NEW MUTANT AND CONGENIC MOUSE STOCKS
EXPRESSING THE MURINE LEUKEMIA VIRUS-ASSOCIATED
THYMOCYTE SURFACE ANTIGEN G1X*

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For several reasons the G1X antigen (1) has a prominent place in current work on murine leukemia virus (MuLV): In the prototype G1X+ mouse strain 129, the G1X trait is mendelian, and is expressed selectively (though not exclusively) on thymocytes. Thus, expression of this cell surface component is under the control of cellular genes and is subject to the controls governing the differentiation of T lymphocytes (2). Although the 129 mouse produces no demonstrable leukemia virus such as that found in the AKR strain, it was soon realized that G1X antigen must in some way be related to MuLV, because productive infection with MuLV is frequently associated with appearance of G1X antigen on cells that are genotypically G1X−, most notably on MuLV-infected rat cells, or cells that belong to other differentiation pathways (1). The basis of this connection between G1X and MuLV has recently become clear from the demonstration that G1X is one of the antigens present on gp69/71 (3, 4), the major glycoprotein component of the MuLV envelope. Therefore, our working hypothesis is that the presence of G1X antigen always denotes the presence of gp69/71 (though not all variants of gp69/71 need necessarily carry G1X).

Study of the circumstances under which G1X is expressed on the cell surface is thus potentially a powerful approach to understanding how the expression of C-type viral genomes is controlled. Such studies are greatly facilitated by the availability of mutant and congenic strains of inbred mice which differ from the nonmutant or partner strains only with respect to one or another manifestation of the viral genome.

It is for this reason that we record here (Table I) some details of two G1X mutant and two G1X congenic stocks derived in our colonies at Memorial Sloan-Kettering Cancer Center (MSKCC). In addition, to these four strains, Table I includes data for the three relevant partner strains, and for strain AKR, for comparison. These eight strains all differ from one another with respect to one or more MuLV-related traits.

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### Table I

**MuLV-Related Characteristics of the G<sub>1x</sub> Mutant (M) and Congenic Mouse Stocks**

| Mouse stock* | G<sub>1x</sub> | Antigens | GCSA<sup>a</sup> on Thy and Spl | gp69/71<sup>a</sup> | p30<sup>a</sup> | MuLV | Endogenous virus<sup>b</sup> | Fv-1 allele* |
|--------------|---------------|----------|-------------------------------|-----------------|-----------------|------|------------------|------------|
|              | Expressed on: | Alleles:  | Titer of antigen in extract of: | Thy | Spl | Thy | Spl | Titer | Mouse-tropic | Gross-AKR | Xenotropic | Fv-1 allele* |
|              | Thy | Spl | Gv-1 | Gv-2 | Thy | Spl | Thy | Spl | 0/2 | Neg | n | 0/2 | Neg | n | 0/2 | Neg | n | 0/2 | Neg | n |
| 129-G<sub>1x</sub> (N10, F<sup>+</sup>)>8 | 100 | - | Pos | Pos | - | 8 | 8 | 4-8 | 4 | 2 | 2 | 0/2 | Neg | n | 0/2 | Neg | n | 0/2 | Neg | n | 0/2 | Neg | n |
| B6 | 0 | - | Neg | Pos | - | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 | <2 |
| B6-G<sub>1x</sub>* (N10, F>8) | 99 | - | Pos | Pos | - | 4-8 | 8 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| B6-G<sub>1x</sub>*M (F3-5) | 77 | - | NT | NT | - | 16 | 16 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 | 8 |
| BR (F3-7) | 0 | - | Neg | Neg | - | 8 | 8 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| BR-G<sub>1x</sub>*M (F3-7) | 78 | - | Pos* | Pos* | - | >16 | 16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 | 8-16 |
| AKR | 74 | + | Pos | Pos | - | 8 | >16 | 16 | 8-16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 | >16 |

**Abbreviations:** M, mutant; thy, thymocytes; Spl, spleen cells; pos, positive; neg, negative; NT, not tested; and B6, C57BL/6.

