A MONOCLONAL ANTIBODY THAT DETECTS A V\(\kappa\)-TEPC15 IDIOTYPIC DETERMINANT CROSS-REACTIVE WITH A Thy-1 DETERMINANT*  

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Receptors on lymphocytes play a crucial role in antigen recognition and effector function during an immune response. In the case of the B cell, this antigen receptor has been conclusively identified as immunoglobulin (1, 2). In contrast, despite intensive investigation, the exact identity of the T cell receptor for antigen remains unknown. Some studies using conventional anti-idiotypic antisera have suggested similarities between T cell receptors and the V\(\kappa\) region of immunoglobulin (3, 4), whereas other experiments involving anti-isotypic antisera indicate the presence of \(\kappa\)- (5, 6) and \(\mu\)-like (7, 8) molecules on the surface of T lymphocytes. However, because all such studies suffer from the difficulties associated with the use of poorly defined conventional antisera, the relationship between classical immunoglobulin and the T cell receptor remains a subject of great controversy.

Previous studies on the T cell receptor have used anti-idiotypic or anti-isotypic antiserum prepared against purified immunoglobulin, and subsequently tested against T cells. To allow for a more precise characterization of immunoglobulin-cross-reactive molecules on T cells, we chose an alternative approach: first to prepare monoclonal antibodies against T cells, and then to test these for binding to purified immunoglobulin. In this report, we describe the isolation of one such monoclonal antibody, 42-21, which detects an antigenic determinant shared by the V\(\kappa\) light chain of the TEPC15 (T15)\(^1\) myeloma protein and the Thy-1 antigen on all T cells.

Materials and Methods

Antibodies. 42-21 is a rat IgM hybridoma prepared by immunization against the AKR spontaneous T lymphoma KKT-2. 31-11 is a rat IgG\(\kappa\) monoclonal antibody specific for the constant portion of mouse Thy-1. The preparation of these two antibodies has been described elsewhere (9). 71-11 is a rat monoclonal prepared against the purified T15 myeloma protein. It recognizes a cross-reactive idiotypic determinant present on all phosphorylcholine (PC)-binding myelomas (E. Pillemer and I. Weissman, manuscript in preparation). Monoclonal anti-Lyt-1 (subclone 53.7.13) and anti-Lyt-2 (subclone 53.6.72)-producing hybridoma cell lines were a generous gift of J. Ledbetter, Stanford University (10).

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1 Abbreviations used in this paper: BSA, bovine serum albumin; FACS, fluorescence-activated cell sorter; FCS, fetal calf serum; FITC, fluorescein isothiocyanate; MEM, minimum essential medium; PBS, phosphate-buffered saline; PC, phosphorylcholine; T15, TEPC15.
Myeloma Proteins, Recombined Molecules, Hybridoma Immunoglobulins, and Isolated Heavy and Light Chains. Column-purified myeloma proteins were a generous gift of R. Coffman and C. Nottenburg, Stanford University. The hybridoma anti-PC antibodies M2, M3, and G1 were provided by P. Gearhart, Carnegie Institution of Washington; the chain composition of these antibodies has been determined by amino acid sequence analysis (11). All three hybridomas possess the T15 H chain; M2 has the T15 L chain, M3 has the McPC603 L chain, and G1 has the MOPC167 L chain. Hybridoma anti-PC antibodies representing the two kappa chain markers were a gift of L. Claflin, University of Michigan (12). The three hybridomas are all of the IgM isotype; 221A4 is from an AKR animal (Igk-), 991G2 is from a C58 animal (Igk-), and 552D3 is from a BALB/c animal (Igk-). The isolated heavy and light chains of T15 and W3207 and the McPC603 H/T15 L recombined molecules were obtained from A. Goetze, California Institute of Technology (13). BALB/c immunoglobulin was prepared by 50% ammonium sulfate precipitation from normal serum.

Cell Lines. All cell lines are maintained in culture in our laboratory in RPMI-1640 medium supplemented with 10% fetal calf serum (FCS). The T lymphomas used in this study include: the spontaneous AKR lymphoma KKT-2, the RadLV-induced C57BL/Ka lymphoma VL-3 (obtained from H. Kaplan, Stanford University), the chemical-induced C57BL/6 tumor EL4, and the radiation-induced C57/L lymphoma L691. The non-T tumors used in this study include: the DBA/2 mastocytoma P815, the BALB/c nonproducer myeloma NS-1, and the Abelson virus-induced pre-B cell tumors L1-2 and RAW112, of C57L and BALB/c origin, respectively. The BW5147 parental line (Thy-1+) and the Thy-1− mutants, B5147.6.1 and BW5147.3, were obtained from R. Hyman at The Salk Institute, San Diego, Calif. (14).

