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# Identification of Specificity *H-2.7* as an Erythrocyte Antigen: Control by an Independent Locus, *H-2G*, between the *S* and *D* Regions

Chella S. David, Jack H. Stimpfling, and Donald C. Shreffler

*Department of Human Genetics, University of Michigan, Ann Arbor, Michigan 48104 and McLaughlin Research Institute, Columbus Hospital, Great Falls, Montana 59401*

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*Abstract.* Specificity *H-2.7* is expressed predominantly on erythrocytes and controlled by a gene that maps within the *H-2* gene complex at a locus, designated as *H-2G*, which apparently lies between regions *S* and *D*. Three phenotypes have been observed with respect to this antigen: a) positive by direct test and absorption (haplotypes $H\text{-}2^f$, $H\text{-}2^j$, $H\text{-}2^p$, $H\text{-}2^s$); b) positive only by absorption ($H\text{-}2^k$); and c) negative ($H\text{-}2^b$, $H\text{-}2^d$, $H\text{-}2^q$). New crossover positions have been established for several *H-2* recombinants based on classifications for the *H-2G* locus.

## Introduction

Specificity H-2.7 was first defined by Hoecker *et al.* (1959) with an A anti-A.CA antiserum tested against A.SW ($H\text{-}2^s$) cells in the hemagglutination test. It was later shown that A anti-A.SW serum tested against A.CA ($H\text{-}2^f$) detects H-2.7 as well (Shreffler and Snell 1969). Further analysis based on hemagglutination tests showed that H-2.7 is also determined by haplotypes $H\text{-}2^p$ and $H\text{-}2^j$. Several other antisera were shown to detect H-2.7 by the hemagglutination test. The same antisera used against the same target cells in the cytotoxic test were assumed also to detect H-2.7. Several intra-*H-2* recombinants have been classified for H-2.7. Recombinant B10.M(11R) [$H\text{-}2^{ap1}$], which is $K^fD^d$, was found to be +H-2.7, and B10.M(17R) [$H\text{-}2^{aq1}$], which is $K^kD^f$, was −H-2.7. Both strains were typed by hemagglutination (Stimpfling and Richardson 1965). This suggested localization to the *K* region. Recombinants A.TL ($H\text{-}2^{t1}$) and A.TH ($H\text{-}2^{t2}$), which are $K^sD^d$, were classified as −H-2.7 on the basis of cytotoxic tests (David and Shreffler 1972). This suggested localization to the *D* region. Stimpfling *et al.* (1971) suggested that H-2.7 may be located to either side of *S* due to duplicated genes. In an attempt to resolve this question we recently reanalyzed several of the *H-2* haplotypes for H-2.7 specificity by both the hemagglutination and cytotoxic tests. In this paper we present evidence which indicates that H-2.7 is expressed predominantly on erythrocytes and is controlled by an independent locus, designated *H-2G*, which maps between regions *S* and *D* of the *H-2* gene complex.

## Materials and Methods

Most of the mouse strains used in this study were obtained either from the colony maintained in the Department of Human Genetics, University of Michigan, or the colony in the McLaughlin Research Institute, Great Falls, Montana. Strains BSVS and LG/Ckc were kindly provided by Dr. M. Dorf and Dr. G. Snell, respectively.

All testing was done by the PVP hemagglutination test (Stimpfling 1961) as well as by the $^{51}$Cr-release method, with lymph node target cells (Snell *et al.* 1971). In vivo absorptions were performed by injecting 0.1 ml of the antiserum, intraperitoneally, bleeding the animal 3 hours later, and checking for residual antibody activity (Shreffler *et al.* 1966). In vitro absorptions were performed by incubating appropriate dilutions of antisera with varying volumes of packed erythrocytes or varying cell numbers of lymphocytes, centrifuging the suspension, and checking the supernatant for residual antibody activity against selected target cells in the hemagglutination test.

