of the disease consistently revealed no hyperplasia. This observation, coupled with the findings described above, points strongly to the spleen as the main source of the erythroblastosis, increased red cell count, and hematocrit in peripheral blood. The very early rise of Fe^{59} uptake by the spleen suggests that the virus may exert a direct stimulating effect on erythroid elements in the spleen. Continued stimulation results eventually in a markedly increased hematocrit with many immature erythrocytes in peripheral blood. Increase in leucocytes, without evidence of increased medullary activity, suggests that the myeloid elements of the spleen may also be stimulated.

As the disease progresses there is decreased uptake of Fe^{59} by the spleen on a weight basis. This may be due to malignant proliferation of reticulum cells in the spleen so that the ratio of blood forming elements to reticulum cells diminishes progressively with advancing disease.

Unlike Polyoma Virus(8,9,10), the Friend Virus is apparently strain specific(1,2,3,7). Adult mice of Swiss and DBA/2 strains are highly susceptible whereas mice of PRI, C_{57} Bl/6, A, C_3H and (C_{58} $\times$ BALB/c) F_1 strains are nonreactive. Our data indicate that DBA/1 strain is susceptible to the Friend Virus, to the same magnitude as HA/ICR Swiss mice.

Summary. 1. These studies demonstrate that Friend Virus produces in HA/ICR Swiss and DBA/1 mice very soon after infection, a marked increase in selective uptake of Fe^{59} by the spleen. This selective uptake is not found in other tissues in Friend Virus Disease. 2. Friend Virus Disease characteristically produces a malignant proliferation of splenic reticulum cells, but is further characterized by a progressive elevation of hematocrit and rbc count which is related to splenic involvement in the disease. 3. The DBA/1 strain is shown to be susceptible to Friend Virus to the same magnitude as HA/ICR Swiss mice.

We wish to acknowledge the assistance of Dr. Paul Trudel, André Bulba, Ruth Deckert, Edward Dywinski, Helen Fox and A. G. Mirand.

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Received December 19, 1960. P.S.E.B.M., 1961, v106.

Thymectomy and Thymic Grafts in Mouse Viral Leukemia.*† (26359)

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Thymectomy has been found to decrease greatly the incidence of spontaneous leukemia in AK(1) and C58 mice(2) and that induced by whole body irradiation in C57B1 mice(3), by administration of methylcholanthrene to DBA mice(4), or by neonatal injection of leukemia virus in C3H or AK mice(5,6,7).

* Supported by grant of the N.C.I.

† This work was done in collaboration with Dr. Jacob Furth.

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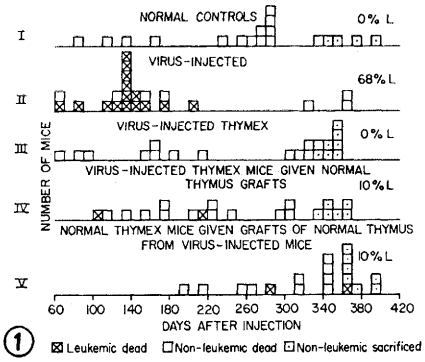
The following studies were carried out to determine the influence of thymic grafts from normal donors in restoring capacity of virus-injected, thymectomized mice to develop leukemia and to determine the origin of the leukemic cell under such conditions.