Synthetic Labeling of Monoclonal Antibodies. Hybridoma cell lines were obtained from tissue culture and washed twice in Hanks’ balanced salt solution without serum. The cells were resuspended in leucine-free minimum essential medium (MEM) plus 5% dialyzed FCS (MEM-5) and adjusted to 3 × 10⁶/ml. 5 μCi/ml of [³H]leucine was added together with 0.01 ml of 10⁻⁵ M cold leucine to give a final leucine concentration of 10⁻⁵ M. 3 ml of cell solution was plated in petri dishes at 37°C plus 7% CO₂. After 5 h incubation, 0.09 ml of complete MEM-5 was added to each plate. After an additional 30 min, the cell suspension was spun for 6 min at 1,200 rpm to pellet the cells. The supernatant was dialyzed three times against 100× volume of phosphate-buffered saline plus 1 mM sodium azide (PBS-Az), and then clarified by spinning for 10 min at 10,000 rpm.

Myeloma-binding Assays. Solid-phase immunoassays were performed in 96-well flexible microtiter plates (Dynatech Laboratories, Inc., Dynatech Corp., Alexandria, Va.). Wells were coated for 16 h at 4°C with 0.05 ml of a 100 μg/ml solution of [³H]leucine was added together with 0.01 ml of 10⁻³ M cold leucine to give a final leucine concentration of 10⁻³ M. 3 ml of cell solution was plated in petri dishes at 37°C plus 7% CO₂. After 5 h incubation, 0.09 ml of complete MEM-5 was added to each plate. After an additional 30 min, the cell suspension was spun for 6 min at 1,200 rpm to pellet the cells. The supernatant was dialyzed three times against 100× volume of phosphate-buffered saline plus 1 mM sodium azide (PBS-Az), and then clarified by spinning for 10 min at 10,000 rpm.

Cell-binding Assay. Assays were performed in 96-well microtiter plates. Cell suspensions were prepared in cell suspension medium plus 5% FCS (CSM-5) and adjusted to 3 × 10⁶/ml. 2.5 × 10⁶ cells were added to each well together with 0.04 ml of [³H]-labeled hybridoma supernate (25,000–50,000 cpm) was added to each well. After incubation for 2 h at 4°C, the wells were washed and the suspension was spun for 6 min at 1,200 rpm to pellet the cells. The supernatant was dialyzed three times against 100× volume of phosphate-buffered saline plus 1 mM sodium azide (PBS-Az), and then clarified by spinning for 10 min at 10,000 rpm.

Fluorescence-activated Cell Sorter (FACS) Analysis. Cell suspensions of normal lymphoid tissues or tissue culture lines were prepared in CSM-5 at a concentration of 10⁶/ml. 0.01 ml of cells was added to a conical tube together with 0.04 ml of hybridoma antibody. After incubation for 15 min on ice, the cells were washed once over an FCS underlayer. The pellet was resuspended in 0.01 ml of fluorescein isothiocyanate (FITC)-labeled rabbit anti-rat Ig second stage and incubated for 15 min on ice. After a final wash over FCS, the cells were resuspended in 1 ml of PBS-Az for FACS analysis.
Inhibition of Antibody Binding to T15. 0.05 ml of T15 (100 µg/ml) was added to each well of a microtiter plate and incubated for 16 h at 4°C, and the wells were washed three times with PBS-5. For hapten inhibition of 42-21 binding, a PC-bovine serum albumin (BSA) conjugate was prepared using p-diazonium phenylphosphorylcholine (Biosearch, San Rafael, Calif.) according to the method of Cosenza et al (15). Various dilutions of PC-BSA were added to the T15-coated wells and incubated for an additional hour. For antibody inhibition of 42-21 binding, 0.05 ml of unlabeled hybridoma supernate was added to each well. In either case, after 1 h at 4°C, the wells were washed three times with PBS-5, and 0.025 ml of 3H-labeled antibody (25,000-50,000 cpm) was added. After an additional 1 h incubation, the plates were again washed, and the wells were cut with a hot wire and transferred to scintillation vials for counting.

