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Immunology

Characterization of the interleukin 2 receptor β chain using three distinct monoclonal antibodies

(Tac peptide/crosslinking)

MITSURU TSUDO, FUJIKO KITAMURA, AND MASAYUKI MIYASAKA

Department of Immunology, The Tokyo Metropolitan Institute of Medical Science, Bunkyo-ku, Tokyo 113, Japan

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ABSTRACT **The human high-affinity receptor for interleukin 2 (IL-2) has been proposed as being a membrane complex composed of at least two distinct polypeptide chains: p55 (α chain), recognized by the anti-Tac monoclonal antibody (mAb), and p75 (β chain), both of which are capable of binding IL-2. Whereas the α chain itself has been shown to be nonfunctional, the β chain appears to be pivotal in the IL-2 signal transduction, although the β chain is otherwise poorly characterized. Three β chain-specific mAbs, designated Mik-β1, -β2, and -β3, were developed. Mik-β1 and -β2 completely inhibited the IL-2 binding to the β chain, whereas Mik-β3 immunoprecipitated the β chain crosslinked with ^{125}I-labeled IL-2. The β chain immunoprecipitated by these mAbs was revealed to have a M_r of 68,000–72,000. High-affinity IL-2 binding was completely abolished by Mik-β1. Although IL-2-dependent T-cell growth at high IL-2 concentrations was not inhibited by the anti-Tac, it was almost completely inhibited by Mik-β1 in the presence of the anti-Tac. These results clearly indicate that the β chain is an indispensable component to the high-affinity IL-2 receptor and is responsible for the IL-2 signal transduction. The β chain was found to be constitutively expressed without the α chain on the surface of peripheral blood Leu-19$^+$ natural killer cells.**

The growth of T cells is regulated by interaction between interleukin 2 (IL-2) and its receptor (IL-2R) (1, 2). Recent chemical crosslinking studies (3–5) using ^{125}I-labeled IL-2 (^{125}I-IL-2) have suggested that the high-affinity receptor for IL-2 having a dissociation constant (K_d) of ≈10 pM is a membrane complex composed of at least two distinct polypeptide chains; one is the M_r 55,000 peptide (p55 or α chain) recognized by the monoclonal antibody (mAb) anti-Tac (6, 7), and the other is a peptide with a putative M_r of 75,000 (p75 or β chain). Unlike many multisubunit receptors, each of the two subunits of the IL-2R can be individually expressed in the absence of the other. Moreover, individually existing α and β chains are capable of binding IL-2 with low (K_d ≈ 10 nM) and intermediate (K_d ≈ 1 nM) affinities, respectively (8, 9). Only when both chains are expressed together on the same cell are high-affinity receptors detectable (8–10).

cDNA transfection experiments (11), in which non-T cells were made to express α chains alone in the absence of β chains, have shown that the α chain itself is nonfunctional. In contrast, the interaction of IL-2 with the β chain alone has been shown to be sufficient to trigger cell differentiation and proliferation in the absence of the α chain (12, 13). Furthermore, binding of IL-2 to the β chain, but not the α chain, allows the internalization of IL-2 (14). Thus, the β chain appears to play an important role in the IL-2 signal transduction. Nevertheless, the β chain has been neither purified nor well characterized primarily because of the absence of specific reagents.

We report herein the direct identification and characterization of the β chain by utilizing three mAbs that we generated; furthermore, we provide evidence that the β chain is an indispensable component of the high-affinity IL-2R and is responsible for the IL-2 signal transduction.

MATERIALS AND METHODS

Cell Lines. The YTS cell line, a high expressor of the IL-2R β chain, was established by a cell-sorting of the human natural killer (NK)-like cell line YTU14 (gift from Y. Tagaya and J. Yodoi; ref. 15), which expressed the β chain alone. The YTU14 cells were stained with biotinylated IL-2 and fluorescein isothiocyanate conjugated avidin, and the top 5% of the brightly stained cells was sorted with a fluorescence-activated cell sorter (EPICS-CS; Coulter). When examined by the ^{125}I-IL-2 binding assay, YTS cells expressed ≈20,000 binding sites per cell, twice as many as the original YTU14 cells, with an intermediate affinity (K_d = 1.8 nM). The Kit 225 cell line is an IL-2-dependent T-cell line derived from a patient with chronic T-lymphocytic leukemia (16).