## Results

### *Hemagglutination Tests*

Three antisera were tested against all the available haplotypes: a) (B6 × A)$F_1$ anti-B10.P [anti-H-2.7,16]; b) (C3H.SW × B10.A)$F_1$ anti-C3H.NB [anti-H-2.7,16]; c) A anti-A.SW [anti-H-2.7,19]. Table 1 shows the results of direct tests with selected haplotypes. As previously demonstrated, haplotypes *H-2$^f$*, *H-2$^p$* and *H-2$^s$* were all positive for H-2.7. Among the five *H-2$^t$* haplotypes with identical *K* and *D* regions, only *H-2$^{t2}$* gave a positive H-2.7 hemagglutination reaction. (All five *H-2$^t$*'s were positive with serum A anti-A.SW, which contained anti-19 antibodies). *H-2$^{t3}$* and *H-2$^{t4}$* differ from *H-2$^{t2}$* only in the *I-C* and *D* regions, while *H-2$^{t5}$* differs from *H-2$^{t2}$* only in the *S* and *D* regions. Since *H-2$^s$* strains are positive for H-2.7 by hemagglutination and *H-2$^d$* and *H-2$^k$* strains are negative, this reaction is apparently due to a gene between the *I* and *D* regions. The region controlling H-2.7 was further narrowed by the positive reaction of strain A.TFR1 (*H-2$^{an1}$*), which is *S$^k$* and *D$^f$*. Since other *S$^k$* strains fail to give direct hemagglutination and the results cited above place the determinant of this reactivity to the left of *D* on the basis of direct hemagglutination, H-2.7 appears to be controlled by an independent locus between *S* and *D*. This region is tentatively designated *H-2G*, since Snell *et al.* (1973) had previously hypothesized a locus (designated *H-2G*) in this interval to account for some observations with another antiserum. Several other recombinants [A.TFR3, A.TFR5, LG/Ckc, A.QSR1] provide additional evidence for this map location. Previous results with B10.M(11R) and B10.M(17R) also are in agreement with this map position. B10.M(11R) is apparently *K$^f$G$^f$D$^d$* (+ H-2.7) while B10.M(17R) must be *K$^d$G$^d$D$^f$* (− H-2.7), since *H-2$^f$* is positive and *H-2$^d$* is negative for H-2.7.

### *Cytotoxic Tests*

Cytotoxic tests with anti-H-2.7 gave some anomalous results, suggesting involvement of other specificities. Antiserum (B6 × A)$F_1$ anti-B10.P was tested by the $^{51}$Cr-release method against all of the haplotypes in Table 1. Among

Table 1. Reactions of Selected *H-2* Haplotypes against Anti-H-2.7 Sera

| Strain | Haplotype | Haplotype Origin of Regions | | | | | | Antiserum Titer by | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | K | I-A | I-B | I-C | S | D | Hemagglutination | | | Cytotoxicity |
| | | | | | | | | $(B6 \times A)F_1$ anti-B10.P (anti-H-2.7, 16) | (C3H.SW × B10.A) anti-C3H.NB (anti-H-2.7, 16) | A anti-A.SW (anti-H-2.7, 19) | $(B6 \times A)F_1$ anti-B10.P |
| B10.A | *a* | *k* | *k* | *k* | *d* | *d* | *d* | 0 | 0 | 0 | 0 |
| B10.S | *s* | *s* | *s* | *s* | *s* | *s* | *s* | 640 | 160 | 1280 | 0 |
| B10.D2 | *d* | *d* | *d* | *d* | *d* | *d* | *d* | 0 | 0 | 0 | 0 |
| A.TL | *t1* | *s* | *k* | *k* | *k* | *k* | *d* | 0 | 0 | 2560 | 0 |
| A.TH | *t2* | *s* | *s* | *s* | *s* | *s* | *d* | 2560 | 1280 | 2560 | 0 |
| B10.S(7R) | *t2* | *s* | *s* | *s* | *s* | *s* | *d* | 640 | 640 | 2560 | 0 |
| B10.HTT | *t3* | *s* | *s* | *s* | *k* | *k* | *d* | 10 | 0 | 640 | 0 |
| B10.S(9R) | *t4* | *s* | *s* | *s* | *d* | *d* | *d* | 0 | 0 | 1280 | 0 |
| BSVS | *t5* | *s* | *s* | *s* | *s* | *d* | *d* | 0 | 0 | 320 | 0 |
| B10.K | *k* | *k* | *k* | *k* | *k* | *k* | *k* | 0 | 0 | 0 | 0 |
| A.CA | *f* | *f* | *f* | *f* | *f* | *f* | *f* | 160 | 320 | 2560 | 320 |
| A.TFR1 | *an1* | *s* | *k* | *k* | *k* | *k* | *f* | 80 | 160 | 2560 | 0 |
| A.TFR3 | *ap3* | *f* | *f* | *f* | *f* | *s* | *d* | 160 | 320 | 640 | 320 |
| A.TFR5 | *ap5* | *f* | *f* | *f* | *f* | *k* | *d* | 0 | 0 | 0 | 320 |
| LG/Ckc | *ar* | *d?* | *f?* | *f?* | *f?* | *f?* | *f?* | 320 | 640 | 640 | 320 |
| C3H.Q | *q* | *q* | *q* | *q* | *q* | *q* | *q* | 0 | 0 | 0 | 320 |
| A.QSR1 | *sq1* | *s* | *s* | *s* | *s* | *q* | *q* | 0 | 0 | 1280 | 0 |
| B10.P | *p* | *p* | *p* | *p* | *p* | *p* | *p* | 2560 | 2560 | 640 | 640 |
| A.AL | *a1* | *k* | *k* | *k* | *k* | *k* | *d* | 0 | 0 | 0 | 0 |
| C3H.OL | *o1* | *d* | *d* | *d* | *d* | *k* | *k* | 0 | 0 | 0 | 0 |
| C3H.OH | *o2* | *d* | *d* | *d* | *d* | *d* | *k* | 0 | 0 | 0 | 0 |
| KBR | *h3* | *k* | *k* | *k* | *k* | *k* | *b* | 0 | 0 | 0 | 0 |
| B10.AKM | *m* | *k* | *k* | *k* | *k* | *k* | *q* | 0 | 0 | 0 | 0 |
| AQR | *y1* | *q* | *k* | *k* | *d* | *d* | *d* | 0 | 0 | 0 | 0 |
| B10.T(6R) | *y2* | *q* | *q* | *q* | *q* | *q* | *d* | 0 | 0 | 0 | 320 |

