

damage. The possible exception to this is for irradiation in nitric oxide where both yields are of similar magnitude.

It is possible that the appearance of the unmasked tyrosyl groups accompanies the breakage disulphide bridges; that is, the three tyrosyl groups are unmasked as a result of the breakage of a single disulphide bridge. Tanford has suggested that the tyrosyl groups themselves might be involved in hydrogen bonding and that such bonds could be important in maintaining the tertiary structure of the molecule.

This work was supported by the National Cancer Institute of Canada and the Medical Research Council of Canada.

J. F. WILLIAMS  
J. W. HUNT

Department of Medical Biophysics,  
University of Toronto, and  
Ontario Cancer Institute.

- <sup>1</sup> Lea, D. E., *Actions of Radiations on Living Cells* (Camb. Univ. Press, 1946).  
<sup>2</sup> Pollard, E. C., Guild, W. R., Hutchinson, F., and Setlow, R. B., *Prog. Biophys.*, **5**, 72 (1955).  
<sup>3</sup> Hunt, J. W., Till, J. E., and Williams, J. F., *Rad. Res.*, **17**, 703 (1962).  
<sup>4</sup> Gordy, W., and Shields, H., *Proc. U.S. Nat. Acad. Sci.*, **46**, 1124 (1960).  
<sup>5</sup> Hirs, C. H. W., Moore, S., and Stein, W. H., *J. Biol. Chem.*, **235**, 633 (1960).  
<sup>6</sup> Ryle, A. P., and Anfinsen, C. B., *Biophys. Biochim. Acta*, **24**, 633 (1957).  
<sup>7</sup> White, F. H., and Anfinsen, C. B., in *Sulphur in Proteins*, 279 (Academic Press, New York, 1959).  
<sup>8</sup> White, F. H., and Sandoval, A., *Biochem.*, **1**, 938 (1962).  
<sup>9</sup> Ray, D. K., Hutchinson, F., and Morowitz, H. J., *Nature*, **186**, 312 (1960).  
<sup>10</sup> Yalow, R. S., *Proc. First Nat. Biophys. Conf.*, Columbus, Ohio, 169 (Yale Univ. Press, New Haven, 1959).  
<sup>11</sup> Boyer, P. D., *J. Amer. Chem. Soc.*, **76**, 4331 (1954).  
<sup>12</sup> Tanford, C., Hauenstein, J. D., and Rands, D. G., *J. Amer. Chem. Soc.*, **77**, 6409 (1955).  
<sup>13</sup> Cha, C.-Y., and Scheraga, H. A., *J. Amer. Chem. Soc.*, **82**, 54 (1960).

### An Alpha-globulin Allotype in the Mouse (MuB1)

POLYMORPHISM, detected by antibody from individuals of the same species (allotropy), has so far been observed only in the  $\gamma$ - (ref. 1) and  $\beta$ -globulins (ref. 2). In man, a class of  $\alpha$ -globulin *Gc1* and *Gc2* is subject to genetic variations and has been revealed by antibodies of heterologous origin<sup>3</sup>. Polymorphism of other  $\alpha$ -globulins, the haptoglobins in several mammalian<sup>1</sup> species and recently the trypsin inhibitors in man<sup>4,5</sup> have been detected by other means. So far, the polymorphism of serum proteins has been most thoroughly examined in man and rabbit. In the mouse the genetic variation of the transferrins<sup>6</sup> and of an allelic pair of  $\gamma$ -globulins (*MuA1* and *MuA2*) (refs. 7-10) have been examined. We wish to report here a new allotypic specificity among the  $\alpha$ -globulins of the mouse.

Antibody was induced in *DBA/2J* and *A/HeJ* mice immunized with the serum of *DBA/1J* and *C57L/J* mice, respectively. Mice were given subcutaneously, or into the foot pad, six injections of serum incorporated in complete Freund's adjuvant. On the fifth day after the last injection, the animals were bled from the tail, and the sera from the two groups of animals (*DBA/2J* and *A/HeJ*) were pooled separately. Both pools of sera reacted in an identical manner, giving one zone of precipitation in double diffusion with the serum of *BALB/cJ*, *CBA/J*, *C57BL/10J*, *C57Br/cdJ*, *C58/J*, *C57L/J*, *129/J*, *SJL/J*, *C3H/HeJ* and *DBA/1J* mice. The specificity was absent in mice of the *A/HeJ*, *A/J*, *AKR/J*, *RF/J*, *SWR/J* and *DBA/2J* strains. A number of hybrid strains were also tested: *AKR/J*  $\times$  *DBA/2J*, *C57BL/6J*  $\times$  *A/J*, *C57BL/6J*  $\times$  *DBA/2J*, *BALB/cJ*  $\times$  *A/J*, *C57L/J*  $\times$  *A/HeJ*, *C3H/HeJ*  $\times$  *DBA/2J*, *C3H/HeJ*  $\times$  *C57BL/6J*, and only the hybrid *AKR/J*  $\times$  *DBA/2J* was found to lack the allotypic specificity under investigation. In short, all hybrids having in their ancestry one or two of the strains containing the allotypic specificity also possessed the allotypic specificity in their serum.

