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Cross-resistance to the Transplantation of Syngeneic Friend, Moloney, and Rauscher Virus-induced Tumors

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SUMMARY

Immunofluorescence data are presented which confirm the previously reported cytotoxic antibody studies suggesting that Friend, Moloney, and Rauscher (FMR) virus-induced lymphomas possess similar surface antigens, not shared by Gross lymphomas. It is not known, however, whether the antigenic relationship demonstrable serologically is, or should necessarily be, demonstrable by transplantation studies. To determine if tumors induced by FMR and Gross virus share transplantation antigens, C57BL/6 mice were pretreated with either infectious FMR viruses, FMR lymphomas, or a Gross lymphoma and challenged with syngeneic FMR and Gross lymphomas. Mice pretreated with any one infectious FMR virus or its induced lymphoma became resistant to the transplantation of all FMR lymphomas, but not to the Gross lymphoma. Immunization with the Gross lymphoma failed to induce resistance to the transplantation of the FMR lymphomas, but also failed to induce resistance to itself. The results strongly suggest that FMR lymphomas possess related or identical transplantation antigens.

INTRODUCTION

Friend, Moloney, and Rauscher (FMR) virus-induced lymphomas are antigenic to syngeneic mice (1, 4, 6, 9, 10, 12). Whether the critical immunogen for the induction of immunity is viral or cellular is not known, since all FMR lymphoma cells release infectious virus (7, 9). Using viable or X-irradiated syngeneic FMR lymphoma cells as immunogens, serologic and transplantation studies have demonstrated that lymphomas induced by a given FMR virus possess common antigens (6, 9, 12). In addition, serologic studies by Old *et al.* (12), using murine antisera prepared against syngeneic FMR cells, have shown that FMR lymphoma cells are antigenically indistinguishable from each other, but may be distinct from tumors induced by Gross virus. However, it cannot be assumed that the antigens responsible for serologic immunity are necessarily the ones operative in the induction of transplantation resistance. Although transplantation studies involving immunization with tumor cells suggest that tumors induced by Moloney leukemia virus share antigens with tumors induced by Rauscher virus (1), but not with those induced by Gross virus (6), a complete cross-resistance study involving FMR and Gross lymphomas has not been reported. Similarly, although infectious FMR viruses readily induce resistance to the transplantation of their homologous FMR lymphoma cells (9, 10, 14), it is not known whether one FMR virus can induce resistance to the transplantation of tumors induced by the other FMR viruses.

This investigation was conducted to determine whether any one FMR virus or its induced tumor can render the host resistant to the transplantation of tumors induced by the other FMR viruses or by Gross virus. The transplantation studies were also serologically supported by immunofluorescence tests.

MATERIALS AND METHODS

Mice. Adult C57BL/6 mice, 7–12 weeks of age, were obtained from the production colonies of Microbiological Associates, Inc.

Tumors. The basic data on the transplantable tumors are presented in Table 1. The FMR lymphomas were induced by inoculation of FMR viruses into C57BL/6 newborn mice. The Gross leukemia, E ♂ G-2 (13), was obtained from Dr. Lloyd J. Old. The tumors were maintained by serial transplantation at weekly or biweekly intervals.

Preparation of Cell Suspensions. Solid lymphomas were forced through an 18 gauge needle into 2 volumes of Hank's balanced salt solution (HBSS). Uniform cell suspensions were obtained by filtering the brei through 4–6 layers of cotton gauze. Ascites lines were prepared by serially diluting ascites fluid in HBSS. Cells were counted after dilution with trypan blue and the cell concentration was adjusted in HBSS.

Table 1

Designation	Etiology	Genotype	Form	No. of transplant generations prior to use
MBL-1	Moloney Virus	C57BL/6	Solid	32
MBL-2	Moloney Virus	C57BL/6	Ascites	4
RBL-3	Rauscher Virus	C57BL/6	Ascites	25
RBL-4	Rauscher Virus	C57BL/6	Ascites	16
FBL-1	Friend Virus	C57BL/6	Ascites	10
FBL-3	Friend Virus	C57BL/6	Ascites	15
LSTRA	Moloney Virus	BALB/c	Ascites	320
E ♂ G-2	Gross Virus	C57BL/6	Splenic	48
EL-4	7,12-Dimethyl-benzanthracene	C57BL/6	Ascites	187

Basic data on the transplantable lymphomas used in this study.