Table 2. Direct Cytotoxic Reactions of Antiserum (B6 × A)$F_1$ Anti-B10.P Against Recombinant Haplotypes[a]

| Strain | Haplotype | H-2 Region | | | | Cytotoxic Titer |
|---|---|---|---|---|---|---|
| | | *K* | *I* | *S* | *D* | |
| A.TFR1 | *an1* | *s* | *k* | ←*k*] | *f* | 0 |
| A.TFR3 | *ap3* | *f* | ←*f*] | *s* | *d* | 320 |
| A.TFR5 | *ap5* | *f* | ←*f*] | *k* | *d* | 320 |
| A.QSR1 | *sq1* | *s* | ←*s*] | *q* | *q* | 0 |
| B10.AKM | *m* | *k* | *k* | ←*k*] | *q* | 0 |
| AQR | *y1* | *q* | [→*k* | *k* | *d* | 0 |
| B10.T(6R) | *y2* | *q* | *q* | ←*q*] | *d* | 320 |
| LG/Ckc | *ar1* | *d* | [→*f* | *f* | *f* | 320 |

[a] Arrows indicate map position of reactivity.

the independent haplotypes, C3H.Q(*H-2*$^q$) was positive, while B10.S(*H-2*$^s$) was negative. With these target cells, a 40–50% release was obtained, compared with a background of 20%. In the hemagglutination test *H-2*$^s$ was positive, while *H-2*$^q$ was negative. Examination of the recombinant hyplotypes clarified these results.

Three strains mapped the cytotoxic reactivity to the left of *S* (Table 2). Strains A.TFR3 and A.TFR5 were positive, while A.QSR1 was negative. Since parental haplotypes *H-2*$^s$, *H-2*$^k$ and *H-2*$^d$ were negative and *H-2*$^f$ and *H-2*$^q$ were positive, the cytotoxic reactivity must be controlled by a gene(s) to the left of *S*. Strain AQR was negative and LG/Ckc was positive, indicating that the *K* region does not control this reaction. This confines the reactivity to a position between *K* and *S* in the *I* region. The reactivity is due to antibodies against an Ia specificity determined by $I^p$, which crossreact with products of $I^f$ and $I^q$. It is designated Ia.13. Further data on this specificity will be reported elsewhere.

These results show that specificity H-2.7 was not involved in the cytotoxic reaction. Some of the difficulties in previous classifications of certain strains for H-2.7 arose from interpreting the cytotoxic reaction as due to anti-7. H-2.7 was originally defined as a hemagglutinating specificity and apparently cannot be detected on lymphocytes by direct cytotoxicity.