Inhibition of Myeloma Binding to 42-21. In some cases, microtiter plates were coated for 16 h at 4°C with 0.05 ml of purified rabbit anti-rat Ig serum (100 µg/ml) in PBS-Az. The wells were washed three times with PBS-5, and 0.05 ml of hybridoma supernate was added. After incubation for 1 h at 4°C, the wells were washed and then incubated with 0.05 ml of 1% normal mouse serum to block nonspecific binding sites. For specific blocking, dilutions of purified unlabeled hybridoma immunoglobulin were added to the wells for 1 h at 4°C. The wells were again washed and 0.025 ml of 3H-labeled M2 protein (25,000-50,000 cpm) was added to each well for 1 h. After a final wash in PBS-5, the wells were cut and transferred to scintillation fluid for counting.

Antibody Absorption. Cell suspensions for antibody absorptions were prepared by passing normal C57BL/6J mouse tissues through a wire mesh. Labeled monoclonal antibodies to be absorbed were either mixed vol:vol with packed cells, or with a pellet containing a known number of cells. After incubation for 1 h at 4°C, the cells were pelleted again for 6 min at 1,200 rpm. The absorbed supernate was counted directly, or in some cases was tested for residual binding activity on T15-coated microtiter wells. For antibody absorption with myelomas, T15 and normal mouse Ig coupled to Sepharose 4B were obtained from R. Coffman. 0.1 ml of labeled antibody was mixed with 0.05 ml of packed Sepharose and incubated for 1 h at 4°C. The Sepharose was removed by pelleting at 1,200 rpm for 6 min, and the supernate was tested for residual binding to the KKT-2 T cell lymphoma.

Results

Isolation of the 42-21 Monoclonal Antibody. Fisher rats were immunized against the spontaneous AKR T lymphoma, KKT-2, to produce a series of monoclonal antibodies reactive with normal and neoplastic lymphocytes. From the original hybridization, 100 wells were obtained that produced supernatant fluids reactive with KKT-2 cells. Further screening against purified KKT-2 SL virus resulted in 50 of these, which were found to react with viral structural proteins; these were excluded from this study. All but two of the remaining hybrids were found to react with normal T cells and thymocytes, and thus were candidates for further screening against a panel of purified mouse myeloma proteins representing all immunoglobulin isotypes.

In the initial screening, only three hybridoma supernatant fluids bound to any of the myeloma proteins, and all of these showed the same pattern of reactivity: binding to the T15 myeloma (α, κ, PC binding), but to no other myeloma in the panel. Of the three T15-binding hybrids, two were lost before subsequent testing was possible. The remaining hybrid, 42-21, was grown to larger volume in flasks and then subcloned by limiting dilution on a thymocyte feeder layer. Of the 15 subclones of 42-21, only one bound to KKT-2 cells; this same clone was also the only one to bind to the T15 myeloma protein.

Characterization of the 42-21 Antigen on the T15 Myeloma. Preliminary evidence on the nature of the 42-21 antigen was obtained by examining the binding of this antibody to a complete panel of purified myeloma proteins (Table I). Binding was observed for
TABLE I

42-21 Binding to Purified Myelomas

| Myeloma   | Isotype | Binding Specificity | cpm/well $^\S$ |
|-----------|---------|--------------------|---------------|
| T15       | IgA$\kappa$ | PC                | 1530 ± 50     |
| S107      | IgA$\kappa$ | PC                | 1150 ± 50     |
| HOPC8     | IgA$\kappa$ | PC                | 1190 ± 70     |
| MOPC511   | IgA$\kappa$ | PC                | 160 ± 0       |
| McPC603   | IgA$\kappa$ | PC                | 150 ± 20      |
| MOPC167   | IgA$\kappa$ | PC                | 170 ± 20      |
| W3207     | IgA$\kappa$ | PC                | 150 ± 0       |
| W3129     | IgA$\kappa$ | 1,6D              | 150 ± 10      |
| MOPC315   | IgA$\lambda$ | DNP              | 140 ± 0       |
| J558      | IgA$\lambda$ | 1,3D              | 130 ± 0       |
| MOPC21    | IgG$\kappa$ | --                | 150 ± 0       |
| GPC7      | IgG2$\kappa$ | --                | 170 ± 20      |
| MOPC141   | IgG2$\kappa$ | --                | 100 ± 20      |
| J606      | IgG$\kappa$ | 2,1F              | 150 ± 0       |
| W3469     | IgM$\kappa$ | --                | 144 ± 30      |

$^*$ Myeloma binding was determined by solid-phase immunoassay using $[^3H]$leucine-labeled 42-21 antibody.

$^\S$ Mean cpm bound/well ± SD.

