Friend murine leukemia virus (F-MuLV) induces in certain strains of newborn mice an acute erythroleukemia characterized by hepatosplenomegaly and severe anemia (1, 2). Mink cell focus-inducing (MCF) viruses, which are recombinants between ecotropic viruses and endogenous envelope sequences present in the mouse genome (for review, see reference 3), can be isolated from the spleens of these mice (4), and are thought to play a critical role in pathogenicity (5). Although strains of mice such as NIH Swiss and BALB/c are very susceptible to the development of early erythroleukemia after infection with F-MuLV, most other strains of mice, including DBA/2 and C57BL/6, are completely resistant (5–7). Resistance appears to be under control of at least one dominant gene, and is not correlated with the expression of known resistance genes such as Fv-1, Fv-2, or H-2-linked immune response genes (5, 6). Although F-MuLV was shown to replicate equally well in all strains, MCF viruses were found to replicate only in the spleens of mice from strains that are susceptible to F-MuLV-induced disease (5). Injection of pseudotypes of Friend MCF virus into various strains of mice results in the development of erythroleukemia only in strains susceptible to F-MuLV-induced disease (5). Thus, the resistance of certain strains of newborn mice to F-MuLV-induced disease appears to be due to the failure of MCF viruses to replicate and spread in these mice.

When tissues from various strains of uninfected animals were examined for the endogenous expression of viral envelope glycoproteins, it was found that resistant but not susceptible strains of mice endogenously express an MCF virus–related envelope glycoprotein that may be responsible for resistance by blocking receptors for MCF viruses (5). This hypothesis was strengthened by in vitro data indicating that MCF viruses fail to replicate in fibroblasts derived from resistant strains of mice, and that this block can be overcome by mechanisms known to overcome viral interference (8). Thus, certain strains of mice appear to be resistant to F-MuLV-induced erythroleukemia due to the constitutive expression of an MCF virus–related envelope glycoprotein that interferes with the replication and spread of MCF viruses. Hartley et al. (9) also observed that fibroblasts derived from certain strains of mice, including DBA/2, were not able to replicate the MCF virus.
MCF viruses, and they mapped the responsible gene, designated Rmcf*, to chromosome 5.

Although DBA/2 and other strains of mice are resistant to the development of early erythroleukemia when injected with F-MuLV, they can develop tumors (>4 mo after virus infection) that involve not only erythroid cells, but also cells of the myeloid and lymphoid lineages (6, 7, 10, 11). There is no evidence that replication of MCF viruses is necessary for the development of these late diseases.

To better understand the genetic basis for the differences in susceptibility of mice to F-MuLV-induced early erythroleukemia and the role of MCF viruses in this disease, we studied BALB/c and DBA/2 first generation backcross mice, as well as a series of BALB/c × DBA/2 (C × D2) congenic mice.

Materials and Methods

Animals and Leukemogenicity Assays. BALB/c AnPt mice (previously designated BALB/c π) were originally obtained from H. Andervont, NCI, in 1964. The DBA/2Pt mice were derived from DBA/2N mice. The partial C × D2 congenic stocks were originally developed to determine whether genetic markers of DBA/2 origin were linked to genes that control resistance to plasmacytomas induced by intraperitoneal injection of pristane. The methods used in deriving several of the stocks have been previously described: Pep-3* and Idh-1*/Pep-3* (12); Fv-1*, Rmcf*/Fv-1*/C, Rmcf*, and Qa2* (13). The Pgm-1*, Lyt-2*, C, C/a, C/d, Es-3*/Hba*, and Igβ-1* stocks were developed by introgressively backcrossing the respective DBA/2-derived markers onto BALB/c AnPt. The phenotypes were detected by the appropriate enzymatic assays, using starch gel electrophoretic separations, or cytotoxic immunoassays. At the designated backcross generation (n), mice were mated to each other, and homozygotes for the respective markers were used to found the stock. These mice are C × D2 partial congenic stocks, and are not considered to be true congenic mice, because the number of backcrosses is <14.