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Virus. Friend and Moloney viruses were obtained from Dr. Moloney. The Friend virus (Lot No. FV-5) was a one-gram-equivalent preparation from spleens of BALB/c mice infected with Friend virus. The Moloney virus (Lot No. 3030-238) consisted of a 10 gram equivalent preparation from plasma of Moloney virus-infected BALB/c mice. Rauscher virus (Lot No. P-972) was prepared as a 10% suspension from spleens of BALB/c mice infected with Rauscher virus.

X-irradiation. Tumor cells were X-irradiated with 5000 R under the conditions previously described (4).

Immunization. Mice received 2 injections of X-irradiated syngeneic FMR or Gross leukemia cells (3×10^5 to 4×10^6 cells/mouse) i.p., two weeks apart. They were challenged s.c. with 10-fold dilutions of viable syngeneic tumor cells 2 weeks later. Immunization with infectious FMR viruses consisted of a single i.p. injection of 0.1 ml of a 10^{-1} dilution of each virus followed by tumor challenge eight weeks later.

Preparation of Antisera. Specific anti-Moloney, anti-Rauscher, and anti-Friend sera were obtained by i.p. inoculation of adult C57BL/6 mice with X-irradiated histocompatible FMR leukemia cells, followed by progressively increasing doses of viable cells s.c. The mice were bled from the retroorbital sinus 7–10 days after each immunization. The anti-FMR sera used were obtained after 6–12 immunizations with viable tumor cells. The sera were distributed into 0.5 ml aliquots and stored at less than −70°C.

The Indirect Fluorescent Antibody Technic. The technic developed by Möller (11) using suspensions of viable cells was employed; 2×10^6 trypan blue-unstained cells in a volume of 0.05 ml of HBSS were added to 0.05 ml of undiluted serum or to serum serially diluted in HBSS. Following a 20-minute incubation at 37°C, the cells were washed four times, and the resulting pellet was mixed and incubated for 20 minutes at 37°C with 0.05 ml of fluorescein-conjugated goat anti-mouse globulin (Hyland 2-18-66) diluted 1:5. After four more washings, the cells were examined under the fluorescence microscope. Samples were read blind; 100–180 cells per sample were counted. Cells manifesting diffuse fluorescence of the type that Möller has demonstrated to be indicative of dead cells were omitted from calculations. This was not a significant problem as the viability of our cell preparations usually was greater than 90%. Viable cells were counted and classified as stained or unstained. All cells exhibiting bright green granular or sectorial fluorescence, or any other staining pattern except that characteristic of dead cells, were counted as positive. The result of each sample, therefore, was expressed as the number of stained viable cells × 100, divided by the total number of viable cells examined.

Absorption of Sera. Tumor cells were prepared as described above, washed three times, and centrifuged at 1800 rpm (4°C) for 10 minutes. Packed cells were mixed with an equal volume of suitably diluted serum. The mixture was incubated at room temperature for 45 minutes and then at 4°C for 45 minutes. The unabsorbed serum control was kept at the same temperature for the same lengths of time. After incubation, the mixtures were centrifuged at 1800 rpm at 4°C for 10 minutes. The absorbed and unabsorbed sera were tested by the indirect fluorescent antibody technic.

RESULTS

Cross-resistance among Lymphomas Induced by a Given FMR Virus. To determine whether the C57BL/6 FMR lymphomas used in this study were capable of inducing transplantation immunity and responding to an induced resistance, a given FMR lymphoma was used as the immunogen for the induction of transplantation resistance to itself as well as to

Table 2

Tumor challenge	Cell dose	Takes/total: Untreated mice	Takes/total: Treated mice	X^2	P
FBL-3	5×10^5	7/10	0/10		
	5×10^4	4/10	0/10		
	5×10^3	7/10	0/10		
	Total	18/30	0/30	22.2	<0.001
FBL-1	1×10^6	10/10	10/10		
	1×10^5	10/10	4/10		
	1×10^4	9/10	1/10		
	Total	29/30	15/30	17.4	<0.001
E ♂ Gross	1×10^4	10/10	10/10		
	1×10^3	6/10	7/10		
	1×10^2	0/10	0/10		
	Total	16/30	17/30		
EL-4	1×10^5	10/10	10/10		
	1×10^4	5/10	10/10		
	1×10^3	6/10	6/10		
	Total	21/30	26/30		

Resistance to the transplantation of syngeneic Friend lymphomas, a Gross lymphoma, and a chemically-induced lymphoma in C57BL/6 mice immunized with an X-irradiated Friend lymphoma (FBL-3).

