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Histochemical localisation of monoamine oxidase A and B in rat brain

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Summary. The histochemical distribution of monoamine oxidase A and B in rat brain was investigated using a coupled peroxidatic technique with benzylamine and tyramine as substrates and clorgyline and (−)-deprenyl as selective inhibitors. Benzylamine oxidase was absent in all areas. Both forms of monoamine oxidase were present, at low levels, in all areas; in addition several regions showed high activity of one or other form or both. Substantial activity of monoamine oxidase B was identified in the pineal gland, the lining of the ventricles, several hypothalamic regions, and the raphe nuclei. The locus coeruleus and interpeduncular nucleus possessed considerable type A activity. The substantia nigra and striatum showed no staining above the low general level, although the ventral tegmental area showed higher levels of both A and B.

In general, noradrenaline-containing neuronal cell body areas showed monoamine oxidase A, and 5-hydroxytryptamine-rich areas monoamine oxidase B. There was no consistent enrichment of either in corresponding dopamine-rich regions. Monoamine oxidase thus appears to have a different role in these three types of neuron.

The low level of monoamine oxidase B in the nigrostriatal tract may help to explain the resistance of the rat to MPTP toxicity.

Keywords: Histochemistry, monoamine oxidase A and B, MPTP, rat brain, clorgyline, (−)-deprenyl.

Introduction

A knowledge of the precise localisation of the two forms of monoamine oxidase (MAO), termed A and B (Johnston, 1968), is relevant to our understanding of their functional role. MAO A is selectively inhibited by clorgyline and, in vitro, preferentially metabolises 5-hydroxytryptamine (5HT) and noradrenaline (Johnston, 1968). MAO B is selectively inhibited by (−)-deprenyl and its preferred substrates include phenylethylamine and benzylamine. The latter is also metabolised by a distinct enzyme, benzylamine oxidase, which is particularly

concentrated in the smooth muscle of blood vessels (Lewinsohn et al., 1980) and is insensitive to (−)-deprenyl (Lewinsohn et al., 1978). Recently, MAO B has also been shown to metabolise l-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) to a neurotoxin which can give rise to a Parkinson's disease model very similar to the idiopathic human illness (Jenner et al., 1984; Salach et al., 1984; Chiueh et al., 1985).

Early studies on the histochemical localisation of MAO in brain did not distinguish the two forms (Hashimoto et al., 1962). Later work concentrated on particular regions such as pineal (de Leo et al., 1983), ependyma (Williams et al., 1979) or hypothalamus (Williams et al., 1975; Kitahama et al., 1984). A comprehensive mapping of MAO in the rat brain using a coupled peroxidatic method has been described very recently (Arai et al., 1986). However, this study did not discriminate between MAO A and B. Immunohistochemical techniques, using monoclonal antibodies for the different forms, have also been developed. Levitt et al. (1982), using an antibody to the B form, mapped MAO B in the rat brain and found it to be present in 5HT-rich neuronal cell bodies; however, catecholamine-rich areas showed no detectable MAO B. Westlund et al. (1985) carried out a detailed mapping of primate brain with antibodies to both forms and identified MAO A within catecholamine cell bodies, both dopaminergic and noradrenergic. MAO B was localized within 5HT-containing cell bodies. Konradi et al. (1987), using immunocytochemistry with human brain, also found MAO B in the dorsal raphe neurones and MAO A in those of the locus coeruleus.

In this paper, we have employed a sensitive peroxidatic histochemical method (Ryder et al., 1979) using fresh unfixed tissue to map out in detail the distribution of both MAO A and B in rat brain.

Methods and materials

Materials

3-Amino-9-ethylcarbazole, horse-radish peroxidase Type II and tyramine hydrochloride were purchased from Sigma Chemical Company Ltd., Poole, Dorset. Dimethylformamide was obtained from BDH Chemicals Ltd., Dagenham, Essex. Benzylamine hydrochloride was prepared from the free base. Clorgyline and (−)-deprenyl were gifts respectively from May and Baker Ltd., Dagenham, Essex, and Professor J. Knoll, Budapest.

Methods

Adult male Wistar rats (180–200 g) were used. After decapitation, brains were rapidly removed and cooled on an ice-cold plate. Each was divided coronally or sagittally into 4 sections which were placed upon metal chucks and frozen on dry ice, to be stored at −20 °C.