All of the mice were bred under conventional conditions at an NCI contract facility (contract N01-CB-25584 to Litton Bionetics). Mice were infected within 72 h of birth by intraperitoneal injection of 5 × 10⁵ XC-plaque-forming units of a stock of F-MuLV. Animals were palpated at intervals, and those judged to have enlarged spleens were killed. Data from some of the animals was obtained at autopsy. Mice were considered positive for disease if the spleen weight was >0.5 grams and the hematocrit was <30%.

Viruses and Cells. The F-MuLV used in this study was the molecularly cloned stock of F-MuLV clone 201 that has been previously described (14). Cell-free homogenates (10%) of splenic tissue were prepared in Dulbecco-Vogt medium containing 10% fetal calf serum and clarified by centrifugation at 2,000 rpm for 10 min. NIH 3T3 cells were then infected with various dilutions of each homogenate, and the supernatants of the infected cells were monitored for virus production by the reverse transcriptase assay.

Pulse-labeling, Immune Precipitation and Polyacrylamide Gel Electrophoresis (PAGE) Virus-infected fibroblasts or uninfected bone marrow cells were pulse-labeled with [35S]-methionine as previously described (15). Extracts of labeled cells were immune precipitated with either goat anti-Rauscher MuLV gp70 (glycoprotein) serum (obtained from the Division of Cancer Cause and Prevention, NCI), goat anti-Moloney MCF gp70–specific serum (15), or normal goat serum. The proteins precipitated were visualized by 7% sodium dodecyl sulfate (SDS)-PAGE.

Results

Occurrence of F-MuLV-induced Disease in BALB/c Mice Carrying Various DBA/2 Genes. BALB/c mice carrying various genes from the resistant DBA/2 mouse were injected as newborns with F-MuLV and observed for the development of early erythroleukemia (within 10 wk), as well as late erythroid and nonerythroid
disease (Table I). When resistant (BALB/c × DBA/2)F₁ hybrid mice are backcrossed to the susceptible BALB/c strain, 36% of the mice develop early erythroid disease. A similar incidence of early erythroleukemia was noted when resistant (NFS × DBA/2)F₁ hybrid mice were backcrossed to the susceptible NFS mouse. These data suggest that more than one gene is involved in resistance of DBA/2 mice to F-MuLV-induced early erythroid disease.

To map the resistance genes in the DBA/2 mouse, we used a series of BALB/c mice partially congenic for a variety of DBA/2 genes. As shown in Table I, BALB/c mice congenic for DBA/2 genes on chromosomes 1, 4-7, 9, 12, and 17 are indistinguishable from BALB/c mice in their susceptibility to early F-MuLV-induced erythroid disease. However, BALB/c mice carrying the *Rmcf<sup>a</sup>* gene (chromosome 5) of the DBA/2 mouse are extremely resistant to the development of early F-MuLV-induced erythroid disease. C × D<sub>2</sub> Pgm-<sup>1b</sup> mice, which carry another marker on chromosome 5 (Pgm-<sup>1a</sup>), remain highly susceptible. BALB/c mice carrying chromosome 11 (indicated by the DBA/2 Es-3 and Hba loci) or chromosome 2 (indicated by the DBA/2 agouti locus) also have a significantly reduced incidence of early disease. The data also indicates that mice carrying the *Rmcf<sup>a</sup>* gene are not resistant to the development of late, multiple-lineage tumors induced by F-MuLV. This indicates that other genes control susceptibility to these diseases, as suggested by earlier studies with DBA/2 and C57BL mice (6, 7, 10, 11). Interestingly, the incidence of late malignancies increases as *Rmcf<sup>a</sup>*

