Some Interrelated Properties of A and B Form Monoamine Oxidase in Monkey Brain Mitochondria

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Abstract—The multiplicity of monoamine oxidase (MAO) in monkey brain was studied by comparing the relationship between the selective substrates of MAO and the pH-activity curves obtained using these substrates. When mitochondrial and A-form MAO were used as the enzyme preparations with serotonin (5-HT) and norepinephrine (NE), preferential substrates for A-form MAO, the pH optima were 8.8 and 7.8 with 5-HT and 8.5 and 7.2 with NE. These substrates were also oxidized by B-form MAO after changing the pH of the incubation medium (shift to alkaline); these pH optima were 9.0 and 8.2, respectively. When common substrates of MAO were used (tyramine, octopamine, dopamine and tryptamine), the pH activity curves obtained were all broad and bell-shaped with pH optima for the 3 species of enzyme (mitochondria, A-form and B-form MAO) at 8.0, 7.8, and 8.0 with tyramine; 8.3, 7.5, and 8.5 with octopamine; 7.8, 7.5, and 8.5 with dopamine; and 8.0, 8.3, and 6.9 with tryptamine, respectively. The pH optima were 6.6 with β-phenylethylamine (β-PEA) and 9.0 with benzylamine, preferential substrates for B-form MAO, for either mitochondria or B-form MAO. The $K_m$ values obtained for tryptamine and β-PEA were lower than those for the other substrates of MAO, regardless of the enzyme preparations. The $K_m$ and $V_{max}$ values of both forms MAO for 5-HT and NE were similar to those of the A-form MAO. The differences in the $K_m$ and $V_{max}$ values of the A-form MAO and B-form MAO for common substrates were comparable. Tyramine, octopamine and dopamine were substrates for both forms MAO, with only a slight preference for B-form MAO over A-form MAO. However, tryptamine may be deaminated predominantly by A-form MAO.

Monoamine oxidase [monoamine: oxygen oxidoreductase (deaminating), EC 1.4.3.4], commonly designated MAO, is responsible for metabolizing many endogenous monoamines, including serotonin (5-HT), norepinephrine (NE), dopamine, octopamine, tyramine, tryptamine and β-phenylethylamine (β-PEA). A large number of xenobiotic compounds as well as endogenous monoamines are inhibitors of MAO; the elevation of mood by these compounds probably related to the accumulation of one or more of these amines.

Recently, potent and selective irreversible inhibitors of MAO have been used to detect two forms of the enzyme in preparations from several sources (1, 2). A-form MAO is very sensitive to clorgyline (3), but B-form MAO is only inhibited by a high concentration of clorgyline. In contrast, the drug (-)deprenyl (4) inhibits B-form MAO at a lower concentration than A-form MAO. 5-HT (3, 5) and NE (6) are preferential substrates for A-form MAO, and β-PEA (7) and benzylamine (8) are preferential substrates for B-form MAO. Dopamine (5), tyramine (7), tryptamine (1, 9) and octopamine (10, 11) have been reported to be substrates in vitro for both forms of the enzyme. Although several workers (12–16) have attempted the separation of A and B-form MAO, the nature of the two forms and the relationship between these forms are still obscure.
Our previous studies (17, 18) demonstrated the existence of two different functional forms of MAO (A-form and B-form MAO) in monkey brain, by specific drug inhibitors or solubilization and separation by electrofocusing. Accordingly, we have attempted to characterize further these multiple forms; in this study, we investigated the relationship between these multiple forms and speculated about the physiological roles they may play in vivo.

**Materials and Methods**

**Enzyme preparation:** Five adult monkeys (Macaca fuscata) were used. The monkeys were anesthetized with ketalar given s.c., and the brains were quickly removed and stored at -60°C until used. Homogenates (10%) were prepared in 0.32 M sucrose, pH 7.2. Pure monkey brain mitochondria were prepared by purifying crude mitochondrial fractions from monkey brain utilizing sucrose density gradient centrifugation (19). All operations were carried out at 0°-4°C.

