response to NE that were used in the calculation of K_A were invariably blocked completely by prazosin ($10^{-7}M$).

There was a positive correlation between pD_2 and the agonist dissociation constant (pK_A) for 12 types of rabbit arteries (slope ± SEM = 0.88 ± 0.26; $P < 0.01$). This slope was not statistically significantly different from unity (Fig. 1). Only with the superior mesenteric artery did endothelium removal increase the pD_2, and then by a factor of 3.5. For this artery, the value after endothelium removal was used. The percentage of available receptors occupied influences tissue sensitivity (*1, 9*). In a blood vessel the smaller the percentage of available receptors occupied, the greater the sensitivity. When the results from three arteries—the ear artery and common and external iliac arteries—with a high receptor reserve [defined as antilog($-\log EC_{50} - pK_A$)] were excluded, the correlation between pD_2 and pK_A was higher, and its slope was not statistically significantly different from unity. There was no statistically significant correlation between receptor reserve and pD_2 (slope ± SEM = 15.8 ± 14.3; $P > 0.05$). Thus, at least in these arteries, although there was some suggestion that receptor occupancy influences tissue sensitivity, the data were consistent with the conclusion that the dominant factor is the agonist dissociation constant.

Variation in K_A values for NE in various in vitro preparations—for example, $1.3 \times 10^{-7}M$ in the rabbit aorta (*10*) and $6.3 \times 10^{-6}M$ in the rat vas deferens (*3*)—has been taken as an indication of heterogeneity of α_1-adrenoceptors. However, our demonstration of a relation between agonist potency and dissociation constant of the same full agonist in anatomically different but structurally similar systems, indicates that the α_1-adrenoceptors in the different arteries are similar, but that they are present in the smooth muscle cells in different affinity states. This variation probably is determined by endogenous factors in the receptor microenvironment.

Recent studies of α_1-adrenoceptor ligand binding associated with Ca^{2+} efflux in cultured vascular smooth muscle cells dispersed from rabbit aorta suggest a population of receptors that exists in both high- and low-affinity states. With this assumption, 40% of the receptors in the rabbit aorta are in the high-affinity state (*11*). However, the ratio of affinities of the two states is only 86. This is small in comparison to differences of more than 500 between receptor systems represented by the 12 arteries in this series. Thus, unless there are additional influences that increase the range between the two states, this explanation would not account for the variation we have found. In the series of arteries that we studied, intracellular regulation of α-adrenoceptor affinity could be responsible for this diversity.

The NE sensitivities of a number of rabbit regional arteries—ear, pulmonary, and mesenteric—are markedly different, and these differences are maintained as they branch (*1*). Thus it seems that α-adrenoceptor affinity is a locally regulated characteristic of vascular smooth muscle which can account for regional differences in sensitivity to NE. As in some instances, patterns of regional differences can be related to embryological development (*1*), such differences in these regulating systems may be established early in development.

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Common Mechanism of Chromosome Inversion in B- And T-Cell Tumors: Relevance to Lymphoid Development

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An inversion of chromosome 14 present in the tumor cells of a patient with childhood acute lymphoblastic leukemia of B-cell lineage was shown to be the result of a site-specific recombination event between an immunoglobulin heavy-chain variable gene and the joining segment of a T-cell receptor α chain. This rearrangement resulted in the formation of a hybrid gene, part immunoglobulin and part T-cell receptor. Furthermore, this hybrid gene was transcribed into messenger RNA with a completely open reading frame. Thus, two loci felt to be normally activated at distinct and disparate points in lymphocyte development were unified and expressed in this tumor.

