sponses to space segment requirements for dual-polarization capability at 6/4 GHz, and capability at 14/11 GHz. In a recent development, these two technologies have been combined; the new U.S. earth station at Roaring Creek has an antenna system that provides dual polarization at 6/4 GHz and at 14/11 GHz.

Another development, made by COMSAT, is a multibeam earth station antenna (torus antenna), which can operate with several satellites simultaneously, using a single reflector structure. Substantial improvements have been achieved in the radiating efficiency of earth station antennas and in the suppression of unwanted side-lobes, which are a major source of interference between geostationary satellite networks.

Transmission technology. In this area the major advance was the development of time division multiple access coupled with digital speech interpolation (DSI). TDMA allows a mix of low- and high-capacity digital signals to be routed dynamically to any desired satellite beam, eliminating the multicarrier penalties and carrier-splitting requirements associated with frequency division multiple access (FDMA). DSI is a capacity multiplication technique in which pauses in human speech during one telephone conversation are used to transmit speech elements from other conversations. An earlier transmission mode known as SPADE (*s*ingle channel per carrier, *p*ulse code modulation, multiple *a*ccess, *de*mand assignment *e*quipment) applied speech statistics collectively to a transponder entirely loaded with speech-activated single-voice channel transmissions through global coverage beams, providing a fully interconnective multiple access capability for small traffic links.

While FDMA, for years the standard access mode in the INTELSAT system, is capable of providing a transmission density of about 15 voice channels per megahertz of bandwidth, SPADE and TDMA/DSI can provide more than 40 equivalent voice channels per megahertz. TDMA/DSI operation will begin in 1984 and is expected to become the predominant transmission mode. Equivalent voice channel capacity will be further increased through the introduction of bandwidth compression techniques (source coding), which will strip speech and video signals of redundant content and allow operation with a lower demand on bandwidth (lower bit rate) and no undue sacrifice in transmission quality. The present coding rate for speech, 64 kilobits per second, is expected to be reduced by a factor of 2 and later in this decade by a factor of 4.

Systems technology. These advances have been mainly in the area of computer software development for the analysis and synthesis of systems planning alternatives. The increasing interference between networks engendered by multiple frequency reuse required extensive measurements and modeling to develop analysis programs. Similarly, the growing complexity of INTELSAT system transmission planning led to a need for automation. Satellite designs require consideration of actual traffic and loading scenarios; computer programs have become indispensable for system design and planning and for determining actual frequency assignments and optimizing satellite deployment sequences.

Current plans to increase system capability emphasize more efficient access and multiplication techniques rather than added satellite capacity. However, traffic growth is expected to require expanded satellite capacity in the 1990's. If a trend toward smaller and more customized spacecraft is followed, this would fundamentally alter the architecture of the INTELSAT system.

Conclusions

The technology base from which INTELSAT embarked to develop a global communications system was substantial, and each new generation of satellites has incorporated new technological advances. The continued demand for growth and diversification of services is reflected in a broad range of ongoing studies, which include areas of investigation such as 30/20-GHz technology, intersatellite link technology, onboard signal processing, advanced modulation and multiple access techniques, higher frequency-reuse techniques, interference suppression and cancellation, dynamic satellite beam coverage generation, and advanced coding techniques for information compression and extension of performance.

References and Notes

1. B. I. Edelson and L. Pollack, *Science* **195**, 1125 (1977).
2. Hodocentric means "as seen from a point on the orbit."

RESEARCH ARTICLE

Chromosome 4 *Jt* Gene Controls Murine T Cell Surface I-J Expression

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In vertebrates the immune system protects the animal from disease-causing organisms, such as viruses and bacteria, by producing an immune response to eliminate foreign intruders. Certain lymphocytes regulate both the magnitude and type of immune response. Helper T lymphocytes enhance and suppressor T lymphocytes diminish immunity in a highly selective manner. These regulatory lymphocytes are crucial to survival.

The I-J structure on a membrane glycoprotein distinguishes suppressor T lymphocytes from all other cells (*1*). This same structure comprises part of secreted glycoproteins with suppressive activity (*1*). Antibodies have been produced that react with the I-J structure; these bind to the suppressor T cell surface and to the secreted glycoproteins (*1*).

