The antigenic products of the major histocompatibility complex (MHC)\(^1\) play a crucial role in the stimulation of immune responses of thymus-derived lymphocytes (T cells) (1-4). A T-cell response to foreign antigen (X) is apparently stimulated not by X alone, but by an antigenic pattern dependent upon both X and an appropriate MHC-coded antigen. In the mouse, H-2K or H-2D antigens are involved in cytotoxic T-cell (T\(_c\)-cell) responses (3-6), and I-region-dependent antigens are involved in helper (7, 8) and delayed hypersensitivity responses (9).

Thus far, MHC-linked control of T-cell responses which determines high- or low-responder status of a particular mouse strain for a particular antigen, has been interpreted in terms of qualitative differences between different K, D, or I gene products (10-14). For example, T\(_c\) cells of female inbred mice may or may not respond to the HY antigen of otherwise identical inbred males depending upon whether or not the K, D, or I region gene products are permissive for the T\(_c\) cell or helper T-cell responses (11-14). In this report, we demonstrate quantitative differences in expression of the same K and D antigens between cells of parental strain and F\(_1\) hybrid mice, and also show that these differences markedly influence the ability of the cells to stimulate T\(_c\)-cell responses, and to act as targets for T\(_c\)-cell-mediated lysis.

Materials and Methods

**Animals.** Mice were bred in the John Curtin School of Medical Research and used when 6-10 wk old. Mice of the same age were used in each experiment. F\(_1\) hybrid mice were obtained from six different matings over a 4-mo period.

**Priming of Mice.** Mice were primed by i.v. injection of 2 \(\times\) 10\(^4\) plaque-forming units (PFU) of ectromelia virus (attenuated Hamspead egg strain) and used from 2 wk to 6 mo postpriming.

**In Vitro Generation of Cytotoxic T Cells.** One-way mixed lymphocyte reactions (MLR), utilizing spleen cell responders and \(^{60}\)Co \(\gamma\)-irradiated (2,000 rads) spleen stimulators were set up at a concentration of 2.5 \(\times\) 10\(^4\) responder cells/ml. Cells were cultured for 5 d in Eagle's minimal essential medium (F15, Grand Island Biological Co., Grand Island, N. Y.) containing 10\(^{-4}\) M 2-mercaptoethanol and supplemented with 10% fetal calf serum (FCS) in an atmosphere of 10% CO\(_2\), 7% O\(_2\), and 83% N\(_2\) for 5 d. When a mixture of stimulators was used, they were irradiated before mixing. Responder:stimulator ratio was always 4:1. The in vitro secondary response to ectromelia virus has been previously described in detail (15). Briefly, spleen cells from primed mice were cultured at 39\(^\circ\)C (nonpermissive for ectromelia virus) with syngeneic

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\(^1\)Abbreviations used in this paper: FCS, fetal calf serum; LRT, likelihood ratio test; MHC, major histocompatibility complex; MLR, mixed lymphocyte reaction; PFU, plaque-forming unit; T cells, thymus-derived lymphocytes; T\(_c\), cytotoxic T cells; X, foreign antigen.
infected spleen cell stimulators at a ratio of 10:1. The virulent Moscow strain was used to infect stimulators and targets.

Each experiment was repeated several times and involved stimulator cells from either individual mice or a pool of two. Either males were used as responders, or females were used throughout the experiment to eliminate possible effects due to the HY antigen.

Cytotoxic Assay. The $^{51}$Cr release assay with macrophage targets has been previously described (16). Briefly, $10^8$ $^{51}$Cr-labeled peritoneal macrophages collected from a pool of at least four mice were used as targets, and were incubated for 6 h with effector cells at a wide range of killer:target ratios. Percent maximum releasable $^{51}$Cr was calculated by water lysis of cells. Spontaneous release was about 3% per hour. The corrected percent lysis was calculated by the formula:

$$\text{percent specific } ^{51}\text{Cr release} = \frac{\text{percent releasable by killers} - \text{percent medium release}}{\text{percent maximum releasable} - \text{percent medium release}}.$$

The assay was done in triplicate.

