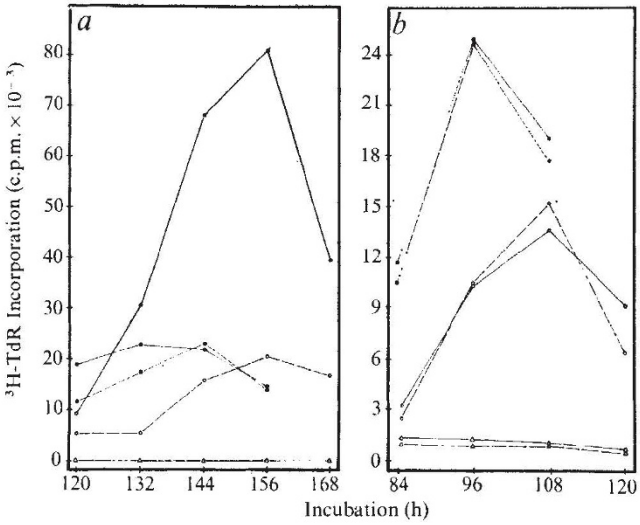

Fig. 2 MLC activation of thymocytes and spleen cells by H-2 and non-H-2 alloantigens in medium supplemented with serum from normal or nude mice. B10.D2 thymocytes (*a*) or spleen cells (*b*) were stimulated with X-irradiated C57BL/10 (○) or DBA/2 (●) spleen cells in medium supplemented with serum from normal (——) or nude (– – –) mice. 2.0 μCi ^{3}H-TdR were added to each culture 8 h before termination of MLC at the times indicated.

that some reactivity occurs using the serum from nude mice in the reaction of B10.D2 thymocytes against C57BL/10 cells.

Results of this study suggest the presence in mouse serum of a factor (or factors) which confers a state of immunocompetence on a class of cell which resides in the thymus of young mice and which is highly reactive against the products of H-2. The serum component permitting mouse thymocytes to respond against H-2 alloantigens is the subject of much speculation, as is the mode of action. Results of experiments showing the effects of serum from normal and from homozygous nude mice on the thymocyte responses to H-2 and non-H-2 antigens strongly supports the concept that this is a 'thymus factor'.

At present, it seems most probable that mouse serum acts on the T-cell precursors or immature T cells of the thymus and induces the state of immunocompetence (for example, development of specific receptor sites on the surface of the T cell). This possibility is supported by studies[20] in which a serum factor secreted by humans and mouse thymus confers on immature cell populations rosette forming capacity or a state of responsiveness to concanavalin A (con A).

In addition to H-2 reactive cells, the thymus also contains a class of cell reactive against products of the non-H-2 loci. On the basis of their MLC responsiveness in the serum-free medium these are probably a different class of cell. It is unlikely that responses against non-H-2 alloantigens in serum-free medium are due to their being more immunogenic than the H-2 alloantigens, because in the serum supplemented medium responses to H-2 are usually stronger than to non-H-2 antigens (Fig. 1).

H-2 alloantigens represent a special class of antigens to the immune system—they serve as highly effective target antigens for T-cell mediated cytotoxicity whereas non-H-2 antigens do not[8]. Other studies (A. B. Peck, unpublished data) show that thymocytes develop cytotoxic potential after their strong MLC activation by H-2 alloantigens only in the presence of mouse serum; no CML is observed in serum-free medium. Thymocytes also fail to develop cytotoxic potential against non-H-2 alloantigens despite strong MLC activation in strain combinations possessing genetic differences at only non-H-2 loci in either medium.

This culture system may define two classes of T cells by their *in vitro* response patterns to H-2 and non-H-2 antigens, and provides an easy method for identifying T cell functions of the two classes. Studies are in progress to separate and isolate the two cell types.

This study was supported by grants from the National Institutes of Health. A.B.P. is a Research Fellow of the Wisconsin Heart Association.

AMMON B. PECK*
FRITZ H. BACH

Departments of Medical Genetics and Surgery and the Immunobiology Research Center,
University of Wisconsin,
Madison, Wisconsin 53706

Received August 19, 1974.