Materials and Methods. Virus. Passage virus, Agent A of Gross, has been carried in our laboratory in 7 serial passages since 1957 (6). Preparation and storage of filtrates has been described. *Mice.* C3H/f/Bi/G/L,§ hereafter designated Z mice, were used in the first experiment, and hybrids of this line with AKR/Jax in the second. Males and females were caged together. Litters from virus-injected females were discarded; those from uninjected controls were raised. *Injection of virus* to newborn mice (less than 1 week of age) in 0.025-0.07 cc amounts was done intraperitoneally using a No. 30 needle. *Thymectomy* was performed under nembutal-ether anesthesia at 4 to 8 weeks of age by incising the sternum and removing the thymic lobes with curved iridectomy forceps. Mice developing thymic tumors due to incomplete thymectomy were excluded from experimental groups. *Intercurrent infections* were treated by addition of veterinary terramycin to drinking water. *Fighting* among AKR males used in bioassays of induced leukemias was diminished by addition to drinking water of a suspension of meprobamate (Miltown), 100-200 mg/liter. *Thymic grafts* (one lobe of thymus from a healthy 5 to 8 week old donor) were implanted subcutaneously in the axillary region. *Bioassays* of leukemias were made by injecting intramuscularly 0.1 cc of a 10% suspension of minced tissue (5-13 million cells), unless otherwise indicated. *Sections* were taken from most leukemic and many non-leukemic animals with special attention to thymectomy sites. The latter was removed *en bloc* and sectioned longitudinally. Kidney and thyroid were routinely examined for polyoma lesions.

Results. Thymus grafts in thymectomized virus-injected Z mice. The experimental groups are indicated in Fig. 1. None of 18 normal controls developed leukemia (Group I). Of 22 virus-injected intact mice, 15 (68%) developed leukemia with an average latent period of 135 days (Group II). Of 17 virus-injected mice thymectomized at 4 to 6 weeks of age none developed leukemia (Group III). Of 20 virus-injected mice thymectomized at 4 to 6 weeks of age and grafted with normal thymus (Group IV), 2 developed leukemia, one generalized with tumor in the thymic graft at 225 days, and the other generalized without gross tumor in the graft at 109 days. Of 19 uninjected thymectomized mice grafted with thymus from virus-injected mice (Group V), 2 developed leukemia with tumor in the grafts at 274 and 369 days.

Evidence for *persistence of virus in thymectomized virus-injected* non-leukemic mice is presented in Table I. Six infected thymuses removed at 38 days after injection (i.e., during the latent period of leukemia)

§ C3H mice, foster-nursed, originally obtained from Bittner, carried for many generations by Gross, and subsequently by Levinthal.



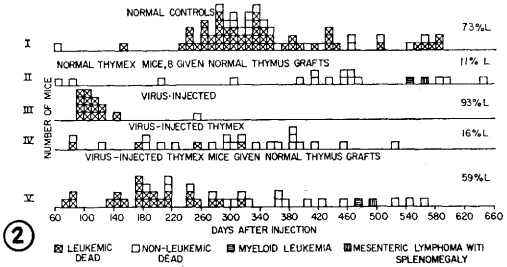


FIG. 1. Effects of grafts of normal thymus from virus-injected or uninjected mice on incidence of leukemia in normal and virus-injected thymectomized Z mice.

FIG. 2. Effects of normal AK and Z thymus grafts on incidence of leukemia in virus-injected and uninjected thymectomized AK/Z mice.

TABLE I. Viral Assay of Non-leukemic Virus-injected Thymectomized Z Mice by Means of Tissue Grafts on Normal Z and AK/Z Mice.

Tissue grafted	Mice assayed: Age, days	Mice assayed: No.	Recipients: No.	Recipients: Strain	Recipients: Age	Recipients: Leuk.	Recipients: Latency, days
			Normal host (control)				
Lymph nodes	254	1	1	Z	newborn	0	
			7	AK/Z	"	1	365
			Virus-injected host				
Thymus	38	6 ind.*	6	Z	adult	2	200, 300
Spleen, liver, lymph nodes	273	3 pooled*	1	Z	newborn	0	
			4	AK/Z	"	4	167, 184, 214, 230
Idem	254	2 "	3	Z	"	0	
			2	AK/Z	"	1	142
"	240	2 "	2	Z	"	1	187
			2	AK/Z	"	2	145, 157
		Total	12	Z		3	187, 200, 300
			8	AK/Z		7	142–230 (177)

* Grafts were assayed individually or pooled.

and grafted individually on 6 intact young adult male Z mice produced 2 thymic lymphomas at 200 and 300 days without tumor at site of the graft, which suggests viral induction of leukemia.

