in the cerebellum, a rare location for such dystrophic changes even in those cases where cerebral cortex alterations are numerous[13,15].

Although wisps of material with the ultrastructural characteristics of amyloid fibrils could be frequently seen among neurites, the so-called primitive type of plaque[19] was the one observed. These dystrophic neurite aggregates are different from spheroids seen in gracilis and cuneatus nuclei following vitamin E deficiency or in aged animals[6,28,29]. They are like the experimental neuritic plaques seen in animals in which aluminium injection within the hemispheres was combined with cortical undercutting[7]. The fact that there was no well defined amyloid core, and also no microglial reaction in these lesions, supports those authors[8] who suggest that microglia play an important role in the processing and production of amyloid in neuritic plaques. To our knowledge, no neuritic plaques of any type have yet been described in the rat cerebellar cortex following prolonged alcohol consumption. Although a discussion of the pathogenesis and associated functional implications of these findings are beyond the scope of this report, it must be stressed that these lesions are probably the end-result of a dying-back process of parallel fiber boutons due to alcohol-induced granule cell dysfunction[16] and of other alterations of the cerebellar cortex milieu which create the environmental characteristics which predispose to neurite formation, as has been suggested under different circumstances[30].

Moreover, keeping in mind that neuritic plaques are related to aging and that a precocious and progressive accumulation of lipofuscin granules in the cytoplasm of Purkinje cells has been recently described under the same experimental conditions[31,32], it is tempting to suggest that prolonged alcohol intake might interact with, and probably accelerate, those mechanisms involved in the normal biological aging of the cerebellar cortex.

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Acute changes in dopamine metabolism in the medio-basal hypothalamus following adrenalectomy

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Summary. During the first hour following adrenalectomy the α-MPT-induced disappearance of dopamine was increased in the arcuate nucleus compared to that in sham-operated rats. In a number of other brain regions of both adrenalectomized and sham-adrenalectomized rats only stress-induced changes were observed in catecholamine utilization. These data suggest that corticosterone selectively modulates dopamine utilization in the medio-basal hypothalamus.

One h after bilateral adrenalectomy the turnover of serotonin in the dorsal hippocampus is significantly reduced when compared to that of sham-operated rats[3]. A low dose of corticosterone, given immediately after adrenalectomy, restores both serotonin turnover and steroid receptor occupancy in the dorsal hippocampus, whereas dexamethasone fails to do so[3,4]. The specificity of the serotonin response in the dorsal hippocampus corresponds to the properties of the glucocorticoid receptor system in rat hippocampal neurons[3,4]. The present experiments were carried out to

Table 1. Effect of bilateral adrenalectomy (ADX) and sham-adrenalectomy (S-ADX) on α-MPT-induced catecholamine disappearance in microdissected brain regions of the rat

Treatment	−90 min: ← α-MPT → −60 min:			−150 min: ← α-MPT → −120 min:		
	Control	S-ADX	ADX	Control	S-ADX	ADX
	Catecholamine content (pg/μg protein)	% of control		Catecholamine content (pg/μg protein)	% of control	
Noradrenaline						
Dorsal hippocampus	6.7 ± 0.6[a]	94 ± 7	104 ± 10	4.6 ± 0.4	95 ± 8	92 ± 6
Periventricular thalamus	11.8 ± 0.4	92 ± 7	91 ± 9	7.6 ± 0.6	88 ± 5	105 ± 6
Lateral septal nucleus	9.8 ± 0.9	84 ± 10	75 ± 13	7.2 ± 0.9	54 ± 6[b]	69 ± 5[b]
Paraventricular nucleus	55.3 ± 15.5	111 ± 21	68 ± 11	32.8 ± 4.9	72 ± 14	53 ± 6[b]
Arcuate nucleus	7.5 ± 1.0	60 ± 11[b]	53 ± 10[b]	4.7 ± 1.2	86 ± 10	82 ± 11
Median eminence	24.2 ± 1.3	70 ± 10[b]	41 ± 9[b]	15.3 ± 1.5	95 ± 14	97 ± 16
Dorsomedial nucleus	45.7 ± 9.0	141 ± 26	107 ± 17	30.7 ± 2.4	32 ± 4[b]	58 ± 16[b]
Dopamine						
Periventricular thalamus	3.0 ± 0.3	103 ± 20	103 ± 30	1.9 ± 0.4	79 ± 11	84 ± 16
Lateral septal nucleus	16.8 ± 0.7	79 ± 11	75 ± 11	7.4 ± 1.0	91 ± 14	96 ± 23
Paraventricular nucleus	6.5 ± 1.8	94 ± 17	65 ± 8	4.3 ± 0.9	69 ± 12	62 ± 15
Arcuate nucleus	5.8 ± 1.1	118 ± 17	37 ± 8[b,c]	4.3 ± 0.7	80 ± 26	46 ± 8[b]
Median eminence	44.5 ± 6.7	91 ± 19	73 ± 19	23.9 ± 5.4	70 ± 12	121 ± 18[c]
Dorsomedial nucleus	5.5 ± 1.3	134 ± 42	69 ± 11	4.3 ± 0.6	68 ± 23	93 ± 15
Adrenaline						
Paraventricular nucleus	2.03 ± 0.49	111 ± 23	78 ± 12	1.96 ± 0.34	76 ± 10	56 ± 6[b]
Arcuate nucleus	1.00 ± 0.28	62 ± 31	117 ± 34	0.83 ± 0.20	68 ± 24	40 ± 16[b]
Median eminence	1.36 ± 0.29	77 ± 19	44 ± 12[b]	1.07 ± 0.23	n.d.	n.d.
Dorsomedial nucleus	1.34 ± 0.21	138 ± 19	113 ± 22	1.25 ± 0.21	42 ± 9[b]	58 ± 13[b]