IL-2 Binding Assay. The binding of ^{125}I-IL-2 to various types of cell lines was measured as described (4). Human recombinant IL-2 (Takeda, Osaka, Japan) was radioiodinated with Enzymobead (Bio-Rad), and its specific activity was 40,000–70,000 cpm/ng.

Production of mAbs. A BALB/c mouse was immunized i.p. at 1- to 2-wk intervals with 1 × 10^7 YTS cells. Three days after the fourth booster immunization, spleen cells were fused with PAI mouse myeloma cells (17) using polyethylene glycol 4000 (Merck). Twelve days after the fusion, hybridoma supernatants were assayed for the presence of anti-β chain antibody by two distinct methods: the ^{125}I-IL-2 binding inhibition assay and the immunoprecipitation of the β chain-radiolabeled IL-2 complex. With the former assay, after 30 min of incubation of 1 × 10^6 YTS cells in 50-μl samples with 50 μl of culture supernatant, 25 μl of 5 nM ^{125}I-IL-2 was added and incubated for 1 hr. Cell-bound radioactivity was then separated as described (4) and counted in a γ-counter. With the latter assay, the β chain-radiolabeled IL-2 complex was prepared. Under low-affinity conditions (i.e., 5 nM ^{125}I-IL-2), YTS cells were crosslinked with 2 mM disuccinimidyl suberate (Pierce) as described (4) and then solubilized with lysing buffer (10 mM Tris·HCl, pH 7.4/0.15 M NaCl/1% Nonidet P-40/2 mM phenylmethylsulfonyl fluoride) at 3 × 10^7 cells per ml. Fifty microliters of supernatant from each of the six culture wells was pooled and incubated with 100 μl (10,000 cpm) of YTS lysate, and the immune complex was precipitated by 10 μl of protein A-Sepharose (Pharmacia) coated with rabbit anti-mouse IgG (Cappel Laboratories). After washing the beads with lysing buffer, bound radioactivity was counted.

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Abbreviations: IL-2, interleukin 2; IL-2R, IL-2 receptor; mAb, monoclonal antibody; 2D, two-dimensional; NK, natural killer.

Hybridoma cell lines producing anti-β chain mAbs (Mik-β1, -β2, and -β3) were obtained, cloned twice by limiting dilution, and grown in ascites fluid. The antibodies were purified by protein A-Sepharose chromatography.

Immunoprecipitation and Gel Electrophoresis. YTS cells were radioiodinated by a glucose oxidase lactoperoxidase method. In the crosslinking experiments, conditions were identical to those reported (4), except that high- (100 pM ^{125}I-IL-2) or low- (5 nM) affinity IL-2 binding conditions were used. Cells were extracted with the lysing buffer and immunoprecipitated with the indicated antibodies and protein A-Sepharose. In the case of Mik-β3 mAb (IgG1), rabbit anti-mouse IgG (Cappel Laboratories) was used as the second antibody. For the sequential immunoprecipitation, YTS extracts were precleared five times with either Mik-β1 or -β3 and then immunoprecipitated by an alternative antibody. The immunoprecipitates were boiled in the presence of 5% 2-mercaptoethanol and analyzed by sodium dodecyl sulfate/polyacrylamide gel electrophoresis (SDS/PAGE) using an 8.5% acrylamide gel. Molecular standards (Amersham) were myosin (M_r 200,000), phosphorylase b (M_r 92,500), bovine serum albumin (M_r 69,000), and ovalbumin (M_r 46,000). Two-dimensional (2D) gel electrophoresis was performed as described by O'Farrell *et al.* (18). ^{125}I-labeled YTS (^{125}I-YTS) extract was immunoprecipitated by Mik-β1 as described above. The immunoprecipitate was eluted by 20 μl of 9.5 M urea/2% Nonidet P-40/2% Ampholine (pH 3.5–10, LKB)/5% 2-mercaptoethanol. The first dimension was a nonequilibrium pH gradient electrofocusing gel and the second dimension was an 8.5% SDS/polyacrylamide gel.