ASSOCIATION OF A SPECIFIC CHROMOSOMAL abnormality with a specific tumor type is well established and may reflect mechanisms of oncogenesis peculiar to that tumor (*1*). Alternatively it may be that these associations reflect the particular differentiated state of the malignant cell, consistent with the model that rearrangements occur only within chromatin in an "active" configuration (*2*). We and others recently reported the molecular analysis of a chromosomal abnormality in the cell line SUP-T1 that was derived from a pediatric patient with T-cell lymphoma (*3*). The SUP-T1 cell line contained a paracentric inversion of chromosome 14, inv(14)(q11.2q32.3), which is commonly seen in T-cell malignancy (*4, 5*). We demonstrated that this abnormal rearrangement was the result of site-specific recombination that could directly produce the inversion by uniting a T-cell receptor α-chain joining segment (TCR J_α) from band q11.2 with the immunoglobulin heavy-chain variable gene (Ig V_H) from band q32.3. This finding demonstrated that not only are the Ig and TCR loci similar in genomic structure but on occasion their component parts can be shuffled to produce a transcriptionally active

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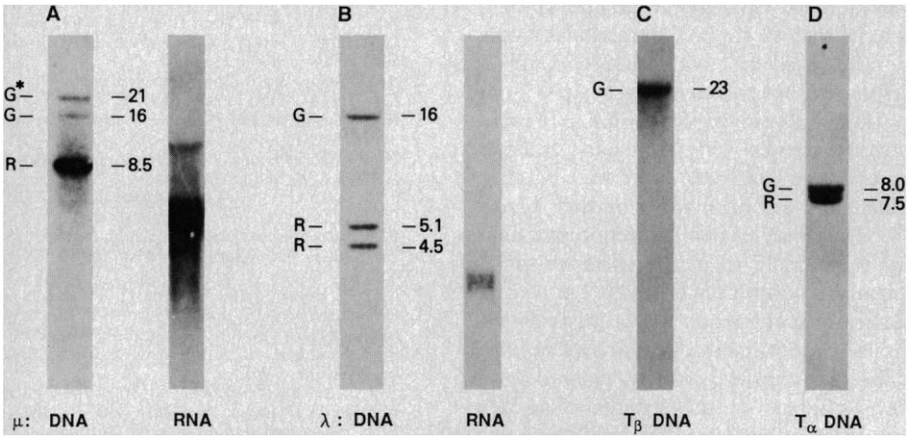

Fig. 1. Southern and Northern blots of Ig and TCR loci in the DNA and RNA from tumor VP. (A) Eco RI–digested genomic DNA was probed with a fragment from the human Ig μ heavy-chain J locus (*16*) that detects a rearranged band (R), plus germline bands from nonmalignant cell contamination (G) and a cross-reacting band (G*). A primary transcript from the Ig heavy-chain locus of approximately 2.5 kb was demonstrated by hybridization of VP total RNA with a probe from the Ig μ constant coding region (*16*). (B) Eco RI–digested genomic VP DNA was probed with a fragment containing the Ig λ constant coding region (*17*). This probe when hybridized to total RNA revealed a transcript of approximately 1.2 kb from the Ig λ locus. (C) Eco RI–digested VP genomic DNA hybridized to a probe from the TCR β-chain constant coding region (*18*). (D) Hind III–digested genomic VP DNA hybridized with a probe from the TCR J_α locus. Differences in band intensities in Northern blots are caused by differing autoradiographic conditions and do not reflect relative levels of transcript.

chimeric gene. These studies imply that during T-cell development Ig and TCR loci are simultaneously accessible for recombination. We were interested in investigating whether this accessibility was restricted to lymphocytes of T-cell lineage or could also be demonstrated in B cells.

We now report the molecular analysis of tumor cells from a child (VP) with acute lymphoblastic leukemia of B-cell lineage. We found that the malignant B cells had an inversion karyotypically identical to that in the previously analyzed T-cell line, SUP-T1. This inversion also was caused by site-specific recombination between a TCR J_α and an immunoglobulin V_H. As with SUP-T1, nuclease S1 analysis of VP RNA reveals expression of a potentially functional transcript of this hybrid gene. Thus by combining this study with our previous effort (*3*), we have been able to molecularly analyze a morphologically identical chromosomal aberration in two different cell types. These results emphasize an unpredicted commonality between B cells and T cells.

We began our studies with a direct chromosome examination of VP peripheral blood lymphocytes that showed a paracentric inversion on chromosome 14. In addition we noted a missing chromosome 11, a novel insertion of part of chromosome 11 into chromosome 4 and an extra marker chromosome of uncertain origin. The complete chromosomal formula is 46,XX,−11,dir ins(4;11)(q21;q11q23), inv(14)(q11q32),+mar.

