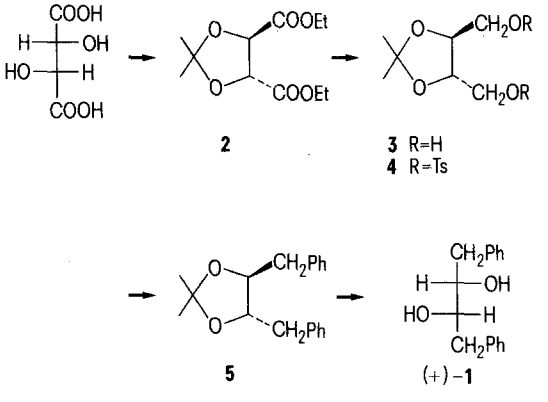
extracts were washed with saturated brine solution, dried, and concentrated. The yellow oily residue was chromatographed on 20 g of silica gel, eluting first with hexane to remove biphenyl, then with hexane-ethyl acetate (3:1) to elute the acetonide. Distillation at 140 °C (0.1 mm) yielded 650 mg (47%) of colorless product. NMR ($CDCl_3$) δ1.4 (s, 6H, CH_3), 2.8 (m, 4H, CH_2), 4.0 (m, 2H, O-CH), 7.25 (s, 10H, aromatic). IR (neat) 3080, 3060, 3010, 2940, 2880, 1620, 1500, 1460, 1380, 1370, 1240, 1215, 1160, 1075, 1050, 750, 695 cm^{-1}.

1,4-Diphenyl-2,3-butanediol (**1**). The acetonide **5** (250 mg) was mixed with 4 ml of 1 N HCl and just enough ethanol to make the mixture homogeneous, then stirred at room temperature overnight. The solid which had filled the flask was filtered and recrystallized from acetone-petroleum ether to give 138.9 mg (81%) of colorless diol, m.p. 146–147 °C (lit.[2] m.p. 146–147 °C); $[\alpha]^{23}D+4.7\pm0.5°$ ($CHCl_3$, c 11); lit. $[\alpha]^{26}D+4.6\pm0.9°$ ($CHCl_3$, c 1.10). IR 3500–3150, 3030, 2960, 2910, 1450 cm^{-1}; NMR ($CDCl_3$) δ2.9 (m, 4H, CH_2), 3.72 (m, 2H, O-CH), 7.3 (s, 10H, aromatic).

Analysis. Calculated for $C_{16}H_{18}O_2$: C, 79.31; H, 7.49. Found: C, 79.09; H, 7.52.



Synthesis of (+)-1,4-diphenyl-2,3-butanediol from L-(+)-tartaric acid.

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The activity of monoamine oxidases A and B in gamma-irradiated rabbit brains[1]

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Summary. The activities of monoamine oxidases A and B towards 5-hydroxytryptamine and β-phenethylamine, respectively, were compared in the left and right caudatus, hippocampus, parietal cortex, cerebellum and frontal cortex 6 months after gamma-irradiation (single dose of 23 Gy) of either the right hemisphere or of the whole rabbit brain (in which case, a dose of 16 Gy). No difference in monoamine oxidase A or B activities were found in any of the brain regions.

In the rabbit brain, the activity of monoamine oxidase (MAO, monoamine O_2:oxidoreductase, EC 1.4.3.4) appears to exist as 2 forms, termed MAO-A and MAO-B, where the A form of the enzyme is responsible for the deamination of 5-hydroxytryptamine (5-HT), and is inhibited by low concentrations of clorgyline, and the B form, responsible for the deamination of β-phenethylamine (PEA), is inhibited by low concentrations of deprenil[2–5]. Although the exact functions of the 2 forms of MAO are not as yet known, the activity of MAO-A appears to be confined, in the main, to the neuronal tissue of the brain, whereas the activity of MAO-B is more non-neuronal in nature[6–8].