### Table I

**F-MuLV-induced Disease in BALB/c Mice Carrying Various DBA/2 Genes**

| Strain                  | Marker locus | DBA/2 chromosome | Incidence of disease<sup>a</sup> |
|------------------------|--------------|------------------|----------------------------------|
|                        |              |                  | 4-10 wk | 11-16 wk | 16-24 wk | Total  |
| Parental               |              |                  |         |          |          |        |
| BALB/c                 |              |                  | 90 (19/21) | 0 (0/19) | 16 (3/19) | 79     |
| DBA/2                  |              |                  |          |          |          |        |
| Backcross              |              |                  | 36 (20/56) | 34 (19/56) | 9 (5/56) | 77     |
| (BALB/c × DBA/2)F₁ × BALB/c |        |                  | 55 (7/20) | 40 (8/20) |          | 72     |
| (NFS × DBA/2)F₁ × NFS  |              |                  |          |          |          |        |
| C × D<sub>2</sub> congenics |        |                  |          |          |          |        |
| 9                      | Pgp-<sup>3b</sup> | 1 | 75 (9/12) | 25 (3/12) |          | 100    |
| 6                      | Idh-<sup>1b</sup>/Pep-<sup>3b</sup> | 1 | 85 (11/15) | 15 (2/15) |          | 100    |
| 6, 10                  | Fe-<sup>1a</sup> | 4 | 70 (21/30) | 23 (7/30) |          | 93     |
| 5                      | Rmcf<sup>a</sup>/Fe-<sup>1a</sup>/Pgp-<sup>1a</sup> | 5, 4, 7 | 7 (3/41) | 59 (24/41) | 7 (3/41) | 73     |
| 5                      | Rmcf<sup>a</sup> | 5 | 0 (0/101) | 7 (7/101) | 11 (11/101) | 18     |
| 9                      | Rmcf<sup>a</sup> | 5 | 0 (0/21) | 29 (6/21) | 19 (4/21) | 48     |
| 6, 10                  | Pgp-<sup>1a</sup> | 5 | 94 (35/56) |          |          | 97     |
| 5                      | Lyt-<sup>2a</sup> | 6 | 80 (12/14) |          |          | 86     |
| 10                     | C            | 7 | 77 (30/39) | 15 (6/39) |          | 92     |
| 6                      | C<sub>17</sub> | 7, 2 | 27 (4/15) | 33 (5/15) |          | 60     |
| 6                      | C<sub>17</sub> | 7, 9 | 86 (12/14) | 14 (2/14) |          | 100    |
| 6                      | Es-<sup>1b</sup>/Hba<sup>a</sup> | 11 | 22 (10/46) | 55 (15/46) | 26 (12/46) | 83     |
| 6                      | Ig<sub>1b</sub> | 12 | 60 (9/15) | 27 (4/15) |          | 87     |
| 6, 10                  | Qa<sub>2</sub><sup>a</sup> | 17 | 62 (16/26) | 38 (10/26) |          | 100    |

<sup>a</sup> Number of generations backcrossed to BALB/c.

<sup>b</sup> Animals were palpated at the indicated intervals, and those judged to have enlarged spleens were killed. Data from some animals were obtained at autopsy. The disease occurring in mice within 10 wk after virus infection was always an erythroleukemia associated with splenomegaly and anemia. The diseases developing later than 10 wk after virus infection were all associated with splenomegaly, but did not exclusively affect the erythroid lineage. Data given as percent, with numbers of diseased mice and numbers of mice in test sample in parentheses.

<sup>c</sup> Designated Fe-<sup>1b</sup> at N6.
congenic mice are backcrossed further to BALB/c (see N5 vs. N9), suggesting that BALB/c genes may favor the development of these late F-MuLV-induced diseases.