A gene cluster termed the major histocompatibility complex has been preserved throughout vertebrate evolution. In the mouse, it is known as the H-2 complex, and the products of these genes function by permitting the animal to discriminate between self and nonself (*2*). Genetic mapping experiments placed

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Summary. Data are presented suggesting a resolution to the paradox concerning the murine response subregion I-J, which encodes a suppressor T cell marker. The controversy arose when sequences corresponding to I-J DNA were not found in the central immune response region described by immunogeneticists. New evidence is presented that T cell surface I-J expression results from the action of at least two complementing genes. One gene is within the H-2 region on chromosome 17; the second gene, termed *Jt*, is on chromosome 4. The two recombinant mouse strains B10.A(3R) and B10.A(5R) originally used to define the I-J subregion apparently differ not within the H-2 region but elsewhere.

the I-J controlling gene in the central I region of H-2 (*1*). In the most refined of these genetic studies, H-2 recombinant strains B10.A(3R) (3R) and B10.A(5R) (5R) were used. Since these strains are H-2 congenic with their parent strain C57BL/10, they are ostensibly identical throughout the portion of their genome other than H-2 (*3*). The 5R cells express I-J^k molecules (k refers to a set of H-2 alleles termed a haplotype), whereas the 3R cells do not. This observation was taken to imply that the locus encoding I-J molecules mapped in a discrete I subregion, its boundaries formed by the crossover points in strains 3R and 5R (*1*).

A controversy arose when DNA from the central I region of H-2 was cloned and analyzed. No difference between 3R and 5R DNA was found (*2, 4*). Thus, I-J DNA sequences could not be identified; the molecular map of the I region could not be reconciled with the genetic map.

Several hypotheses have been proposed to resolve this paradox. One denies the existence of I-J (*5*), while others propose complex molecular options (*2*). The critical assumption that 3R and 5R differ only within H-2 is not justifiable. The two strains apparently differ in their "non–H-2 genes." We discovered a locus required for I-J^k expression in the C57BL/10 background and we have mapped this locus to chromosome 4. The chromosome 4 locus, termed Jt, together with a locus in H-2 (probably I-E) controls I-J^k expression on T lymphocytes.

Table 1. Monospecific antiserum and monoclonal antibodies react with an I-J^k T cell determinant. The T cells were prepared by passing nonadherent lymph node cells through a column of Sepharose coupled with goat antibody to immunoglobulin (Ig) (*8*). A two-stage, dye-exclusion microcytotoxicity assay was performed as described (*7*), except that with WF8.C12.8 monoclonal antibodies, antibody to mouse IgG_1 was added after excess monoclonal antibody had been removed by washing. The percentage of specific lysis was corrected for background lysis by complement (less than 10 percent). Specific antiserum to Thy-1.2 was produced by immunizing AKR mice with C3H thymocytes (*9*). Specific antiserums to I-J^k was produced by immunizing B10.A(3R) mice with B10.A(5R) T cells (*8*). Monoclonal antibody 1 was WF8.C12.8 (*10*); monoclonal antibody 2 was Jk.18 (*11*).

T cell donor strain	I region A J E	Percentage of cells lysed*			
		Thy-1.2	Specific antibody to I-J^k		
			Anti-serum	Mono-clonal 1	Mono-clonal 2
B10.A	k *k* k	81 ± 5	23 ± 3	14 ± 3	17 ± 1
C57BL/10	b b b	78 ± 9	0 ± 1	1 ± 1	0 ± 0
B10.A(3R)	b b k	85 ± 6	1 ± 2	0 ± 1	0 ± 0
B10.A(5R)	b *k* k	79 ± 6	13 ± 1	14 ± 6	11 ± 1
B10.HTT	s s k	82 ± 3	0 ± 0	0 ± 1	0 ± 1
B10.S(9R)	s *k* k	86 ± 2	13 ± 3	10 ± 2	15 ± 3
A.TH	s s s	79 ± 4	1 ± 1	0 ± 0	0 ± 0

*Mean ± standard error of the mean (S.E.M.); three or more experiments.

Table 2. *H-2*^{k} genes are not sufficient to produce I-J^k determinants. T cells were prepared and assayed as in Table 1. Specific antiserum to Thy-1.1 was used with strains AKR/J and RF/J. Antiserum to Thy-1.2 was used with other strains (*9*). Antibodies to H-2K^k and to H-2D^k were produced by immunizing (A.TL × B10.D2)F_1 mice with B10.BR lymphocyte (*8*). Antibodies to I-J^k were as described in Table 1.

