synaptic events, and, therefore, somehow constitute one of the physiological parameters of bioelectrogenesis in excitable tissues.

If the two (cytosolic and membrane) stores of the enzyme are exchangeable, a view not too far-fetched (see discussion in [2]), a rate of the replenishment of them could exist in the excitable cells and be paced in a scheduled fashion. Should this prove to be true, one would like to know whether the ratio of bound to free AChE could reflect the dynamic of interconversion in excitable cells of the two native states of the enzyme.

One may further wonder whether during nerve activity such an interconversion takes place; the phenomenon could be strictly regulated and contribute to some features of the biopotentials, such as the differences noted in nicotinic as compared to muscarinic conduction and transmission. In the pace of such interconversion, the second cholinesterasic ratio (R_2) could be involved. Hypotheses concerning a possible role of pseudocholinesterases, viz. BChE, in nerve activity have been successively advanced and rejected, and it should be recognized that the function of this enzyme remains unknown. We shall try to test the above assumption.

The third ratio (R_3) was found to have different values ($p<0.001$) in the muscarinic tissues studied, but no significant differences between brain and muscle ratios were observed (Table II). Remembering that all these muscarinic tissues differ in their cholinergic innervation[8], parasympathetic of inhibitory (heart) and excitatory type (intestine) or sympathetic of excitatory type (uterus), such differences could, if anything, be expected, but the lack of significance between the values of brain and muscle is surprizing.

The observation that a significant (bound to free) AChE ratio also occurs in excitable tissues of the chick, and that it seems to be attained at a specific period of development (VARELA and MANDEL, unpublished) renders the foregoing views worthy of consideration.

Lastly, the enzymic values found in nerve reminds one of the debate about the participation of AChE in conduction besides its role in synaptic transmission[9].

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Genotype Dependence of Monoamine Oxidase in Inbred Strains of Mice[1]

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Summary. MAO activity was found to be influenced by the genotype or strain of mouse up to 20 days of age. The strain differences observed may derive from different rates of brain development. A number of neurological mutations comprizing three pathological classes had no effect on MAO.

Monoamine oxidase (E.C. 1.4. 3.4.; MAO) is a flavoprotein-linked deaminating enzyme of biogenic amines[2–4]; it is present in mitochondria of mammalian liver, kidneys, intestines and brain. In the brain it occurs in both glial cells and synaptosomes[5,6]. Apparently, multiple forms of MAO exist in the brain although there is still controversy as to the existence of true isozymes or binding of a single enzyme protein to different combinations of phospholipids[7–10]. The probable existence of multiple enzymic forms was suggested by studies, mostly in rats, on the role of MAO in brain maturation. In rats[11,12] and mice[13–15] MAO activity is clearly age-dependent; it increases to about 18 days of age but changes little thereafter in rats, but continues to fluctuate in mice until 6 weeks of age. Differences in MAO activity as related to brain regions and estrus cycle have been noted in the rat[16]; regional changes have also been reported in mice following fighting[17].

In our continuing studies of neurological mutations of mice we determined brain and liver MAO of a number of mutants belonging to 3 different clinico-pathological classes. These were mutants with a) cerebellar malformations and ataxia, b) cerebellar malformations, ataxia and epileptiform seizures, and c) tremors and fatal seizures without anatomical changes of the cerebellum.

Material and methods. The following mutations were studied, leaner (C57BL/6J – tg^{la}/tg^{la}) and reeler (C57BL/6J – *rl/rl*) belonging to class (a), weaver ($B6CBAF_1$ – *wv/wv*) of class (b), and jimpy ($B6CBAF_1$ – jp^{msd}/y) and wobbler-lethal (WLHR/J – *wl/wl*) of class (c). They were compared with their respective normal (+/–) littermate controls. Since several of these mutations are maintained on different genetic backgrounds we also analyzed the effect of the genotype or inbred strain on MAO. Due to the shortened life span of the mutants, they and their controls were generally 18 to 20 days old but we also studied the different inbred strains at 6 weeks of age.