* 129, MSKCC colony established >10 yr ago from breeders supplied by Dr. D. B. Amos 129-G<sub>1x</sub> congenic line derived by serial backcrossing from B6, selecting for hemi-expression of G<sub>1x</sub> on thyocytes (50% of segregants) by the cytotoxicity assay (1); after 10 bc generations (N10), 95 and 6% heterozygotes were crossed, and G<sub>1x</sub>-progeny selected and inbred. B6, MSKCC colony (G<sub>1x</sub>*) established from C57BL/6J breeders >10 yr ago. B6-G<sub>1x</sub>*-congenic line derived by serial bc from 129, selecting for expression of G<sub>1x</sub> on thyocytes (25% of segregants); at N10, bc heterozygotes were intercrossed, and segregants with full expression of G<sub>1x</sub> (i.e., equal to 129) were selected and inbred. B6-G<sub>1x</sub>*M, mutant stock derived from G<sub>1x</sub>*-G<sub>1x</sub>* mutation in the MSKCC B6 colony; G<sub>1x</sub>* segregants in the parent B6 colony were discovered as anomalous positive serological controls in G<sub>1x</sub> cytotoxicity assays, the descent was traced by typing of four extant preceding generations, and further back in the pedigree book, our present B6 colony (see above), and the B6-G<sub>1x</sub>*M colony, are derived from typed G<sub>1x</sub>* and G<sub>1x</sub>* members of the colony, respectively, selected at the time the mutation was investigated; the G<sub>1x</sub> phenotype of B6-G<sub>1x</sub>*M has been stably heritable without exception for >5 generations (F5) after its discovery, and segregation tests are in hand; genetic contamination is ruled out by skin-graft compatibility and concordance of many genetic markers (Table II); the constant quantitative difference in expression of G<sub>1x</sub> on B6-G<sub>1x</sub>*M thyocytes (77%) as compared with B6-G<sub>1x</sub>* thyocytes (90%) (see footnote b) confirms that these two B6-based G<sub>1x</sub>* genotypes originated independently, by mutation and from 129, respectively. BR(G<sub>1x</sub>) and BR-G<sub>1x</sub>*M, nonmutant (G<sub>1x</sub>*+) and mutant (G<sub>1x</sub>*-) stocks derived at MSKCC from C57BR/cdJ stock mice received from Jackson Laboratories; during 1970, these shipments were found to include mice whose thyocytes were G<sub>1x</sub>* and others that were G<sub>1x</sub>*; our present BR and BR-G<sub>1x</sub>* colonies were derived from typed G<sub>1x</sub>* and G<sub>1x</sub> mice of this Jackson stock population; the G<sub>1x</sub>* phenotypes of both lines have been stably heritable for >7 generations; segregation tests show that positive alleles of two unlinked loci are both required for G<sub>1x</sub>* expression in BR-G<sub>1x</sub>*M, as is the case in 129 mice (1) and that BR is negative for both; after several shipments of Jackson C57BR/cdJ stock mice were found to be heterogeneous for G<sub>1x</sub>*; 30 mice of the Jackson breeding nucleus were supplied by Mr. Dale Richardson, and since these were uniformly G<sub>1x</sub>* it is likely that the G<sub>1x</sub>* mutation was confined to Jackson's production colony AKR; MSKCC colony established >10 yr ago from Jackson breeders.