RESULTS

Development and Characterization of mAbs Against the IL-2R β Chain. YTS cells, a human NK-like cell line that highly expresses the human IL-2R β chain, were used as an immunogen to develop mAbs to the β chain. Hybridomas generated from immunized mice were screened for production of anti-β chain antibody by their ability either to inhibit the ^{125}I-IL-2 binding to the β chain on YTS cells or to immunoprecipitate the β chain-radiolabeled IL-2 complex chemically crosslinked with disuccinimidyl suberate. Two hybridoma cell lines, each producing IgG2a mAbs, designated Mik-β1 and -β2, were determined by the IL-2 binding inhibition assay, and another cell line producing IgG1 mAb, designated Mik-β3, was determined by the immunoprecipitation assay.

As shown in Fig. 1*A*, Mik-β1 and -β2 inhibited dose dependently the ^{125}I-IL-2 binding to YTS cells, which express the β chain alone, with a 50% binding inhibition at 0.6 and 3 μg/ml, respectively. Mik-β3 did not inhibit the IL-2 binding to any extent. Reciprocally, a 200 molar excess of IL-2 blocked >90% of the ^{125}I-labeled Mik-β1 (^{125}I-Mik-β1) binding to YTS cells (Fig. 1*B*), indicating that Mik-β1 specifically recognizes the IL-2 binding site on the β chain. Since Mik-β2 completely blocked the ^{125}I-Mik-β1 binding (not shown), these two antibodies appear to recognize the same epitope or very closely related epitopes on the β chain.

On the other hand, Mik-β3, although not capable of inhibiting IL-2 binding to the β chain, precipitated from the extract of YTS cells crosslinked with radiolabeled IL-2 the same M_r 90,000 protein as did polyclonal rabbit anti-IL-2 antibody (Fig. 2). Since Mik-β3 did not precipitate free ^{125}I-IL-2 (not shown), this antibody was judged to react with the β chain, precipitating the M_r 90,000 complex protein consisting of IL-2 (M_r 15,000) and the β chain (M_r ≈75,000). Inability of Mik-β3 to inhibit IL-2 binding indicates that this mAb recognizes a β chain epitope not involved in IL-2 binding, thus precipitating the β chain even when IL-2 binding sites are already occupied. Mik-β3 did not precipitate any material from the crosslinked MT-1, a human T-cell lymphotrophic virus I (HTLV I)-induced T-cell line that expresses α chains alone (8). However, when HUT 102 cells (also a HTLV I-induced T-cell line but expressing α and β chains) were used, not only the M_r 90,000 band (β chain + IL-2) but also a M_r 70,000 band (α chain + IL-2) were observed (Fig. 2, lane 5), suggesting that the α chain is closely associated with the β chain on the membrane and thus coprecipitated by Mik-β3. In contrast to Mik-β3, neither Mik-β1 nor -β2 immunoprecipitated the β chain-radiolabeled IL-2 complex (not shown), confirming the notion that Mik-β1 and -β2 recognize the IL-2 binding epitope on the β chain.