Expression of Viral Proteins in C × D2 Congenic Mice Infected with F-MuLV. Since the replication of MCF viruses is believed necessary for the development of the early erythroleukemia induced by F-MuLV, we examined the spleens from F-MuLV-infected mice for replication of both the input F-MuLV and the generated MCF virus. This was carried out by preparing cell-free spleen homogenates from infected mice, passing onto NIH 3T3 fibroblasts, and then examining the fibroblasts for expression of viral proteins by pulse-labeling, immune precipitation, and SDS-PAGE. As previously shown (5, 15), the envelope proteins of F-MuLV and F-MCF virus can be distinguished by size as well as by precipitation with an MCF gp70–specific antiserum. As shown in Fig. 1 and summarized in Table II, NIH 3T3 cells infected with cell-free spleen homogenates from all mice tested express high levels of the F-MuLV envelope precursor, gPr85
env.

The MCF viral envelope precursor, gPr80 env, was also expressed in cells infected with the spleen homogenates from all mice susceptible to early erythroleukemia (Fig. 1, A–F, and J) but was not expressed in cells infected with spleen homogenates from Rmcf
env mice (Fig. 1, G–I), indicating that MCF viruses were not replicating in the latter mice. This is consistent with previous data indicating that the Rmcf
env gene restricts the replication of MCF viruses (9). Thus, BALB/c mice that are congenic for the Rmcf
env gene and fail to replicate MCF viruses are resistant to the early erythroleukemia induced by F-MuLV. Failure to replicate MCF viruses, however, is not the only cause of resistance, since mice carrying
TABLE II

Infectious MuLV in Cell-free Spleen Homogenates From F-MuLV-infected Mice

| Strain         | Marker locus | DBA/2 chromosome | Early erythroleukemia | Viral proteins expressed in NIH 3T3 cells infected with spleen homogenates from infected mice* |
|----------------|--------------|------------------|-----------------------|----------------------------------------------------------------------------------|
| Parental       |              |                  |                       |                                                                                   |
| BA1B/c         |              | +                | +                     | +                                                                                 |
| DBA/2          |              | -                | -                     | -                                                                                 |
| C x D2 congenics |             |                  |                       |                                                                                   |
| *Pep-3         | 1            | +                | +                     | +                                                                                 |
| *1dh-1*/Pep-3  | 1            | +                | +                     | +                                                                                 |
| *1Tv-l**       | 4            | +                | +                     | +                                                                                 |
| *Rmcf*/1Tv-l*/C | 5, 4, 7      | -                | +                     | -                                                                                 |
| *Rmcf*(N5)     | 5            | -                | +                     | -                                                                                 |
| *Rmcf*(N9)     | 5            | -                | +                     | -                                                                                 |
| *Rmcf*(N5)     | 5            | -                | -                     | -                                                                                 |
| *Pgm-1*        | 5            | +                | +                     | +                                                                                 |
| *Lyt-2*        | 6            | +                | +                     | +                                                                                 |
| *C             | 7            | +                | +                     | +                                                                                 |
| *C/a           | 7, 2         | +/-              | +                     | +                                                                                 |
| *C/d           | 7, 9         | +                | +                     | +                                                                                 |
| *Es-3*/Hba     | 11           | +/-              | +                     | +                                                                                 |
| *Igh-1*        | 12           | +                | +                     | +                                                                                 |
| *Qu2*          | 17           | +                | +                     | +                                                                                 |

* Cell-free spleen homogenates from diseased mice were passaged onto NIH 3T3 cells, and protein expression was determined by pulse-labeling, immune precipitation, and SDS-PAGE, as previously described (15).

the DBA/2 Es-3/Hba (Fig. 1 F) and C/a (E) loci have no defect in their ability to replicate F-MuLV or MCF viruses, yet they have a considerably reduced incidence of early erythroleukemia. Thus, other DBA/2 genes in these mice must be controlling the development of erythroleukemia by a different mechanism.

