that H-Y antigen would be found in reptiles, and so we next investigated its possible occurrence in Amphibia, a more primitive class of vertebrates. In *R. pipiens* the male is heterogametic as in mammals whereas in *X. laevis* the female is heterogametic as in birds (Table 1). Our serological tests (Fig. 1 and Table 2) demonstrate that cells from both these species contain a surface component identical or cross reactive with H-Y antigen of the mouse. Moreover, the antigen is found in the heterogametic sex of both these amphibian species. Thus it would seem that the cell surface component conferring H-Y antigenicity in the mouse has been conserved through some 300 Myr of evolution (spanning the Carboniferous radiation of amphibians and the Pleistocene emergence of man).

Although the phylogenetic conservation of a particular polypeptide sequence (indicated here by common antigenicity) suggests some essential or highly advantageous role, the reason for the conservation of H-Y antigen is not at all obvious. Nevertheless the association of H-Y antigen with either one sex or the other in those species tested so far seems to indicate a sex-related function of some kind, perhaps one concerned with the recognition of one sex by the other in nature (see discussion by Thomas[8]).

As to whether or not H-Y antigen expression is hormone dependent[9], several experiments involving male←→female transplantation and/or hormonal manipulation have given conflicting results[10–16]. Sex-limited, hormone-dependent traits affecting the cell surface are known however. Indeed the 'Hi' red blood cell agglutinogen of chickens is normally confined to females although this trait can be induced in males by treatment with diethylstilboesterol[17]. Thus it seems wise to leave open the question of the extent to which expression of H-Y antigen may be influenced quantitatively by sex hormones, although our finding that H-Y may occur in females implies that its expression is not qualitatively hormone-dependent. In this context it remains to be determined whether or not the *H-Y* structural locus is situated on the Y chromosome. An exceptional species in which H-Y antigen were found in the homogametic sex would suggest that the *H-Y* structural locus is autosomal (or even X-linked). We are now investigating what may prove to be such an exception.

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STEPHEN S. WACHTEL
GLORIA C. KOO
EDWARD A. BOYSE

Memorial Sloan-Kettering Cancer Center, New York, New York 10021

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Production of lymphoid tumours in hamsters by direct implantation of normal human leukocytes

LEUKOCYTES from patients with various lymphoproliferative diseases can be serially transplanted as malignant lymphoid tumours in immunosuppressed newborn hamsters[1]. We report that normal human leukocytes can also be transplanted into newborn hamsters treated with antilymphocyte serum.

Three human leukocyte samples were used; one from peripheral blood of an Epstein–Barr virus (EBV) antibody-positive normal male and two from umbilical cord blood with and without EBV infection. Leukocytes were separated by Dextran sedimentation from 20 ml of peripheral or cord blood and $0.5–1 \times 10^7$ viable leukocytes were implanted intraperitoneally into Syrian golden hamsters less than 24 h of age. This was followed by twice weekly intraperitoneal inoculation of 0.1 ml rabbit antilymphocyte serum against hamster thymocytes. The leukocytes from cord blood were incubated with 1 ml of a filtered freeze–thaw extract of the (EBV+) B95 cell line[2] at 37 °C for 2 h, before implantation.

All three hamsters transplanted with peripheral leuckocytes and two out of three hamsters transplanted with cord leukocytes infected with EBV were found to have lymphoid tumours involving the lymph nodes, liver, lungs and kidneys, when killed on days 11–21. No tumours were observed, however, in hamsters transplanted with cord leukocytes not infected with EBV.

Enlarged inguinal lymph nodes resulting from transplantation of peripheral leukocytes or EBV-infected cord leukocytes were cultured in medium RPMI 1640 supplemented with 20% foetal calf serum, and two lymphoblastoid cell lines were established from a hamster of each group. The cells began to grow vigorously in about 3–4 weeks and have been maintained for 6 months. These cell lines have a normal diploid human chromosome constitution and are positive for EBV nuclear antigen[3]. Electron microscopy revealed EBV particles in lymphoid cells.

Our observation seems to be analogous with the establishment of lymphoblastoid cell lines *in vitro* from normal human peripheral and cord leukocytes, for which an essential role of EBV has been demonstrated[4,5]. Successful heterotransplantation of normal human leukocytes into hamsters may be based on the same mechanism as in the *in vitro* system. It is possible that normal human lymphocytes can be transformed *in vivo* by EBV and grow progressively in the immunosuppressed heterologous hosts.

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ISAO MIYOSHI
ICHIRO KUBONISHI
TAKEHIKO HAYASHI
SHINJI ABE
HIROSHI UCHIDA
TERUHIKO TSUBOTA
TOSHIO TANAKA

Department of Medicine, Okayama University Medical School, Okayama, Japan

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