Cell surface immunophenotyping was done by means of fluorescence-activated cell sorting (FACS) and a battery of monoclonal antibodies against B- and T-cell antigens. VP tumor cells tested strongly positive for B-cell antigens Leu 12, BA-1, BA-2, 6A4, and Coulter B4 but negative for T-cell antigens Leu 1, Leu 2a, Leu 3a, Leu 4, Leu 5, Leu 6, and Leu 9. There was strong reactivity against HLA-DR and Leu M1, weak reactivity against the common ALL antigen (cALLa), and no reactivity against surface immunoglobulins of any type.

This tumor's B-cell lineage was confirmed genotypically by examination of Ig and TCR loci. By means of DNA hybridization with probes from the Ig μ and Ig λ loci, novel bands were detected consistent with VDJ and VJ rearrangement (Fig. 1, A and B). Active transcription of these loci was demonstrated by Northern blotting experiments (Fig. 1, A and B). Similar Southern blot experiments with a probe to the constant coding region of the TCR β-chain locus showed only the germline configuration (Fig. 1C). Northern blot analysis did not show any evidence of transcription from the T_β locus in this tumor cell population. When a TCR J_α probe located approximately 7 kb 5′ of C_α (see schematic Fig. 2A) was hybridized to Hind III–cut genomic DNA, a 7.5-kb rearranged band was seen in addition to the 8.0-kb germline band (Fig. 1D).

This rearranged fragment was cloned from a VP genomic DNA library in the phage vector λJ1 (*6*). The position of the rearrangement was determined by comparison with a germline clone (Fig. 2A), and its nucleotide sequence was determined (Fig. 2B). This sequence analysis revealed that a site-specific recombination event had occurred between a completely intact immunoglobulin V_H gene and a TCR J_α. The predicted amino acid sequence of this Ig V_H gene shows it to be a member of subgroup III (*7*). Translation of the nucleotide sequence indicated that an in-frame VJ join had occurred, resulting in an open reading

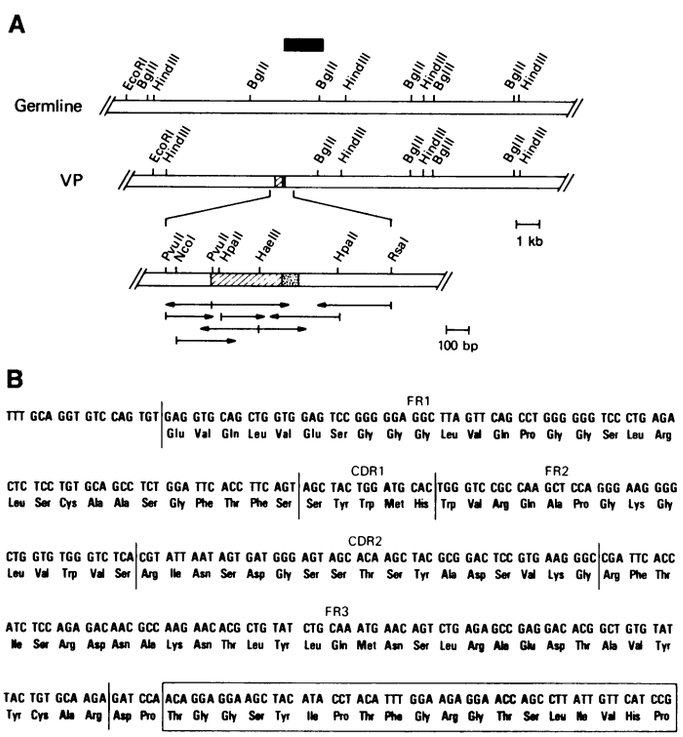

Fig. 2. Molecular analysis of the inv(14) in VP tumor DNA. (A) Schematic representation of germline and VP TCR J_α loci. A germline probe from approximately 7 kb 5′ of TCR C_α is indicated by the black bar. A magnified view of the rearrangement with sequencing strategy is shown in the lower schematic. Hatched area represents Ig V_H; stippled area, TCR J_α. (B) Nucleotide and predicted amino acid sequence of VP rearrangement. Ig V_H framework (FR) and complementarity-determining regions (CDR) are indicated. TCR J_α sequence is boxed.

frame from the start of the Ig V_H gene through the TCR J_α.