T cell donor strain	Percentage of cells lysed*				
	Thy-1	K^{k}D^k	I-J^k		
			Anti-serum	Monoclonals	
				1	2
AKR/J	83 ± 1	98 ± 2	1 ± 1	0 ± 0	0 ± 0
B6-H-2^k	80 ± 3	98 ± 2	13 ± 5	12 ± 2	14 ± 2
B10.BR	78 ± 3	97 ± 1	16 ± 4	10 ± 1	12 ± 4
CBA/J	89 ± 3	97 ± 2	22 ± 4	11 ± 3	12 ± 3
CE/J	85 ± 3	96 ± 2		0 ± 0	0 ± 0
C3H/HeJ	86 ± 3	97 ± 2	20 ± 7	12 ± 3	12 ± 1
C58/J	85 ± 3	97 ± 1		15 ± 2	13 ± 1
MA/MyJ	90 ± 1	97 ± 1		13 ± 2	10 ± 3
MRL/MpJ$^+$	84 ± 1	97 ± 1	13 ± 5	11 ± 2	13 ± 2
RF/J	81 ± 9	96 ± 2		11 ± 2	11 ± 1

*Mean ± S.E.M.; three or more experiments.

Non–H-2 Genes and I-J^k

The original definition of the I-J^k structure depended on a characteristic reactivity pattern of specific antibodies with H-2 recombinant strain T cells (*1*). The I-region genes of these strains and their reaction with a monospecific antiserum and two monoclonal antibodies are indicated in Table 1. Both the antiserum and the monoclonal antibodies are I-J^k–specific in that they exhibit the accepted strain reactivity pattern (*1*).

Molecular cloning experiments raised the possibility that the *I-J*^{k} structural gene might actually be outside H-2 (*2*). To test this possibility, we examined I-J^k determinant expression on T cells from ten strains; each strain has a k haplotype H-2 complex (*H-2*^{k}), but they all derive their non–H-2 genes from different sources. If the *I-J*^{k} structural gene is in *H-2*^{k}, these strains should not differ in their ability to produce an I-J^k molecule. In contrast, if the *I-J*^{k} gene is outside H-2, some strains might not produce I-J^k structures. Two strains, AKR/J and CE/J, did not express I-J^k determinants on their T lymphocytes (Table 2). The *H-2*^{k} genes are therefore not sufficient to produce I-J^k molecules.

Four H-2 congenic strains permitted examination of H-2 and non–H-2 contributions to I-J^k expression (Fig. 1A). Strain AKR was backcrossed 20 times to strain C57BL/6 (B6) (*6*). At each step of the backcross, an animal with the AKR H-2^k complex was selected for further backcrossing. The resulting strain, B6-H-2^k, derives its *H-2*^{k} genes from AKR and its non–H-2 genes from B6 (Fig. 1A). Similarly, strain AKR-H-2^b derives its *H-2*^{b} genes from B6 and its non–H-2

genes from AKR. The B6-H-2^k T cells express I-J^k molecules, whereas the other three members of this group of four strains do not (Table 3). We conclude that at least two genes are necessary for the I-J^k positive phenotype: the first gene is in *H-2*^{k} and is present in AKR and B6-H-2^k, the other gene or genes are outside the H-2 complex and are present in C57BL/6 and B6-H-2^k.

Whereas I-J^k expression is not governed by a single dominant *H-2*^{k} gene, expression does depend on some k haplotype gene in H-2. We previously tested seven other H-2 haplotypes (b, d, f, p, q, r, and s), each having non–H-2 genes from C57BL/10; these seven haplotypes do not express I-J^k determinants (*7*). Conversely, all strains that express I-J^k derive some portion of H-2 from the k haplotype (Table 1) (*1, 7*). Results obtained with strain B10.A(4R) (I-A^k, I-E^b) (*8, 11*), together with the observation that all strains expressing I-J^k are *I-E*^{k}, suggest that I-E rather than I-A subregion genes control I-J^k expression. Thus I-J^k expression results from the concerted action of an *H-2*^{k} gene and a non–H-2 gene or genes.