Rats received α-MPT (250 mg/kg, i.p.) either 90 or 150 min prior to decapitation. 30 min after α-MPT treatment, i.e., 60 or 120 min before decapitation, ⅓ of the rats was adrenalectomized (ADX); ⅓ was subjected to sham-adrenalectomy (S-ADX); ⅓ was left undisturbed for 90 and 150 min respectively after α-MPT administration (Controls). Catecholamines were assayed according to Van Der Gugten et al.[17]; protein was assayed according to Lowry et al.[18]. [a] Mean ± SEM (number of animals = 6–8); [b] $p < 0.05$ for difference with respective controls; [c] $p < 0.05$ for difference between ADX and S-ADX (Student's t-test, two-tailed); n.d. = not detectable.

investigate whether catecholamine-containing systems in the brain might show changes in activity due to the absence of corticosterone shortly after bilateral adrenalectomy.

Methods. The disappearance of catecholamines following inhibition of their synthesis by the tyrosine hydroxylase inhibitor α-methyl-*p*-tyrosine was chosen as a measure of catecholaminergic activity. Rats received an i.p. injection of 250 mg/kg α-methyl-*p*-tyrosine methylester HCl (α-MPT; Labkemi, Göteborg) and were, 30 min later, subjected either to bilateral adrenalectomy under ether anesthesia (ADX) or to sham-adrenalectomy (S-ADX). Half of the rats of both groups were decapitated 60 min, the other half 120 min after operation. Control rats were left undisturbed for 90 and 150 min respectively after α-MPT administration.

Results. In a pilot experiment the brains of the rats were rapidly taken out and septum, medio-basal hypothalamus, dorsal hippocampus and periventricular thalamus were dissected on ice from 2-mm-thick brain slices. In this experiment it was found that catecholamine utilization in the dorsal hippocampus and periventricular thalamus of S-ADX and ADX rats was not altered (table 1) as compared to that in the control rats, in contrast to that in the other 2 regions (data not shown). In a 2nd experiment 3 septal and 5 hypothalamic nuclei were obtained by microdissection[5]. The results are summarized in table 1. No effects were found on the utilization of any of the 3 catecholamines in the dorsal and medial septal nuclei and in the periventricular nucleus (data not shown). An enhanced noradrenaline utilization was found in the arcuate nucleus and median eminence of ADX rats at 1 h after operation and in the lateral septal, paraventricular and dorsomedial nuclei at 2 h after operation as compared to that in controls. Adrenaline utilization was enhanced in the median eminence, arcuate nucleus and paraventricular nucleus of ADX rats. In all these cases, however, catecholamine utilization was not different from that is S-ADX rats. The only exception is found in the utilization of dopamine which was faster in the arcuate nucleus and slower in the median eminence of ADX rats as compared to that in S-ADX rats (table 1). The data on plasma corticosterone at the time of decapitation are summarized in table 2.

Table 2. Effect of treatment with α-MPT and of adrenalectomy (ADX) and sham-adrenalectomy (S-ADX) on plasma corticosterone levels

	Plasma corticosterone (μg/100 ml)
Resting value	4.7 ± 0.9[a]
30 min after α-MPT	52.0 ± 3.6[b]
90 min after α-MPT/--	58.1 ± 3.7
90 min after α-MPT/S-ADX	48.7 ± 4.1
90 min after α-MPT/ADX	6.7 ± 1.1[c,d]
150 min after α-MPT/--	20.0 ± 4.9
150 min after α-MPT/S-ADX	34.1 ± 1.8[c]
150 min after α-MPT/ADX	3.7 ± 0.3[c,d]

[a] Mean ± SEM (n = 6); [b] $p < 0.05$ for difference with resting value; [c] $p < 0.05$ for difference with unoperated rats decapitated at the same time (α-MPT/--); [d] $p < 0.05$ for difference between α-MPT/ADX (Student's t-test, two-tailed). Corticosterone levels in plasma were measured with a radioimmunoassay. The antiserum was raised against corticosterone 25-hemisuccinate (a gift from Dr Th. J. Benraad, Catholic University, Nijmegen, The Netherlands).