**MAO activity:** MAO activity was measured with an ammonia selective electrode as described earlier (20). MAO activity was expressed as nmoles of NH₃ formed/min/mg of protein. Substrate concentrations used were 1 mM tyramine and dopamine; 200 nM 5-HT, NE, tryptamine and octopamine; and 100 nM β-PEA and benzylamine; these are expressed as final concentrations.

When MAO inhibitors were used, the enzymes were preincubated for 60 min at 37°C with 10⁻³ M to 10⁻¹⁰ M of inhibitor, clorgyline or (-)deprenyl, before addition of the substrate.

To determine the MAO activity at various pH values, the following buffers were used: McIlvain buffer (0.2 M Na₂HPO₄ and 0.1 M citric acid, pH 5.0–pH 6.0); phosphate buffer (0.1 M KH₂PO₄ and 0.1 M Na₂HPO₄, pH 6.0–pH 7.5); tris-HCl buffer (0.02 M tris-hydroxymethyl aminomethane and 0.1 M HCl, pH 7.5–pH 9.0); and borate buffer (0.2 M boric acid and 0.1 M NaOH, pH 9.0–pH 10.5).

**Mitochondrial treatments with clorgyline and (-)deprenyl:** Mitochondrial preparations were preincubated with either clorgyline (0.1 μM) or (-)deprenyl (1 μM) in a total volume of 1.9 ml at 37°C for 2 hr (19, 21), and then MAO activities of samples of the mixtures were estimated. Estimated MAO activity in preparations treated with clorgyline had 90% of the control activity toward β-PEA and no activity toward 5-HT. Conversely, preparations treated with (-)deprenyl had 2% and 95% of the control activities toward β-PEA and 5-HT, respectively. Thus, A-form and B-form MAO were irreversibly inhibited by clorgyline and deprenyl (21), respectively; and in subsequent experiments, preparations treated with (-)deprenyl or clorgyline were used as preparations of A-form or B-form MAO, respectively.

**Estimation of protein concentration:** Protein content was estimated by the method of Lowry et al. (22) with bovine serum albumin as the standard; protein concentrations of the enzyme preparations were adjusted to 2.0 mg/ml.

**Results**

The effects of increasing concentrations of clorgyline and deprenyl on the MAO activity in monkey brain mitochondria were investigated using 8 different substrates. The MAO activity with 5-HT was strongly inhibited by clorgyline, but was less inhibited by (-)deprenyl (Fig. 1, top). MAO activity toward NE decreased proportionally to the concentration of deprenyl and was inhibited completely at a concentration of 1 X 10⁻⁴ M (-)deprenyl. However, clorgyline at a concentration of 1 X 10⁻⁴ M inhibited MAO activity about 60% with NE as substrate (Fig. 1, bottom).

When benzylamine and β-PEA were used as the substrate, high sensitivity of MAO activity to inhibition by (-)deprenyl was observed, and the inhibition curves were also sigmoidal at a low concentration of (-)deprenyl. However, clorgyline at a concentration of 1 X 10⁻⁴ M inhibited MAO activity about 60% with NE as substrate (Fig. 1, bottom).

When benzylamine and β-PEA were used as the substrate, high sensitivity of MAO activity to inhibition by (-)deprenyl was observed, and the inhibition curves were also sigmoidal at a low concentration of (-)deprenyl. However, the MAO activities toward these two substrates were only inhibited by a high concentration of clorgyline (Fig. 2).

The activities with tyramine, dopamine, tryptamine and octopamine gave plateau-shaped inhibition curves by clorgyline and (-)deprenyl, although the plateau inhibition curves varied widely among substrates used,
indicating that these substrates were oxidized by both A and B-forms of the enzyme. Thus both forms of MAO may be present in monkey brain mitochondria (Fig. 3).

The rates of oxidation of 8 substrates at various pH values were investigated using various buffers (see Materials and Methods). The curves obtained with monkey brain mitochondria were all bell-shaped, but the pH optima were remarkably different: pH 6.6 with 3-PEA, pH 7.8 with dopamine, pH 8.0 with tyramine and tryptamine, pH 8.3 with octopamine, pH 8.5 with NE, pH 8.8 with 5-HT and pH 9.0 with benzylamine (Fig. 4).