C57BL/10 H-2 Congenic Strains

Alerted to the fact that non–H-2 genes also regulate I-J^k expression, we asked why the 3R strain fails to produce I-J^k structures, and whether 3R lacks the requisite *H-2*^{k} locus or expresses inappropriate alleles among its non–H-2 genes. Accordingly, we made F_1 hybrids between I-J^k negative strains (Table 3). Expression of I-J^k by (C57BL/10 × AKR)F_1 hybrid T cells indicated successful gene complementation between two heterozygous loci (Table 3); C57BL/10 contributed the requisite non–H-2 gene (or genes), whereas AKR provided

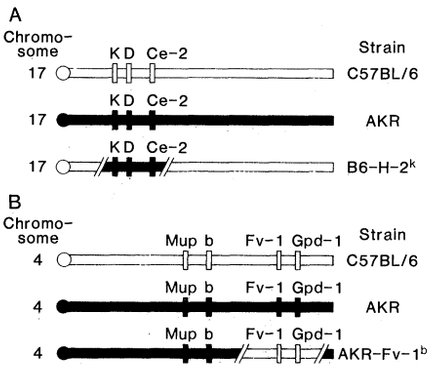

Fig. 1. Chromosome structure of AKR–C57BL/6 congenic strains. (A) Chromosome 17; (B) chromosome 4.

the *H-2*^{k} locus. Since (3R × C57BL/10)F_1 T cells produce I-J^k molecules, whereas (3R × AKR)F_1 T cells do not, 3R can contribute the needed *H-2*^{k} gene (lacking in C57BL/10), but lacks the non–H-2 I-J^k–controlling gene or genes (present in C57BL/10). This experiment suggests that non–H-2 genes of 3R may not be identical (that is, not congenic) to C57BL/10. Recombinant strains 3R and 5R arose separately when A/WySn was backcrossed to C57BL/10 (*3*). The 3R animal was discovered among progeny of the seventh backcross (*3*). As few as three backcrossings of recombinant 3R to C57BL/10 preceded intercrossing to establish the homozygous inbred 3R line (*12*). The 3R may retain A/WySn background genes which do not permit I-J^k expression that have been replaced by C57BL/10 genes in 5R. Strain A/WySn does not express I-J^k molecules.

Usually H-2 congenic strains are produced by crossing the H-2 donor (such as A/WySn) with the background strain (C57BL/10); progeny expressing the donor H-2 type are subsequently backcrossed to the background strain (*13*). Backcrossing is usually terminated after ten backcross generations (*13*). Some "passenger genes" of donor strain origin, unlinked to H-2, may be retained in such congenics. After ten backcrosses, the probability of retaining an unlinked donor gene is 0.2 percent; an average of 3 centimorgans (cM) of donor DNA in addition to the H-2 gene complex would be retained in the H-2 congenic line (*13*). Genes in quasi-linkage with *H-2* would show a higher probability of retention (*14*). The 3 cM of persisting donor DNA in an H-2 congenic may contain active loci, and cannot be ignored in interpreting experiments in which H-2 congenics are used. That 3R may not be fully H-2 congenic with C57BL/10 may account for the conclusion that a single autosomal dominant gene in the I subregion controls I-J expression (*1*).

The Chromosome 4 *Jt* Gene

Using AKXL recombinant inbred mice, we investigated the non–H-2 locus regulating I-J^k expression. These 18 strains derive genetic information from AKR and C57L parent strains (*15*). The parental chromosomes are randomly assorted in AKXL strains; each locus is homozygous as a result of generations of intercrossing (*16*). Neither parent strain AKR nor C57L expresses I-J^k molecules (Table 4), whereas the (AKR × C57L)F_1 does (not shown). The H-2 types reported in Table 4 agree with published results (*17*). The AKXL *Thy-1* alleles (Table 4) are concordant with unpublished experiments (*18*). Of 18 strains tested, only strains 13, 14, 28, and 38 expressed the I-J^k molecule on the T cell surface (Table 4). These four animals are H-2^k; no H-2^b mice express I-J^k determinants.