* Present address: Uppsala Universitet Wallenberglaboratoriet, Dag Hammerskjölds Väg 21, 751 22 Uppsala 1, Sweden.

1 Cerrotini, J. C., Nordin, A. A., and Brunner, K. T., *Nature*, **228**, 1308–1309 (1970).
2 Blomgren, H., and Svedmyr, E. A. J., *Cell. Immun.*, **2**, 285–299 (1971).
3 Wagner, H., Harris, A. W., and Feldman, M., *Cell. Immun.*, **4**, 39–50 (1972).
4 Cerrotini, J. C., Nordin, A. A., and Brunner, K. T., *Nature*, **227**, 72–73 (1970).
5 Goldstein, P., Wigzell, H., Blomgren, H., and Svedmyr, E. A. J., *J. exp. Med.*, **135**, 890–906 (1972).
6 Eijsvoogel, V. P., du Bois, R., Melief, C. J. M., Zeylemaker, W. P., Raat-Koning, L., and de Groot-Kooy, L., *Transplant. Proc.*, **5**, 415–420 (1973).
7 Bach, F. H., Segall, M., Zier, K. S., Sondel, P. M., Alter, B. J., and Bach, M. L., *Science*, **180**, 403–406 (1973).
8 Alter, B. J., Schendel, D. J., Bach, M. L., Bach, F. H., Klein, J., and Stimpfling, J. H., *J. exp. Med.*, **137**, 1303–1309 (1973).
9 Schendel, D. J., Alter, B. J., and Bach, F. H., *Transplant. Proc.*, **5**, 1651–1655 (1973).
10 Festenstein, H., Sachs, J. A., Abbasi, K., and Oliver, R. T. D., *Transplant. Proc.*, **4**, 219–222 (1972).
11 Peck, A. B., Bach, F. H., and Boyse, E. A., *Transplant. Proc.*, **5**, 1611–1613 (1973).
12 Peck, A. B., and Bach, F. H., *J. Immun. Methods*, **3**, 147–163 (1973).
13 Katz-Heber, E., Peck, A. B., and Click, R. E., *Eur. J. Immun.*, **3**, 379–385 (1973).
14 Peck, A. B., and Click, R. E., *Eur. J. Immun.*, **3**, 385–392 (1973).
15 Bach, F. H., Widmer, M. B., Bach, M. L., and Klein, J., *J. exp. Med.*, **136**, 1430–1444 (1972).
16 Bach, F. H., Widmer, M. B., Segall, M., Bach, M. L., and Klein, J., *Science*, **176**, 1024–1027 (1972).
17 Klein, J., and Shreffler, D. C., *J. exp. Med.*, **135**, 924–937 (1972).
18 Stimpfling, J. H., and Reichert, A. E., *Transplant. Proc.*, **2**, 39–41 (1970).
19 Scheid, M. P., and Boyse, E. A., *Ann. N.Y. Acad. Sci.* (in the press).
20 Bach, J. F., Dardenne, M., Pleau, J. M., and Bach, M. A., *Ann. N.Y. Acad. Sci.* (in the press).

Erratum

In the article "Science and works of art" by J. P. Brommelle and N. S. Brommelle (*Nature*, **250**, 767; 1974) the address given for N. S. Brommelle was incorrect. N. S. Brommelle is at the Victoria and Albert Museum.

Corrigendum

In the article "Radiological mapping of the ribosomal RNA transcription unit in *E. coli*" by P. B. Hackett and W. Sauerbier (*Nature*, **251**, 639; 1974) the authors erroneously referred to the work of Bleyman *et al.* (*J. Bact.*, **99**, 535; 1969) (ref. 20) as being conducted in *E. coli*. These studies of Bleyman *et al.* utilised *B. subtilis* in which the transcriptional order of the ribosomal RNA genes seems to be the same as in *E. coli* (Pace, N. R., *Bact. Rev.*, **37**, 562; 1973) (ref. 1).