Using the A-form or B-form MAO preparations (see Materials and Methods), pH-activity curves were investigated. When A-form MAO was used as the enzyme preparation, typical bell-shaped curves were obtained with all substrates except NE. In the case of NE, MAO activity was very low and the pH-activity curve was broad. The pH optima were remarkably similar, that is, pH 7.2 with NE, pH 7.5 with octopamine and dopamine, pH 7.8 with 5-HT and tyramine and pH 8.3 with tryptamine (Fig. 5).

As can be seen in Fig. 6, markedly different pH-activity curves were obtained when B-form MAO was used as the enzyme preparation. Moreover, it was difficult to determine the optimum pH toward the respective substrates. Apparent pH optima were pH 6.6 with 3-PEA, pH 6.9 with tryptamine, pH 8.0 with tyramine, pH 8.2 with NE, pH 8.5 with octopamine and dopamine and pH 9.0 with 5-HT and benzylamine.

The $K_m$ and $V_{max}$ values of various substrates for the untreated mitochondria (both forms), the (−)-deprenyl-treated (A-form) and clorgyliline-treated (B-form)
Fig. 3. Inhibition of mitochondrial MAO activity in monkey brain by clorgyline and (-)depreriy toward tyramine, dopamine, octopamine and tryptamine. Experimental conditions and symbols are as for Fig. 1. The control values for MAO activity were 5.46±0.42 for 1 mM tyramine (A), 3.95±0.30 for 1 mM dopamine (B), 1.95±0.03 for 200 μM octopamine (C) and 2.13±0.05 nmol/min/mg of protein for 200 μM tryptamine (D). Each point represents the mean value obtained from triplicate experiments.

Fig. 4. pH-activity curves of monkey brain mitochondrial MAO. pH-activity curves were obtained from the rates of oxidation of 8 substrates at various pH values. McIlvain buffer (pH 5.0 – pH 6.0), phosphate buffer (pH 6.0 – pH 7.5), tris-HCl buffer (pH 7.5 – pH 9.0), borate buffer (pH 9.0 – pH 10.5), tyramine (○○○), dopamine (●●●), octopamine (□□□), tryptamine (×××), β-PEA (■■■), 5-HT (▲▲▲), benzylamine (△△△), norepinephrine (○○○). The results are means of triplicate assays.

Fig. 5. pH-activity curves of A-form MAO toward A and common substrates of MAO. Experimental conditions and symbols are as for Fig. 4. Monkey brain (-)deprenyl-treated mitochondria (A-form MAO) were used as the enzyme source (see Materials and Methods). The results are means of triplicate assays.
mitochondria were calculated from Lineeweaver-Burk plots at pH 7.2. MAO in monkey brain showed lower $K_m$ values for tryptamine and $\beta$-PEA than for the other substrates. The $K_m$ and $V_{max}$ values of both forms of MAO for 5-HT and NE were similar to those of the A-form MAO. However, when B-form MAO was used as the enzyme preparation, lower $K_m$ values for NE and 5-HT were observed at pH 8.0 and at pH 9.0, respectively, in comparison with those of A-form MAO.

The differences in the $K_m$ and $V_{max}$ values of the A-form MAO and B-form MAO for substrates of both forms of MAO were comparable. The $K_m$ values of the B-form MAO for substrates of both forms of MAO were smaller than those of A-form MAO, and the $V_{max}$ values were greater than the corresponding values of A-form MAO, except with tryptamine as the substrate (Table 1).