Sections (30 μm) were prepared from frozen brain tissue using a cryostat. Sections were mounted on glass slides and left at room temperature for 30 min prior to incubation. The incubations used to demonstrate MAO A and B were adapted from the method of Ryder et al. (1979). The incubation medium was prepared by dissolving 3-amino-9-ethylcarbazole (20 mg) in dimethylformamide (5 ml), followed by addition of 0.05 M phosphate buffer, pH 7.6 (95 ml), mixing and filtering and finally adding peroxidase (25 mg) and substrate (tyramine as free base, or benzylamine), 120 mg. Slides were immersed in this medium,

modified for controls by the absence of substrate. All incubations were carried out at 37 °C for 3 hours in a covered water bath. Some sections were pre-incubated in phosphate buffer (0.05 M, pH 7.6) containing either 10^{-7} M clorgyline, 10^{-6} M (−)-deprenyl, or both, for 30 min at room temperature. Such concentrations provided optimal selective inhibition. After incubation in the carbazole-containing medium, sections were washed in 0.9% (w/v) NaCl solution, post-fixed for 2 hours in 10% formalin, and mounted in glycerol jelly. Results given are the combined findings from 4 rats, 3 of which were sectioned coronally and one sagitally. Localisation and staining in specific regions was determined by comparing carbazole stained sections with parallel sections stained with cresyl violet. Reference was made to the rat brain atlas of Paxinos and Watson (1982) for anatomical orientation. The evaluation of intensity of staining was by eye and graded according to intensity above general background, i.e., + + + + high intensity and + low intensity.

Results

With both benzylamine and tyramine as substrates, there was a low level of fairly evenly distributed staining throughout the brain, together with several discrete areas of intense activity. With no substrate, both types of staining were absent. The overall level of staining with benzylamine (low level plus "high spots") was considerably greater than that with tyramine. All benzylamine-related staining was abolished by 10^{-6} M (−)-deprenyl. All tyramine-related staining (low level plus "high spots") was abolished by preincubation with 10^{-6} M (−)-deprenyl plus 10^{-7} M clorgyline. Certain of the areas staining intensely with tyramine as substrate remained unstained after preincubation with (−)-deprenyl, others by preincubation with clorgyline. Low level tyramine-related staining was reduced to a greater extent by (−)-deprenyl than by clorgyline. All areas that stained with tyramine as substrate and were sensitive to (−)-deprenyl, were also stained with benzylamine as substrate. However, some of the areas that stained more faintly with benzylamine showed no staining with tyramine, e.g. raphe pallidus and obscuris. All areas that stained using benzylamine and tyramine as substrates and which were inhibited by (−)-deprenyl were designated MAO B. Tyramine staining areas inhibited by clorgyline were designated MAO A. Table 1 shows the distribution of the intensely staining areas of MAO A and B found in this study.

The most intensely staining areas were the pineal gland, the lining of the ventricles and the locus coeruleus. Figure 1 a and b shows sagittal sections stained for MAO A and B respectively, and illustrates the higher overall staining obtained with benzylamine (Fig. 1 b). This figure also illustrates some of the different levels of staining obtained in different regions. In Fig. 1 a, the locus coeruleus stained intensely while the habenular nucleus stained with low intensity. In Fig. 1 b, the lining of the ventricles and the pineal gland stained intensely, the dorsal raphe was intermediate, and the habenular nucleus with lower intensity. Figure 2 a shows the staining in the locus coeruleus and lining of the ventricles; Fig. 2 b shows the selective inhibition of locus coeruleus staining by clorgyline and Fig. 2 c that of the lining of the ventricles by (−)-deprenyl. The staining in the locus coeruleus at a higher magnification (Fig. 2 d) is also compared with

Table 1. Areas of intense monoamine oxidase staining in rat brain

Regions	MAO A	MAO B
Pineal gland	–	+ + + +
Ventricular lining	–	+ + + +
Locus coeruleus and subcoeruleus	+ + + +	
Interpeduncular nuclei		
Apical	+ +	+ +
Paramedial	+ + +	+ + +
Central	+ + +	+ + +
Inner posterior	+ +	+ +
Raphe nuclei		
Dorsal	–	+ + +
Median	–	+ +
Caudal	–	+ +
Pallidus	–	+ +
Obscuris	–	+ +
Habenular nuclei		
Median	+	+ +
Lateral	+	+ +
Hypothalamic nuclei		
Arcuate	+	+ + +
Median eminence	+	+ + +
Dorsomedial	+	+
Periventricular	–	+ +
Paraventricular	–	+ +
Lateral	–	+
Anterior	–	+ +
Ventromedial	–	+ +
Ventrolateral	–	+ +
Caudomagnocellular	–	+
Reticular nuclei		
Lateral	+ +	–
Nucleus of the solitary tract	+ +	–
Other		
Ventral tegmental region	+	+ +
Cerebral aqueduct (lining)	–	+ + +
Central canal (lining)	–	+ + +
Olfactory ventricle	–	+ +
Inferior olive	–	+ + +
Corpus callosum	–	+ + +
Paragigantocellular nucleus	+ +	+
Rhinal fissure	–	+ +
Hippocampus	–	+ +

Table 1 *(continued)*

Regions	MAO A	MAO B
Paraventricular thalamic nucleus	−	+
Pontine nucleus	−	+
Paranigral nucleus	+	+
Lateral mammillary nucleus	−	+
Median mammillary nucleus	−	+
Area postrema	−	+
Substantia nigra	−	−
Caudate/putamen	−	−

− No significant staining above an even (MAO A and MAO B) background
\+ Low intensity staining
\+ + Low to medium intensity staining
\+ + + Medium intensity staining
\+ + + + High intensity staining

a parallel cresyl violet section (Fig. 2e). Figure 3 shows staining in some of the different regions of the raphe, all of which stained exclusively for MAO B, the dorsal raphe being the most intense.