Table 3

Tumor challenge	Cell dose	Takes/total: Untreated mice	Takes/total: Treated mice	X^2	P
MBL-1	3×10^5	8/8	0/8		
	3×10^4	7/8	0/8		
	3×10^3	1/8	0/8		
	Total	16/24	0/24	24.1	<0.001
MBL-2	3×10^5	7/8	1/8		
	3×10^4	4/8	0/8		
	3×10^3	1/8	0/8		
	Total	12/24	1/24	11.8	<0.001
E ♂ Gross	1×10^4	8/8	7/8		
	1×10^3	7/8	6/8		
	1×10^2	1/8	0/8		
	Total	16/24	13/24	1.15	<0.100
EL-4	1×10^5	8/8	8/8		
	1×10^4	7/8	8/8		
	1×10^3	0/8	4/8		
	Total	15/24	20/24		

Resistance to the transplantation of syngeneic Moloney lymphomas, a Gross lymphoma, and a chemically-induced lymphoma in C57BL/6 mice immunized with an X-irradiated Moloney lymphoma (MBL-1).

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Table 4

Tumor challenge	Cell dose	Takes/total: Untreated mice	Takes/total: Treated mice	X^2	P
RBL-3	4×10^6	7/7	1/7		
	4×10^5	7/7	0/7		
	4×10^4	6/7	0/7		
	Total	20/21	1/21	43.2	<0.001
RBL-4	4×10^6	5/7	0/7		
	4×10^5	3/7	0/7		
	4×10^4	1/7	0/7		
	Total	9/21	0/21	9.15	<0.005
E ♂ Gross	1×10^4	7/7	7/7		
	1×10^3	2/7	3/7		
	1×10^2	0/7	0/7		
	Total	9/21	10/21		
EL-4	1×10^5	7/7	7/7		
	1×10^4	7/7	7/7		
	1×10^3	5/7	5/7		
	Total	19/21	19/21		

Resistance to the transplantation of syngeneic Rauscher lymphomas, a Gross lymphoma, and a chemically-induced lymphoma in C57BL/6 mice immunized with an X-irradiated Rauscher lymphoma (RBL-3).

another syngeneic lymphoma induced by the same virus. Syngeneic lymphomas induced by Gross virus or by a chemical were also used to challenge mice pretreated with the various FMR lymphomas. The results presented in Table 2–4 show that a given FMR virus-induced lymphoma is capable of inducing resistance to itself and to another tumor induced by the same virus, but not to a Gross or chemically-induced lymphoma.

Cross-resistance among FMR Lymphomas Induced by Friend, Moloney, and Rauscher Viruses. To determine whether tumors induced by FMR viruses share transplantation antigens, and whether Gross lymphomas have transplantation antigens in common with FMR lymphomas, C57BL/6 mice were immunized with infectious FMR viruses, X-irradiated FMR, or Gross cells, and were challenged with graded doses of syngeneic Friend (Table 5), Moloney (Table 6), Rauscher (Table 7), or Gross (Table 8) lymphomas. FMR viruses or tumors induced resistance to the transplantation of all FMR lymphomas but not to a Gross lymphoma. Pretreatment with Gross cells failed to induce resistance to the transplantation of either FMR or Gross lymphomas.

Serologic Cross-reactivity of Anti-FMR Sera with FMR Cells as Detected by Immunofluorescence. Serially diluted serum from C57BL/6 mice immunized with lymphomas in-

Table 5

Type of immunization	Tumor takes in normal mice (Cells × 10^{-3}/mouse): 500	50	5	Tumor takes in immunized mice (Cells × 10^{-3}/mouse): 500	50	5	Sum of takes/total: Control	Immunized	X^2	P
MBL-1	10/10[a]	10/10	7/10	7/10	4/10	2/10	27/30	13/30	13.7	<0.001
RBL-3	7/7	7/7	6/7	0/7	0/7	0/7	20/21	0/21	31.0	<0.005
FBL-3	9/10	7/10	5/10	0/10	0/10	0/10	21/30	0/30	25.0	<0.001
E ♂ G-2	8/8	7/8	2/8	5/8	4/8	6/7	17/24	15/23		
MV	10/10	8/10	9/10	5/10	4/10	1/9	27/30	10/29	13.9	<0.001
RV	9/10	8/10	8/10	4/10	4/10	2/10	25/30	10/30	22.7	<0.001
FV	4/5	5/5	5/5	0/5	0/5	0/5	14/15	0/15	20.5	<0.001

Resistance to the transplantation of a syngeneic Friend lymphoma in C57BL/6 mice pretreated with Friend, Moloney, and Rauscher viruses or lymphomas.