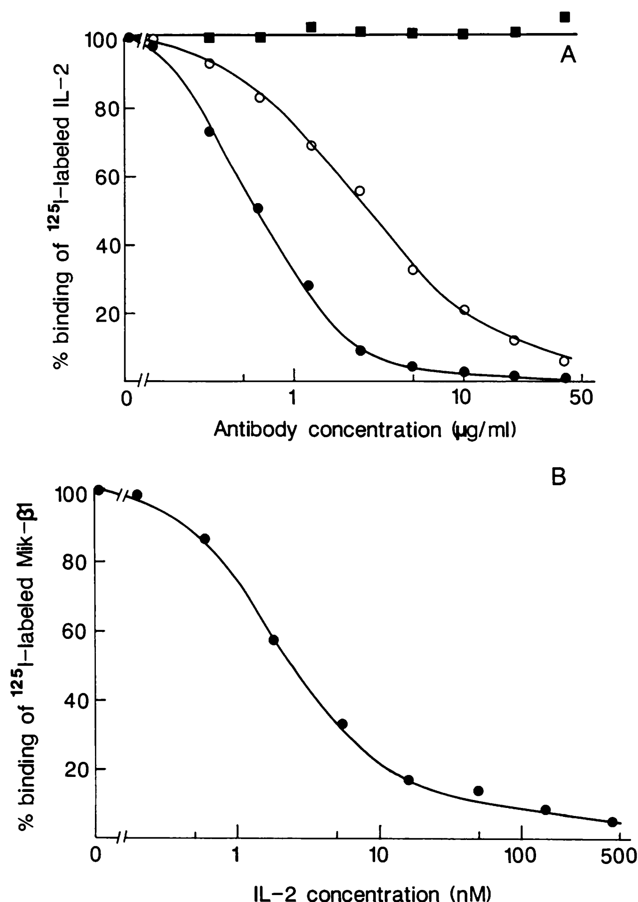

FIG. 1. (*A*) Inhibition of ^{125}I-IL-2 binding to YTS cells by mAbs: Mik-β1 (●), -β2 (○), or -β3 (■). Two million YTS cells in 75-μl samples were incubated with various concentrations of antibodies for 30 min at 4°C. Then, 25 μl of 4 nM ^{125}I-IL-2 was added and incubated for 1 hr at 4°C. Cell-bound radioactivity was measured as described (4). Nonspecific binding was determined in the presence of a 500-fold excess of unlabeled IL-2. The percent binding of ^{125}I-IL-2 = 100 × [(experimental − nonspecific binding)/(antibody-free control − nonspecific control)], where the control was 7910 cpm, and nonspecific binding was 749 cpm. (*B*) Inhibition by IL-2 of ^{125}I-Mik-β1 to YTS cells. Two million YTS cells in 75-μl samples were incubated with various concentrations of IL-2 for 1 hr at 4°C. Then, 25 μl of 2 nM ^{125}I-Mik-β1 (specific activity, 30,000 cpm/ng) was added and incubated for 30 min at 4°C. The percent binding was calculated as above; the IL-2-free control was 17,753 cpm, and nonspecific binding in the presence of a 300-fold excess of unlabeled Mik-β1 was 3445 cpm.

Immunoprecipitation of the β Chain from ^{125}I-YTS Cell Extracts. When YTS cells were radioiodinated and cell extracts were immunoprecipitated by the anti-β chain mAbs, a single broad band with a M_r of 68,000–72,000 was observed with all three antibodies on SDS/PAGE (Fig. 3*A*). To investigate the identity of these bands, sequential immunoprecipitation experiments were performed. Preclearing the

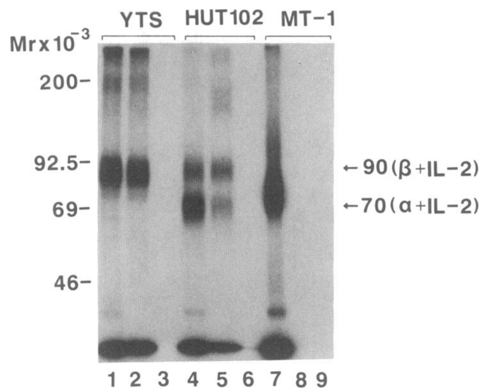

FIG. 2. SDS/PAGE analysis following the immunoprecipitation of proteins crosslinked with ^{125}I-IL-2. Lanes 1, 4, and 7, rabbit anti-human recombinant IL-2; lanes 2, 5, and 8, Mik-β3; lanes 3, 6, and 9, control UPC10 mAb (IgG2a, Sigma). Cells were crosslinked with ^{125}I-IL-2 under high-affinity conditions (100 pM ^{125}I-IL-2) for HUT 102 or low-affinity conditions (5 nM) for YTS and MT-1, subsequently solubilized, and immunoprecipitated by the indicated antibodies.

cell extracts by either Mik-β1- or Mik-β3-coated protein A-Sepharose completely removed M_r 68,000–72,000 molecules precipitable by the alternative antibody (Fig. 3A, lanes 5 and 6), demonstrating that Mik-β1 and -β3 recognize different epitopes of the same protein—i.e., the β chain. Although the β chain has occasionally been demonstrated as a doublet in crosslinking experiments (19, 20), our 2D gel analysis showed that the β chain immunoprecipitated with Mik-β1 migrated at a single spot (Fig. 3B).