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Monoamine oxidase activities in different strains of mice

Strain	Age	Specific activity, SE				Relative activity, SE			
		Brain		Liver		Brain		Liver	
C57B1/6J (4)	18–20 days	11.82	0.44[a]	166.67	5.75[b]	0.336	0.012[b]	5.74	0.20[b]
CBA/J (8)	18–20 days	9.26	0.25[a]	97.80	3.40[a]	0.249	0.015[b]	2.58	0.20[a]
$B6CBAF_1$ (8)	20 days	9.37	0.52	104.65	7.77	0.256	0.02	3.23	0.30
C57B1/6J (7)	6 weeks	13.40	1.48	104.03	16.03	0.387	0.043	4.26	0.75
CBA/J (6)	6 weeks	11.37	0.66	93.65	5.93	0.317	0.023	3.37	0.36
DBA/2J (6)	6 weeks	12.80	0.56	133.52	8.02	0.349	0.011	4.84	0.17

Specific activity = nmole Kynuramine deaminated/mg protein/30 min. Relative activity = μmole Kynuramine deaminated/g tissue/30 min. SE, standard error; ( ) number of mice; [a] Student t-test, $p < 0.05$; [b] $p < 0.01$.

The mice were killed by decapitation. The brains and livers were then excised and frozen immediately at –70 °C. On the day of assay the organs were weighed and homogenized in 9 volumes of 0.25 M sucrose. The homogenates were then centrifuged at $1500 \times g$ for 10 min to remove cellular debris and the supernatant again centrifuged at $18{,}000 \times g$ for 20 min to pellet the mitochondria. The mitochondria were then resuspended in a volume equal to 3× the wet weight of the organ. Protein was determined by the LOWRY method[18] and MAO according to WEISSBACH et al[19]. Since interference occurred with mitochondrial suspensions from brain materials at the adsorption peak of the substrate, kynuramine dihydrobromide (360 nm) we read the adsorptions at 328.5 nm which is the peak for the reaction product, 4-hydroxyquinoline. As the reaction is equimolar we express all the results as the amount of substrate utilized. Enzyme activity was halted either by boiling the homogenate for 10 min or the addition of 0.6 ml of 10 mM iproniazid phosphate to the reaction mixture, but was unaffected by the addition of 10 μmoles of potassium cyanide.

Results and discussion. None of the mutations tested affected MAO as we found no differences in MAO between any of the mutants and their respective littermate controls (data not shown). However, there were significant differences ($p < 0.05$) between some of the mutants i.e. reeler (*rl*), jimpy (*jp*^{msd}) and wobbler lethal (*wl*). Since these differences did not result from an effect of the mutant genes themselves they must have resulted from differences in the genetic background or inbred strains. Our results are presented in the Table. For example strain C57BL/6J has significantly higher levels of MAO in both brain and liver than strain CBA/J. The hybrid between them ($B6CBAF_1$) is like CBA/J suggesting dominance for low over high MAO activity. The fact that differences in MAO between strains occurred only up to 20 days of age but not at 6 weeks may reflect genotype-dependent differences in rates of organ development and maturation. Thus, our data confirm the age differences in levels of MAO observed by others[13–15].

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Serum Enzyme Activities in the African Elephant (*Loxodonta africana*)

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Summary. The serum activities of aspartate aminotransferase, alanine aminotransferase, α-hydroxybutyrate dehydrogenase and creatine phosphokinase have been measured in the African elephant. In general, the values were broadly comparable with those of man except that alanine aminotransferase was much lower and creatine phosphokinase higher. No variation due to age, sex, season or location was observed.

Measurements of the levels of the enzymes found in blood serum are widely used in clinical laboratories as diagnostic aids in the treatment and assessment of human disease. Increasing use of such assays is also being made in veterinary laboratories in the care and management of domestic animals. The African elephant, *Loxodonta africana*, is an animal of some commercial importance and, since we are currently involved in a wide ranging study of the elephant in the game parks of Uganda, we decided to measure the serum activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT), α-hydroxybutyrate dehydrogenase (α-HBD, lactate dehydrogenase-1-isoenzyme) and creatine phosphokinase (CPK) and to find out what factors, if any, caused variations in these levels.

Material and methods. Blood was obtained immediately after death from elephants shot in the Rwenzori and Kabalega National Parks in Uganda. Serum samples were frozen as soon as practicable and then flown to the United Kingdom for analysis.

AST, ALT, α-HBD and CPK were all estimated using optimized standard methods[3] obtained in kit form