The intensely staining areas were not all of the same appearance. In some, the staining appeared as discrete spots but in others it was more diffuse. The areas with the most obvious spotting were the locus coeruleus, the subcoeruleus, the reticular nuclei, the nuclei of the hypothalamus (except the arcuate nucleus and the median eminence), and the dorsal, median and caudal raphe nuclei. The nature of the staining in the corpus callosum was neither in spots nor diffuse and resembled the pattern traced by blood vessels (Fig. 4).

Some areas stained for both MAO A and B, including interpeduncular nuclei, hypothalamus, habenular nuclei and ventral tegmental area. In the ventral tegmental area, staining for both A and B was evenly distributed whereas, within the hypothalamus, there were individual high points for each. No staining for either MAO A or B above background was found in the substantia nigra (Fig. 5) or the striatum.

Discussion

This study confirms that, whilst in general, both MAO A and B are fairly evenly distributed throughout the whole rat brain, there exist in addition small areas very rich in one or other, or both forms. The overall picture of MAO staining in rat brain that we have found in monoamine neurons is similar to that of Arai et al. (1986), who used a histochemical method with fixed sections. It also correlates well with the results of Rainbow et al. (1985), who localised ^{3}H-pargyline and ^{3}H-MPTP binding in rat brain. The pattern they obtained suggests

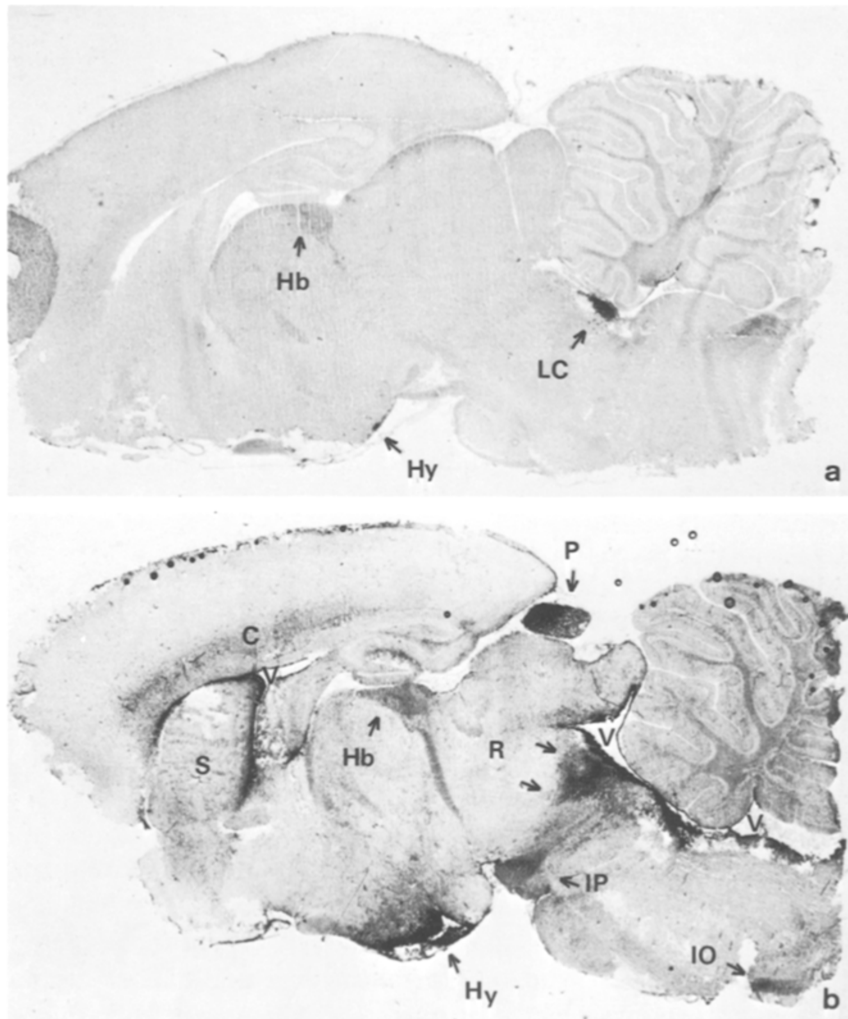


Fig. 1. MAO A and B staining in sagittal sections of rat brain (magnification × 7.5). **a** MAO A staining in sections stained with tyramine pretreated with (−)-deprenyl; *LC* locus coeruleus, *Hb* habenular nucleus, *Hy* hypothalamus. **b** MAO B staining in sections stained with benzylamine; *P* pineal gland, *V* ventricular lining, *IP* interpeduncular nucleus, *Hy* hypothalamus, *R* raphe nucleus, *C* corpus callosum, *IO* inferior olive, *Hb* habenular nucleus, *S* striatum

