resistance against *LZ* cells could be detected in animals treated with an extract of spontaneous mammary tumour of strain *A* female or with extracts of spleen and liver of a healthy *A* mouse.

Antigens related to, or identical with, antigens induced by MCLV infection were detected not only in leukæmia strains originally induced by the same virus but also in cells of *NK/Ly* strain. This established lymphoma strain did not grow in MCLV infected mice after inoculation of 10^4 cells, while in untreated animals LD_{50} was $< 10^3$. Nemeth and Kellner obtained the *NK/Ly* strain by transplantation of a spontaneous *C3* mouse leukæmia[8]. It is quite possible that spontaneous leukæmias in *C3* mice, which develop approximately in 10 per cent of mice of this strain, are induced by MCLV or by a related virus. Pre-treatment by MCLV had no effect on the transplantability of Ehrlich-tumour and of the established mammary carcinoma of mice *A-OMG*. It should be noted that MCLV contamination of our sub-line of Ehrlich carcinoma, repeatedly demonstrated in our experiments[6], did not induce new cellular antigen(s). The presence of antigens peculiar to a certain oncogenic virus indicates that the tumour must have been induced by the same agent.

Resistance-phenomenon was also used for titration of MCLV in leukæmic-tissue extracts. The mice were inoculated by one of consecutive ten-fold dilutions of the extract, 10^{-1}–10^{-8}, and the resistance of the animals to *LZ*-leukæmia transplantation was determined by the method already mentioned. The titration results were expressed in virus doses inducing resistance in 50 per cent of mice—RID_{50}. As seen from Table 3, virus preparations contained $10^{4.6}$ and $> 10^{6.0}$ RID_{50}. The method of MCLV titration by its resistance-inducing capacity proved to be sensitive enough. This method allows determination of the concentration of the virus in the material investigated in a reasonably short time.

Thus the resistance-phenomenon can be used for identification and titration of MCLV and other leukæmogenic mice viruses as well as for the studies of the ætiology of long-transplantable and primary tumours of various types.

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Protective Effect of Encephalitogenic Factor in Experimental Allergic Encephalomyelitis

It has long been known and confirmed that a course of inoculation with nervous tissue affords some protection against subsequent challenge with a potent brain–Freund adjuvant mixture[1–4]. Moreover, Freund's adjuvant alone has also been shown to exert a protective effect[5].

Following the isolation of a purified and highly active encephalitogenic factor (E.F.) from human brain[6], attempts to protect with this material seemed appropriate.

Guinea pigs (400–600 g) of either sex were given three intracutanous injections of 100 E.F.[6] in either: (*a*) saline or (*b*) incomplete Freund's adjuvant (that is, lacking tubercle), at 10-day intervals. Ten days following the last injection they were challenged with a known potent encephalitogenic mixture (15 mg human white matter with Freund's adjuvant) by injecting 0·1 ml. intracutaneously in the right foot.

Response to this challenge was assessed on the scoring system proposed by Kies *et al.*[7] and the results are set out in Table 1. For comparison purposes the protective effects of similar pretreatment with Freund's adjuvant and also incomplete adjuvant are included.

While E.F. in saline solution exerted little protective effect, in incomplete Freund it showed a highly significant activity.

Table 1

	No. of animals	Score		*P* (*t* test)
Controls	17	4·12		
Exp. I 100μ E.F. in saline	12	4·75	4·38	
Exp. II 100μ E.F. in saline	10	4·00		
Exp. I 100μ E.F. in incomplete Freund	11	1·45		
Exp. II 100μ E.F. in incomplete Freund	12	0·00	0·55	< 0·001
Exp. III 100μ E.F. in incomplete Freund	10	0·20		
Freund complete	9	1·33		< 0·001
Freund incomplete	6	4·66		

Protection afforded by E.F. in incomplete Freund's adjuvant is comparable with that given by complete Freund.

If human multiple sclerosis is indeed a disease resulting from an auto-immune response to the patient's own brain material (perhaps degraded by a prior virus or other infection), then the activity of this human purified brain derivative might be harnessed for therapeutic purposes.

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Tetracyclin Fluorescence as a Marker for Detection of Viral Lesions *in vitro*

Tetracyclin has been shown to be incorporated into different tissues undergoing necrobiotic changes, and the presence of the drug can be detected by its fluorescence properties. Consequently, tetracyclin fluorescence has been seen in neoplastic tissues[1], in gastric and cutaneous ulcers[2,3], and in bacterial foci[4]. Our recent investigations on viral infection in organ rudiments *in vitro* seemed to offer a good possibility for following tetracyclin incorporation into viral lesions.

When embryonic mouse kidneys are infected with *SE*-polyoma virus, both the morphologically detectable viral lesions and the antigenic viral proteins show a characteristic pattern; the viral lesions and antigenicity are invariably found in the undifferentiated mesenchyme, whereas the epithelial elements seem to resist the viral infection[5,6]. Hence, this model seemed to provide a good tool for examining incorporation of tetracyclin into cells with and without viral lesions.

Twelve-day mouse kidney rudiments were cultured for various periods of time in a Trowell-type culture as described earlier[5]. Immediately after setting up the cultures, they were infected with *SE*-polyoma virus by adding 0·1 c.c. of the stock solution to 12 c.c. of medium. The stock solution had an HA-titre of 1 : 1024 on guinea pig red cells[7]. 24 h prior to collecting the cultures, purified tetracyclin ('Tetracyn', Pfizer) was added to the culture medium in a final concentration of 0·05 mg/c.c.