The 42-21 antibody does not bind to other myelomas, including similar $\alpha$, $\kappa$, PC-binding proteins such as W3207 and MOPC603. This pattern of reactivity is consistent with that of an anti-T15 idiotype, and thus the determinant recognized by 42-21 must lie within the T15 variable region.

Using a panel of isolated heavy and light chains, recombined molecules and hybridomas, the antigenic site on the T15 molecule was localized further (Table II). The 42-21 antibody binds to isolated T15 light chains, but not to T15 heavy chains, and selectively interacts with recombined or hybridoma immunoglobulins containing the T15 light chain. In an inhibition study, the M2 hybridoma (T15 L, T15 H, as determined by amino acid sequence) was found to inhibit completely the binding of labeled M2 to 42-21, whereas the M3 hybridoma (MOPC603 L, T15 H, by amino acid sequence) had no effect (Fig. 1). These findings are consistent with the conclusion that 42-21 detects a T15-specific idiotype on the $\lambda$ light chain. The 42-21 antigen was not detected on normal immunoglobulin from unimmunized BALB/c mice. In addition, this $\lambda$ determinant is distinct from the $\kappa$ chain marker described by Claflin (12), because the 42-21 hybridoma binds equally well to both types of light chains (Table II).

To test whether the 42-21 antigen is binding-site related, blocking studies were performed with the PC hapten. Antibody binding is only weakly inhibited by high concentrations of free hapten (data not shown), but even trace amounts of a PC-BSA conjugate significantly block 42-21 binding to T15 (Fig. 2); 71-11, a monoclonal antibody that recognizes a cross-reactive idiotype present on all PC-binding myelomas,
Table II

| Ig source | Isotype | V<sub>H</sub> | V<sub>L</sub> | cpm/well ± SD |
|-----------|---------|--------------|--------------|---------------|
| Myelomas  |         |              |              |               |
| T15       | IgA<sub>x</sub> | T15          | T15          | 1,530 ± 50    |
| McPC603   | IgA<sub>x</sub> | McPC603      | McPC603      | 150 ± 20      |
| Recombined molecules |         |              |              |               |
| T15/McPC603 | IgA<sub>x</sub> | McPC603      | T15          | 1,820 ± 70    |
| Normal BALB/c Ig |     |              |              | 128 ± 4       |
| Hybridomas |         |              |              |               |
| M2        | IgM<sub>x</sub> | T15          | T15          | 1,301 ± 28    |
| M3        | IgM<sub>x</sub> | T15          | McPC603      | 85 ± 10       |
| G1        | IgG<sub>x</sub> | T15          | MOPC167      | 107 ± 14      |
| (V<sub>x</sub>-P<sub>c</sub>) |         |              |              |               |
| 221A4     | IgM<sub>x</sub> | T15          | T15          | 1,083 ± 8     |
| (V<sub>x</sub>-P<sub>c</sub>) |         |              |              |               |
| 991G2     | IgM<sub>x</sub> | T15          | T15          | 1,213 ± 31    |
| (V<sub>x</sub>-P<sub>c</sub>) |         |              |              |               |
| 552D3     | IgM<sub>x</sub> | T15          | T15          | 1,085 ± 100   |
| Isolated chains |     |              |              |               |
| T15 H     | IgA<sub>-</sub> | T15          | --           | 206 ± 1       |
| T15 L     | IgM<sub>-</sub> | --           | T15          | 832 ± 10      |
| W3207L    | --       | --           | W3207        | 155 ± 12      |

* Myeloma binding was determined by solid-phase immunoassay using [<sup>3</sup>H]leucine-labeled 42-21 antibody.

† Mean cpm bound/well ± SD.

Fig. 1. Inhibition of <sup>3</sup>H-labeled M2 binding to 42-21 antibody. 42-21 antibody was immobilized in microtiter wells using a rabbit anti-rat Ig first stage. Various dilutions of unlabeled M2 (T15 H, T15 L) or M3 (T15 H, McPC603 L) hybridoma immunoglobulins were used to block the binding of <sup>3</sup>H-labeled M2 protein to the plate-bound 42-21.

is not blocked by PC-BSA. This implies that the antigen recognized by 42-21 is close to the PC binding site of T15.

Characterization of the 42-21 Antigen on T Lymphocytes. After the characterization of
Fig. 2. Hapten inhibition of 42-21 antibody binding to T15. Various dilutions of a PC-BSA conjugate were preincubated in T15-coated microtiter wells to inhibit the binding of synthetically labeled antibody. 71-11 is a hybridoma that recognizes a cross-reactive idiotype present on all PC-binding myelomas.