Quantitative Antiserum Absorption. The method has been previously described by McKenzie et al. (17). Antisera were generously provided by Dr. Ian McKenzie (University of Melbourne, Australia). They were produced in the following mouse strains: anti-H-2.2 (K b) in [B10.D2 × A]F1; anti-B10.A(2R); anti-H-2.23 (K b) in [B10.D2 × A]F1 anti-B10.A(3R); anti-H-2.4 (D b) in [B10.AKM × 129]F1 anti-B10.A; anti-H-2.23 (K b) in [AQR × B10]F1 anti-B10.A. The medium used for all procedures was F15 supplemented with 5% FCS. Spleen cells, purified by 14% Isopaque-Ficoll separation (<4% dead cells) were used for titrations and absorptions. A two-stage cytotoxic assay was carried out in V-bottomed Linbro 6-mm microtiter trays. Antisera were titered at least twice on control cells. For quantitative absorption, 50 $\mu$L of cells (doubling dilutions) were added to 50 $\mu$L of antiserum (used at a dilution which gave 80% lysis of control cells in a routine titration) for 30 min at room temperature. After centrifugation, each sample of absorbed antiserum was titered on control cells. Preabsorbed rabbit serum diluted one in four was used as a complement source. The percentage of dead cells was measured by trypan blue staining. Unabsorbed antiserum and rabbit complement were titered as controls and results were plotted as percent dead cells versus number of absorbing cells (log 2 scale). The likelihood ratio test was used to compare data sets in each experiment, and maximum likelihood estimates of the 50% end points were calculated.

Results

Absorption of Anti-H-2 Sera by Spleen Cells from Parental Strain and F1 Hybrid Mice. Spleen cells from B10.A(5R) (K b, D b) and B10.A(2R) (K b, D b) (henceforth referred to as 5R and 2R) were compared with cells from the (5R × 2R)F1 hybrid for their ability to absorb out activity from specific anti-H-2 serum. Individual mice of each sex and from different matings were used in three experiments with similar results. Representative data (Fig. 1) indicate that 2R and F1 cells were indistinguishable in their ability to absorb anti-K b and anti-D b sera, but F1 cells were significantly inferior to 5R in absorbing anti-K b and anti-D b sera. The data suggested that F1 cells expressed approximately fourfold less K b and D b surface antigen than 5R cells, and that the difference was possibly more pronounced with D b than K b.

Sensitivity of Macrophages from Parental Strain and F1 Hybrid Mice to Lysis by Alloreactive and H-2 Restricted Tc Cells. Tc cells from B10.G(H-2 b) mice were stimulated in MLR by either 2R or 5R cells. The effector Tc cells thus generated were assayed on macrophage target cells from 2R, 5R, and (5R × 2R)F1 mice (pools of four mice for each strain). Representative results from one out of four similar experiments (Table I) show that lysis of 2R and F1 targets by anti-2R Tc cells was similar, but that 5R targets were lysed significantly more than F1 targets by anti-5R Tc cells. This latter
DIFFERENCES IN H-2 EXPRESSION IN F1 HYBRIDS

FIG. 1. Quantitative absorption of anti H-2 serum by spleen cells of the F1 hybrid [B10.A(5R) x B10.A(2R)] in comparison with B10.A(5R) and B10.A(2R) cells. The F1 hybrid was compared with 5R for absorption of anti-K\textsuperscript{b} and anti-D\textsuperscript{d} serum and with 2R for anti-K\textsuperscript{k} and anti-D\textsuperscript{b} serum. F1 (●); 2R (○); 5R (▲). Lysis by unabsorbed antiserum plus rabbit complement (AS) and by rabbit complement alone (RC) are shown as controls. Each experiment was repeated thrice, but one representative result is shown. The number of cells absorbing was plotted on a log2 scale. The likelihood ratio test (LRT) was used to compare data sets in each experiment. The 50% end point of titration was calculated from the curve of best fit (maximum likelihood estimation) and the results are summarized below.

| Antiserum specificity | Dilution used for absorption | LRT (\ χ^2_0) | Cell number required to reduce cytotoxicity of antiserum by 50% |
|-----------------------|-----------------------------|--------------|---------------------------------------------------------------|
|                       |                             |              | (5R × 2R)\textsuperscript{F1} | 5R                     | 2R                     |
| K\textsuperscript{b}  | 1/300                       | 24.36        | 11.3 × 10\text{5} | 3.5 × 10\text{5}       |
| D\textsuperscript{d}  | 1/120                       | 44.12        | 19.1 × 10\text{5} | 4.0 × 10\text{5}       |
| K\textsuperscript{k}  | 1/16                        | 8.48*        | 6.0 × 10\text{5} | 8.8 × 10\text{5}       |
| D\textsuperscript{b}  | 1/50                        | 12.57*       | 4.6 × 10\text{5} | 4.6 × 10\text{5}       |

* Not significantly different at the 95% level.

difference was of the order of 10-fold, i.e., up to 10 times more T\text{c} cells were required to lyse F\text{1} cells to the same extent as 5R cells.