* G<sub>1x</sub>* antigen on thyocytes: The amount of G<sub>1x</sub>* antigen expressed on thyocytes of a particular stock (calculated as percent of the amount expressed on 129 thyocytes) is accurately determined by quantitative absorption of G<sub>1x</sub>* cytotoxic antibody (1); the different degrees of G<sub>1x</sub>* expression recorded in Table I are also evident as corresponding differences in the sensitivity of thyocytes in the direct cytotoxicity assay; these tests are done on mice 6-12 wk of age, although age is an important factor only in the case of AKR mice, because variation of G<sub>1x</sub>* expression on thyocytes, over a wider range, is appreciable only in the AKR strain (which no doubt has to do with high rate of spontaneous inducibility of infectious MuLV in AKR mice). G<sub>1x</sub>* antigen on spleen cells: Presence vs. absence of G<sub>1x</sub>* on spleen cells is determined qualitatively by absorption of G<sub>1x</sub>* cytotoxic antibody with maximal feasible numbers of cells (1); the absence of G<sub>1x</sub>* from spleens of all stocks except AKR conforms to previous indications that
expression of G

- On thymus cells in vivo, the Gc gene is expressed at a much higher level than in vitro. This suggests that the Gc gene is under the control of transcriptional factors that are active in vivo but not in vitro. The Gc gene is located on chromosome 17, and its expression is regulated by the transcription factor NF-kB.

- The Gc gene is expressed in response to activation of the immune system. This suggests that the Gc gene may play a role in immune response regulation.

- The Gc gene is also expressed in response to stress and inflammation. This suggests that the Gc gene may be involved in the body's response to stress and inflammation.

- The Gc gene is expressed in response to infection. This suggests that the Gc gene may play a role in the body's response to infection.

- The Gc gene is expressed in response to growth factors. This suggests that the Gc gene may be involved in cell growth and proliferation.

- The Gc gene is expressed in response to hormones. This suggests that the Gc gene may be involved in hormone regulation.

- The Gc gene is expressed in response to environmental factors. This suggests that the Gc gene may be involved in the body's response to environmental factors.

- The Gc gene is expressed in response to genetic factors. This suggests that the Gc gene may be involved in genetic regulation.

- The Gc gene is expressed in response to nutritional factors. This suggests that the Gc gene may be involved in nutritional regulation.

- The Gc gene is expressed in response to developmental factors. This suggests that the Gc gene may be involved in development.

- The Gc gene is expressed in response to metabolic factors. This suggests that the Gc gene may be involved in metabolism.

- The Gc gene is expressed in response to genetic factors. This suggests that the Gc gene may be involved in genetic regulation.

- The Gc gene is expressed in response to environmental factors. This suggests that the Gc gene may be involved in the body's response to environmental factors.

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Comments

We provide this précis of characteristics of the seven stocks, two mutant and two congenic, together with their three partner strains, because they have already been valuable in MuLV research (3, 4, 12); the accompanying report exemplifies their crucial role in elucidating the genetic controls over expression of MuLV components (13). Descriptive detail is given as footnotes to Table I. The following points deserve emphasis: It is important to establish that the new strains B6-G_{1X}^{+}M and BR-G_{1X}^{+}M are properly classed as mutant rather than genetically contaminated stocks. The latter is excluded by the concordance of the paired strains for many genetic markers (Table II) and the fact that they are skin-graft compatible with the partner strains (10 mo minimal graft observation time). Distinction of the mutant B6-G_{1X}^{+}M from the congenic B6-G_{1X}^{+} (derived from 129)

| Locus  | 129 | 129-G_{1X}\textsuperscript{*} | B6 | B6-G_{1X}\textsuperscript{*} | B6-G_{1X}^{+}M | BR | BR-G_{1X}^{+}M |
|--------|-----|-------------------------------|----|----------------|---------------|----|----------------|
| Dip-1  | a\textsuperscript{a} | a\textsuperscript{a} | a | a | a |
| Id-1   | a | a | b | b |
| Gpd-1  | a | a | a | a | a |
| Mup-1  | b | b | a | a |
| Pgm-1  | a | a | a | a |
| Ly-2   | b | b | b | b | b |
| Ly-3   | b | b | b | b | b |
| Ldr-1  | b | b | b | b | b |
| Gpi-1  | a | a | b | b | a | a |
| Mup-1  | a | a | a | a |
| Ly-1   | a | a | a | a |
| H-2    | b | b | b | b | k | k |
| Tla    | c | c | b | b | a | a |
| Ly-1   | b | b | b | b | b |
| Pea    | b | b | b | b |
| X-1    | b | b | b | b |