The locations and orientations on chromosome 14 of the Ig heavy-chain and the TCR α-chain genes are known. Ig heavy-chain genes at 14q32 are oriented with V_H genes telomeric to C_H (*8*), while TCR α-chain genes at 14q11 are oriented with V_α genes centromeric of C_α (*9*) (Fig. 3). Thus a site-specific recombination between Ig V_H and TCR J_α would result in the precise paracentric inversion of chromosome 14 seen in this tumor (Fig. 3).

To ascertain the transcriptional activity of this hybrid gene, an S1 nuclease experiment was performed. A ^{32}P-labeled single-stranded M13 probe containing Ig V_H, TCR J_α, and 3′ intervening sequence was hybridized to total RNA from the VP tumor. In this analysis, two fragments were protected from S1 nuclease digestion. The larger fragment corresponds to transcripts from the hybrid Ig V_H–TCR J_α (Fig. 4). The smaller fragment most likely reflects lower-level transcription of a closely related Ig V_H not associated with this TCR J_α.

This study complements our earlier analysis of a chromosome 14 inversion in the T-cell line SUP-T1. In SUP-T1 and in the B cells from VP this karyotypic abnormality was the result of site-specific recombination juxtaposing immunoglobulin V_H genes to TCR α-chain J segments. In the VP tumor the recombination is to a TCR J_α located approximately 4 kb further upstream from the TCR J_α involved in SUP-T1. In addition, the Ig V_H gene employed in the VP rearrangement belonged to subgroup III in contrast to the Ig V_H gene rearranged in the SUP-T1 cell line, which is a member of the subgroup II family. Thus, although the molecular mechanism of rearrangement in these two tumors is the same, the sites of recombination differ. In both instances the join is in-frame and transcription of the hybrid gene occurs.

Lymphocytes have traditionally been separated into two broad classes, B cells and T cells. Each subset is thought to serve different though interrelated roles in the immune system. Critical to these functions is the ability to specifically recognize and respond to antigen. This is mediated in large part by the distinctive cell surface receptors of lymphocytes: the Ig molecule in B cells and the TCR in T cells. Both Ig and TCR molecules achieve the diversity necessary to react to a broad array of antigens by site-specific VDJ or VJ recombination. In both systems these rearrangements are mediated by signal sequences that are structurally similar (*10*) and as shown by our analysis functionally interchangeable. Yancopoulos *et al.* recently provided evidence that TCR and Ig recombinations can be catalyzed by a common recombinase system (*11*). The specificity then, of which locus rearranges in which cell is unlikely to depend on the recombination machinery itself but rather on the activational state of the TCR and Ig loci. Ig heavy-chain rearrangements occurring in T cells and TCR β-chain rearrangements in B cells have been observed by others (*12*). Our analyses clearly show that both the TCR and Ig loci can be activated and united to form hybrid genes by VJ recombination in both B cells and T cells.