In another series of 10 similar experiments T\text{c} cells specific for K\textsuperscript{b}, D\textsuperscript{d}, K\textsuperscript{k}, or D\textsuperscript{b} antigens were generated in MLR and assayed against pools of 5R, 2R, and F\text{1} macrophage targets. Representative data (Table II) show again that 2R and F\text{1} macrophages were lysed similarly by anti-K\textsuperscript{b} and anti-D\textsuperscript{b} \text{T\text{c} cells}, although 5R targets were lysed more efficiently than F\text{1} cells by anti-K\textsuperscript{b} and anti-D\textsuperscript{b} \text{T\text{c} cells}. However, the difference between 5R and F\text{1} targets was less pronounced than in Table I, and was
Table I

Comparison of 5R, 2R, and F1 Macrophage as Targets for MLR Killers

| Spleen* responders | Spleen stimulators | K:T | [B10.A(5R) × B10.A(2R)] K<sup>d</sup> × K<sup>d</sup> | B10.A(2R) K<sup>d</sup> | B10.A(5R) K<sup>d</sup> |
|-------------------|-------------------|-----|---------------------------------|----------------|----------------|
| B10.G             | B10.A(2R)         | 3:1 | = 100                           | = 100          |                |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 1:1 | 82.9 ± 1.0                      | 54.2 ± 3.9     |                |
|                   |                   | 0.3:1| 37.0 ± 3.6                      | 32.5 ± 1.6     |                |
|                   |                   | 0.1:1| 6.1 ± 0.7                       | 6.0 ± 0.6      |                |
| B10.G             | B10.A(5R)         | 3:1 | 42.9 ± 3.3                      | = 100          |                |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 1:1 | 16.4 ± 1.0                      | 78.3 ± 1.2     |                |
|                   |                   | 0.3:1| 3.3 ± 1.2                       | 31.4 ± 3.8     |                |
|                   |                   | 0.1:1| 0                              | 16.1 ± 2.1     |                |

* MLR set up as in Materials and Methods.
‡ Values are means ± SE of triplicates of ⁵¹Cr release over a 6-h period.

Table II

Differential H-2 Antigen Expression on (5R × 2R)F<sub>1</sub> Macrophage Targets

| Spleen* responders | Spleen stimulators | K:T | [B10.A(5R) × B10.A(2R)] K<sup>d</sup> × K<sup>d</sup> | B10.A(2R) K<sup>d</sup> | B10.A(5R) K<sup>d</sup> |
|-------------------|-------------------|-----|---------------------------------|----------------|----------------|
| B10               | B10.A(2R)         | 9:1 | 96.4 ± 1.1                      | 89.9 ± 0.4     | 14.0 ± 1.0     |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 3:1 | 82.8 ± 1.6                      | 84.0 ± 1.3     | 86 ± 0.4       |
|                   | Anti-K<sup>b</sup> | 1:1 | 68.5 ± 3.2                      | 61.2 ± 3.7     | 48 ± 1.2       |
|                   |                   | 0.3:1| 19.3 ± 3.9                      | 30.9 ± 0.8     | 0              |
| B10.BR            | B10.A(2R)         | 9:1 | = 100                           | = 100          | 19.2 ± 1.4     |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 3:1 | 97.0 ± 1.9                      | 90.6 ± 0.7     | 7.2 ± 2.3      |
|                   | Anti-D<sup>d</sup> | 1:1 | 46.9 ± 1.1                      | 43.3 ± 2.0     | 14.1 ± 2.1     |
|                   |                   | 0.3:1| 28.8 ± 1.9                      | 17.5 ± 1.0     | 0              |
| B10.D2            | B10.A(5R)         | 9:1 | 59.0 ± 0.6                      | 76.5 ± 1.0     | 6.2 ± 0.2      |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 3:1 | 53.8 ± 1.1                      | 63.3 ± 2.6     | 5.5 ± 0.6      |
|                   | Anti-K<sup>b</sup> | 1:1 | 26.4 ± 0.6                      | 30.4 ± 0.4     | 2.3 ± 0.7      |
|                   |                   | 0.3:1| 97.2 ± 1.5                      | 17.2 ± 1.5     | 0              |
| B10               | B10.A(5R)         | 9:1 | 65.6 ± 0.5                      | 82.8 ± 1.2     | 9.3 ± 0.9      |
| H-2<sup>n</sup>   | K<sup>d</sup>     | 3:1 | 40.6 ± 1.3                      | 73.2 ± 2.7     | 28 ± 0.8       |
|                   | Anti-D<sup>d</sup> | 1:1 | 35.7 ± 1.3                      | 54.0 ± 4.3     | 48.8 ± 1.0     |
|                   |                   | 0.3:1| 7.2 ± 1.8                       | 15.8 ± 1.9     | 2.1 ± 0.9      |