In 1973, Pausescu et al.[9] reported that exposure of rabbits to a low dose of gamma-irradiation (4 Gy) produced an increased activity of brain monoamine oxidase. A similar result was found for rat brain MAO after gamma-irradiation[10], whereas neutron-irradiation produced a decrease in the activity of MAO. In contrast, however, a single dose of 100 Gy gamma-irradiation was found to be without effect on the activity of rat brain MAO-A activity, the rats being sacrificed 24 h after the irradiation[11], which may suggest that the increases found by other authors might have been due to the effects of stress rather than the effect of gamma-irradiation, since stress is known to produce short-term increases in MAO activity[12–14].

Of long-term effects of irradiation upon MAO, it has been reported that the catalytic properties of MAO are 'transformed' to properties resembling those of diamine oxidase upon radiation injury (7 Gy) of experimental animals[15,16], due to a build-up in the concentration of lipid-peroxides[15–17]. In consequence, it was felt to be of importance to see whether long-term changes in the activity of MAO-A and -B of rabbit brain could be brought about by a single dose of gamma-irradiation.

Materials and methods. Rabbits (2.0 ± 0.1 kg) were irradiated over either the right hemisphere alone (radiation dose 23 Gy) or over both hemispheres (radiation dose 16 Gy).

The irradiation was performed with the rabbits under mebumal anesthesia, using ^{60}Co gamma-radiation from a linear accelerator (Climac 4). The radiation dose was calculated 3 mm benath the skin at the midline of the skull, using a Siemens Sondenfingerhut chamber dosimeter. Mebumal anesthetized non-irradiated rabbits were used as controls.

6 months after irradiation, the rabbits were killed by decapitation and the brain regions caudatus, hippocampus, parietal cortex, cerebellum and frontal cortex dissected from the left and right sides. The brain regions were frozen at −80 °C immediately after dissection. The specimens were homogenized 1:20 (w/v) in 0.5 M potassium phosphate buffer, pH 7.4, and assayed for monoamine oxidase activity by the method of Wurtman and Axelrod[18], with 50 µM 5-HT as substrate for MAO-A and 12.83 µM PEA as substrate for MAO-B. Activities were calculated as pmoles (of substrate metabolized) · (mg wet weight)$^{-1}$ · min^{-1}.

The remaining portions of the brains were used for histopathology. After fixation, slices were stained with hematoxylin-eosin, van Gieson, Nissel stain, and with McMahon's myelin stain.

Results and discussion. The rabbits were studied in the 6-month period between irradiation and sacrifice. Of the rabbits irradiated on the right side of the brain, 2 of the 6 animals showed right rotational behaviour. A further 2 animals displayed a drooped right ear together with drooping of the right side of the head. These 2 animals also showed classical symptoms of radiation injury: hair loss, keratitis and conjunctivitis. Among the 6 rabbits with whole-brain irradiation, 1 rabbit had the head turned constantly to the right. No neurological or other symptoms were observed for the 6 control rabbits. The mean weight gain of the rabbits 6 months after the irradiation were: control, 2 kg; right hemisphere irradiated: 1.25 kg; both hemispheres irradiated: 1.55 kg.

Macroscopically, in 5 of the 6 one-side-irradiated brains petechial hemorrhages and adherences where found, mainly in the walls of the lateral ventricle and in the hippocampus on the irradiated side. In 1 of the animals the hippocampus was totally necrotic. Microscopically, no morphologic abnormality was observed in the non-irradiated part of the brain. Parts of the irradiated regions showed, however, necroses and perivascular inflammation and sometimes dilated vessels with thrombosis together with cystic degeneration of the surrounding brain tissue. In the animals where the whole brains were irradiated the changes were less marked and bilaterally distributed. The brains from the control rabbits showed no morphological abnormalities. The macroscopical and light microscopical findings were consistent with the presence of significant radiation damage, particularly for the rabbits irradiated on the right side of the brain.