In all cases, 3 or more males and 3 or more females were tested for the presence of the allotypic specificity and in

all cases the distribution of the allotypic specificity in males and females was identical.

In addition to inbred mice examined, forty 'Swiss' mice, from the Connaught Laboratories and from our own colony, were found to possess the allotypic specificity under investigation. Five serum samples of woodland deer mouse (*Peromyscus maniculatus gracilis*) were also examined and found to give a reaction of partial identity with the allotypic specificity of strains such as *DBA/1J*. These samples did not produce any detectable reaction with a previously described *MuA2* antiserum.

The antibody from *DBA/2J* and *A/HeJ* mice was shown by immunoelectrophoresis to react with an  $\alpha$ -globulin; the mobility of the  $\alpha$ -globulin was located by comparison with that of some well-defined human proteins (Fig. 1). Since it has been previously proposed to call the  $\gamma$ -globulin allotypes *MuA1*, *MuA2*, *MuA<sub>n</sub>*<sup>8,9</sup>, we now propose to call the  $\alpha$ -globulin family of allotypic specificities *MuB* and the specificity here described *MuB1*.

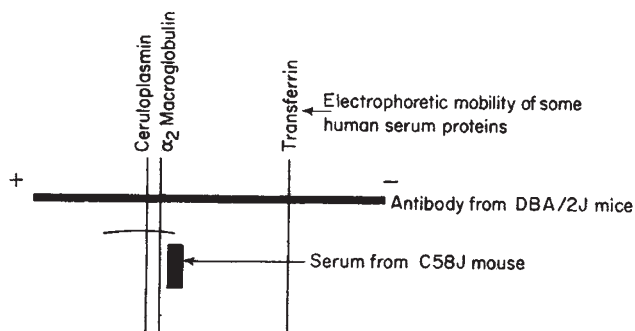


Fig. 1. Electrophoretic mobility of the  $\alpha$ -globulin *MuB1* in relation to human ceruloplasmin, transferrin and  $\alpha$ -macroglobulin

Thanks for financial support are due to the Banting Research Foundation, the National Institutes of Health (grant No. 571 GM-506-03), the Medical Research Council (grant No. MT-832) and the National Cancer Institute of Canada. We also thank Mr. W. A. McKinlay for blood samples of the woodland deer mice.

B. CINADER  
S. DUBISKI

Subdivision of Immunochemistry,  
Division of Biological Research,  
Ontario Cancer Institute, and  
Departments of Medical Biophysics and  
Pathological Chemistry,  
University of Toronto.

- <sup>1</sup> Cinader, B., and Dubiski, S., in *Colloq. Intern. Centre Nat. Rech. Sci.*, Paris, edit. by Bussard, A., **116**, 255 (1963).  
<sup>2</sup> Blumberg, B. S., *Ann. N.Y. Acad. Sci.*, **103**, 1052 (1963).  
<sup>3</sup> Hirschfeld, J., *Sci. Tools*, **8**, 17 (1961).  
<sup>4</sup> Allison, A. C., *Proc. Biochem. Soc.*, April 1963 (*Biochem. J.*, in the press).  
<sup>5</sup> Landerman, N. S., Webster, M. E., Becker, E. L., and Ratcliffe, H. E., *J. Allergy*, **33**, 330 (1963).  
<sup>6</sup> Cohen, B. L., and Schreffler, D. C., *Genet. Res.*, **2**, 306 (1961).  
<sup>7</sup> Kelus, A., and Moor-Jankowski, J. K., *Nature*, **191**, 1405 (1961).  
<sup>8</sup> Dubiski, S., and Cinader, B., *Nature*, **197**, 705 (1963).  
<sup>9</sup> Dubiski, S., and Cinader, B., *Canad. J. Biochem. Physiol.*, **41**, 1311 (1963).  
<sup>10</sup> Wunderlich, J., and Herzenberg, L. A., *Proc. U.S. Nat. Acad. Sci.*, Wash., **49**, 592 (1963).

## BIOCHEMISTRY

### X-Irradiation of Deoxyribonucleic Acid

ONE of the biochemical effects of X-irradiation is the inhibition of synthesis of DNA. Although Okada and Hempelmann<sup>1</sup> showed that, after ionizing radiation, there is an inhibition of the activities of enzymes essential for DNA synthesis, the experiments of Bollum *et al.*<sup>2</sup> indicate that regenerating rat liver can still synthesize thymidylate kinase and DNA polymerase in tissues where DNA syn-