* ‡ As for Table I.

seen most prominently at high killer:target ratios. Also the difference between 5R and F<sub>1</sub> was greater with anti-D<sup>d</sup> than with anti-K<sup>b</sup> T<sub>c</sub> cells, a result which conforms with the antisera absorption results (Fig. 1).

Differences between F<sub>1</sub> and parental strain macrophage were also investigated in four similar experiments with H-2 restricted T<sub>c</sub> cells. For example, secondary anti-ectromelia T<sub>c</sub>-cell responses were generated in vitro using 2R and 5R responders and their reactivity tested on infected and uninfected targets (Table III). 2R T<sub>c</sub> cells which recognize K<sup>k</sup> and D<sup>b</sup> plus viral antigens killed 2R infected cells as efficiently as F<sub>1</sub> cells over a 10-fold range of killer:target ratios. In contrast, there was approximately three times less lysis of F<sub>1</sub> than 5R infected targets by 5R T<sub>c</sub> cells. Similar results have
Differences in H-2 Expression in F\textsubscript{1} Hybrids

### Table III

**Recognition of H-2 Antigens in (5R × 2R)F\textsubscript{1} Targets by H-2-Restricted Anti-Ectromelia Tc Cells**

| Secondary* anti-ectromelia Tc cells | Specific \(^{51} \text{Cr} \) release from macrophage targets‡ | B10.A(2R) | B10.A(5R) | [B10.A(5R) × B10.A(2R)] |
|-----------------------------------|-------------------------------------------------|-------------|-------------|--------------------------|
|                                   | K\textsuperscript{d} \times K\textsuperscript{d} | K\textsuperscript{d} \times K\textsuperscript{d} | K\textsuperscript{d} \times K\textsuperscript{d} | K\textsuperscript{d} \times K\textsuperscript{d} |
| Infected                          | Uninfected                                      | Infected    | Uninfected  | Infected                |
| BI0.A(2R)                         | 3:1                                             | 57.1 ± 0.2  | 123 ± 0.8   | 61.4 ± 1.1              |
|                                  | 1:1                                             | 63.0 ± 0.7  | 80 ± 1.7    | 45.8 ± 0.7              |
|                                  | 0.3:1                                           | 34.0 ± 1.0  | 0           | 37.8 ± 1.4              |
| BI0.A(5R)                         | 1:1                                             | 60.2 ± 2.1  | 12.9 ± 1.7  | 62.9 ± 2.0              |
|                                  | 0.3:1                                           | 62.5 ± 2.3  | 6.6 ± 0.5   | 41.7 ± 1.7              |
|                                  | 0.1:1                                           | 48.2 ± 1.8  | 4.1 ± 1.6   | 36.4 ± 1.5              |
| BI0.A(2R)                         | 1:1                                             | 68.2 ± 2.1  | 12.9 ± 1.7  | 62.9 ± 2.0              |
|                                  | 0.3:1                                           | 62.5 ± 2.3  | 6.6 ± 0.5   | 41.7 ± 1.7              |
|                                  | 0.1:1                                           | 48.2 ± 1.8  | 4.1 ± 1.6   | 36.4 ± 1.5              |

* 10\textsuperscript{7} ectromelia-infected spleen stimulators were cultured for 5 d with 10\textsuperscript{8} syngeneic spleen responders from mice preprimed intravenously with 10\textsuperscript{4} PFU hamptoned egg strain virus.

† As for Table I.