that both these ligands were binding to both forms of the enzyme. The present paper extends their findings, as we have discriminated between MAO A and B, and provides the first comprehensive mapping of both forms of the enzyme in rat brain.

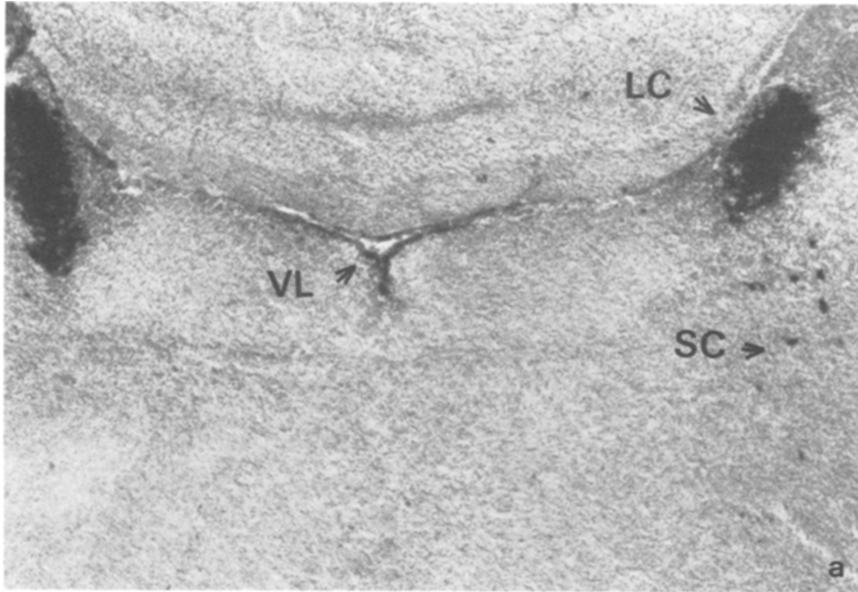
The histochemical method described here works very well for MAO B and demonstrates more localised areas of intense staining than histochemical or immunohistochemical methods have previously identified. It appears to be rather less sensitive for MAO A. Although the locus coeruleus stained as clearly

as any MAO B "high spot", there appeared to be fewer clorgyline-sensitive tyramine-related areas of intense enzyme activity and a lower level of uniform staining than would have been predicted from in vitro biochemical measurements which show a predominance of MAO A in rat brain (Waldmeier et al., 1976; Garrick et al., 1979). Most of the histochemical methods have used fixatives to treat the tissue to improve resolution. However fixatives have been shown by us (unpublished) and others (Barrand et al., 1984) to inhibit MAO activity severely. In our method, no fixatives were used. This approach gives higher activity, but less resolution. We are, therefore, more confident about negative results, particularly with respect to MAO B.

The different type of staining pattern from area to area was quite striking. The staining pattern characterized by spots seems likely to correspond with neuronal cell bodies. However, because of the thickness of the cryostat sections and possibly also some diffusion, the diameter of the spots was somewhat larger than that of neuronal cell bodies. The more diffuse staining seen elsewhere is presumably due to glia or nerve fibres. The staining in some structures resembling blood vessels (Fig. 4) is also notable. Benzylamine oxidase is rich in large blood vessels. However, the staining we observed in the corpus callosum was inhibited by 10^{-6} M (−)-deprenyl, unlike benzylamine oxidase. It was also not detected in the absence of tyramine or benzylamine and so is unlikely to be due to endogenous peroxidase activity.

It is of interest to try to relate the MAO A and B intense-staining pattern to the neurotransmitters found in particular regions. The major source of noradrenergic neurons in the rat brain is the locus coeruleus and sub-coeruleus (areas A4, A6; Dahlstrom and Fuxe, 1964), and these areas were found to stain densely for MAO A, with no intense staining for MAO B. Nakamura and Vincent (1986) made a similar observation, using 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) as substrate and showing that the staining in the locus coeruleus was clorgyline sensitive. Kishimoto et al. (1983), using a coupled peroxidatic histochemical method, localised both MAO A and B in guinea pig locus coeruleus. However, this difference could well be due to variation between species. MAO A but not MAO B was also found to be present in high activity in two further noradrenergic cell groups, areas A_1 and A_2, the lateral reticular nuclei and the nucleus of the solitary tract. No enrichment of either MAO A or B was found in other noradrenaline cell body-containing regions.