### *In Vivo Absorptions*

To check the positive hemagglutination results, in vivo absorptions were done with antiserum (B6 × A)$F_1$ anti-B10.P in mice of all independent *H-2* haplotypes. The absorbed sera were tested by hemagglutination against cells from B10.P (H-2.7,16;Ia.13), B10.K (negative), B10.S(H-2.7), and B10.M (H-2.7; Ia.13) (Table 3). As expected, B10.P cleared reactivity for all test cells, while

Table 3. In Vivo Absorptions of Antiserum (B6 × A)$F_1$ Anti-B10.P in Strains Carrying Independent Haplotypes

| Absorbed In | Haplotype | Tested Against[a] | | | |
|---|---|---|---|---|---|
| | | B10.P | B10.K | B10.S | B10.M |
| B10 | *b* | >80 | 0 | 40 | 20 |
| B10.D2 | *d* | >80 | 0 | 80 | 40 |
| A.CA | *f* | >80 | 0 | 0 | 0 |
| B10.K | *k* | >80 | 0 | 0 | 0 |
| B10.P | *p* | 0 | 0 | 0 | 0 |
| C3H.Q | *q* | >80 | 0 | >80 | >80 |
| B10.S | *s* | >80 | 0 | 0 | 0 |

[a] Titer by hemagglutination.

Table 4. In Vivo Absorptions of Antiserum (B6 × A)$F_1$ Anti-B10.P in Strains Carrying Recombinant Haplotypes

| Absorbed In | *H-2* Regions | | | | Tested Against[a] | | | |
|---|---|---|---|---|---|---|---|---|
| | *K* | *I* | *S* | *D* | B10.P | B10.K | B10.S | B10.M |
| A.TL | *s* | *k* | *k* | *d* | >80 | 0 | 0 | 0 |
| A.TH | *s* | *s* | *s* | *d* | >80 | 0 | 0 | 0 |
| B10.HTT | *s* | *s* | *k* | *d* | >80 | 0 | 0 | 0 |
| A.AL | *k* | *k* | *k* | *d* | >80 | 0 | 0 | 0 |
| C3H.OH | *d* | *d* | *d* | *k* | >80 | 0 | >80 | >80 |
| C3H.OL | *d* | *d* | *k* | *k* | >80 | 0 | 0 | 0 |
| B10.AM | *k* | *k* | *k* | *b* | >80 | 0 | 0 | 0 |
| B10.AKM | *k* | *k* | *k* | *q* | >80 | 0 | 0 | 0 |
| A.TFR5 | *f* | *f* | *k* | *d* | >80 | 0 | 0 | 0 |

[a] Titer by hemagglutination.

B10 and B10.D2 failed to clear. Strains A.CA and B10.S cleared anti-H-2.7. However, unexpectedly B10.K cleared the anti-H-2.7 reactivity against B10.S and B10.M test cells. This was repeated several times, and haplotype $H\text{-}2^k$ was found to be consistently positive for H-2.7 by absorption, but negative by direct test (HANAP-hemagglutination negative, absorption positive).

In vivo absorptions were also done in mice of a number of different recombinant haplotypes to check the results of direct tests (Table 4). As expected, the C3H.OL strain, which is $S^kD^k$, absorbed the antiserum. Strain A.AL ($S^kD^d$) also absorbed the antiserum, suggesting that it carries an $H\text{-}2G^k$ allele and placing the crossover point to the right of *H-2G*. Three strains, which derived their *D* end from A.AL [A.TL, B10.HTT, and A.TFR5], also absorbed for H-2.7. Strains B10.AM ($S^kD^b$) and B10.AKM ($S^kD^q$) also absorbed, again suggesting crossover positions to the right of *H-2G*. Strain C3H.OH ($S^dD^k$) failed to absorb, indicating an $H\text{-}2G^d$ allele and a crossover to the right of *H-2G*. As expected, all recombinants positive by direct test [A.TH, B10.S(7R), A.TFR1, A.TFR3, and LG/Ckc] absorbed for H-2.7.

### *In Vitro Absorptions*

*Erythrocytes:* Quantitative absorptions were performed with erythrocytes from strains B10.S, B10.D2, and B10.K and tested by hemagglutination against target cells from B10.S, B10.M, and B10.P (Table 5). B10.D2 red blood cells failed to absorb for any of the target cells. A volume of 0.05 ml of packed B10.S erythrocytes was required to absorb for B10.S and B10.M (H-2.7) target cells but complete absorption was not obtained for B10.P (H-2.7,16) targets. In contrast, 0.5 ml of packed B10.K erythrocytes was required to absorb for B10.S and B10.M target cells, a volume 10 times greater than in the previous case. This is consistent with the failure of direct hemagglutination with B10.K erythrocytes, suggesting a low dinsity of antigenic sites on these cells.