### Table IV

**Tc Cell Stimulation by H-2 Antigens of the (5R × 2R)F\textsubscript{1}**

| Spleen* responders | Spleen stimulators | Fraction of culture assayed | Specific \(^{51} \text{Cr} \) release from macrophage targets‡ | B10.A(5R) | B10.A(2R) |
|--------------------|--------------------|-----------------------------|-------------------------------------------------|-------------|-------------|
|                    |                    |                             | K\textsuperscript{d} \times K\textsuperscript{d} | K\textsuperscript{d} \times K\textsuperscript{d} | K\textsuperscript{d} \times K\textsuperscript{d} |
| BI0.A(2R)          | BI0.A(2R) × BI0.A(2R) | 0.15                        | 22.0 ± 3.2  | 65 ± 1.6     | 0           |
|                    | [K\textsuperscript{d} \times K\textsuperscript{d}] | 0.08                        | 10.6 ± 1.7  | 21 ± 2.1     | 1.8 ± 0.3   |
|                    |                    | 0.04                        | 9.5 ± 1.1   | 20 ± 0.8     | 0.5 ± 0.7   |
| BI0.A(5R)          | BI0.A(5R)          | 0.15                        | 38.4 ± 0.6  | 39.0 ± 1.8   | 5.0 ± 1.7   |
|                    | [K\textsuperscript{d} \times K\textsuperscript{d}] | 0.08                        | 32.6 ± 2.7  | 18.6 ± 1.8   | 2.9 ± 1.5   |
|                    |                    | 0.04                        | 16.9 ± 2.3  | 5.1 ± 0.6    | 0           |
| BI0.A(5R)          | BI0.A(2R) × BI0.A(2R) | 0.15                        | 0           | 0           | 31.2 ± 2.4  |
|                    | [K\textsuperscript{d} \times K\textsuperscript{d}] | 0.08                        | 0           | 0           | 16.6 ± 0.7  |
|                    |                    | 0.04                        | 0           | 0           | 7.1 ± 1.5   |
| BI0.A(5R)          | BI0.A(2R)          | 0.15                        | 0           | 0           | 30.3 ± 0.5  |
|                    | K\textsuperscript{d} \times K\textsuperscript{d} | 0.08                        | 0           | 0           | 29.5 ± 2.2  |
|                    |                    | 0.04                        | 0           | 0           | 9.2 ± 0.9   |

* ‡ As for Table I. Cells from a pool of two mice were used as both responders and stimulators in MLR.

§ Cultures employ splenic responders at a concentration of 2 × 10\textsuperscript{5} c/ml and diluting numbers of γ-irradiated spleen stimulators. Triplicate 2-ml cultures were pooled after 5 d and a given fraction of input cells assayed.

## Ability of Spleen Cells from Parental Strain and F\textsubscript{1} Hybrid Mice to Stimulate Production of Tc Cells in MLR.

Irradiated spleen cells from 2R, 5R, and the (5R × 2R)F\textsubscript{1} hybrid were used as stimulator cells in an MLR using either 2R or 5R responders. F\textsubscript{1} cells were compared with either 2R or 5R cells, for their ability to stimulate a response in the opposite parent. The stimulating ability of limiting numbers of cells was investi-
gated in terms of the effector T-cell activity generated for a given initial responder cell population. F₁ cells were as efficient as 2R cells in producing an anti-2R response, but they were significantly inferior to 5R cells in stimulating an anti-5R T-cell response (Table IV). The data shown is from one out of two similar experiments.

Discussion

The results of this investigation indicate that less 5R-derived H-2K and H-2D antigenic determinants are expressed on the surfaces of peritoneal macrophages and spleen cells of heterozygous (5R × 2R)F₁ hybrid mice than similar cells of 5R homozygotes. The difference is selective and not due to heterozygosity per se, because H-2K and H-2D antigens coded by the 2R haplotype were apparently expressed in similar concentrations on cells from (5R × 2R)F₁ and 2R mice. Other preliminary results show that some mouse strain combinations in F₁ hybrids exhibit this differential expression of parentally-derived H-2 antigens, (e.g. CBA/H × Balb/c) while others show even-handed expression (e.g. CBA/H × C57B1/6J). Galfre and co-workers (18) have seen a similar difference in MHC antigen expression between DA and (HO × DA)F₁ hybrid rats. In their case, the two strains of rats had different genetic backgrounds, so that the location or nature of the genes which regulate expression of MHC genes is unknown. In this report, two congenic mouse strains which differ only in the region of chromosome 17 carrying the H-2 gene complex have been used, which raises the possibility of H-2-linked regulation of H-2 gene expression.