### TABLE III

| Cell source       | Cell type  | 42-21  | 31-11  |
|-------------------|------------|--------|--------|
|                   |            | Percent positive | Median fluorescence | Percent positive | Median fluorescence |
| Normal tissue§    | Thymus     | 95.6   | 23     | 94.3   | 100     |
|                   | Lymph node | 61.1   | 27     | 64.9   | 100     |
|                   | Spleen     | 20.5   | 16     | 22.1   | 100     |
|                   | Bone marrow| 4.2    | 9      | 4.5    | 7       |
| Tumor cells       | KKT2       | —      | 24     | —      | 75      |
|                   | VL-3       | —      | 30     | —      | 100     |
|                   | EL-4       | —      | 49     | —      | 100     |
|                   | L691       | —      | 35     | —      | 100     |
|                   | P815       | —      | 0      | —      | 0       |
|                   | NS-1       | —      | 0      | —      | 1       |
|                   | L1-2       | —      | 0      | —      | 0       |
|                   | RAW112     | —      | 0      | —      | 0       |

* Suspensions of the various cells were incubated with the indicated antibody, followed by a FITC-labeled rabbit anti-rat Ig second stage. Analysis was performed on a FACS.

§ Relative fluorescence standardized to a scale with 31-11 staining on thymocytes arbitrarily assigned a value of 100.

§ Cell suspensions were prepared from normal lymphoid tissues of 6-wk-old male C × B/J mice.

The 42-21 antigen on T15 as a V₄ idiotype, the antibody was investigated for binding to a panel of normal lymphoid cells and tumors (Table III). The 42-21 antibody binds to all T cell tumors, but not to non-T tumors, and shows a pattern of binding...
to normal lymphoid cells identical to that of an anti-Thy-1 hybridoma, 31-11 (16). The reduced median fluorescence on T cells observed with 42-21 when compared with 31-11 is probably related to differences in the ability of the FITC-labeled second stage to react with different rat isotypes (IgM vs. IgG). In addition, staining of frozen lymphoid sections is confined to T-dependent areas (R. Rouse, personal communication). By absorption, the 42-21 antigen is found on all lymphoid tissues, but not on other organs, such as liver (Fig. 3). Brain tissue was almost as effective as thymus in absorbing out the 42-21 antibody.

Because the Thy-1 antigen is found also on the brain and thymus, well-characterized anti-Thy-1 antibodies were examined for their ability to block the binding of 42-21 to thymocytes (Fig. 4). The anti-Thy-1 monoclonal, 31-11, blocks 42-21 binding, whereas other monoclonals specific for Lyt-1, Lyt-2, or a cross-reactive PC idiotype (71-11) have no effect. The relationship between the Thy-1 molecule and the 42-21 antigen is supported by several additional independent lines of evidence: (a) the failure of the 42-21 antibody to bind to Thy-1 mutant cell lines (Fig. 5); (b) the localization of 42-21 and anti-Thy-1 antibodies to the same cells in the brain, as determined by immunohistochemical analysis on frozen sections (R. Rouse, personal communication); and (c) the precipitation of a 25,000–28,000 mol wt glycoprotein from the surface of labeled T lymphomas and lymphocytes (9). Thus, the 42-21 antibody appears to recognize the Thy-1 antigen on all T lymphocytes.

**Demonstration of the Cross-Reaction between T15 and Thy-1.** Although the 42-21 cell line has been subcloned by limiting dilution, the binding of this antibody to both the T15 myeloma protein and the Thy-1 molecule theoretically could be due to two antibody populations present in the same hybridoma supernate. To rule out this possibility, it was necessary to investigate the ability of one of these antigens to absorb out antibody activity against the other antigen. Absorption of the 42-21 antibody

![Figure 3](image)

**Fig. 3.** Absorption of 42-21 antibody with mouse tissues. *3H*-labeled 42-21 antibody was incubated vol:vol with cell suspensions of various mouse tissues. After centrifugation, residual antibody in the supernate was determined by scintillation counting.
with thymocytes removes all activity against the T15 myeloma, whereas similar absorption of another monoclonal antibody recognizing a cross-reactive PC idiootype not present on T cells (71-11) has no effect (Fig. 6). Similarly, absorption of 42-21 with T15-Sepharose removes binding to thymocytes, whereas normal mouse Ig-Sepharose has no effect (Fig. 7). Thus, it is evident that the binding of 42-21 to both
Fig. 6. Absorption of 42-21 antibody with thymocytes. $^3$H-labeled antibody was absorbed with various numbers of C×B/J normal thymocytes and then tested for residual binding activity against T15 in a solid-phase immunoassay. 71-11 is a hybridoma that recognizes a cross-reactive idiootype present on all PC-binding myelomas.