*Lymphocytes:* Quantitative absorptions were also done with lymphocytes from lymph nodes, spleen and thymus (Table 6). No absorption was seen with B10.D2 lymphocytes, when the absorbed serum was tested by hemagglutination against B10.S, B10.M and B10.P target cells. The concentration of lymph node cells from strain B10.S required to absorb reactivity against B10.S and B10.M target cells was $10^7$, but up to $200 \times 10^6$ B10.S cells failed to absorb for B10.P. In comparison, only $10^6$ B10.P cells were required to absorb anti-H-2.16 reactivity from the same volume and concentration of antiserum when tested by hemagglutination against B10.P erythrocytes (Data not included in Table 6). As many as $10^8$ B10.S thymus and spleen cells were required to absorb the anti-H-2.7 reaction with B10.S and B10.M erythrocytes. Lymph node cells (50 million)

Table 5. In Vitro Absorptions of $(B6 \times A)F_1$ Anti-B10.P (Anti-7.16) with Erythrocytes

| Target Cells | Absorbed In[a] | | |
|---|---|---|---|
| | B10.S | B10.D2 | B10.K |
| B10.S | 0.05 ml | NA | 0.5 ml |
| B10.M | 0.05 ml | NA | 0.5 ml |
| B10.P | NA | NA | NA |

[a] Volume of cells (packed) required to completely absorb 0.1 ml of antiserum diluted 1/100. NA indicates no absorption with up to 1 ml packed erythrocytes.

Table 6. In Vitro Absorptions of $(B6 \times A)F_1$ Anti-B10.P (Anti-7,16) with Lymphocytes

| Target Cells | Absorbed In[a] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | B10.S | | | B10.K | | | B10.D2 | | |
| | LN | TH | SP | LN | TH | SP | LN | TH | SP |
| B10.S | 10 | 100 | 100 | 50 | NA | NA | NA | NA | NA |
| B10.M | 10 | 100 | 100 | 50 | NA | NA | NA | NA | NA |
| B10.P | NA | NA | NA | NA | NA | NA | NA | NA | NA |

[a] Number of cells (million) required to completely absorb 0.1 ml of antiserum 1/100. NA indicates no absorption with up to 200 million lymphocytes.

from B10.K absorbed the anti-H-2.7, but no absorption could be detected with up to 200 million thymus and spleen cells. These results suggest that specificity H-2.7 is expressed on H-$2^s$ lymph node lymphocytes at a density 10 to 20 times less than that of classical H-2 antigens, and the density on *H-2*$^k$ lymph node cells is even lower. The expression on thymic and spleen cells is still weaker.

## Discussion

Most of the initially defined H-2 specificities were identified by the hemagglutination method. When the antisera which defined these specificities were tested by the cytotoxic method, they were assumed to be detecting the same specificities. Specificity H-2.7 is an example of such a case. Several anti-H-2.7 sera gave good reactions in both the hemagglutination and cytotoxicity assays. However, discrepancies between the results of these assays when these sera were tested against intra-*H-2* recombinants suggested that we were dealing with two different specificities, mapping in separate regions. This raises the possibility that still other H-2 specificities apparently localized to the *D* or *K* regions might be separable from the *H-2D* and *H-2K* genes. Such reactivity in the *D* region could be due to other alleles at the *H-2G* locus.

We have only preliminary data on the histocompatibility properties of the H-2G antigen. Skin grafts between strains carrying haplotypes differing at this locus do not show a classical H-2 (K or D) rejection pattern [12–14 days]. Rejection is obtained 60–90 days after grafting, if the recipient is presensitized (Stimpfling, unpublished). This pattern is similar to that of the weaker histocompatibility loci. However, since the haplotype combinations tested so far also differ in the *S* and *I-C* regions, whether genes determining the erythrocyte antigens are identical to those determining these minor histocompatibility differences cannot be determined as yet.

