



Fig. 1 Daily group parasitaemia levels (top); weekly fluorescent antibody titres (bottom).

until the fifth week of infection. By day 14, the untreated control and the NRS-treated mice showed significant increases in antibody titre, and peak titres were attained by day 28. At day 14, the ALS-treated mice showed no increase in titre. By day 21 they showed modest increases, and not until day 35 had they attained titres comparable to the other two groups.

This study shows that, in mice, ALS can suppress the antibody response to a replicating agent just as it suppresses the response to non-replicating antigens such as sheep red cells. As already noted, ALS treatment did not alter the humoral response of rodents to several other infectious agents. On the other hand, Wenner *et al.* have recently reported a delay in the antibody response to a monkey pox infection in ALS-treated cynomolgous monkeys¹⁰, a result similar to our own in mice with a malaria infection. It is reasonable to suppose that ALS may act similarly in man and that the suppression of the humoral immune response by ALS might interfere with the diagnosis of or recovery from some infections.

It is apparent from the appearance of antibody and recovery from the *P. berghei* infection that the effects of ALS were transient. This was probably because of the formation of antibody to the ALS, known to occur in rodents receiving frequent ALS injections^{2,11}.

The close temporal relationship of antibody synthesis and decrease in parasitaemia in all three groups of mice suggests that antibody participates in the recovery process. This possibility is supported by previous studies which showed that malarial antibody can passively protect infected humans and rodents¹²⁻¹⁴, and impair the *in vitro* proliferation of plasmodia¹⁵.

There is evidence from studies using neonatal thymectomy and passive transfer of lymphocytes that cell-mediated immunity may also participate in recovery from *P. berghei*¹⁶⁻¹⁸. Phagocytic activity by the reticuloendothelial system is essential to recovery¹⁹, and recently it has been suggested that interferon may be involved²⁰. In addition to antibody synthesis, each of these defence mechanisms may be impaired by

ALS^{21,22}, making it difficult to delineate the critical factors in recovery from infection by the use of ALS.

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- ¹ James, K., *Clin. Chim. Acta*, **22**, 101 (1968).
- ² Barth, R., Southworth, J., and Burger, G., *J. Immunol.*, **101**, 282 (1968).
- ³ Muschel, L. H., Gustafson, L., and Atai, M., *Immunology*, **14**, 285 (1968).
- ⁴ Sadun, E., *Proc. Annual Meeting Amer. Soc. Parasitologists, Washington* (1969).
- ⁵ Hirsch, M., and Murphy, F. A., *Lancet*, ii, 37 (1968).
- ⁶ Hirsch, M., *J. Exp. Med.*, **128**, 121 (1968).
- ⁷ Landau, I., and Chabaud, *AGCR Acad. Sci.*, **260**, 230 (1965).
- ⁸ Levey, R. H., and Medawar, P. B., *Proc. US Nat. Acad. Sci.*, **56**, 1130 (1966).
- ⁹ Kuvin, S. F., Tobie, J. E., Evans, C. B., Coatney, G. R., and Contacos, P. G., *Science*, **135**, 1130 (1962).
- ¹⁰ Wenner, H. A., Bolano, C., Cho, C. T., and Kamitsuka, P. S., *J. Inf. Dis.*, **120**, 318 (1969).
- ¹¹ Jasin, H. E., Lourie, S. H., Currey, H. L. F., and Ziff, M., *J. Immunol.*, **100**, 654 (1968).
- ¹² Cohen, S., McGregor, I. A., and Carrington, S., *Nature*, **192**, 733 (1961).
- ¹³ Diggs, C., and Osler, A., *J. Immunol.*, **102**, 298 (1969).
- ¹⁴ Stechschulte, D. J., Briggs, N. T., and Wellde, B. T., *Mil. Med.*, suppl., 1140 (1969).
- ¹⁵ Cohen, S., Butcher, G. A., and Crandall, R. B., *Nature*, **223**, 368 (1969).
- ¹⁶ Brown, I., Allison, A. T., and Taylor, R. B., *Nature*, **219**, 292 (1968).
- ¹⁷ Stechschulte, D. J., *Proc. Soc. Exp. Biol. Med.*, 748 (1968).
- ¹⁸ Stechschulte, D. J., *Mil. Med.*, suppl., 1147 (1969).
- ¹⁹ Goble, F., *Ann. NY Acad. Sci.*, **88**, 149 (1960).
- ²⁰ Jahiel, R., Vilcek, J., Nussensweig, R., and Vanderberg, J., *Science*, **802** (1968).
- ²¹ Sheagren, J., Barth, R. F., Edelin, J. B., and Malmgren, R. A., *Lancet*, ii, 297 (1969).
- ²² Barth, R., Friedman, R. M., and Malmgren, R. A., *Lancet*, ii, 723 (1969).

Xg Blood Groups of Thais

SAMPLES of blood from 181 normal unrelated Thais from Bangkok have been tested for the X-linked blood group antigen Xg^a. The results are shown in Table 1.

Table 1 Frequency of Xg^a in Thais

	Xg (a +)	Xg (a -)	Total
Male	73	48	121
Female	46	14	60

The number is small but the results are sufficient to show that antigen is less common in Thais than in Europeans¹ and Indians², but more common than in Chinese³. Thai gene frequencies calculated from the male and female results are: Xg^a 0.57 and Xg 0.43.

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- ¹ Gavin, J., Tippett, P., Sanger, R., and Race, R. R., *Nature*, **200**, 82 (1963).
- ² Bhatia, H. M., *Indian J. Med. Sci.*, **17**, 491 (1963).
- ³ Boon, W. H., Noades, J., Gavin, J., and Race, R. R., *Nature*, **204**, 1002 (1964).