Effects of Mik-β1 on the High-Affinity IL-2 Binding and IL-2-Induced T-Cell Growth. To study the role of the β chain in the high-affinity IL-2R complex, Mik-β1, which specifically inhibits IL-2 binding to the β chain, was employed. An IL-2 binding study was performed in the presence or absence of Mik-β1 using an IL-2-dependent human T-cell line, Kit 225 (16), which manifests high- and low-affinity IL-2R. As displayed in Fig. 4A, Scatchard analysis showed that Kit 225 expresses 3400 high-affinity IL-2 binding sites with a K_d of 12 pM. This high-affinity binding, however, was completely abrogated by Mik-β1, clearly indicating that blocking of the IL-2 binding to the β chain leads to a complete disruption of the high-affinity binding, whereas the α chain binding is left intact.

Having obtained the data described above, we anticipated that Mik-β1 would inhibit the IL-2-dependent T-cell growth. Kit 225 cells were cultured with a wide range of IL-2 concentrations (0.1 pM to 10 nM) in the presence of Mik-β1 [40 μg/ml; a sufficient concentration to compete with 1 nM ^{125}I-IL-2 for binding to the β chain (see Fig. 1A) and also to abrogate the high-affinity binding (Fig. 4A)] or in the presence of anti-Tac (40 μg/ml) alone, or in combination with anti-Tac. Surprisingly, however, addition of Mik-β1 did not inhibit the IL-2-induced proliferation at any of the IL-2 concentrations examined (Fig. 4B). The IL-2 dose-dependent [^{3}H]thymidine incorporation with an IL-2 concentration of 15 pM for a half-maximal response was identical with that obtained in the absence of antibody, indicating that IL-2 utilized high-affinity receptors even in the presence of Mik-β1. Nor did the anti-Tac antibody that blocks the IL-2 binding to the α chain prevent on its own IL-2-dependent growth. Rather, the IL-2 concentrations required for growth were increased 20 times. However, when both antibodies were present, the proliferation was almost completely inhibited.

Distribution of the β Chain. Flow cytometric analysis showed that cellular distribution of the β chain, as determined by Mik-β1 antibody staining, was consistent with that obtained by ^{125}I-IL-2 crosslinking studies (8) (Fig. 5A). In particular, Mik-β1 was reactive with MLA 144, a gibbon ape T-cell line that expresses the β chain alone (4), but was unreactive with MT-1 cells expressing the α chain alone. A small but significant subpopulation (≈10%) of unstimulated peripheral blood lymphocytes bore detectable levels of the β chain. Two-color flow cytometric analyses revealed that the β chain-bearing population was composed mainly of Leu-19$^+$ NK cells (Fig. 5B). CD8$^+$ cells constituted a minor population among the β chain-bearing cells. Interestingly, there were no β chain-positive cells among the CD4$^+$ subset. Although ≈3% of peripheral blood lymphocytes expressed the α chain when stained with the anti-Tac antibody, this population and the β chain-bearing population were mutually exclusive. However, lymphocytes activated with phytohemagglutinin expressed α and β chains simultaneously, and ≈60% of the β chain-bearing cells (≈40% of the activated lymphocytes) were CD4$^+$ and the remainder were CD8$^+$ (data not shown).

DISCUSSION

The human high-affinity receptor for IL-2 has been proposed as being a membrane complex composed of at least two peptide chains: p55 α and p75 β chains, both of which are capable of binding IL-2. Although the identity of the α chain has been definitively made (21, 22), the existence of the β chain has only been suggested by crosslinking studies using ^{125}I-IL-2. We have now directly identified the IL-2R β chain using three β chain-specific mAbs designated Mik-β1, -β2, and β3.

Two of our antibodies, Mik-β1 and -β2, recognize the IL-2 binding site on the β chain, since these antibodies completely inhibit the ^{125}I-IL-2 binding to the β chain, and, reciprocally, unlabeled IL-2 inhibits the binding of ^{125}I-Mik-β1. On the other hand, Mik-β3 recognizes the β chain epitope not involved in IL-2 binding, since this antibody immunoprecipitates the β chain that is already crosslinked with ^{125}I-IL-2.