To determine the linkage group of the required non–H-2 gene (or genes), we

Table 3. Genes in the C57BL/6 and C57BL/10 background control I-J^k determinant expression. The T cells were prepared and assayed as in Table 1. Antibodies to Thy-1.1, Thy-1.2, K^{k}D^k, and I-J^k were as described in Tables 1 and 2. Antibodies to H-2K^b and to I-A^b were produced by immunizing (A × B10.D2)F_1 mice with B10.A(5R) lymphocytes (NIH typing serum). The F_1 hybrid T cells were treated with neuraminidase, which increases the percentage of T lymphocytes lysed by I-J^k-specific antibodies three- to fivefold, without altering the strain distribution pattern shown in Table 1 (*11*).

T cell donor strain	Genes		Percentage of cells lysed						
	H-2	Non–H-2	Thy-1.1	Thy-1.2	K^{k}D^k	K^{b}A^b	I-J^k		
							Anti-serum	Mono-clonal 1	Mono-clonal 2
C57BL/6	b	+	0 ± 0	77 ± 3	0 ± 0	98 ± 1	0 ± 0	2 ± 1	0 ± 1
AKR/J	k	−	83 ± 3	0 ± 1	98 ± 2	0 ± 0	1 ± 1	0 ± 0	0 ± 0
B6-H-2^k	k	+	0 ± 0	80 ± 3	98 ± 2	0 ± 0	13 ± 5	12 ± 2	12 ± 1
AKR-H-2^b	b	−	82 ± 3	0 ± 1	0 ± 0	98 ± 1		0 ± 0	0 ± 0
(B10 × AKR)F_1	b/k	+/−		88 ± 2				17 ± 3	25 ± 4
(3R × B10)F_1	i3/b	−/+		89 ± 3				21 ± 4	23 ± 1
(3R × AKR)F_1	i3/k	−/−		89 ± 3				0 ± 2	0 ± 3

*Mean ± S.E.M.; three or more experiments.

searched for a previously reported gene that is C57L-type in each AKXL animal that expresses I-J^k, but AKR type in each nonexpressor. None of the chromosome 1, 2, 6, 9, 11, 12, or 17 loci show the same distribution pattern as the I-J^k determinant (*19–25*). In contrast, the strain distribution patterns for the *b* coat color locus on chromosome 4 and for the *Akv-1* gene on chromosome 7 match the I-J^k positive strain distribution pattern (*26–28*). Animals 13, 14, 28, and 38 are C57L-type at *b* and *Akv-1* and express I-J^k determinants. Animals 6, 8, and 21 are AKR-type at these loci and do not express I-J^k. Thus, the I-J^k–controlling locus map position was narrowed to chromosome 4, chromosome 7, or some chromosome for which strain distribution patterns in AKXL mice have not been published.

Further experiments allowed us to place an I-J^k–controlling locus on chromosome 4. An AKR congenic strain, AKR-Fv-1^b, was produced by crossing AKR with C57BL/6 and then backcrossing to AKR (Fig. 1B) (*29*). This strain has a segment of B6 DNA on chromosome 4; the *Fv-1*^{b} locus served as a marker for this B6 segment since AKR carries the *Fv-1*^{n} allele (*23*). We tested I-J^k expression in AKR-Fv-1^b mice from the 11th backcross generation (Fig. 2); the AKR-Fv-1^b genome exclusive of the chromosome 4 segment is presumably from AKR. The AKR-Fv-1^b T cells produce I-J^k molecules, whereas the parent strains AKR and B6 do not (Fig. 2).

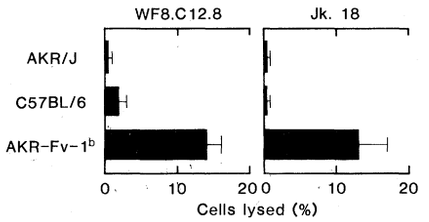

Fig. 2. T lymphocytes from mouse strain AKR-Fv-1^b express I-J^k determinants. The T cells were prepared and assayed as described in Table 1; WF8.C12.8 and Jk.18 are I-J^k-specific monoclonal antibodies.

These mice should have approximately 10 cM of B6 DNA on either side of *Fv-1*^{b} (Fig. 1B) (*30*); this DNA segment is sufficient to produce I-J^k expression. We conclude that the non–H-2 gene required for I-J^k expression is on chromosome 4 in linkage group VIII. We think it is likely that a single autosomal dominant chromosome 4 gene regulates expression of T cell I-J^k, and have named this gene *Jt*. The *Jt*$^+$ allele is exemplified by strains C57BL/6, C57BL/10, and C57L; it permits I-J^k expression. The *Jt*$^-$ allele is carried by strain AKR [and perhaps B10.A(3R)] and does not permit I-J^k expression.

