Brodie and Maickel[10] felt that the goldfish could not metabolize drugs *in vivo* in part because of low liver TPNH activity. Because fish liver alcohol dehydrogenase (ADH) does not use TPN[11], Brodie and Maickel's conclusions are not relevant at least with alcohol metabolism in goldfish.

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Aldolase C in Brain Tumour

Three molecular species of aldolase have been observed in mammalian tissues: aldolase *A* in muscle, aldolase *B* in liver and aldolase *C* in brain[1-3]. We have already reported that the aldolase *B* found in the normal rat liver disappeared in the fast growing and highly malignant rat hepatomas and, instead, aldolase *A* was produced. In slowly growing rat hepatomas, aldolase *A*, aldolase *B* and their hybrids were observed[4]. The existence of aldolase *C* in the brain attracted our attention and led us to investigate the aldolase isozyme patterns of human brain tumours.

Fresh tumour tissue removed at the operation room was homogenized with one volume of 20 mM *tris* HCl buffer (*p*H 7·4) containing 0·154 M KCl and 5 mM EDTA. The aldolase in the supernatant fraction obtained after centrifugation at 105,000*g* for 60 min was assayed for activities, separated electrophoretically and stained on a cellulose acetate membrane as described before[4].

Fig. 1 shows representative cases of aldolase isozyme patterns from cerebral cortex and brain tumour. The normal cerebral cortex showed bands of aldolase *C*, and aldolase *A* with three hybrid bands. The pattern was the same in white matter. It is clear that glioblastoma possessed aldolase *C*, aldolase *A* and their hybrids, and meningioma lacked aldolase *C* or its hybrids with aldolase *A*.

Table 1. ALDOLASE ACTIVITIES OF BRAINS

Tumours	FDP (units/g protein)	F1P	FDP/F1P ratio
Cerebral cortex	120·8	6·3	19·2
Glioblastoma	54·1	2·4	20·8
Meningioma	78·6	2·8	47·6

Average values from several determinations.

The aldolase activities of brain tumours toward fructose-1,6-diphosphate (FDP) and F1P are listed in Table 1. The average ratio of FDP/F1P was about 50 for meningioma but those of cerebral cortex and glioma were far lower than 50. These data also show that aldolase in glioblastoma consisted of aldolase *C* and aldolase *A* peptides and that aldolase in meningioma was chiefly aldolase *A*. Nerve cells and glia cells originate from the ectodermal component[5] and it seems reasonable to suppose that glioblastoma has the same aldolase pattern as cerebral cortex, and that meningioma, which is thought to be of mesodermal origin, is different in its aldolase pattern.

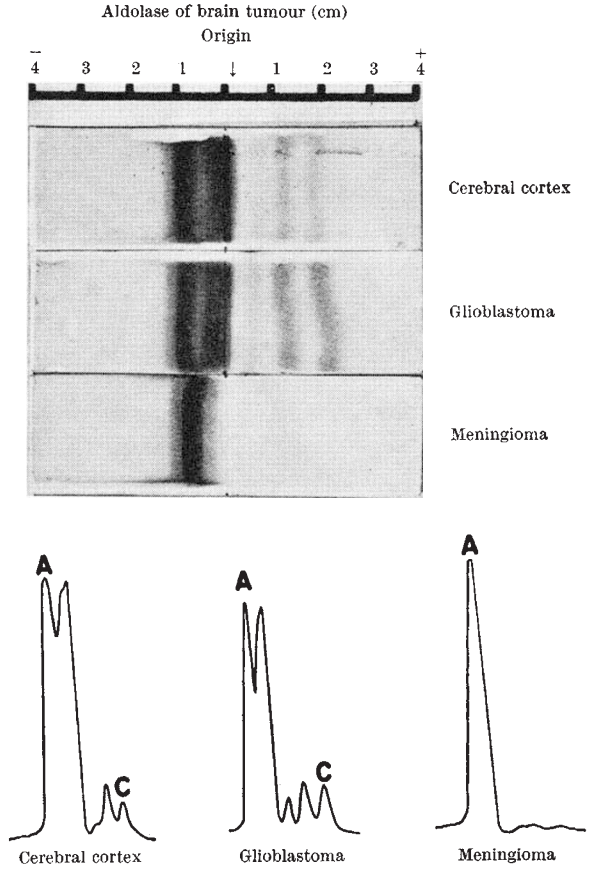

Fig. 1. Isozyme patterns of aldolase and their densitometric tracings.

Studies on relationships between patterns of aldolase isozymes and other types of tumours are now being carried out. Aldolase isozyme pattern in brain tumour may offer a new biochemical tool for the differential diagnosis of the brain tumours, the histological diagnoses of which are equivocal.

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