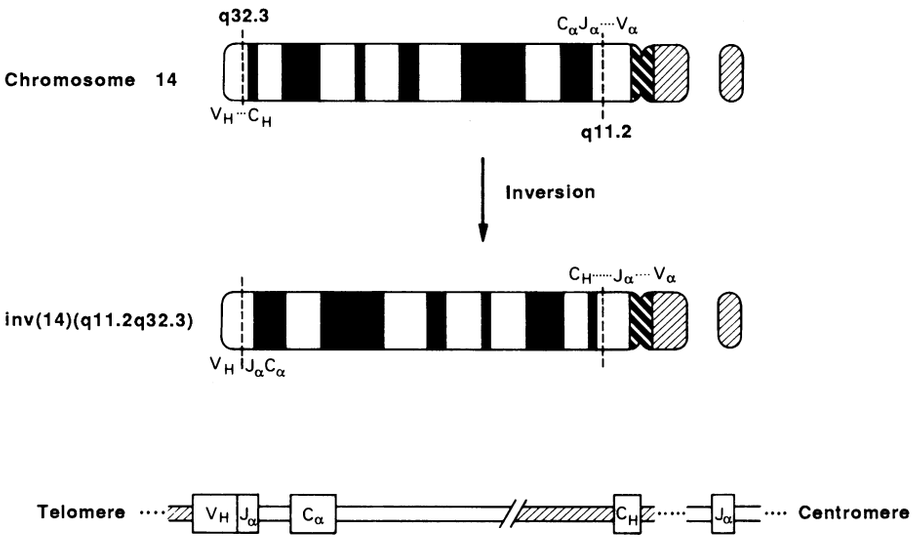

Fig. 3. A model for the mechanism of chromosomal inversion in the VP tumor. Idiograms of normal and inverted chromosomes 14 are depicted. Orientations and locations of Ig heavy-chain and TCR α-chain loci in both germline and inverted states are illustrated. Schematic representation is shown below: chromosomal band 14q11.2 sequences are open; band 14q32.3 sequences are hatched.

In the two cases we have described, the stage during lymphocyte development when these mixed TCR-Ig rearrangements occurred is unclear. It could be that early in lymphocyte development both Ig and TCR loci are open and accessible and that functional rearrangement of one or the other locus is an important step in determining whether a B-cell or T-cell differentiation pathway will be followed. However, the involvement of the TCR α-chain locus in VP and SUP-T1 complicates this concept because it has been speculated that the α-chain is the last of the TCR loci to rearrange and be expressed during T-cell development (*10*). We might not expect it to be "accessible" to recombination in a pre-B or pre-T stage of lymphocyte development. Thus, the rearrangement of the Ig loci may have occurred in SUP-T1 after commitment to a T-cell lineage. Similarly, late activation and rearrangement of the TCR loci may have occurred in the B cell in patient VP.

It is possible that these TCR-Ig recombinations are a result of the malignant state of these cells rather than of normal differentiative processes. However, an inversion of chromosome 14 identical to that seen in VP and SUP-T1 has been observed commonly in nonmalignant lymphocytes of patients with ataxia-telangiectasia (*13*). Although these patients are at risk for developing

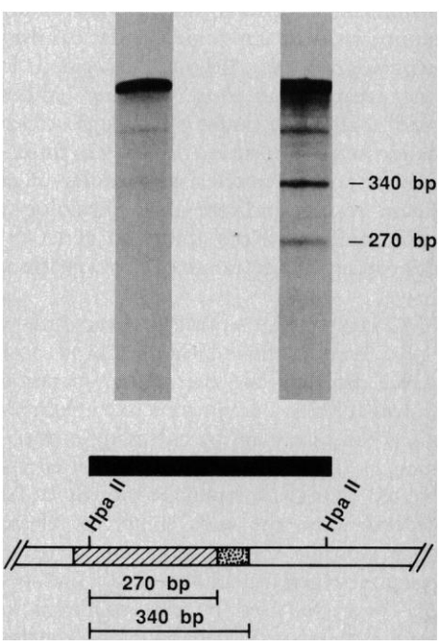

Fig. 4. Nuclease S1 analysis of VP tumor RNA. A ^{32}P-labeled single-stranded M13 probe (black bar) was hybridized to VP total RNA and subjected to S1 nuclease digestion (right panel). VP RNA affords protection to a 340-bp band whose size corresponds to a message containing Ig V_H–TCR J_α, and to a 270-bp band corresponding to transcription of another Ig V_H. Control hybridization and digestion in the absence of VP tumor RNA is shown in the left panel. In the lower schematic hatched areas represent Ig V_H; stippled, TCR J_α.

lymphocytic neoplasms, the inverted chromosome 14 (when present in normal lymphocytes) is not invariably present in the subsequent malignant clone (*14*). It may be that the Ig V_H–TCR J_α rearrangement must fulfill additional criteria of functionality or specificity in order to contribute to malignant transformation. Both SUP-T1 and VP tumors contain other karyotypic abnormalities that could provide the primary event in malignant transformation (*5, 15*). The inv(14) in these two tumors might confer an additional selective advantage. It is striking that in both cases a hybrid Ig V_H–TCR J_α transcript with an open reading frame is generated. The message for this hybrid receptor could be translated into a protein capable of providing an abnormal mitogenic stimulus to the cell resulting in deregulated proliferation.