The major sites of 5HT-rich neurons in the brain are the raphe nuclei. Most such cell groups [B_1, B_2, and B_7 regions of Dahlstrom and Fuxe (1964)] stained intensely for MAO B, with no staining for MAO A greater than background level. Another 5HT-containing cell body region, B_8 (the interpeduncular nucleus), also stained for MAO B and, in addition, showed the presence of significant MAO A activity. The most intense areas of MAO B staining in the brain were the pineal gland and the lining of the ventricles, with no enrichment of MAO A staining in these regions. The pineal has a rapid turnover of 5HT, presumably as a precursor of melatonin, whilst the ventricular lining is rich in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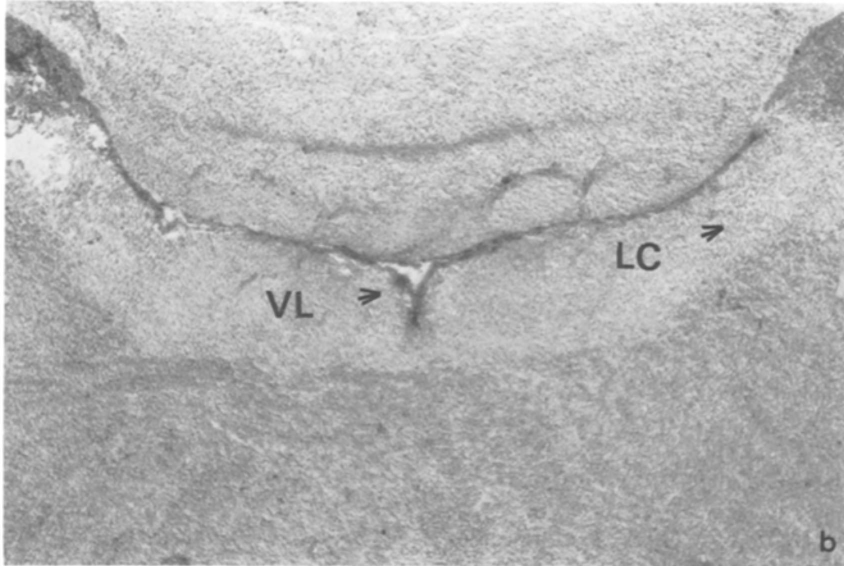
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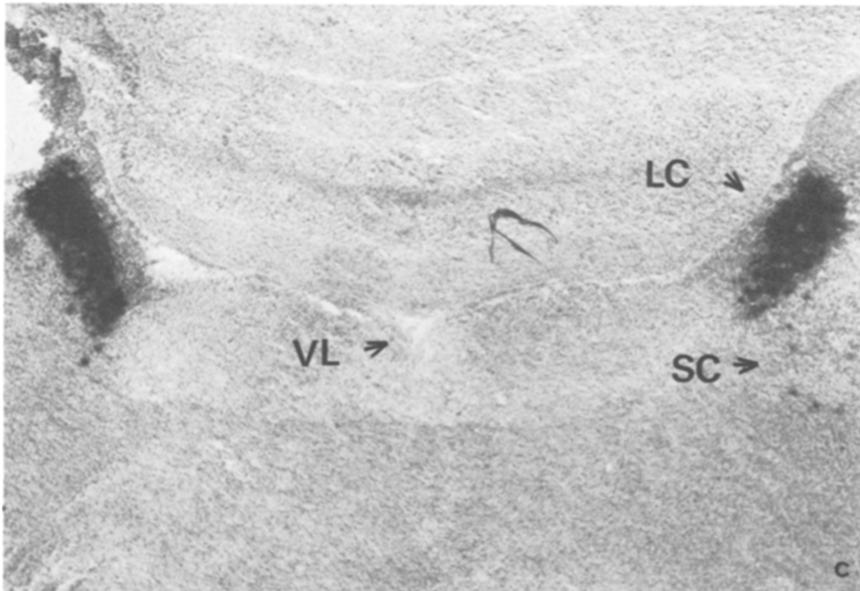
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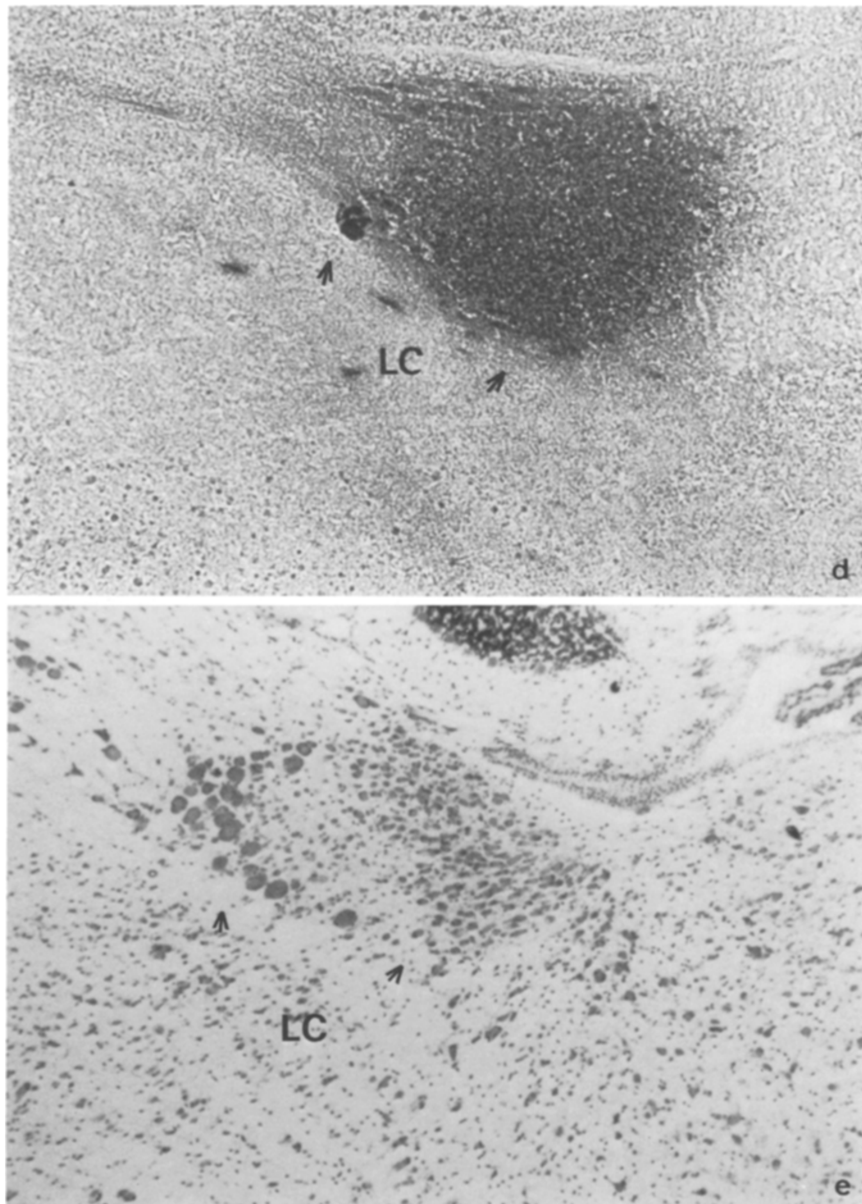