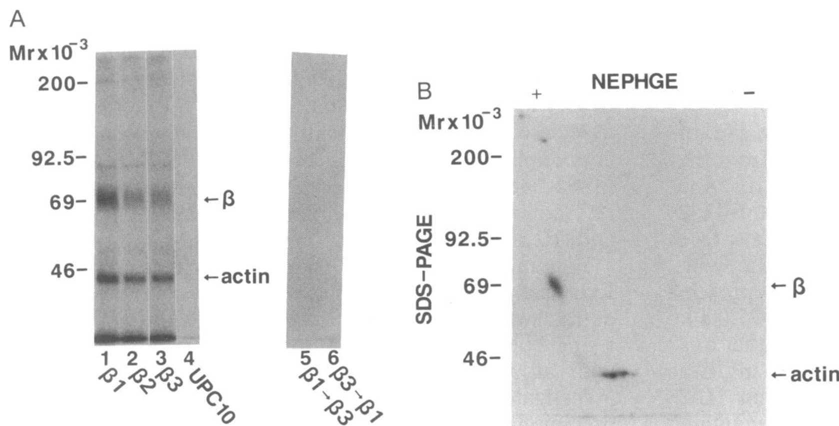

FIG. 3. (A) Immunoprecipitation and sequential immunoprecipitation from ^{125}I-YTS extracts. Lane 1, Mik-β1; lane 2, Mik-β2; lane 3, Mik-β3; lane 4, UPC10; lane 5, Mik-β3 after preclearing with Mik-β1; lane 6, Mik-β1 after preclearing with Mik-β3. YTS cells were radioiodinated, solubilized, and then immunoprecipitated by the indicated antibodies. The immunoprecipitates were analyzed by SDS/PAGE under reducing conditions. (B) 2D gel electrophoresis of the β chain. The β chain immunoprecipitated by Mik-β1 from radioiodinated YTS extract was analyzed on the 2D gel. The first dimension was nonequilibrium pH gradient gel electrofocusing (NEPHGE) and the second was SDS/PAGE.