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Brominating Oxidants Generated by Human Eosinophils

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Eosinophils are white blood cells that in humans are found in association with helminthic infections and various inflammatory disease processes. These cells contain a unique lysosomal peroxidase that oxidizes halides to generate highly reactive and toxic hypohalous acids. Although chloride is found in vivo at concentrations at least 1000-fold greater than those of other halides, human eosinophils did not preferentially oxidize chloride under physiologic conditions. Instead, eosinophils used bromide, a halide with a hitherto unknown function in humans, to generate a halogenating oxidant with characteristics similar, if not identical, to those of hypobromous acid. These results indicate that physiological concentrations of bromide arm human eosinophils with the ability to generate and release an unusual oxidant capable of destroying a wide range of prokaryotic and eukaryotic targets.

THE HUMAN EOSINOPHIL CAN PLAY A unique and beneficial role in host defense by destroying parasitic worms and also a detrimental role in inflammatory disease states by damaging host tissues (*1*). In both cases the destructive effects exerted by the eosinophil are thought to be dependent on the cell's ability to release toxic lysosomal components and to generate reactive oxygen metabolites (*1, 2*). The specific processes used by the eosinophil to carry out its specialized functions are not known, but increasing attention has focused on one of the major lysosomal proteins of the cell, the heme-enzyme eosinophil peroxidase (*1, 2*). In cell-free systems, purified eosinophil peroxidase utilizes H_2O_2 to catalyze the peroxidation of halides to highly reactive halogenating intermediates capable of destroying a host of targets ranging from multicellular worms to mammalian cells (*2–9*).

Recent studies have directly demonstrated that purified eosinophil peroxidase can oxidize chloride to the powerful oxidant hypochlorous acid (*10*).

$$H_2O_2 + Cl^- + H^+ \xrightarrow{\text{eosinophil peroxidase}} HOCl + H_2O$$

Because chloride is found in vivo at concentrations at least 1000-fold higher than those of any other halide (*11*), eosinophils might be expected to generate primarily HOCl under physiologic conditions. The ability of intact eosinophils to generate HOCl would allow the cell to mediate a variety of toxic effects (*12*), but chloride is oxidized less efficiently by purified eosinophil peroxidase than by myeloperoxidase, a lysosomal haloperoxidase that is found in the human neutrophil (*2, 13–16*). This information and the fact that these two peroxidases are distinct gene products with different heme and protein moieties (*17, 18*) have led to the suggestion that the true function of eosinophil peroxidase in the intact cell is still unknown (*18*). We have now shown that human eosinophils selectively utilize physiologic concentrations of bromide, a halide with a hitherto unknown function in humans, to generate a highly reactive and toxic oxidant with characteristics similar if not identical to those of hypobromous acid (HOBr).

In cell-free systems, purified eosinophil peroxidase can catalyze the H_2O_2-dependent oxidation of halides, but the ability of intact eosinophils to generate oxyhalides under physiologic conditions is unknown. Thus, purified eosinophils were isolated from the venous blood of normal volunteers (*19*), suspended in a physiologic, chloride-containing buffer (Dulbecco's phosphate-buffered saline, *p*H 7.4), and triggered to generate oxygen metabolites and release lysosomal components by the addition of either phorbol myristate acetate (PMA) (Consolidated Midland) or serum-opsonized zymosan particles (Sigma). As shown in Table 1, triggered eosinophils were able to generate significant quantities of HOCl after the addition of either stimulus. However, under identical conditions, triggered neutrophils produced even larger amounts of HOCl (Table 1). The attenuated ability of the eosinophil to generate HOCl relative to the neutrophil was not due to differences in the magnitude of the cells' respiratory burst.

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