[a] Tumor takes/total inoculated with viable FBL-3 cells.

Table 6

Type of immunization	Tumor takes in normal mice (Cells × 10^{-3}/mouse): 300	30	3	Tumor takes in immunized mice (Cells × 10^{-3}/mouse): 300	30	3	Sum of takes/total: Control	Immunized	X^2	P
MBL-1	10/10[a]	9/10	6/10	2/10	1/10	2/10	25/30	5/30	24.5	<0.001
RBL-3	7/7	7/7	5/6	2/6	3/7	3/6	19/20	8/19	9.7	<0.005
FBL-3	10/10	9/10	9/10	0/10	0/10	0/10	28/30	0/30	46.4	<0.001
E ♂ G-2	8/8	8/8	6/8	8/8	8/8	8/8	22/24	24/24		
MV	7/10	9/10	8/10	5/10	0/10	0/10	24/30	5/30	21.1	<0.001
RV	10/10	9/10	9/10	4/9	8/9	4/10	28/30	16/28	13.5	<0.001
FV	4/5	4/5	5/5	0/5	0/5	0/5	13/15	0/15	18.7	<0.001

Resistance to the transplantation of a syngeneic Moloney lymphoma in C57BL/6 mice pretreated with Friend, Moloney, and Rauscher viruses or lymphomas.

[a] Tumor takes/total inoculated with viable MBL-1 cells.

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Table 7

Type of immunization	Tumor takes in normal mice (Cells × 10^{-4}/mouse)			Tumor takes in immunized mice (Cells × 10^{-4}/mouse)			Sum of takes/total		X^2	P
	400	40	4	400	40	4	Control	Immunized		
MBL-1	9/10[a]	7/10	8/10	3/10	1/10	1/10	24/30	5/30	23.7	<0.001
RBL-3	7/7	7/7	6/7	1/7	0/7	0/7	20/21	1/21	29.0	<0.001
FBL-3	10/10	6/10	0/10	0/10	0/10	0/10	16/30	0/30	23.2	<0.001
E ♂ G-2	6/6	8/8	6/8	10/10	9/9	5/8	20/22	24/27		
MV	7/10	1/9	5/10	1/10	0/10	1/10	12/30	2/30	13.8	<0.001
RV	10/10	7/10	4/10	0/10	1/10	0/10	21/30	1/30	26.4	<0.001
FV	4/5	4/5	5/5	1/5	1/5	0/4	13/15	2/14	15.2	<0.001

Resistance to the transplantation of a syngeneic Rauscher lymphoma in C57BL/6 mice pretreated with Friend, Moloney, and Rauscher viruses or lymphomas.

[a] Tumor takes/total inoculated with viable RBL-3 cells.

Table 8

Immunogen	Tumor takes in normal mice (Cells × 10^{-2}/mouse)				Tumor takes in immunized mice (Cells × 10^{-2}/mouse)				Sum of takes/total	
	1000	100	10	1	1000	100	10	1	Control	Immunized
MBL-1	10/10[a]	7/10	0/10		10/10	8/10	2/10		17/30	20/30
FBL-3		5/5	0/5			5/5	0/5		5/10	5/10
Rauscher virus	10/10	10/10	9/10		10/10	10/10	9/10		29/30	29/30
E ♂ G-2		8/8	7/8	0/8		8/8	8/8	0/8	15/24	16/24

Resistance to the transplantation of a syngeneic Gross lymphoma in C57BL/6 mice immunized with Friend, Moloney, Rauscher, and Gross lymphomas.

[a] Tumor takes/total inoculated with viable E ♂ G-2 cells.

duced by each FMR virus was tested by the indirect fluorescent antibody test against the immunizing tumor, against tumors induced by the other FMR viruses, and against a chemically-induced lymphoma (EL-4). Low viability of E ♂ G-2 and MBL-1 cell suspensions precluded their use as target cells. Since Moloney lymphomas possess similar antigens, regardless of the strain of origin (9), a Moloney lymphoma of BALB/c origin was employed as a target. All samples were examined blind. Table 9 reveals that each antiserum reacted strongly with all 3 FMR lymphomas, but not with the chemically-induced EL-4.