Analysis of Cells from Uninfected Mice for Viral Protein Expression. Normal cells from DBA/2 mice have previously been shown to constitutively express an MCF virus–related envelope glycoprotein that cannot be detected in cells from susceptible strains such as BALB/c (5). This protein is thought to bind to the receptor for MCF viruses and block incoming MCF viruses from infecting the cell (8). To determine whether C x D2 congenic mice carrying the Rmcf* gene also constitutively express an MCF-related envelope protein, bone marrow cells from uninfected mice were examined. As shown in Fig. 2, bone marrow cells from C x D2 mice carrying the Rmcf* gene express an 80 kilodalton (kD) protein that is precipitable with an MCF gp70-specific antiserum (Fig. 2A). No such protein could be detected in cells from other congenic mice examined (Fig. 2, B–D), all of which replicate MCF viruses well.
FIGURE 2. Expression of envelope proteins in bone marrow cells from uninfected C × D~ congeneric mice. Bone marrow cells were removed from the femurs of uninfected C × D~ congeneric mice carrying the following DBA/2 markers: Rmcf" (A), Pgm-1^b (B), C/a (C), and Es-3//Hba~ (D). The cells were labeled for 2 h with [35S]methionine, and the labeled extracts were immune precipitated with an MCF gp70-specific goat antiserum (lanes 1) or normal goat serum (lanes 2). Precipitated proteins were visualized by 7% SDS-PAGE and autoradiography.

Discussion

The studies reported here establish that DBA/2 mice carry a gene on chromosome 5 at or near the Rmcf locus that plays a major role in resistance to early F-MuLV-induced erythroleukemia. The fact that this gene controls the replication of MCF viruses (9) strengthens the case for these viruses playing a crucial role in the disease, since failure to replicate these viruses results in resistance to early erythroleukemia (5 and this study). The data also indicate that additional genes on other chromosomes may contribute to resistance to F-MuLV-induced early erythroleukemia, although their mechanisms are unknown.

Normal cells from DBA/2 mice have previously been shown (5) to constitutively express an MCF-related envelope glycoprotein on the cell surface that cannot be detected on the surface of cells from susceptible strains such as BALB/c. This protein apparently binds to the receptor for MCF viruses and blocks incoming MCF viruses from infecting the cell (8). Cells from BALB/c mice congenic for the Rmcf" gene also express an MCF-related envelope glycoprotein, which is most likely responsible for the failure of MCF viruses to replicate in this strain. No such protein could be detected on the surface of cells from the other congenic mice studied, all of which replicate MCF viruses well. Thus, the Rmcf" gene may represent a unique viral envelope gene, or may be a regulatory locus that governs the expression of this envelope gene. It is not known whether the BALB/c mouse completely lacks the Rmcf gene or carries a different allele of this gene (Rmcf") that is not expressed.

There are other examples of resistance to retrovirus-induced diseases being associated with expression of endogenous envelope genes. In the mouse, replication of ecotropic virus is blocked in mice carrying the Fv-4' allele, which is associated with the constitutive expression of an ecotropic viral envelope gene whose product blocks the replication of ecotropic viruses by viral interference
Also, chickens expressing defective, endogenous avian leukosis viral subgroup E envelope genes were shown to be resistant to infection with subgroup E viruses (19).

While it is clear that the \textit{Rmcf}\textsuperscript{+} gene plays a major role in resistance of DBA/2 mice to early F-MuLV-induced erythroleukemia, the basis for resistance in C57BL mice is unclear, and may be under control of yet another resistance gene (10, 11). These mice do not carry the \textit{Rmcf}\textsuperscript{+} gene (9), yet MCF viruses cannot be detected after infection with F-MuLV. Endogenous MCF virus-related envelope glycoproteins can be detected in cells derived from C57BL mice (5), but they are apparently not the same as the protein expressed in \textit{Rmcf}\textsuperscript{+} mice that blocks the receptor for MCF viruses. Perhaps C57BL mice carry a genetic defect that prevents the generation of MCF viruses or affects the number of target cells available for MCF virus infection.