Several workers have reported selective depression of MHC-coded antigen expression on the surfaces of cell lines cultured in the presence of anti-MHC antibody (19, 20). It is interesting that antibody specific for one out of four H-2K and H-2D antigens expressed on murine F₁ tumor cells depressed the expression not only of that antigen, but in some cases also depressed the antigen coded by the K and D gene in the cis position. This observation conforms with ours in that both involve reduced expression of the K and D genes of one parental chromosome of the F₁ hybrid, but whether this reflects a common mechanism is unknown. One obvious area under investigation is the expression of maternal versus paternal antigens.

Apart from its intrinsic interest as a case study for the regulation of H-2 antigen expression, the phenomenon reported here has implications for the induction and expression of T-cell responses in which specific recognition of antigens coded by the MHC is crucial. For example, experiments in which macrophages were used as targets for lysis by alloreactive or H-2-restricted Tc cells showed that there was less lysis of (5R × 2R)F₁ targets than the 5R targets by anti-5R effector Tc cells, but similar lysis of F₁ and 2R targets by anti-2R Tc cells. The differences between 5R and F₁ were variable, but generally conformed with the differences in H-2K and H-2D antigen concentrations between spleen cells and 5R and (5R × 2R)F₁ absorption experiments with specific anti-H-2 sera.

There was also a clear effect on the ability of spleen cells to stimulate Tc-cell responses. The data in Table IV show that alloreactive Tc cells specific for H-2 antigens coded by the 5R haplotype were less efficiently stimulated by (5R × 2R)F₁ cells than by 5R cells, whereas 2R and (5R × 2R)F₁ cells were of similar efficiency in stimulating Tc cells specific for the H-2 antigens coded by the 2R haplotype. The ability of F₁ hybrid cells to stimulate H-2 restricted Tc-cell responses against various viruses is being investigated. F₁ hybrids may not necessarily be at a selective advantage
as proposed by Doherty and Zinkernagel (21, 22) simply because they express a wider repertoire of H-2 antigens than homozygotes. An F1 hybrid with some weakly expressed H-2 antigens could be a poor responder with respect to H-2 restricted Tc cells which recognize these antigens.

The concept that H-2 antigen concentration on stimulator cell surfaces is a crucial variable determining the strength of T-cell responses is relevant to two other phenomena. First, Egorov et al. (23) have reported several examples of graft versus-host reactions against H-2 antigens coded by a given haplotype that were weaker when the reaction was stimulated by a semihistocompatible heterozygote rather than a homozygote. This otherwise puzzling result is readily explained by a decreased concentration of parentally-derived H-2 antigens on the cells of F1 hybrids. Second, Lafferty and colleagues (24) have observed that survival of allografted thyroid tissue can be spectacularly prolonged if measures, such as organ culture, are taken to remove passenger lymphomyeloid cells from the tissue before grafting. Since histocompatibility antigens are generally expressed in higher concentrations on lymphomyeloid cells than other cell types (25), our results (Table IV) may partly explain why lymphomyeloid cells are potent stimulators of allogeneic T-cell responses (26), and why the response to precultured thyroid grafts is weak or absent.

Summary
Quantitative absorption with specific anti-H-2 sera has shown that the H-2K^b and H-2D^d antigens coded by the B10.A(5R) haplotype are expressed in about fourfold lower amount on the spleen cells of [B10.A(5R) × B10.A(2R)]F1 hybrids than on parental B10.A(5R) cells. In contrast, the H-2K^k and H-2D^b antigens of B10.A(2R) are expressed equally on parental and F1 cells. These quantitative differences are reflected in cytotoxic T-cell (Tc-cell) function. Macrophage target cells from F1 mice are killed less efficiently than B10.A(5R) targets by alloreactive or H-2-restricted Tc cells specific for H-2K^b or H-2D^d, and spleen cells of F1 mice are less efficient stimulators of alloreactive Tc cells specific for B10.A(5R) H-2 antigens, whereas F1 and B10.A(2R) cells are equal as targets and stimulators for Tc cells recognizing B10.A(2R) H-2 antigens.

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