Other tissues from virus-injected mice in which leukemia was prevented by thymectomy were assayed at 240 to 273 days in newborn Z and AK/Z mice. One of 6 Z, and 7 of 8 AK/Z mice developed leukemia. The short latent period in AK/Z mice compared with uninjected controls (Fig. 2) and the high per cent of leukemia indicate superinfection with leukemia virus. However, the latent period was long enough to exclude the likelihood of leukemia due to presence of a few leukemic cells in the assay material. Lymph nodes from an 8 month old uninjected Z mouse assayed as a control had no leukemogenic activity (Table I).

Persistence of virus in tissues of infected thymectomized animals that fail to develop leukemia is to be expected from observations with AK mice showing that non-leukemic parents (non-thymectomized (8) or thymectomized(1)) transmit the capacity to develop leukemia, as do leukemic parents. Also presence of virus in non-leukemic tissues of AKR embryos was demonstrated by Gross(9).

Thymus grafts in thymectomized virus-injected AK/Z mice. Experimental groups are indicated in Fig. 2. Incidence of spontaneous leukemia in normal AK/Z mice was about 60% with a latent period of 10 to 12 months (Group I). Of 18 uninjected mice thymectomized at 6 to 8 weeks of age (8 were later grafted with normal AK and Z thymus), none developed lymphoid leukemia (Group II). Of 15 virus-injected intact mice, 14 (93%) developed leukemia with an average latency of 110 days (Group III). Of 25 virus-injected mice thymectomized at 6 to 8 weeks of age, 4 (16%) developed generalized leukemia (Group IV).

In Group V, 44 virus-injected thymectomized mice (22 males, 22 females) were grafted within a week after thymectomy with one lobe of normal AK and Z thymus.

The donors were about the same age and sex as recipients, with the following exceptions: 14 females were grafted from male donors, and received a second graft (2 to 4 weeks later) from female mice; 2 males received only one female thymus graft (one Z and one AK), and one female only 2 male grafts.

Of 44 mice of this group, 24 (55%) developed generalized lymphoid leukemia at less than one year of age. In 6 of these there was a large lymphoma at site of the Z graft, and in one at the site of the AK graft also. In 7 of the leukemias there was a large lymphoma at site of the AK graft only. In 3 there was a large tumor at site of the

TABLE II. Bioassay for Histocompatibility of Virus-induced and Spontaneous Leukemias in AK/Z, Z and AK Mice.

Leukemia assayed			Latency (days) of leukemia in recipients		
Mouse	No. of Cells assayed (approx.)	Passage	AK/Z	Z	AK
			Spontaneous AK		
1	10^7	I	16, 16, 16, 16	N N N N N*	16, 16, 16
2	10^7	I	27, 27, 27	N N N N	20, 23, 24, 24
3	10^6	I	89, N	N N N	N N N
			Virus-induced Z		
48	10^7	I		14, 14, 28, 32, 35	N N N
6	10^7	I		15, 15, 15, 18, 21	N N N
7, 24, 25†	10^7	I		18, 25, 26, 27, 28, 28, 28, 30	N N N N
31	2×10^5	I	31, 31		N N N
			Virus-induced AK/Z		
25	1.3×10^7	I	13, 15, 15	15, 19, 20, N N	13, 13, 15
	9×10^5	II	15, 16, 18	28, 41, N	15, 33, 34
	2.5×10^4	III	16, 17, 20	23, 23, 23	17, 20, 23
34	10^7	I	16, 16, 17	39, 39, N N N N	N N N
	5×10^5	II	21, 21, 21	38, 47, 54	N N N
18	4×10^6	I	22, 22, 22	50, 59, N N N	28, 28, 49
	9×10^5	II	23		28, 28, 40
5	5×10^4	I	14	N N N	N N N

* N = No growth. † Pooled.