Discussion The present data show that, in contrast to the activity of the raphe-hippocampal serotonin system[3], the activity of coeruleo-hippocampal noradrenaline neurons is not modulated by adrenal corticosterone. Since it was found that dopamine utilization in the arcuate nucleus and median eminence of ADX rats is different from that of S-ADX rats, it is likely that dopaminergic activity in the medio-basal hypothalamus is subject to modulation by corticosterone. The only data on effects of acute treatment

with corticosterone are those of Fekete et al.[6]. Unfortunately, these authors measured effects on dopamine concentrations only, which they found to be unaltered in a number of hypothalamic nuclei including the arcuate nucleus and the median eminence. Furthermore, they carried out their study in intact rats. In intact rats corticosterone occupies its receptors in the brain to an extent more than 80%[4]. An involvement of brain corticosterone receptors, therefore, seems to be less likely in the experiments of Fekete et al.[6]. There is no evidence in favor of a role of brain dopamine in the regulation of ACTH secretion, though this catecholamine participates in the control of the release of other pituitary hormones[7]. Increases in the utilization of noradrenaline and adrenaline in the arcuate nucleus, median eminence, lateral septal nucleus, paraventricular nucleus and dorsomedial nucleus were found in ADX as well as in S-ADX rats. This indicates that these increases are due to the stress of the exposure to ether and the operation and not to differences in the amount of circulating corticosterone, which is low in ADX rats and high in S-ADX rats (see table 2). These increases were found in hypothalamic nuclei, in which changes in catecholamine concentrations have already been observed after various stresses[8–11], and are in accordance with the hypothesis that stress causes an activation of various catecholamine systems in the brain[8–16].

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Composition and novel pattern of emission of defensive scent oils in the larva of the cotton seed bug *Oxycarenus hyalinipennis* (Costa) (Heteroptera: Lygaeidae)[1]

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Summary. Differences in composition and pattern of emission in the scent oils from the two abdominal scent glands in the larva of *Oxycarenus hyalinipennis* are reported. The scent oils contain hex-2-enal, oct-2-enal, dec-2-enal and 4-oxo-oct-2-enal.

Defensive scent glands are widely present in Heteroptera[2,3]. Volatile fatty aldehydes, ketoaldehydes, esters and other biosynthetically related materials, sometimes together with isoprenoids, have been identified as components of their often complex secretions[4]. However, there has been as yet little investigation of the factors conditioning intraspecies and interspecies variation in scent oil composition. In this contribution to the problem we correlate differences in scent oil composition with differences in pattern of emission in the 2 abdominal scent glands in the larva of the cotton seed bug *Oxycarenus hyalinipennis* (Costa)[1].

Material and methods. The bugs[5,6] were maintained at 26 °C on dry cotton seeds and drinking water under a 14 L: 10 D photoperiod. Scent glands were excised from chilled larvae under 200 mM NaCl. Gas chromatography-mass spectrometry (GC-MS) was performed in both the electron impact (EI) and chemical ionization (CI) modes. For CI-GC-MS ammonia was used as reagent gas. The 7070H VG mass spectrometer was operated at 70 eV with the ion source at 190 °C, separator 180 °C and 200 μA ionizing current. Separations were achieved with a 2 m × 2 mm i.d. glass column packed with 3% OV 225 on 100–120 mesh Gas Chrom Q: 10 ml helium/min, column isothermal at 70 °C for 5 min and then temperature programmed to 200 °C at 10 °C/min. For routine gas chromatography (GC) a Varian 1440 instrument equipped with flame ionization detector was used. Injection of glandular samples was effected by a solventless open column procedure[7]. Dodecane supplied the external standard (1 μl dodecane in 1 ml of acetone).

Results. GC analysis of the secretions yielded 7 peaks of which 4 peaks (95% of the total) were identified as follows. Peak 2, hex-2-enal; base peak m/z 41, major ions at 55, 69 and 83, prominent M^+ 98 and M-1, matching the mass spectrum (MS) of authentic (E)-hex-2-enal (Aldrich). Peak 5, oct-2-enal; base peak m/z 41, major ions at 55, 70 and 83, matching the MS of authentic (E)-oct-2-enal (PPF International). Peak 6, dec-2-enal; base peak m/z 43, major ions at 55, 70 and 83, matching the MS of authentic (E)-dec-2-enal (PPF International). Peak 7, 4-oxo-oct-2-enal; base peak m/z 98, major ions at 55, 83 and 111, prominent M^+ 140, matching the MS published for (E)-4-oxo-oct-2-enal[8]. Molecular ions for oct-2-enal (M + 126)