The activities of brain MAO-A and -B 6 months after irradiation are shown in tables 1 and 2. There was no difference between the activities of either MAO-A or -B for either the left or right sides between the controls, rabbits irradiated on the right side alone, or for rabbits irradiated on both sides. Thus, in the rabbit, there was no evidence to suggest a 'transformation' of MAO activity, since this would have appeared as a decreased MAO activity with 5-HT and PEA as substrates in the irradiated animals. The data in tables 1 and 2 would also suggest that the short-term effects found in other laboratories[9,10] are temporary in nature, and might possibly be due to stress, rather than to the irradiation procedure.

Table 1. The effect of gamma-irradiation on the activity of rabbit brain monoamine oxidase A

	Monoamine oxidase activity (pmoles · mg wet weight^{-1} · min^{-1})					
	Control rabbits		Right-hemisphere-irradiated rabbits		Both-hemisphere-irradiated rabbits	
	Left	Right	Left	Right	Left	Right
Caudatus	24.5±1.2	26.5±1.5	25.8±1.0	25.1±1.1	25.4±1.0	22.8±1.2
Hippocampus	33.8±2.0	37.1±2.7	34.3±2.0	30.0±1.4*	32.4±2.0	32.8±1.2
Parietal cortex	29.0±0.5	28.9±0.8	27.0±1.3	29.7±1.5	28.7±1.1	28.2±1.5
Cerebellum	17.3±0.5	17.3±0.4	17.5±0.6	17.0±0.5	17.2±0.3	17.3±0.7
Frontal cortex	21.2±1.1	22.2±1.1	19.8±0.9	21.0±1.3	19.8±0.7	19.9±0.8
Thalamus	24.4±0.4	22.9±1.0	24.4±1.2	24.8±1.9	24.7±0.7	25.8±1.3

Values represent the means±SEM of duplicate determinations of activity in the 6 rabbits comprising each group. MAO-A activity was assayed with 50 µM 5-HT as substrate. Significantly lower MAO activity was found in the right hippocampus when the right hemisphere was irradiated compared with the control right, but not the paired left hippocampus, * $p < 0.05$; two-tailed t-test. In no other case were the MAO activities for the irradiated brains significantly different from the control values.

Table 2. The effect of gamma-irradiation on the activity of rabbit brain monoamine oxidase B

	Monoamine oxidase activity (pmoles · mg wet weight^{-1} · min^{-1})					
	Control rabbits		Right-hemisphere-irradiated rabbits		Both-hemisphere-irradiated rabbits	
	Left	Right	Left	Right	Left	Right
Caudatus	59.4±5.1	56.9±5.4	67.5±3.9	61.6±2.7	65.4±3.5	56.6±3.3
Hippocampus	81.2±6.0	95.2±4.2	84.0±4.7	93.5±7.6	87.0±5.1	99.2±3.7
Parietal Cortex	50.9±1.8	54.8±4.3	54.1±2.1	55.9±3.0	55.3±3.3	57.4±3.2
Cerebellum	18.6±2.5	26.4±5.1	24.1±1.8	24.3±1.9	21.7±1.4	34.8±4.1
Frontal Cortex	77.8±5.3	74.1±6.8	70.7±4.3	68.4±4.4	68.2±2.3	85.2±4.5
Thalamus	52.0±4.3	57.2±9.8	60.5±4.1	57.3±2.2	56.0±3.4	51.9±5.9

Values represent the means±SEM of duplicate determinations of activity in the 6 rabbits comprising each group. MAO-B activity was assayed with 12.83 µM PEA as substrate. In no cases were the MAO activities for the irradiated brains significantly different from the control values (two-tailed t-test).

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Comparative studies of murine anionic and cationic arylsulfatase B[1]

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Summary. Murine brain possesses an anionic form of arylsulfatase B which accounts for approximately 12–16% of non-microsomal arylsulfatase activity. This isozyme is antigenically similar to cationic arylsulfatase B, displays a similar developmental profile, and can be converted to a form resembling the cationic species by prior treatment with neuraminidase.