AK and a small tumor at site of the Z graft. The remaining 8 leukemias were generalized; in some of these the small grafts, identified microscopically, were infiltrated with leukemic cells.

Myeloid leukemia is known to occur in thymectomized AK mice at late age(1), and Gross believes that it may be caused by the same virus which causes thymic lymphona (10). It is noteworthy, therefore, that in the present series, one thymectomized control (Group II, Fig. 2) and one virus-injected thymectomized thymus-grafted mouse (Group V, Fig. 2) developed myeloid (chloro-) leukemia at 548 and 476 days respectively.

Two late survivors, one thymectomized control (Group II, Fig. 2) and one virus-injected thymectomized thymus-grafted mouse (Group V, Fig. 2) had greatly enlarged mesenteric lymph nodes and splenomegaly at 569 and 490 days respectively. Earlier studies indicated that this is reticulum cell or lymphoid neoplasia, different from thymic lymphoma. In the present cases, microscopic studies to determine cell type were inconclusive because of autolysis.

Presence of polyoma in filtrates. In the first experiment, one of 22 virus-injected intact Z mice developed typical bilateral salivary gland carcinomas. The leukemia passage virus used in this experiment caused cytopathogenicity in embryo tissue cultures with formation of hemagglutinins. Supernatant fluids of these tissue cultures induced typical polyoma lesions in newborn AK/Z mice.

Immunogenetic character of leukemic tumors arising in thymic grafts. The question whether grafted normal thymus supplies a factor needed by the virus for leukemic transformation of normal non-thymic lymphocytes of the host calls for an immunogenetic characterization of the leukemias arising in thymectomized virus-injected F_1 mice grafted with normal thymus from the parental strains.

As a base line, the immunogenetic relationship of AK, Z and AK/Z leukemic cells was investigated (Table II). a) Spontaneous AK leukemias (using a heavy inoculum containing an estimated 10^7 cells) grew in AK and AK/Z and failed to grow in Z mice.

TABLE III. Summary of Transplantation Assays for Immunogenetic Character of Induced Leukemia and Tumors in Thymic Grafts.

AK/Z donor						
	Tumor in donor*		Transplantation pattern†			
No. assayed	Z	AK	AK	Z	AK/Z	No takes
7	0	+	2 (+1)	0	1 (+1)	2
5	+	0	1	1	2 (+1)	0
4	+	+	(1)	0	3	0
6‡	0	0	1 (+1)	0	2 (+1)	1

* Thymus graft site.

† Numbers in AK column indicate takes in AK and AK/Z; in Z column in Z and AK/Z; and in AK/Z column in AK/Z only. No. in parentheses indicate some uncertainty as to the transplantation pattern because few animals were used and only some took.

‡ Generalized leukemia with no tumor at graft site.

One leukemia tested at a lower inoculum grew only in AK/Z. No spontaneous Z leukemias were available for assay. During 4 years of study, only one spontaneous leukemia was encountered in Z mice. b) Viral induced leukemias in Z mice have uniformly grown in Z and AK/Z but not in AK mice. c) Viral induced leukemias in AK/Z mice always grew in AK/Z, but unexpectedly 3 of 4 grew also in Z, and 2 of those 3 also in AK mice.

The irregularities and failures of cell doses at or below 10^5 are disturbing. This may be due to inadequate inbreeding (homozygocity) of the strain, or to the character of the leukemic cells, or to some unsuspected technical error. Subcutaneous grafts, used here, are known to require more cells than intraperitoneal or intravenous grafts. These observations show a puzzling deviation from the classical histocompatibility pattern and impose limitations on interpretation of the results of grafting in the host systems under study.

Transplantation assays were performed with leukemic cells from 22 of the 24 thymectomized AK/Z mice which received grafts of AK and Z thymuses. The transplantation patterns are indicated in Table III. The donors fall into 4 groups according to presence or absence of lymphoma at site of the grafts as shown in columns 2 and 3.