Since the \textit{Rmcf}\textsuperscript{+} gene controls the replication of MCF viruses, its presence should be associated with resistance to all leukemias that are mediated by MCF viruses. The incidence of spontaneous and MCF virus-induced lymphomas in AKR mice was shown (20, 21) to be greatly reduced when the mice were crossed to DBA/2, which carries the \textit{Rmcf}\textsuperscript{+} gene. On the other hand, the \textit{Rmcf}\textsuperscript{+} gene had no effect on the incidence of pristane-induced plasmacytomas in BALB/c mice (13), indicating that MCF viruses play no role in this disease.

Future studies on the \textit{Rmcf}\textsuperscript{+} gene will focus on cloning the gene to determine whether it represents a structural gene for a unique envelope glycoprotein or a regulatory gene that controls expression of this envelope gene.

\textbf{Summary}

Using a series of BALB/c mice congenic for various DBA/2 genes, we were able to establish that DBA/2 mice carry a gene on chromosome 5, at or near the \textit{Rmcf}\textsuperscript{+} locus, that plays a major role in resistance to early erythroleukemia induced by injection of Friend murine leukemia virus (F-MuLV) into newborn mice. The fact that this gene controls the replication of mink cell focus–inducing (MCF) viruses strengthens the case for these viruses playing a crucial role in the development of erythroleukemia, since failure to replicate MCF viruses results in resistance to early erythroleukemia. The expression of the \textit{Rmcf}\textsuperscript{+} gene is correlated with the constitutive expression of an MCF virus–related envelope glycoprotein that apparently blocks the receptor for MCF viruses, preventing their spread. Thus, the \textit{Rmcf}\textsuperscript{+} gene is either a structural gene for this unique protein, which can block the receptor for MCF viruses, or is a regulatory gene that controls expression of such a structural gene. Although the \textit{Rmcf}\textsuperscript{+} gene is clearly involved in resistance to the early erythroleukemia induced by F-MuLV, it appears to have no effect on the late myeloid, lymphoid or erythroid diseases that appear in DBA/2 and other strains of mice after injection of F-MuLV, consistent with data indicating that replication of MCF viruses is not required for the development of these late diseases. Our studies with congenic and backcross mice also indicate that, in addition to the \textit{Rmcf}\textsuperscript{+} gene, other genes of DBA/2 origin may contribute to resistance to F-MuLV-induced early erythroleukemia by mechanisms other than blocking the replication of MCF viruses.
We thank J. Wax for her expert technical assistance and advice in the development of the congenic strains used in this study, and J. Hoffman for technical assistance with other aspects of this work.

Received for publication 5 July 1985.

References

1. Troxler, D. H., and E. M. Scolnick. 1978. Rapid leukemia induced by cloned strain of replicating murine type-C virus: Association with induction of xenotropic-related RNA sequences contained in spleen focus-forming virus. Virology. 85:17.

2. Troxler, D. H., S. K. Ruscetti, D. L. Linemeyer, and E. M. Scolnick. 1980. Helper-independent and replication-defective erythroblastosis-inducing viruses contained within anemia-inducing Friend virus complex (FV-A). Virology. 102:28.

3. Famulari, N. G. 1983. Murine leukemia viruses with recombinant *env* genes: a discussion of their role in leukemogenesis. Curr. Top. Microbiol. Immunol. 110:76.

4. Troxler, D. H., E. Yuan, D. Linemeyer, S. Ruscetti, and E. M. Scolnick. 1978. Helper-independent mink cell focus-inducing strains of Friend murine type-C virus: Potential relationship to the origin of replication defective spleen focus-forming virus. J. Exp. Med. 148:639.

5. Ruscetti, S., L. Davis, J. Feild, and A. Oliff. 1981. Friend murine leukemia virus-induced leukemia is associated with the formation of mink cell focus-inducing viruses and is blocked in mice expressing endogenous mink cell focus-inducing xenotropic viral envelope genes. J. Exp. Med. 154:907.