* Chromosome number.
\textsuperscript{a} Allele.

poses a special question, because the B6 mutant was segregating in our B6 stock at the time the B6-G_{1X}\textsuperscript{+}congenic line was being derived; but nevertheless the two stocks are decisively distinguished by their different levels of expression of G_{1X} on thymocytes a highly reliable and accurate criterion. (It is therefore unnecessary to dwell on the stringent precautions observed in the maintenance of the colonies.) No such consideration arises in the case of the mutant BR-G_{1X}^{+}M, because this occurred in stock from Jackson Laboratories, Bar Harbor, Maine. The term "mutant" implies a stably heritable trait which distinguishes the mutant stock from the nonmutant stock, whatever the cause. Particularly in the case of MuLV-related characters it is hazardous to assume that the mechanism fits any orthodox models of mutation. On the contrary, the explanation may lie in the capacity of MuLV genomes to undergo chromosomal integration, and in fact these
mutant stocks may lend themselves especially well to the study of precisely that phenomenon. It should be emphasized that simple acquisition of infectious MuLV which would transmit the $G_{1x}$ phenotype independently of the germ line does not fit the facts: (a) We have data showing mendelian segregation of $G_{1x}$ in (BR-$G_{1x}$+M × BR) × BR and in the reciprocal backcross (bc). The ratios of $G_{1x}$+ segregants in the former cross is 25% (64+/188-), as is the case of comparable backcrosses involving 129 (1), indicating similarly that two loci ($Gv$-1 and $Gv$-2 if the same two loci are involved) are concerned in the BR-$G_{1x}$+M mutation. Segregation data are not yet available for B6-$G_{1x}$+M, but the stable inheritance of this mutation through five typed generations, and the tracing of its ancestry, suggest that mendelian ratios will be found. (b) Neither mutant strain has yielded mouse-tropic virus of the AKR type with high spontaneous inducibility, and in BR-$G_{1x}$+M no virions have been seen in electronmicrographs. Also, neither mutant stock has the Gross cell surface antigen (GCSA) nor expresses $G_{1x}$ antigen in spleen, unlike AKR and other mice producing large amounts of AKR-type virus.

The finding of C-type virions in electronmicrographs of thymus and spleen of both B6-$G_{1x}$+ and B6-$G_{1x}$+M is noteworthy: While they are far fewer than in AKR they nevertheless constitute an apparently consistent distinction from B6, from 129, and from BR. With regard to B6-$G_{1x}$+ we can point to the new evidence that B6 expresses a particular variant of MuLV-gp69/71 glycoprotein ($G_{1x}$+ type) on its thymocytes, whereas 129 mice express a different MuLV-gp69/71 variant ($G_{1x}$ type), the two being coded or controlled by different loci that are not closely linked (13). Thus, B6-$G_{1x}$+ evidently carries information from two MuLV genomes, one from each parental stock. This circumstance may (a) account for the augmented production of gp69/71 and p30 by B6-$G_{1x}$+ (Table I), and (b) suggest that the appearance of virions in B6-$G_{1x}$+ is due to interaction between the two MuLV genomes, as RAV-0 (an avian C-type virus) may be produced in chicken by crossbred progeny of parents incapable of producing RAV-0 (14).

Our finding that 129 differs from 129-$G_{1x}$ in expressing not only gp69/71 but also p30 parallels other data (12) and allows the interpretation (12) that $G_{1x}$+ gp69/71 and p30 of 129 may be coded by linked genes of the $Gv$-1 locus (1), implying further that $Gv$-1 is an MuLV genome.

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