Absorption of Anti-Friend Antibody Activity by FMR Lymphomas. To corroborate the above serologic findings, serum from mice immunized with Friend lymphoma cells was absorbed with equal volumes of packed FMR and Gross lymphoma cells or with EL-4. Each absorbed serum, as well as an unabsorbed aliquot, was then serially diluted and tested blind against the immunizing tumor (FBL-3) by the indirect fluorescent antibody test. Table 10 shows that E ♂ G-2 and EL-4 absorbed the least activity; the relatively small absorption by MBL-1 with respect to FBL-3 and RBL-3 is unexplained.

DISCUSSION

Studies by Old *et al.* (12), employing the cytotoxicity test, have demonstrated that murine FMR lymphoma cells are serologically indistinguishable from each other, but may be antigenically distinct from tumors induced by Gross virus. Immunofluorescence and antibody absorption results presented in this study confirm that lymphomas induced by FMR viruses possess antigens in common and suggest that Gross cells do not absorb anti-FMR activity. However, it cannot be assumed that the antigens shared by FMR lymphomas, as demonstrated serologically, are necessarily the transplantation antigens.

Lymphomas induced by Rauscher and Moloney viruses have been reported (1) to have transplantation antigens in common. Mice pretreated with X-irradiated Rauscher lymphoma cells were resistant to the transplantation of some, but not all, Rauscher and Moloney lymphomas. However, the transplantation resistance was weak since the hosts employed, namely BALB/c and CDF_1, are not susceptible to induction of strong transplantation resistance by infectious viruses (1, 4) or by FMR tumors containing infectious virus (9). C57BL/6 mice, in whom strong transplantation resistance to FMR lymphomas is readily induced (9, 10), were, therefore, used in this study.

To determine whether tumors induced by FMR viruses share transplantation antigens, C57BL/6 mice were pretreated with infectious FMR viruses or their induced lymphomas and challenged with syngeneic FMR lymphomas. Mice pretreated with any one infectious FMR virus or its induced lymphoma became resistant to the transplantation of all FMR lymphomas. Thus, lymphomas induced by Friend, Moloney, or Rauscher virus share transplantation antigens. These findings are not inconsistent with the reported ability of a fourth viral agent, namely Moloney sarcoma virus, to induce resistance to the transplantation of FMR lymphomas (2).

Gross lymphomas have been shown to possess weak tumor-

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Table 9

C57BL/6 Serum	Serum Dilution in HBSS	Target cells, percentage staining FBL-3	RBL-3	LSTRA[a]	EL-4
	Undiluted	98	90	78	<5
	1:2	95	65	72	
Anti-FBL-3	1:4	99	60	34	
	1:8	84	37	32	
	1:16	58	15	30	
	Undiluted	80	98	87	<5
	1:2	87	55	58	
Anti-RBL-3	1:4	21	35	35	
	1:8	31	43	12	
	1:16	22	22	10	
	Undiluted	80	82	92	<5
	1:2	38	56	71	
Anti-MBL-1	1:4	29	22	70	
	1:8	40	5	25	
	1:16	17	7	10	
Normal Serum	Undiluted	17	19	9	<5

Serologic cross-reactivity of anti-FMR sera against Friend, Moloney, and Rauscher lymphoma cells as tested by the indirect fluorescent antibody technic. HBSS, Hank's balanced salt solution.

[a] Moloney Leukemia of BALB/c origin.

Table 10

Anti-FBL-3 serum absorbed with:	Dilution of anti-FBL-3 serum 1:4	1:8	1:16	1:32
FBL-3	9[b]	8	8	8
RBL-3	12	7	9	6
MBL-1	62	37	30	30
MBL-1[a]	32	26	25	23
E ♂ G-2	98	67	10	10
EL-4	72	95	24	13
Unabsorbed	88	80	42	10

Absorption of fluorescent antibody activity of anti-Friend serum by syngeneic Friend, Moloney, and Rauscher lymphoma cells. The target cells were obtained from the FBL-3 lymphoma.

[a] Absorbed twice.

[b] % staining.

associated transplantation antigens (8) not shared by Moloney lymphomas (6). In the current study the inability of the Gross lymphoma to induce resistance against itself precluded an examination of whether the FMR lymphomas used possessed Gross transplantation antigens. However, it was clearly demonstrated that the Gross lymphoma did not possess FMR transplantation antigens. The existence of some antigenic similarity between FMR and Gross lymphomas has been suggested by (*a*) the partial absorption of a mouse anti-Gross antibody by Friend leukemias and transplantable Moloney and Rauscher leukemias (13), (*b*) the ability of a rabbit anti-Gross serum to neutralize Moloney virus (5), and (*c*) the ability of a rat anti-Gross serum, as well as mouse anti-FMR sera, to neutralize Moloney sarcoma virus and Moloney leukemia virus (3).