Fig. 7. Absorption of 42-21 antibody with T15. $^3$H-labeled 42-21 was absorbed with T15-Sepharose or normal mouse Ig-Sepharose, and then tested for residual binding activity against the KKT-2 T lymphoma in a cell-binding assay.
Thy-1 and T15 represents a true cross-reaction, rather than a contaminating antibody specificity present in the hybridoma supernate.

Discussion

In an attempt to identify cell surface molecules on T lymphocytes that cross-react with immunoglobulin, we have isolated a monoclonal antibody that detects an antigenic determinant shared by V₆-T15 and the Thy-1 molecule. This unexpected cross-reaction is of interest because it conclusively demonstrates that a unique idiotypic determinant present on a purified immunoglobulin is shared by another normal cellular component present on all T cells. This finding may represent merely a trivial cross-reaction of no structural or functional significance, or it may imply a role for the Thy-1 molecule in antigen and/or mitogen recognition. The Thy-1 molecule has been shown to possess structural homology to an immunoglobulin domain (17), and highly significant sequence homology to Vₓ and Vₓ regions (A. Williams and J. Gagnon, personal communication). In addition, it has been suggested that the Thy-1 antigen plays a functional role in morphogenetic changes in cultured epithelial cells (18), in recirculation of mature T cells (19), and in the in vitro development of myogenic cell lines (20).

It is interesting to note our own experiments using the 42-21 antibody to block normal and neoplastic T cell growth and function. We have shown that the 42-21 antibody significantly inhibits T cell-mediated allogeneic killing (N. Hollander, E. Pillemer, and I. L. Weissman, manuscript in preparation) and also inhibits virus binding to virus receptors on T lymphoma cells (9). Both of these phenomena probably represent steric hindrance of antigen or viral receptors, but it is possible that the Thy-1 molecule itself could be linked to T cell receptors as part of a mitogenic apparatus. In addition, we have demonstrated that anti-Thy-1 antibodies block concanavalin A-induced T cell mitogenesis (21) and T lymphoma cell proliferation (9, 16) in vitro. This provides further support for a direct role for the Thy-1 molecule in normal and neoplastic T cell growth control.

The occurrence of the Thy-1 antigen on both T cells and cells of the central nervous system is a particularly intriguing phenomenon. Although the Thy-1 molecule is found on many different cell types in the brain (22), it has been suggested that it plays a selective role in synaptic transmission. Thus, injection of anti-Thy-1 antibodies into a rat hypothalamus results in selective blockade of cholinergic function (23). Recently, Fuchs et al. (24) have detected a thymic cell surface antigen using antibodies prepared against purified acetylcholine receptors. Although highly speculative, it is interesting to consider the relationship of these observations to our finding of a cross-reactive idiotype on the Thy-1 molecule and the T15 myeloma, a molecule that binds acetylcholine as well as PC (25).

As our studies on the 42-21 antigen have demonstrated, serological identity cannot be equated with structural or functional similarity. A similar unexpected crossactivity between light chains and T cells was found to be due to a determinant on β-2 microglobulin (26). Attempts to isolate and characterize the T cell receptor for antigen will continue to rely heavily on detection of immunoglobulin-related molecules on T cells, using antisera prepared against purified immunoglobulin. However, future studies on the T cell receptor must take into account the possibility that such antisera
or even monoclonal antibodies) may recognize a cell surface antigen on all or a subset of T cells shared by a normal component unrelated to classical immunoglobulin.

Summary

To identify T lymphocyte antigens with immunoglobulin-like determinants, we prepared rat anti-mouse T cell monoclonal antibodies and screened them against a panel of purified mouse myeloma proteins representing all isotypes of immunoglobulin. One hybridoma, designated 42-21, was found to detect a novel antigenic determinant shared by V\textsubscript{\textalpha}-TEPC15 and the Thy-1 molecule on all T lymphocytes. Although several explanations for this unusual phenomenon exist, it may imply some role for the Thy-1 molecule in antigen and/or mitogen recognition. In any event, future studies of idiotypes on T lymphocytes must consider the possibility that anti-idiotypic sera detect cell surface molecules unrelated to classical immunoglobulin.

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