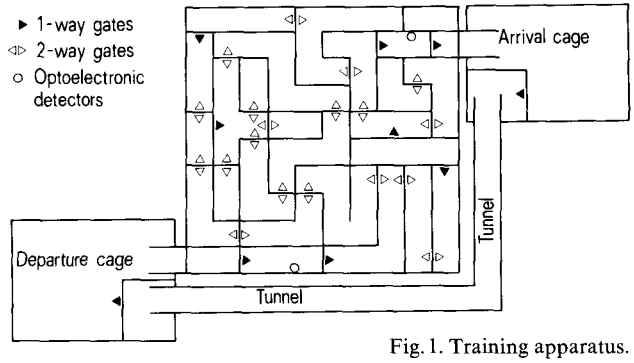


Fig. 1. Training apparatus.