Fig. 2. MAO staining in the locus coeruleus. **a** The locus coeruleus (*LC*), subcoeruleus (*SC*), and ventricular lining (*VL*) stained with tyramine in a coronal section of rat brain. **b** and **c** Pretreated with clorgyline and (−)-deprenyl respectively (magnification ×47). **d** and **e** Higher magnification (×118) photomicrographs of sagittal sections of the brain showing the locus coeruleus. **d** A section stained with tyramine. **e** A similar section stained with cresyl violet

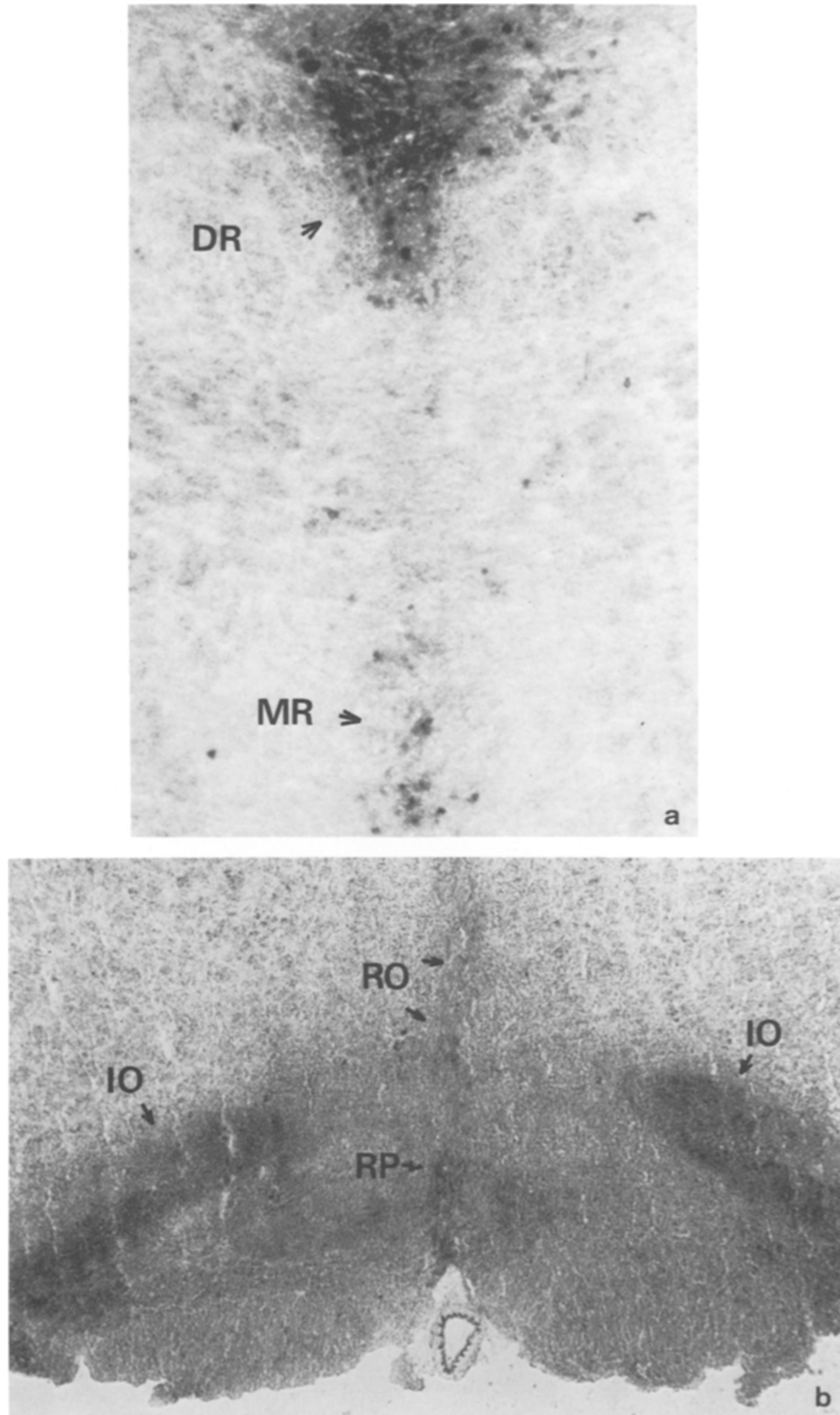

Fig. 3. MAO B staining in the raphe nucleus in coronal sections of rat brain stained with benzylamine (magnification ×47). **a** In the dorsal raphe nucleus (*DR*) and median raphe nucleus (*MR*). **b** The raphe obscuris nucleus (*RO*) and raphe pallidus nucleus (*RP*), shown staining between the inferior olive (*IO*)

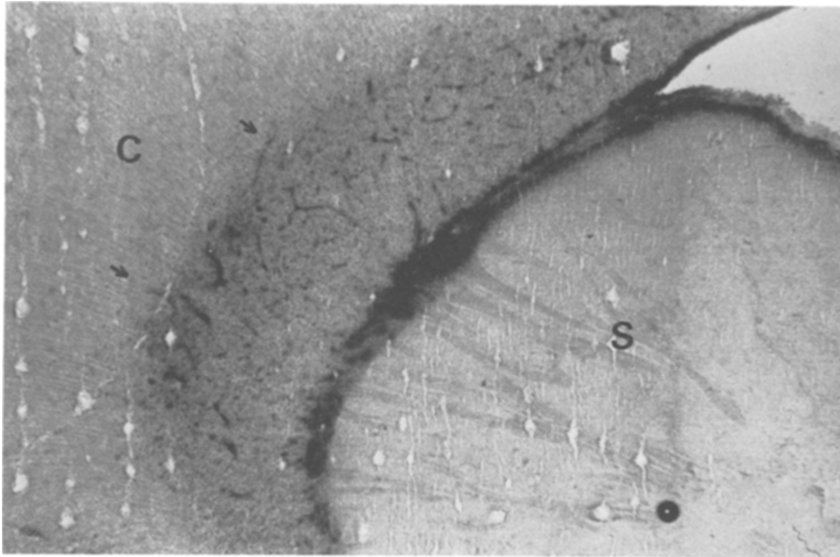

Fig. 4. MAO B staining in the corpus callosum in a sagittal section of rat brain stained with benzylamine (magnification ×47); *C* corpus callosum, *S* striatum

