and hamster sera were entirely free of inhibitory activity. Lyophilized human serum fractions also failed to inhibit the effect of a potent filtrate.

The data cited in the last two paragraphs were obtained from observations on 5500 hamster babies injected since group 1 was done (*7*).

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References and Notes

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3. ——— and R. A. Wallace, *ibid.* **18**, 698 (1958).
4. The abnormality of the hamsters described in this report is physically quite different from the "runting syndrome" described in rats and mice. Baby rats and mice injected with potent fractions developed normally.
5. I am deeply indebted to William Money of the Sloan-Kettering Institute for the I^{131} studies.
6. The relationship of the abnormality described here for hamsters to that of human mongolism is unknown. An extra autosome has been found in the cells of a number of human mongoloid individuals. It does not seem likely that an extra chromosome exists in the animals described here, although this possibility is being investigated. It appears probable that we have here a phenocopy of the mongoloid entity.
7. This work was aided by grant No. E-109-E10 from the American Cancer Society and was supported in part by Public Health Service grant No. C2042 from the National Cancer Institute, Public Health Service.

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Rapid Induction of Allergic Encephalomyelitis in Rats without the Use of Mycobacteria

Abstract. Rats, in contrast to certain other species of animals reported, have a striking capacity to develop allergic encephalomyelitis within 2 to 3 weeks following one injection of spinal cord antigen combined with Freund's incomplete adjuvant—that is, adjuvant prepared without addition of killed mycobacteria.

The necessity of combining nervous tissue inocula with Freund's complete adjuvant (emulsifying agent, paraffin oil, and killed mycobacteria) for rapid and regular induction of allergic encephalomyelitis (AE) in monkeys and guinea pigs has been reported (*1*, *2*). In these studies, injection of nervous tissue emulsions not containing the mycobacteria induced little, if any, disease. Freund and Stone (*2*) have determined the minimal amount of mycobacteria required for induction of characteristic allergic encephalomyelitis in the guinea pig.

Work in our laboratory indicates that mycobacteria are not required for rapid induction of this inflammation in rats. Groups of adult male or female Wistar rats, obtained from two commercial sources, were injected intracutaneously with guinea pig spinal cord (collected aseptically and stored at −20°C for 1 to 7 days) homogenate combined with either complete adjuvant (that is, with added mycobacteria) or incomplete adjuvant (without added mycobacteria) prepared as described by Freund (*3*). Each rat received approximately 115 mg (wet wt.) of spinal cord (an excessive dose used in past work with this host) in 0.7 ml of inoculum distributed among six sites over the upper dorsum and one site on the ventral neck. The animals were given free access to food pellets and water. They were observed daily for neurological signs for 21 to 26 days following injection; then they were killed and their brains and spinal cords were removed for histological studies. A minimum of seven different hematoxylin and eosin stained sections (at levels of thalamus, mesencephalon, cerebellum-pons, medulla, and cervical-thoracic spinal cord) of nervous tissue from each rat were examined microscopically for lesions.

The results of two representative experiments are shown in Table 1. Twenty-four rats received either of two spinal cord homogenates combined with incomplete adjuvant. Nine rats exhibited moderate to severe flaccid paralysis of the hind legs within 14 to 20 days. Numerous and intense lesions were found in these 9 animals as well as in 13 of the remaining 15 rats. As expected, 6 of the 11 control rats which received spinal cord homogenate combined with complete adjuvant developed allergic encephalomyelitis. The data (Table 1) are in agreement with the results of three other experiments, not given in detail here. Additional control rats, which were similarly injected with guinea pig kidney homogenate and incomplete adjuvant or with only the adjuvant, remained clinically well and were subsequently found to have no lesions.

It is of interest that allergic encephalomyelitis may be induced in rats by an intracutaneous injection of the spinal cord antigen alone. For example, 2 of 16 rats used in two experiments were found to have lesions when sacrificed approximately 3 to 4 weeks postinjection. This finding is not unexpected in view of studies of earlier workers (*4*) showing that this inflammation or its equivalent may occasionally be induced in rats, rabbits, and monkeys by often-repeated injections of nervous tissue homogenates or extracts. More recently, Morrison (*5*), Jervis *et al.* (*6*), and Waksman (*7*) have reported that encephalomyelitis occasionally may be induced in rabbits, dogs, and mice after one to several injections of nervous tissue not combined with Freund's adjuvant.

The data (Table 1) indicate that in the rat, emulsifying agent-paraffin oil, without addition of mycobacteria, provides the necessary adjuvant effect for rapid, regular induction of the inflammation. The work reported here has direct bearing on studies of the mode of action of Freund type immunological adjuvants with respect to their capacity to enhance immune responses, of the immediate or the delayed type or both, against a wide variety of antigenic materials, including nervous tissue antigens (*8*).

Table 1. Allergic encephalomyelitis (AE) in rats following an intracutaneous injection of spinal cord combined with Freund's immunological adjuvants.

Expt. No.	Spinal cord antigen	Paraffin oil and emulsifying agent	Killed mycobacteria	No. with signs and lesions of AE	No. with lesions of AE only	Total No. injected
E 7-58	+	+	0	3	9	12
	+	+	+	3	0	6
G11-58	+	+	0	6	4	12
	+	+	+	2	1	5

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