6. Shibuya, T., and T. W. Mak. 1982. Host control of susceptibility to erythroleukemia and to the types of leukemia induced by Friend murine leukemia virus: Initial and late stages. Cell. 31:483.

7. Chesebro, B., J. L. Portis, K. Wehrly, and J. Nishio. 1983. Effect of murine host genotype on MCF virus expression, latency, and leukemia cell type of leukemias induced by Friend murine leukemia helper virus. Virology. 128:221.

8. Bassin, R. H., S. Ruscetti, I. Ali, D. K. Haapala, and A. Rein. 1982. Normal DBA/2 mouse cells synthesize a glycoprotein which interferes with MCF virus infection. Virology. 123:139.

9. Hartley, J. W., R. A. Yetter, and H. C. Morse. 1983. A mouse gene on chromosome 5 that restricts infectivity of mink cell focus-forming recombinant murine leukemia viruses. J. Exp. Med. 158:16.

10. Silver, J. E., and T. N. Fredrickson. 1983. A new gene that controls the type of leukemia induced by Friend murine leukemia virus. J. Exp. Med. 158:493.

11. Silver, J. E., and T. N. Fredrickson. 1983. Susceptibility to Friend helper virus leukemias in C × B recombinant inbred mice. J. Exp. Med. 158:1693.

12. Potter, M., A. D. O'Brien, E. Skamene, P. Gros, A. Forget, P. A. L. Kongshavn, and J. S. Wax. 1983. A BALB/c congenic strain of mice that carries a genetic locus ('Ity') controlling resistance to intracellular parasites. Infect. Immun. 40:1234.

13. Potter, M., J. W. Hartley, J. S. Wax, and D. Gallahan. 1984. Effect of MuLV-related genes on plasmacytogenesis in BALB/c mice. J. Exp. Med. 160:435.

14. Oliff, A. I., G. L. Hager, E. Chang, E. M. Scolnick, H. W. Chan, and D. R. Lowy. 1980. Transfection of molecularly cloned Friend murine leukemia virus DNA yields a highly leukemogenic helper-independent type-C virus. J. Virol. 33:475.

15. Ruscetti, S., D. Linemeyer, J. Feild, D. Troxler, and E. M. Scolnick. 1979. Characterization of a protein found in cells infected with the spleen focus-forming virus that shares immunological cross-reactivity with the gp70 found in mink cell focus-inducing virus particles. J. Virol. 30:787.
16. Ikeda, H., and T. Odaka. 1983. Cellular expression of murine leukemia virus gp70-related antigen on thymocytes of uninfected mice correlates with Fv-4 gene-controlled resistance to Friend leukemia virus infection. Virology. 128:127.

17. Ikeda, H., and T. Odaka. 1984. A cell membrane "gp70" associated with Fv-4 gene: Immunological characterization, and tissue and strain distribution. Virology. 133:65.

18. Kozak, C. A., N. J. Gromet, H. Ikeda, and C. E. Buckler. 1984. A unique sequence related to the ecotropic murine leukemia virus is associated with the Fv-4 resistance gene. Proc. Natl. Acad. Sci. USA. 81:834.

19. Robinson, H. L., S. M. Astrin, A. M. Senior, and F. H. Salazar. 1981. Host susceptibility to endogenous viruses: Defective, glycoprotein-expressing proviruses interfere with infections. J. Virol. 40:745.

20. Chen, S., and F. Lilly. 1982. Suppression of spontaneous lymphoma by previously undiscovered dominant genes in crosses of high- and low-incidence mouse strains. Virology. 118:76.

21. Rowe, W. P., and J. W. Hartley. 1983. Genes affecting mink cell focus-inducing (MCF) murine leukemia virus infection and spontaneous lymphoma in AKR F1 hybrids. J. Exp. Med. 158:353.