The *Jt* gene may be placed more precisely on chromosome 4 by establishing boundaries within which the locus must reside. The I-J^k expression in AKXL mice did not correlate with the *Mup-RF* locus; this gene is about 4 cM to the left (centromeric) of *b* (Fig. 1B) (*31*). Likewise, I-J^k expression did not correlate with *Gpd-1* which is about 33 cM to the right of *b* (Fig. 1B) (*31*). Therefore, *Jt* must be positioned between *Mup-RF* and *Gpd-1*. *Fv-1* is just 1 cM to the left of *Gpd-1* (Fig. 1B) (*31*); the B6 DNA segment in AKR-Fv-1^b mice extends about 10 cM to the left and to the right of *Fv-1* and includes *Jt*. Taken together, these results place *Jt* within a 10 cM DNA segment to the left of *Gpd-1*.

Gpd-1 encodes an enzyme, glucose-6-phosphate dehydrogenase, for which there are electrophoretic variants; strain A carries the *Gpd-1*^{b} allele, and strain C57BL/10 carries the *Gpd-1*^{a} allele. In strains 3R, 5R, and B10 the electrophoretic mobilities of kidney glucose-6-phosphate dehydrogenase were identical (not shown). If 3R and 5R differ in some chromosome 4 segment that includes *Jt*, this segment does not extend to *Gdp-1*.

We also examined the possibility that 3R and 5R might differ at other loci associated with suppressor T cell determinants. The Lym-22 antigen demarcates a suppressor T cell population; it is the only antigen other than I-J known to be restricted to suppressor T cells (*32*). The Lyt-2 marker is found on suppressor and cytotoxic T lymphocytes (*33*). The 5R and 3R T cells were lysed equally well by monoclonal antibodies to Lyt-2 and to Lym-22 (not shown).

Table 4. Expression of I-J^k in AKXL mice. The T cells were prepared and assayed as described in Table 1. Specific antibodies were as described in Tables 1, 2, and 3.

T cell donor strain	Percentage of cells lysed*					
	H-2		Thy-1		I-J^k	
	K^{k}D^k	K^{b}A^b	Thy-1.1	Thy-1.2	Monoclonal 1	Monoclonal 2
AKR/J	98 ± 2	0 ± 0	83 ± 3	0 ± 1	0 ± 0	0 ± 0
C57L/J	0 ± 0	92 ± 0	0 ± 0	88 ± 1	0 ± 1	0 ± 1
5	0 ± 0	98 ± 1	84 ± 2	0 ± 0	0 ± 0	0 ± 0
6	98 ± 1	0 ± 0	1 ± 1	81 ± 1	0 ± 0	0 ± 0
7	0 ± 0	95 ± 2	0 ± 0	87 ± 0	0 ± 0	0 ± 0
8	97 ± 0	1 ± 2	83 ± 3	0 ± 0	3 ± 2	1 ± 1
9	0 ± 0	98 ± 2	0 ± 1	92 ± 1	0 ± 0	0 ± 0
12	0 ± 0	96 ± 1	0 ± 0	89 ± 1	0 ± 1	0 ± 0
13	98 ± 2	1 ± 1	80 ± 4	2 ± 1	13 ± 1	13 ± 1
14	99 ± 1	0 ± 0	2 ± 1	82 ± 3	11 ± 2	11 ± 2
16	1 ± 0	92 ± 2	1 ± 1	86 ± 3	0 ± 1	0 ± 1
17	0 ± 0	97 ± 2	63 ± 5	2 ± 2	0 ± 0	1 ± 2
19	0 ± 1	95 ± 2	0 ± 1	90 ± 0	0 ± 0	0 ± 1
21	98 ± 1	0 ± 0	0 ± 0	88 ± 0	0 ± 0	0 ± 0
24	0 ± 0	91 ± 1	84 ± 2	0 ± 1	0 ± 0	0 ± 0
25	1 ± 1	95 ± 2		90 ± 1	0 ± 1	0 ± 0
28	98 ± 1	0 ± 0		91 ± 0	11 ± 2	12 ± 3
29	0 ± 0	93 ± 5	82 ± 2	4 ± 4	0 ± 1	0 ± 0
37	0 ± 0	90 ± 1	3 ± 3	87 ± 2	0 ± 0	0 ± 1
38	93 ± 3	0 ± 1	82 ± 3	2 ± 2	15 ± 2	10 ± 4

*Mean ± standard deviation; one representative experiment of 2 or 3.