It has been reported that transplantable tumors can acquire surface antigens by virtue of infection with viruses unrelated to their etiology, a condition termed "antigenic conversion" (15). The acquisition of these antigens can be detected serologically and by transplantation tests. During the course of the current studies, the tumors and the C57BL/6 mice were checked for the presence of the following viruses: pneumonia virus (PVM), Reo virus type 3, Thieler's mouse encephalomyelitis (CDVII), Sendai virus, mouse pneumonitis (K virus), mouse hepatitis virus, LCM virus, and polyoma. None of the agents were detected. However, this does not exclude the possibility that some other virus might be common to all the Friend, Moloney, and Rauscher virus preparations and to the transplantable tumors employed. Since the leukemogenic viruses studied were isolated from BALB/c mice at different times, the extraneous hypothetical virus would have to have been endemic in the BALB/c mouse. Furthermore, this virus would have been retained by FMR tumors induced, and serially transplanted, in C57BL/6 mice.

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REFERENCES

1. Bianco, A. R., Glynn, J. P., and Goldin, A. Induction of Resistance against the Transplantation of Leukemias Induced by Rauscher Virus. Cancer Res., *26:* 1722–1728, 1966.
2. Fefer, A., McCoy, J. L., and Glynn, J. P. Antigenicity of a Virus-induced Murine Sarcoma (Moloney). Cancer Res., *27:* 962–967, 1967.
3. Fefer, A., McCoy, J. L., and Glynn, J. P. Neutralization of the Oncogenicity of Moloney Sarcoma Virus and Moloney Leukemia Virus by Anti-gross Serum. Int. J. Cancer, *2:* 647–650, 1967.
4. Glynn, J. P., Bianco, A. R., and Goldin, A. Studies on Induced Resistance against Isotransplants of Virus-induced Leukemia. Cancer Res., *24:* 502–508, 1964.
5. Gross, L. Neutralization *In Vitro* of Mouse Leukemia Virus by Specific Immune Serum. Importance of Virus Titration. Proc. Soc. Exptl. Biol. Med., *119:* 420–427, 1965.
6. Klein, E., and Klein, G. Antigenic Properties of Lymphomas Induced by the Moloney Agent. J. Natl. Cancer Inst., *32:* 547–568, 1964.
7. Klein, E., and Klein, G. Mouse Antibody Production Test for the Assay of the Moloney Virus. Nature, *204:* 339–342, 1964.
8. Klein, G., Sjögren, H. O., and Klein, E. Demonstration of Host Resistance against Isotransplantation of Lymphomas by the Gross Agent. Cancer Res., *22:* 955–961, 1962.
9. McCoy, J. L., Fefer, A., and Glynn, J. P. Comparative Studies on the Induction of Transplantation Resistance in BALB/c and C57BL/6 Mice in Three Murine Leukemia Systems. Cancer Res., *27:* 1743–1748, 1967.
10. McCoy, J. L., Fefer, A., and Glynn, J. P. Influence of Infectious Virus on the Induction of Transplantation Resistance in the Friend Tumor System. Cancer Res., *27:* 2267–2271, 1967.

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11. Möller, G. Demonstration of Mouse Isoantigens at the Cellular Level by the Fluorescent Antibody Technique. J. Exptl. Med., *114:* 415–434, 1961.
12. Old, L. J., Boyse, E. A., and Stockert, E. Typing of Mouse Leukemias by Serological Methods. Nature, *201:* 777–779, 1964.
13. Old, L. J., Boyse, E. A., and Stockert, E. The G (Gross) Leukemia Antigen. Cancer Res., *25:* 813–819, 1965.
14. Sachs, L. The Transplantability of an X-ray-induced and a Virus-induced Leukemia in Isologous Mice Inoculated with Leukemia Virus. J. Natl. Cancer Inst., *29:* 759–764, 1962.
15. Stück, B., Old, L. J., and Boyse, E. A. Antigenic Conversion of Established Leukemias by an Unrelated Leukemogenic Virus. Nature, *202:* 1016–1018, 1964.

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