There was no absolute conformity between the two indicators of cell type: tumor formation at graft site and transplantation pattern. Only one tumor was clearly Z type, as indicated by both tumor formation in the Z graft and transplantability in Z but not in AK mice. Two with AK transplantation pattern were found in mice which had tumor at site of AK graft and not of Z graft, but some occurred also in other groups. Paradoxically, one with AK pattern had a tumor at the Z graft site only. Most tumors appeared to be of AK/Z type, according to the transplantation pattern. It is possible that many donor-leukemias were of mixed cell type.

Discussion. These experiments confirm the effectiveness of thymectomy in preventing virus-induced leukemia(5,6,7) and the capacity of normal thymic grafts to restore partially the leukemogenic action of the virus (11). The low number of leukemias in grafted Z animals may be due to grafting only one lobe of normal thymus and some resistance of Z as compared to AK/Z mice. In most grafted non-leukemic mice, the grafts could not be identified at autopsy. In the second experiment using AK/Z mice the leukemia incidence in the thymectomized group receiving grafts was over 3 times that in the thymectomized littermates not given grafts (55% and 16% respectively). In the first experiment the sex of donor and recipients was not matched. Female mice may reject male grafts(12). In the second experiment most mice received matching grafts and many received 2 grafts in order to insure a match. However, the importance of matching the sex of the grafted thymus with that of recipient is not indicated by these figures. Leukemia occurred in greater frequency (86%) among the 14 females that received a second grafting (4 of 8 females grafted once *vs.* 12 of 14 grafted twice) but females are known to be more susceptible to thymic lymphoma than males(8). The additional amount of grafted thymic tissue may have been more important than the sex of the donor.

The mechanism by which thymic grafts in virus-injected thymectomized mice restore

the leukemogenic action of the virus is still unknown. A similar problem occurs with x-ray induced leukemias, where thymic grafts can restore the capacity for development of leukemia prevented in thymectomized irradiated mice. In Kaplan's work, all leukemias bioassayed appeared to be of the graft origin (13); in studies of Law and Potter(14), 64% appeared to originate in the hybrid host tissue under the influence of some factor supplied by grafted thymus. This is comparable to our findings, suggesting that a thymic factor can affect the virus-injected hybrid host and allow the leukemic transformation to occur in the host's tissue. This factor may be related to the lymphocyte stimulating factor (LSF) of Metcalf(15) which he considers to be a thymic hormone. Attraction exerted by normal thymus grafts and their repopulation by normal as well as malignant lymphocytes of the host have been shown by several workers(14,16,17), as well as in the present studies. Observations that more tumors developed at site of the AK graft than the Z graft, and more of these tumors appeared genetically of AK cell type than of Z type, may be related to the greater genetic susceptibility of AK thymus to malignant transformation, or perhaps to richness in virus of the preleukemic AK thymus.

The results of many transplantation assays here recorded are not clear-cut and further work is needed to explain the puzzling findings. An unknown number of virus-induced leukemias may have been of mixed cell type. This supposition is supported by the observation that on subpassage some of the tumors failed to maintain the initial histocompatibility pattern. However, neoplastic transformation may yield cells with immunological patterns different from that of normal (non-leukemic) cells.

