the particles, whether electrostatically or sterically generated, to permit flocculation to occur.

We thank the Australian Research Grants Committee for financial support.

ROBERT EVANS
D. H. NAPPER

Department of Physical Chemistry,
University of Sydney,
NSW 2006

Received September 3, 1973.

1 Napper, D. H., and Hunter, R. J., *M.T.P. Int. Rev. Sci., phys. Chem. Ser.*, **1**, 7, 294 (1972).
2 La Mer, V. K., and Healy, T. W., *Rev. pure appl. Chem.*, **13**, 112 (1963).
3 Ries, jun., H. E., and Meyers, B. L., *Science, N.Y.*, **160**, 1449 (1968).
4 Gregory, J., *Trans. Faraday Soc.*, **65**, 2260 (1969).
5 Ries, jun., H. E., *Nature*, **226**, 72 (1970).
6 Williams, D. J. A., and Ottewill, R. H., *Kolloid-Z. Z. Polym.*, **243**, 141 (1971).
7 Ottewill, R. H., and Shaw, J. N., *Kolloid-Z. Z. Polym.*, **215**, 161 (1967).
8 Ottewill, R. H., and Shaw, J. N., *Kolloid-Z. Z. Polym.*, **218**, 34 (1967).
9 Shaw, J. N., and Marshall, M. C., *J. Polym. Sci. Pt A-1*, **6**, 449 (1968).
10 Smitham, J. B., Gibson, D. V., and Napper, D. H., *J. Colloid Interface Sci.* (in the press).
11 Evans, R., Davison, J. B., and Napper, D. H., *J. Polym. Sci., Pt. B*, **10**, 449 (1972).
12 Bailey, F. E., Lundberg, R. D., and Callard, R. W., *J. Polym. Sci. Pt. A*, **2**, 845 (1964).
13 Bailey, F. E., and Koleske, J. V., *Nonionic Surfactants* (edit. by Schick, M. J.), **1**, 819 (Dekker, New York, 1967).
14 Kurata, M., Iwama, M., and Kamada, K., *Polymer Handbook* (edit. by Brandrup, J., and Immergut, E. H.), IV-58 (Interscience, New York, 1966).
15 Walles, W. E., *J. Colloid Interface Sci.*, **27**, 77 (1968).

BIOLOGICAL SCIENCES

Genetic Differences in Conformational Changes of Albumin in the House Mouse

THE ability of mouse liver to convert mouse or bovine albumin to a faster migrating band under acid conditions of electrophoresis has recently been found to be controlled by a dominant gene, as observed in the SWR/J and several other strains of mice[1]. Other strains, such as C3H/HeJ, had the recessive allele and were unable to convert albumin. The conditions used included a *p*H at which conformational changes in albumin are known to occur, suggesting that the strains of mice differ in a factor in the liver capable of converting albumin to a different conformational state. I report here evidence that the conversion of albumin can be reversed by lowering the *p*H markedly. This provides further evidence that a conformational change has taken place.

Livers from male C3H/HeJ and SWR/J mice were homogenized and centrifuged, and the supernatant was extracted using procedures described before[1]. One volume of horse serum albumin (HSA, Fraction V, Sigma, 5 mg ml^{-1} in 0.25 M sucrose) was added to 9 volumes of extract. An equal volume of solvent (citric acid, monohydrate: 15% sucrose, 1:5 (w/v), plus 6 M urea) was added to each extracted sample. Before electrophoresis 10 μl was added to a slot, 5 volumes of an aliquot was diluted with 1 volume of concentrated HCl and 20 min later 12 μl was added to another slot, and finally 10 μl of an aliquot untreated with HCl was added to a third slot. Controls were also included in which 0.25 M sucrose replaced HSA or liver supernatant, but were otherwise handled as other

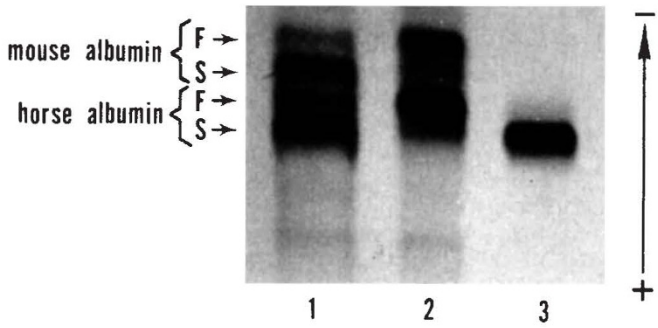

Fig. 1 Electrophoretograms showing the effect of liver extract and HCl on HSA. Channels 1–3 are (1) HSA+SWR/J liver extract+HCl; (2) HSA+SWR/J liver extract, and (3) HSA. Electrophoresis was as previously described[1]. Gel buffer was 0.05 M Tris-citric acid, *p*H 2.4+3 M urea+10% 'Cyanogum'-41. Electrode buffer was 0.2 M glycine-citric acid, *p*H 3.5.

samples, although not treated with HCl. HSA was used because both slow and fast forms were electrophoretically distinct from proteins of mouse liver.

Results are shown in Fig. 1. HSA was present as a single, slower migrating band when not combined with liver (channel 3), whereas most was converted to a faster band when combined with liver extract of the SWR/J strain (channel 2). The latter result was not affected by the time when the sample was added to the slot, that is, before addition of HCl to an aliquot more than 20 min before electrophoresis compared with just before electrophoresis. Treatment with HCl (channel 1) lowered *p*H markedly (from 2.83 to 0.05) and converted much of the HSA to the slower migrating band. In C3H/HeJ mice (results not shown) HSA was not converted to a faster migrating band, but consisted only of a slow band migrating the same distance as in channel 3, with or without added HCl. Most of the mouse albumin of liver was also converted to a faster migrating band in the SWR/J but not the C3H/HeJ strain, as described previously[1]; treatment with HCl converted a large portion to the slower migrating band in the SWR/J strain (Fig. 1), but there was no change in the C3H/HeJ strain.

My data show that liver of one strain of mouse but not of another can convert HSA to a faster migrating form. The genetic difference is clearly not in the primary structure of albumin, that is, amino acid sequence, since the same source of HSA was used in all preparations, but is rather a secondary alteration of the molecule. A conversion to a different conformational state is highly likely in view of the reversal from the faster to the slower migrating band after treatment with excess acid, especially since lowering the *p*H below 3.5 favours the expanded, slower migrating form[2]. The reversibility of the change in albumin renders untenable the hypothesis that conversion of albumin from a slower to faster migrating form results from cleavage of a portion of the albumin molecule. These results point to the existence of two genes which control the structure of albumin, that is a conventional "structural gene" specifying primary structure[3] and a second gene affecting secondary or tertiary structure.

I thank D. E. Grossnickle for technical assistance, and Mr A. J. Brentlinger of the Audio-Visual Center, Indiana State University, for photography.

F. H. WILCOX

Department of Life Sciences,
Indiana State University,
Terre Haute, Indiana 47809

Received July 13, 1973.

1 Wilcox, F. H., *Biochem. Genet.*, **10**, 69 (1973).
2 Sogami, M., and Foster, J. F., *J. biol. Chem.*, **237**, 2514 (1962).
3 Petras, M. L., *Biochem. Genet.*, **7**, 273 (1972).