The inability to detect H-2.7 specificity by direct cytotoxic tests on lymphocytes could be interpreted in two ways: a) either the density of the antigenic sites on the lymphocytes is too sparse for proper complement binding or b) the antibodies are noncomplement fixing. Since *H-2*$^k$ erythrocytes do not give a direct reaction in the hemagglutination or cytotoxic test, the most probable explanation would be the low density of antigenic sites on the cell surface. The almost negligible expression on thymic and splenic lymphocytes is also unlike that of other H-2 antigens. Further studies on the tissue distribution of the H-2.7 antigen are in progress.

It would be interesting to know whether antigens coded by the *H-2G* loci are on molecules independent of H-2D or on the same molecule. If they are on different molecules, do they differ in molecular weight and other chemical properties? Such studies may be difficult due to the low density of H-2.7 on lymphocytes.

The genetic compositions of several recombinant haplotypes with regard to the *H-2* gene complex are shown in Table 7. On the basis of current information on the *H-2G* locus, new crossover points have been assigned to several of the recombinants. The recombinant haplotypes of strains A.AL, C3H.OH, B10.AM,

Table 7. Compositions of the *H-2* Gene Complex in Some Recombinant Haplotypes

| Strain | Haplotype | *H-2* Region[a] | | | | | | | H-2.7 Reactivity | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *K* | *I-A* | *I-B* | *I-C* | *S* | *G* | *D* | Direct Test | ABS |
| B10.A | *a* | *k* | *k* | *k* \| | *d* | *d* | *d* | *d* | – | – |
| A.AL | *a1* | *k* | *k* | *k* | *k* | *k* | *k* \| | *d* | – | + |
| C3H.OL | *o1* | *d* | *d* | *d* | *d* \| | *k* | *k* | *k* | – | + |
| C3H.OH | *o2* | *d* | *d* | *d* | *d* | *d* | *d* \| | *k* | – | – |
| B10.AM | *h3* | *k* | *k* | *k* | *k* | *k* | *k* \| | *b* | – | + |
| B10.AKM | *m* | *k* | *k* | *k* | *k* | *k* | *k* \| | *q* | – | + |
| A.QSR1 | *sq1* | *s* | *s* | *s* | *s* \| | *q* | *q* | *q* | – | – |
| A.TH | *t2* | *s* | *s* | *s* | *s* | *s* | *s* \| | *d* | + | + |
| A.TL | *t1* | *s* \| | *k* | *k* | *k* | *k* | *k* | *d* | – | + |
| B10.HTT | *t3* | *s* | *s* | *s* \| | *k* | *k* | *k* | *d* | – | + |
| B10.S(9R) | *t4* | *s* | *s* | *s* \| | *d* | *d* | *d* | *d* | – | – |
| BSVS | *t5* | *s* | *s* | *s* | *s* \| | *d* | *d* | *d* | – | – |
| A.TFR1 | *an1* | *s* | *k* | *k* | *k* | *k* \| | *f* | *f* | + | + |
| A.TFR3 | *ap3* | *f* | *f* | *f* | *f* \| | *s* | *s* | *d* | + | + |
| A.TFR5 | *ap5* | *f* | *f* | *f* | *f* \| | *k* | *k* | *d* | – | + |
| LG/Ckc | *ar1* | *d* \| | *f* | *f* | *f* | *f* | *f* | *f* | + | + |

[a] Vertical lines indicate crossing over points.

B10.AKM, and A.TH seem to have derived from crossover between the *G* and *D* regions. So far only haplotype $H\text{-}2^{an1}$ (A.TFR1) seems to have a crossover point between *S* and *G*, but four haplotypes, $H\text{-}2^{o1}$ (C3H.OL), $H\text{-}2^{t5}$ (BSVS), $H\text{-}2^{ap3}$ (A.TFR3), and $H\text{-}2^{ap5}$ (A.TFR5), carrying crossovers between the *I-C* and *S* regions, separate the *H-2G* locus from the *I* region.

Since the $H\text{-}2^{b}$, $H\text{-}2^{d}$, and $H\text{-}2^{q}$ haplotypes completely lack the H-2.7 antigen, an interesting question is whether allelic products at the *H-2G* locus are determined by these haplotypes. Likewise, does the $H\text{-}2^{k}$ haplotype produce an *H-2G* product which is qualitatively different from those of $H\text{-}2^{f}$, $H\text{-}2^{j}$, $H\text{-}2^{p}$, and $H\text{-}2^{s}$? Experiments are underway to attempt to detect other antigens controlled by the *H-2G* locus.

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