Models for I-J^k Molecules

A number of options have been proposed to explain how the *H-2*^{k} and *Jt* genes work together to produce the molecule which carries I-J^k determinants (*2*). Our results suggest it is unlikely that DNA rearrangements occur in T lymphocytes to juxtapose the *H-2* and *Jt* genes such that a single transcript might encode a polypeptide with two domains, since these genes are on different chromosomes. Results of other investigators indicate that an association between two separate polypeptides is not required to form I-J^k determinants; I-J^k epitopes occur on single polypeptide chains (*34, 35*).

At least two equally plausible models remain. First, one gene product may be an enzyme that glycosylates, phosphorylates, acylates, or otherwise modifies the second gene product. If this is the case, the *Jt* gene product is most probably enzymatically active, whereas the *H-2* gene is most likely the structural gene. No I-J molecules have been detected on B cells or their products but I-region structural genes are fully active in B lymphocytes (*2*). The *Jt* gene may be active only in differentiated T lymphocytes; I-J^k molecules might then be

formed from E_α or E_β polypeptides (I-E gene products) to which are attached carbohydrate chains that are different in T cells from those in B cells or macrophages. Recent evidence was taken to imply that I-J^k and E^k_β determinants were on the same polypeptide (*36*). However, the monoclonal antibody used in experiments from which this conclusion was drawn binds to a structure formed only when E_α and E_β polypeptides associate (*37*). Furthermore, conditions that would dissociate noncovalently bound polypeptides were not used, and therefore it is uncertain whether a single polypeptide accounted for the results obtained. Extracts from suppressor T cell lines have not yielded messenger RNA (mRNA) for E_α or E_β polypeptides (*38*). However, I-J^k expression in these cells is cell-cycle dependent, and was not demonstrated at the time of mRNA extraction (*38, 39*). Finally, the molecular weights of E_α or E_β polypeptides are not comparable with those reported for I-J^k determinant-bearing polypeptides (*40–43*). Our results suggest that terminal α-D-mannosyl residues associated with protein are required for the integrity of the epitope recognized by I-J^k–specific monoclonal antibodies (*11*). Thus, available evidence does not yet provide convincing support for the posttranslational modification model.

An alternative hypothesis suggests that one gene might regulate synthesis of the other structural gene product. For example, I-J^k epitopes may be on the *Jt* gene product, their synthesis and expression being controlled by the I-E subregion genes. The I-J^k determinants might reside on the T cell receptor molecule, which binds to self $E_\alpha E_\beta$ complexes on other lymphocytes. The receptor is apparently inducible only in the presence of appropriate H-2 molecules. Genetic control of the receptor, and therefore of I-J^k determinants, would then apparently map to the *H-2* gene encoding the inducing ligand. To accommodate allelic forms of I-J, one must speculate that different receptor molecules are generated according to the ligand structure, either through a multigene system or by adaptive modification of a single gene product. This hypothesis cannot be excluded on examination of available data.

Conclusion

Our experiments show that I-J^k expression is not under the control of a single *H-2*^{k} gene. A chromosome 4 locus, *Jt*, together with an *H-2*^{k} locus (probably I-E) regulates production of I-J^k molecules. H-2–recombinant strains 3R and 5R may differ in their non–H-2 genetic makeup, not in H-2, an observation that is consistent with the I-region molecular map (*2*). Two equally viable models might explain how these genes contribute to the I-J^k–bearing polypeptide. One proposes a posttranslational modification of I-E–encoded polypeptides, the other suggests an inducible T cell receptor structure capable of binding I-E–encoded polypeptides. These theories are experimentally distinguishable. Future studies on the biochemistry of I-J^k polypeptides and the molecular biology of the *Jt* locus should provide a final understanding of the mechanism by which these genes contribute to the immunosuppressive T cell molecules.

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