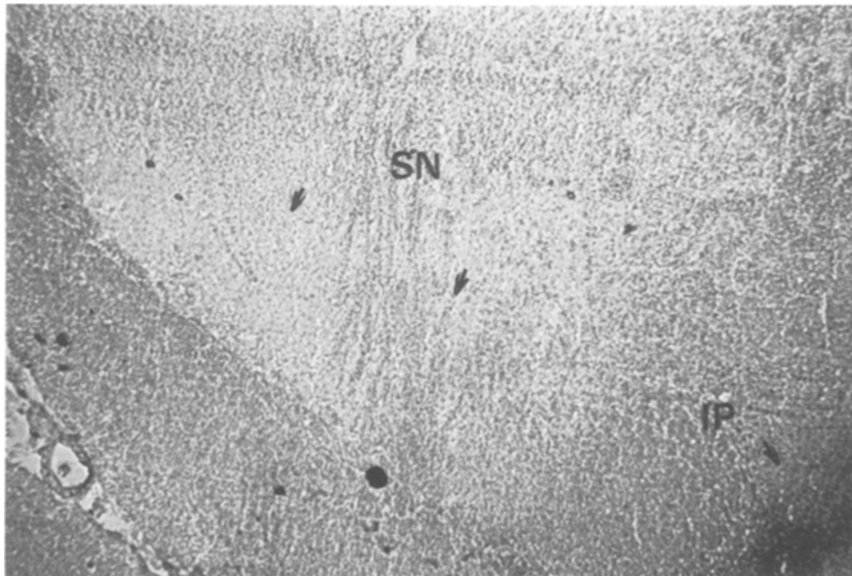

Fig. 5. Substantia nigra of rat brain in coronal section stained with benzylamine (magnification ×47). The photomicrograph shows an absence of significant MAO B staining in the substantia nigra (*SN*), in comparison with the interpeduncular nucleus (*IP*), stained in the bottom right-hand corner

5HT nerve-terminals. We and others have previously noted MAO B in the lining of the ventricles in the rat (Williams et al., 1975; Böhm et al., 1979). However, de Leo et al. (1983) and Uchida and Koelle (1984), using somewhat different staining methods, have also demonstrated the presence of significant amounts of MAO A in the pineal.

The important dopamine cell body regions in the brain showed no consistent pattern. There was no enrichment of either enzyme form in the substantia nigra or striatum, confirming the results of Arai et al. (1986). They questioned whether their result was an artefact due to fixatives inhibiting MAO; in our study this does not apply and suggests that this finding is real. Several hypothalamic regions containing dopamine, cell bodies A_{11}, A_{12}, and A_{14}, stained for MAO B, with the arcuate nucleus and median eminence staining most densely. Some of these regions also showed evidence of MAO A activity (see Table 1). There was some staining of both MAO A and B in the ventral tegmental area.

Several conclusions emerge from our findings: MAO A is located primarily in noradrenergic cell body regions, MAO B in 5HT-containing regions and dopamine cell body aggregations show no consistent pattern. The complete lack of enrichment of either MAO A and B in the nigrostriatal tract was striking. The finding of MAO B in the 5HT regions is similar to that Levitt et al. (1982) using rat brain and Westlund et al. (1985) using monkey brain. The finding of MAO A in some noradrenergic regions is also similar to the results of Westlund et al. (1985). If also present in human brain, it would help to explain why clorgyline administration has a more pronounced effect on cerebrospinal fluid concentration of the noradrenaline metabolite, 4-hydroxy-3-methoxyphenylglycol, than on that of the 5HT metabolite, 5-hydroxyindoleacetic acid (Garrick et al., 1984).

The segregation of MAO A and B in vivo between noradrenaline and 5HT rich neurons, respectively, contrasts with the properties of MAO A and B in vitro in the rat where MAO A selectively metabolises both substrates. Indeed, 5HT is commonly employed as the standard MAO A substrate as it has a lower Km and considerably higher V_{max} with this form than it does with MAO B (Fowler and Tipton, 1982). However, such in vivo localisation makes it tempting to view the human platelet, with its substantial presence of both 5HT and MAO B, as a closer model for 5HT neurons than had previously been realised. Why MAO B rather than MAO A should be present in these neurons is still unexplained. Perhaps its role is primarly protective, to metabolise extraneous amines, such as tyramine, which might otherwise bring about release of neurotransmitter.

Findings in the rat are not dissimilar from those in the primate with respect to MAO A and B distribution in noradrenergic and 5-hydroxytryptaminergic neurons. There is less agreement about the dopamine-rich regions of the different species. Westlund et al. (1985) found dopamine-containing neurons, including those in monkey substantia nigra, to contain MAO A whereas there is no enrichment of either MAO A or B in the rat substantia nigra (Fig. 5). However,

in a preliminary histochemical study of both marmoset and human brain, we have found that both substantia nigra and striatum stained intensely for MAO B, probably located in glia (Willoughby, Glover, Sandler, in preparation).

The finding of an absence of enrichment of MAO B in the nigrostriatal tract of the rat may be relevant to understanding the relative lack of neurotoxicity of MPTP in this species. However, the presence of a proximate source of MAO B in a dopaminergic region is not, by itself, sufficient for MPTP toxicity to be expressed, as MAO B is also present in the ventral tegmental region which is not affected by the toxin (Langston and Irwin, 1986).

Acknowledgements

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