Arylsulfatases (arylsulfate sulfohydrolase, EC 3.1.6.1) occur in mammalian tissues in several forms which differ with respect to their intracellular distribution, substrate affinities, and physical and chemical properties[2]. Lysosomal arylsulfatase B appears to participate in the stepwise degradation of sulfated mucopolysaccharides by acting as an exosulfatase upon N-acetylgalactosamine-4-sulfate residues of chondroitin and dermatan sulfates[3]. Arylsulfatase B is deficient in the human mucopolysaccharidosis, Maroteaux-Lamy syndrome[4].

Multiple forms of arylsulfatase B have been detected in bovine, rabbit, and human tissues[5–7]. Stevens et al.[8] resolved human brain arylsulfatase B into major (cationic) and minor (anionic) species using DEAE-cellulose chromatography. Both species of enzyme exhibited similar substrate specificities and pH optima and were not inhibited by silver ion, an inhibitor of arylsulfatase A; however, the isozymes differed with respect to their apparent isoelectric points, electrophoretic mobilities, and thermolability.

We have detected both cationic and anionic forms of arylsulfatase B in murine tissue. This report describes the tissue distribution, physical and chemical properties, and developmental and immunological relationships of these isozymes.

Methods. Inbred mice were obtained from the Jackson Laboratory, Bar Harbor, Maine, and raised to 45 days of age unless otherwise indicated. Arylsulfatase A and B activities were assayed using p-nitrocatechol-SO_4 as substrate[9].

Isolation of anionic arylsulfatase B. 50% w/v aqueous homogenates of C57BL/6J brain or liver were sonicated, centrifuged at 100,000× g for 60 min, and dialyzed overnight against 0.02 M Tris-acetate buffer, pH 7.4 (start buffer). 1 ml of retentate was applied to a 10×1.5 cm column of DEAE-cellulose preequilibrated with start buffer. Approximately 20 ml of start buffer were run through the column (flow rate: 12–13 ml/h) followed by 40 ml of a linear buffer-chloride gradient formed from 20 ml start buffer and 20 ml of 0.2 M Tris-acetate buffer, pH 7.4, containing 0.4 M NaCl, and 1-ml fractions were collected.

Electrofocusing of arylsulfatase isozymes. Arylsulfatase isozymes from the DEAE-cellulose peaks were electrofocused in narrow gradient (pH 5–8) ampholine-acrylamide slab gels[10]. The isoelectric points of the enzyme bands were estimated by pH measurements of extracts from parallel slices from the same gel. Enzyme bands were visualized by immersion in 10 mM p-nitrocatechol-SO_4 in acetate buffer, pH 5.9, at 37 °C for 30 min and in 1 N NaOH for 5 min.

Characterization of anionic arylsulfatase B. The Michaelis constants and pH optima of anionic and cationic arylsulfatase B and arylsulfatase A were determined by standard methods using p-nitrocatechol-SO_4 as substrate. The respective enzymes were preincubated at 37 °C for 15 min in the presence of 200 µM $AgNO_3$ prior to enzyme assay to measure silver inhibition. Immunological characterization of the 3 arylsulfatases was accomplished by mixing 400 µl of appropriately diluted DEAE-cellulose fraction with increasing quantities of rabbit anti-mouse kidney arylsulfatase B IgG, and the volume of the mixture was adjusted to 600 µl with 0.05 M imidazole-HCl buffer, pH 6.6, containing 0.1 M NaCl. The mixtures were incubated at 37 °C for 1 h and at 4 °C for 16 h, and the immunoprecipitate was pelleted at 6000× g. The effect of neuraminidase upon anionic arylsulfatase B was determined by the method of Farooqui and Srivastava[11]. The 100,000× g supernatant from the sonicated brain extract was incubated with type VI