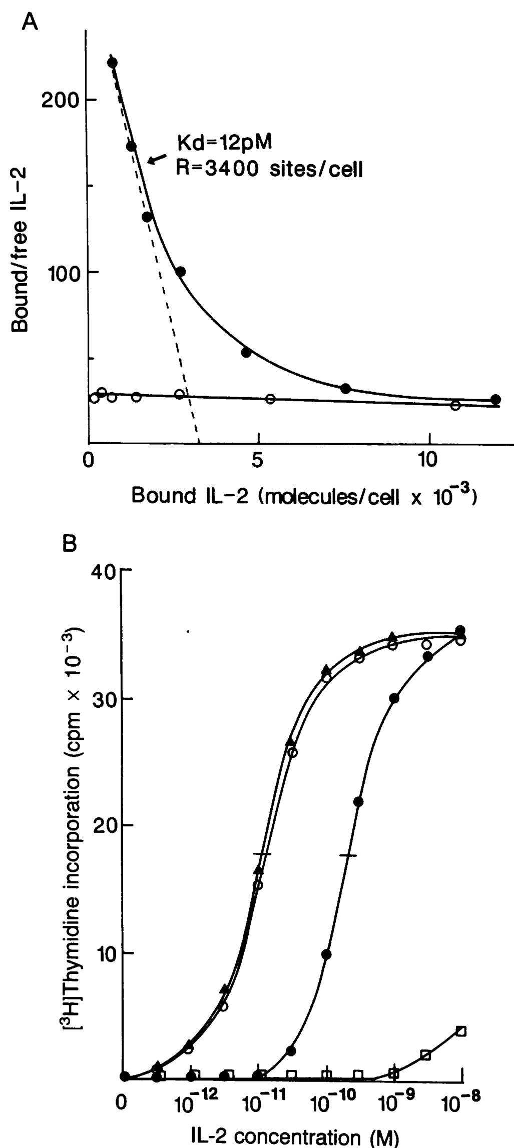

FIG. 4. Effect of Mik-β1 on the high-affinity IL-2 binding and the IL-2-dependent proliferation. (*A*) Scatchard plot analysis of ^{125}I-IL-2 binding to Kit 225 cells. The binding studies were performed under the high-affinity IL-2 binding conditions as described (4) in the presence (○) or absence (●) of 40 μg of Mik-β1 per ml. The number of high-affinity binding sites (R) was 3400 per cell, with a K_d of 12 pM. (*B*) Proliferative response of Kit 225 cells to IL-2. Samples (200 μl) of 2 × 10^4 cells in a 96-well plate were cultured for 72 hr at 37°C in triplicate with various concentrations of IL-2 in the absence (○) or presence of either 40 μg of Mik-β1 per ml (▲), 40 μg of anti-Tac antibody per ml (●), or both (□). Proliferation was measured by the incorporation of 0.5 μCi of [^{3}H]thymidine (6.7 Ci/mmol; 1 Ci = 37 GBq; New England Nuclear) per well during the last 4 hr of the culture.

By using these antibodies, the β chain was demonstrated to have a M_r of 68,000–72,000. As described elsewhere (19, 20), a β chain crosslinked with IL-2 has occasionally been demonstrated as a doublet on SDS/PAGE, and this heterogeneity has been claimed to be due to posttranslational modification or existence of two distinct IL-2 binding peptides with a M_r of 70,000. However, our 2D gel studies indicated that the β chain is of a single species. Furthermore, it is unlikely that there is another IL-2 binding peptide other than the β chain, because all IL-2 binding sites on YTS cells can be blocked by Mik-β1 (Fig. 1*A*).

Mik-β1 completely abolished the high-affinity IL-2 binding to the IL-2-dependent T-cell line Kit 225 (Fig. 4*A*), which thus strongly enforces the view that the β chain is an indispensable component of the high-affinity receptor and that simultaneous IL-2 binding to α and β chains is required for the high-affinity binding. It has been argued whether the high-affinity receptors are formed only when IL-2 is present (23) or whether they exist as a preformed α/β heterodimer without requiring IL-2 for their association (4, 24). Our immunoprecipitation experiments using Mik-β1 favor the latter hypothesis. When HUT 102 cells, which express high- and low-affinity receptors, were radioiodinated and solubilized, a M_r 55,000 protein representing the α chain was coprecipitated by Mik-β1 with the β chain (data not shown).

The IL-2R α chain has been shown to be nonfunctional in transducing IL-2 signals (11), and cells expressing the β chain alone can respond to IL-2 (12, 13, 25). These observations led to the hypothesis that the β chain mediates the IL-2 signal. A surprising observation in the present study examining the role of the β chain was that addition of Mik-β1 did not inhibit the IL-2-induced proliferation of Kit 225 cells at any of the IL-2 concentrations used (Fig. 4*B*). No significant shift in the dose-dependent proliferation curves indicates that an IL-2 signal was transduced by high-affinity receptors even in the presence of Mik-β1, which blocks the β chain and abolishes the high-affinity IL-2 binding. This may be explained in part by kinetic and equilibrium IL-2 binding studies performed by others (9, 26); when Kit 225 cells are cultured at 37°C, β chains could be newly synthesized to form high-affinity receptors in cooperation with free α chains. Then, IL-2 might have associated with α/β heterodimers very rapidly (a half-time for association = 42 sec; ref. 26) and with a much higher affinity (K_d = 12 pM) than Mik-β1 (K_d = 4 nM; not shown). Therefore, IL-2 could have bound to the newly synthesized β chain even if Mik-β1 was continuously present in the culture. The presence of the anti-Tac antibody that blocks the IL-2 binding to the α chain was not found to prevent the IL-2-dependent growth either. Rather, the IL-2 concentrations required for growth were increased 20 times, possibly reflecting the situation whereby IL-2 interacted only with the intermediate-affinity β chains. When both antibodies were present, the proliferation was almost completely inhibited, supporting the view that the β chain is responsible for mediating the IL-2 signal. However, the fact that an IL-2 signal through high-affinity receptors is blocked by Mik-β1 only when the anti-Tac is simultaneously present may also suggest that the α chain is somehow involved in the initial events of the signal transduction through the β chain. The possible presence of an additional receptor subunit has yet to be clarified. Molecular cloning of the β chain utilizing these mAbs is necessary to examine these possibilities.