Summary. 1. Passage leukemia virus A of Gross produced leukemia in 15 of 22 virus-injected intact Z mice, 0 of 17 virus-injected thymectomized, and 2 of 20 virus-injected thymectomized Z mice given one lobe of normal thymus from uninjected donors of the same strain. 2. Thymuses of virus-injected mice removed at thymectomy, when implanted in 19 uninjected thymectomized adult Z mice, produced 2 lymphomas in the grafts. 3. Virus was found by bioassay to persist in tissues of virus-injected thymectomized non-leukemic Z mice sacrificed at 8 to 9 months of age. 4. In similar experiments in AK/Z mice, leukemia occurred in 14 of 15 virus-injected intact mice, 4 of 25 virus-injected thymectomized, and 24 of 44 virus-injected thymectomized AK/Z mice given normal thymus grafts from young AK and Z donors. 5. Bioassays were performed for immunogenetic character of 22 leukemias and tumors arising in thymic grafts on virus-injected thymectomized AK/Z mice. AK, Z and AK/Z recipients were used with the following results: a) 3 leukemias failed to grow in any recipients. b) In 8 cases, grafts took in AK/Z only (excluding those with uncertain typing); in 4 cases, in AK and AK/Z mice; and in one case in Z and AK/Z recipients. 6. These data indicate that a thymic factor, other than cell, supplied by normal thymus grafts may carry to completion viral leukemogenesis inhibited by removal of the thymus.

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Received December 21, 1960. P.S.E.B.M., 1961, v106.

Effect of Oxalate on Turnover of Radioactive Phosphate by the Human Erythrocyte.*†‡ (26360)

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Although oxalate has been reported to be involved in glycolysis of human blood(1,2,3, 4,5,6,7), its specific role has not been completely determined. The present investigation was made to study further the effects of oxalate on uptake, distribution and loss of phosphate by the human erythrocyte using radioactive phosphate and radioactive oxalate. Distribution of oxalate between plasma and erythrocyte, turnover of phosphate by erythrocytes incubated with oxalate, role of potassium, magnesium, and calcium ions on the effect of oxalate upon phosphate metabolism, and the effect of oxalate on distribution of phosphate in some of the acid-soluble phosphate ester fractions of the erythrocyte were determined.

Method and materials. Blood drawn from young adults was stored in sterile bottles containing sodium heparinate. The pH of the fresh blood was measured with a Beckman Model G pH meter with a glass electrode. During incubation the blood was kept mixed and at a nearly constant pH of 7.4 by bubbling a gas mixture containing 95% oxygen and 5% carbon dioxide slowly in through a finebore glass tube.

Since Weinhouse and Friedmann(12) demonstrated that oxalate is not metabolized in whole blood, radioactive oxalate (0.00075 Molar) was added and its distribution in whole blood determined by measuring the radioactivity of plasma, hemolyzed whole blood, and hemolyzed washed red cells. Radioactive analysis of a paper electrophoretic chromatogram from the hemolyzed whole blood was done to establish whether or not the oxalate was bound to protein.

Radioactive phosphate was used to measure turnover of phosphate by erythrocytes as affected by oxalate alone or in conjunction with calcium, magnesium, and potassium ions. Fresh erythrocytes were washed with physiological saline phosphate-buffer solution of pH 7.4, incubated for 30 minutes at 37.5°C in their own serum or the above mentioned physiological solution containing radioactive P^{32} inorganic phosphate, washed, then 1 volume further incubated one hour at 37.5°C in 50 volumes of saline-glycine buffer (0.15 molar NaCl, 0.0024 molar glycine, pH = 7.4). After the second incubation the supernatant solution was withdrawn and erythrocytes were washed 3 times with saline, then hemolyzed with 0.01 Molar sodium carbonate in 27% ethanol. P^{32} activity per volume of supernatant and cell hemolysate was determined. These data were used in the equation below for determining the fraction loss. In view of the constancy of turnover rate at one hour, this single determination was used for the assay. Since loss-rates varied with concentration of the phosphate in the uptake media, a standard physiological saline phosphate-buffer medium [0.30% glucose, 0.09% NaCl, 0.15 molar NaH_2PO_4-Na_2HPO_4, pH 7.4], which was found to give reproducible turnover rates, was used throughout the experiment. All tested uptake media were compared to it. Radioactivity measurements were made on "infinitely thick liquid specimens" using a lead shielded

* Presented in part before Am. Chem. Soc., Miami, April, 1957.

† Aided in part by Grant of Nat. Inst. Health.

‡ Radioactive phosphate used in this study was furnished on allocation by the Atomic Energy Comm. at 20% of cost.