### Discussion

Previous studies have indicated that NE and 5-HT are generally selective substrates for A-form MAO on the basis of the sensitivity to inhibition by low concentrations of clorgyline (6) and the localization of A-form MAO in tissues containing NE (6, 23, 24). However, the investigation by White et al. (25, 26), on the mixed substrate competition and effects of inhibitor on human brain MAO indicated that $\beta$-norepinephrine acts as a common substrate. Garrick (27) also reported that $\beta$-norepinephrine should be regarded as a substrate for both forms of MAO in human and brains of other primates. In this experiment, MAO activity in monkey brain toward NE was not inhibited completely by high concentrations of clorgyline, although MAO activity with 5-HT was strongly inhibited by low concentrations of clorgyline (see Fig. 1).
This finding agrees with the reports by White et al. (25, 26) and Garrick (27) and also suggests that NE is a substrate for both forms of MAO in monkey brain. Moreover, the inhibition pattern with low concentrations of clorgyline and the kinetic constants revealed that 5-HT was deaminated predominantly by A-form MAO in monkey brain, although there have been reports of 5-HT oxidation by B-form MAO (28–30). In the present study, it is possible that 5-HT and NE also are deaminated by B-form MAO with changing the pH range from 7.2 to 8.8. These results indicate that although 5-HT and NE generally are oxidized by A-form MAO, 5-HT or NE oxidation also is carried out by B-form MAO in mitochondria of monkey brain when some changes of physiological conditions occurred such as selective inhibition of A-form MAO, release of a high concentration of 5-HT or change of pH.

Dopamine (5), tyramine (7), octopamine (10, 11) and tryptamine (1, 9) are known as substrates for both forms of the enzyme based on inhibitor sensitivity and substrate specificity. It is of interest to correlate the relationship between the localization of these amines and that of MAO. MAO plays a major role in the catabolism of these amines in central and peripheral nerve tissues, and in spite of relatively low endogenous concentrations, it regulates concentrations of these amines in animal tissues (31–37). Recently, Demarest and Moore (38) suggested that the neuronal pool of dopamine is deaminated only by A-form MAO in dopamine neurons. However, the present studies showed, based on the results obtained from the inhibition pattern seen with clorgyline or (−)-deprenyl and the kinetic data, that tyramine, dopamine and octopamine are substrates for both forms of MAO with only a slight preference for B-form MAO over A-form MAO. However, tryptamine may be deaminated predominantly by A-form MAO. As indicated in Table 1, the $K_m$ value for tryptamine by B-form MAO was 2-fold greater than that of the A-form. The $V_{max}$ value for tryptamine by B-form MAO was about 2-fold smaller than the corresponding value by A-form MAO in monkey brain. This finding agrees with our previous reports for preparations from rat (19) and mouse (39) brain.

When β-PEA and benzylamine were used as substrate, pH-activity curves were markedly different and pH optima were 6.6 and 9.0, although they were both preferential substrates for B-form MAO. It is generally known that β-PEA oxidation is inhibited by high concentration of β-PEA. Suzuki et al. (40, 41) and Kinemuchi et al. (42) reported that the inhibition pattern by clorgyline or (−)-deprenyl change markedly at different concentrations of β-PEA and at different pH values and suggested that these changes are due to the strong substrate inhibition. These observations agree with this report which investigated monkey brain mitochondria using reaction media at pH 6.6 (optimum pH) and pH 7.2 for kinetic analysis. As shown in Figs. 4 and 6, the curves obtained from the rates of oxidation of β-PEA at various pH values were bell-shaped, and the pH optimum was 6.6. The activity was decreased with increasing the pH range from 6.6 to 8.0. Since the active substrate of MAO is the unprotonated amine and the pK value of β-PEA is 9.5, the concentration of the active substrate may increase proportionally while the reaction medium is adjusted to the alkaline side. Thus, the substrate inhibition of MAO probably would occur.

When A-form MAO was used as the enzyme source, pH-activity curves were typically bell-shaped and optimum pH values for substrates of A and both forms of MAO were similar and exhibited a limited range of pH. The activity of A-form MAO may be more dependent on the conditions of the reaction medium. These results indicate that the activity of A-form MAO is affected easily and may be inactivated under some conditions, that is, by effects of pH (43), various anions and heat treatment (44). On the other hand, B-form MAO showed broad pH activity curves and high activity at wide pH ranges. However, optimum pH values for tryptamine and β-PEA were acidic and for 5-HT and NE, highly alkaline. These findings indicate that with B-form MAO, it should be possible to deaminate every substrate in any condition. Moreover, on the basis of mixed substrate experiments (19, 25, 45) and
studies on the effect of pH (this report), we suggest that there is also more than one catalytic site for the deamination of monoamines by monkey brain MAO.

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