Attempts to purify the β chain protein utilizing an IL-2-coupled affinity column have been met with difficulties, which may be in part explained by the following observations. We developed the sandwich enzyme-linked immunosorbant assay (ELISA), in which Mik-β3 was coated on the plate, and biotinylated Mik-β1 plus alkaline phosphatase-conjugated avidin were used as the second reagents to detect the solubilized β chain (unpublished). When we used this assay system to study whether the solubilized β chain could bind IL-2, it was found that IL-2 efficiently competed with biotinylated Mik-β1 for the binding to the solubilized β chain fixed on the solid phase. However, the IL-2 concentration required for the aforementioned competition was much higher than that required for the binding competition of intact β chain present on viable cells, suggesting that affinity of the solubilized β chain to IL-2 is much lower than that of intact β chain on cell membranes. These observations also indicate that the antigen recognized by Mik antibodies is indeed capable of binding IL-2.

We (12) and others (13, 25) have previously suggested that, based on crosslinking studies, large granular lymphocytes

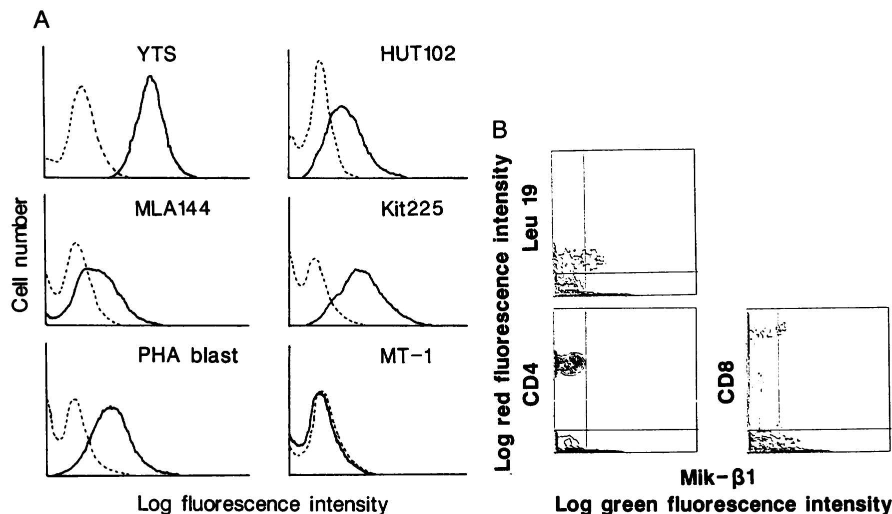

FIG. 5. (*A*) Flow cytometric analysis of the reactivity of Mik-β1. Cell lines and phytohemagglutinin (PHA) blasts obtained by culturing peripheral blood mononuclear cells with 1% PHA-M (Difco) for 3 days were stained with Mik-β1 (solid lines) or control UPC10 antibody (IgG2a, dotted lines) and subsequently stained with fluorescein isothiocyanate (FITC)-conjugated goat anti-mouse IgG (Tago). Cells were analyzed by the Epics-CS (Coulter). (*B*) Two-color analysis of peripheral blood lymphocytes. Following the staining with PE-T4 (CD4, Coulter), T8 (CD8), or Leu-19 (Becton Dickinson), cells were stained with biotinylated Mik-β1 plus avidin-FITC (Tago). Fifty thousand cells bearing the typical lymphocyte scatter were analyzed with the EPICS-CS.

and resting T cells from peripheral blood express the β chain. Two-color flow cytometric analysis using anti-β chain mAbs clearly showed that $\approx$10% of peripheral blood lymphocytes manifest the β chain and that this population is mainly composed of Leu-19^+ NK cells (Fig. 5*B*). These results may account for the previous observation that NK cells lacking the α chain could be induced to proliferate by addition of relatively high concentrations of IL-2 (27–29). Preliminary studies showed that $\approx$10% of human thymocytes are reactive with Mik-β1, indicating that a subpopulation of thymocytes constitutively expresses the IL-2R β chain, as has been inferred by others (30). The functional role of the IL-2R β chain in thymocytes can now be examined by the use of these mAbs.

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