Table 1. AVERAGE PROPORTION OF THE MEDULLA IN THE THYMUS

Strain Age (months) 1 6	Female Age (months)		
	6	1	6
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	$21 \cdot 2$ (8) $38 \cdot 2$ (6) $28 \cdot 5$ (5)	$\begin{array}{c} 14 \cdot 2 & (6) \\ 23 \cdot 8 & (6) \\ 22 \cdot 3 & (7) \end{array}$	$\begin{array}{c} 27 \cdot 7 & (8) \\ 44 \cdot 1 & (6) \\ 32 \cdot 3 & (5) \end{array}$
	Age (mon 1 16·5% (5)* 28·5 (7)	Age (months) 1 6 16.5% (5)* 21.2 (8) 28.5 (7) 38.2 (6)	$\begin{array}{ccc} Age \ (months) & Age \ (n\\ 1 & 6 & 1 \\ 16.5\% \ (5)^{*} \ 21^{-}2 \ (8) & 14^{+}2 \ (6) \\ 28.5 \ (7) & 38^{+}2 \ (6) & 23^{+}8 \ (6) \end{array}$

* Total number examined.

Sections of the thymus were projected on white paper and the outlines of the cortex and the medulla were The percentage of the area occupied by the traced. medulla in the thymus was calculated. The results obtained at one and six months of age are presented in Table 1. The relative size of the medulla seems to increase with advancing age. It was also interesting to find a difference in histology of the thymus between the two strains examined: the relative size of the medulla being larger in strain SL than in AKR, and the values obtained in the F_1 hybrid mice were intermediate between those of the two parental strains.

A detailed study of the histology of the lymphoid tissues from the two strains of mice is now in progress, to investigate preleukæmic lesions and the earliest stage of leukæmic proliferation in the thymus and/or other lymphoid tissues.

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Encephalitogenic Factor in Experimental 'Allergic' Encephalomyelitis

SEVERAL attempts to isolate purified encephalitogenic factor(s) capable of producing experimental 'allergic' encephalomyelitis (with Freund's adjuvants) have been reported¹⁻⁵. Recently, Kies, Gordon, Laatsch and Alvord⁶ were successful in obtaining a derivative from guinea pig brain active in doses of 1-2 µg; and Robertson et al.7 obtained a preparation which showed "high . . . activity even at five gamma doses".

A material capable of producing disease in twelve out of twelve guinea pigs (about 500 g weight) when 1 μ g was injected with Freund's adjuvants has been isolated from fresh human post-mortem brain. It was obtained by a method differing from that of Kies et al.³ only in so far as special precautions were taken throughout to maintain an acid pH (refs. 2-5). On passing through a 'Sephadex G100' column this material gave five components, most of the activity being contained in the second (largest) fraction. Electrophoretic examination showed that the original material comprised a major and a minor moiety migrating towards the cathode at pH 5. The most active eluate from the Sephadex column migrated as a single component in the same direction, provided that the pH was again maintained at 5.0. If the pH were altered to 9.0 then this purified material showed one major and five minor bands.

An analytical ultracentrifuge analysis, kindly carried out by Dr. R. A. Kekwick of the Lister Institute, showed only one component in both the crude and the columnpurified encephalitogenic material. It seems probable that relatively small molecules (20,000-50,000 mol. wt.) are involved.

The non-specific antigens shared by brain with various other tissues^{8,9} can still be demonstrated in both the crude and the purified encephalitogenic factor by the Ouchterlony plate method. Obviously then, despite the high activity of the final material, non-active contaminant vet remains.

An attempt has been made to demonstrate circulating antibody to the encephalitogenic factor both in experimental 'allergic' encephalomyelitis of guinea pigs and in human multiple sclerosis cases by the tanned red blood cell technique. So far, no statistically significant difference from controls has been demonstrated in either case. Nor have serial estimations of the serum titre of encephalitogenic factor-antibody in animals which developed experimental 'allergic' encephalomyelitis after the inoculation of freeze-dried brain plus Freund's adjuvants demonstrated a rise in antibody level.

Since the development of experimental 'allergic' encephalomyelitis has in general been linked with the evolution of a delayed tuberculin type sensitivity to the brain used as antigen, skin sensitivity to encephalitogenic factor has been tested in animals suffering from the disease. This sensitivity has been compared with that developed to the saline supernate from fresh brain emulsion, known to be non-encephalitogenic. While guinea pigs suffering from experimental 'allergic' encephalomyelitis (induced by freeze-dried brain plus Freund's adjuvants) show definite sensitivity to encephalitogenic factor (P < 0.001), they do so also to the inactive supernate referred to here (P < 0.001). Moreover, sensitivity of such animals to supernate is greater than it is to encephalitogenic factor (P < 0.05 - 0.02). These results suggest that many of the skin hypersensitivity observations hitherto published may relate to material which is non-encephalitogenic.

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RADIOBIOLOGY

Effect of Fluoroacetate on Uptake of Yttrium-91 in Rabbits

STUDIES of yttrium-binding by bone powder and bone extracts have indicated that the presence of citrate might explain the uptake of yttrium-91 in bone¹. The experiments recorded here were undertaken to determine whether the citrate content of tissues could be correlated with uptake of yttrium, and for this purpose the uptake of vttrium in the soft tissue of the rabbit was investigated after giving sodium fluoroacetate to the animal prior to the vttrium.

Several workers²⁻⁴ have examined citrate accumulation in the soft tissues of rats at various times after injection with non-lethal doses of fluoroacetate. They were able to observe a 10- to 20-fold increase in concentration of citrate in tissues such as heart, spleen and kidney, but no significant increase in $bone^{4,5}$. Similar investigations of citrate accumulation in the soft tissues of rabbits have not been reported. In my experiments Dutch rabbits received intraperitoneal injections of sodium fluoroacetate; the dose ranged from 0.1 mg/kg to 1.0 mg/kg body-weight.