of cells could be influenced by attention to stimuli throughout at least the central 12° of both the contralateral and ipsilateral visual fields (the maximum distance that could be tested). The magnitude of the effect was somewhat smaller than in V4 (Fig. 2C), possibly because IT neurons generally gave weaker, more variable responses than neurons in V4.

The results from area V4 and the IT cortex indicate that the filtering of irrelevant information is at least a two-stage process. In V4 only those cells whose receptive fields encompass both attended and unattended stimuli will fail to respond to unattended stimuli. In the IT cortex, where receptive fields may encompass the entire visual field, virtually no cells will respond well to unattended stimuli.

In contrast to area V4 and the IT cortex, there was no effect of attention in V1. When relevant and irrelevant stimuli were in a receptive field (typically 0.5° to 0.9° wide), the animal could not perform the task. When one stimulus was located inside the field and one just outside, the monkey was able to perform the task, but, as in V4 under this condition, attention had little or no effect on the cells (Fig. 2D).

Our results indicate that attention gates visual processing by filtering out irrelevant information from within the receptive fields of single extrastriate neurons. This role of attention is different from that demonstrated previously in the posterior parietal cortex (*3*), to our knowledge the only other cortical area in which spatially directed attention has been found to influence neural responses. In the posterior parietal cortex, some neurons show enhanced responses when an animal attends to a stimulus inside the neuron's receptive field compared to when the animal attends to a stimulus outside the field.

Since parietal neurons have large receptive fields with little or no selectivity for stimulus quality, these cells may play a role in directing attention to a spatial location (*4*), but by themselves do not provide information about the qualities of attended stimuli. By contrast, in area V4 and the IT cortex selective attention may allow the animal to identify and remember the properties of a particular stimulus out of the many that may be acting on the retina at any given moment. If so, then the attenuation of response to irrelevant stimuli found in V4 and the IT cortex may underlie the attenuated processing of irrelevant stimuli shown psychophysically in humans (*5*).

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2. Both sample and test stimuli were presented for 200 msec, with a delay between them of 400 to 600 msec. The sample and test were randomly chosen on each trial from a set of two stimuli, and the irrelevant stimuli were chosen from a different set of two. If the animal attempted to perform the task on the basis of the irrelevant stimuli, its performance would be governed by chance. The performance of the animals was 94 percent correct. The cue to the animal to switch the locus of its attention was to delete the test-time stimulus from the previously relevant location for two trials. On the first of these trials, the animals' performance dropped to 65 percent correct and their reaction time increased by 90 msec, indicating that they had been ignoring the irrelevant stimuli. The neural responses on the two cue trials were not counted. The locus of attention was switched frequently enough to achieve a minimum of ten trials per stimulus configuration. Fixation was monitored by a magnetic search coil, and trials were aborted if the eyes deviated from the fixation target by more than 0.5°.
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Locus of the α-Chain of the T-Cell Receptor is Split by Chromosome Translocation in T-Cell Leukemias

Abstract. *Mouse lymphoma cells were hybridized with two human acute T-cell leukemias with a t(11;14) (p13;q11) translocation and the segregated hybrids were examined for the presence of the DNA segments coding for the constant (C) and the variable (V) regions of the α chain (C_α and V_α) of the T-cell receptor. The C_α segment was translocated to the involved chromosome 11 ($11p^+$) while the V_α segment remained on the involved chromosome 14 ($14q^-$). The data indicate that the locus for the α chain of the T-cell receptor is split by the chromosomal breakpoint between the V_α and the C_α gene segments, and that the V_α segments are proximal to the C_α segment within chromosome band 14q11.2.*

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The locus for the α-chain of the T-cell receptor is at band q11.2 of chromosome 14 (*1*), a chromosome region that is involved in inversion and translocations in T-cell tumors (*2*). In Burkitt lymphoma, immunoglobulin gene loci are split by the three chromosome translocations associated with this tumor (*3*) and are involved in rearrangements with the c-*myc* locus (*3*), leading to deregulation of the c-*myc* gene involved (*4*). In this paper, we have tested whether the locus for the α-chain of the T-cell receptor is directly involved in the chromosome rearrangements observed in T-cell neoplasms, since this locus could be similarly involved in proto-oncogene activation (*1*).

Cells from two (8511 and 8508) acute T-cell leukemias (ALL) that are characterized by a t(11;14) (p13;q11) chromosome translocation (*5*) were hybridized with BW5147 mouse T-cell lymphoma cells deficient in hypoxanthine phosphoribosyltransferase (*6*). The hybrids were selected in HAT (hypoxanthine, aminopterin, thymidine) medium containing 10^{-7} *M* ouabain (*7*). Several hybrids were derived from the fusion of BW5147 cells with 8511 leukemia cells (hybrid number 517), while the others were derived from the fusion of BW5147 cells with 8508 leukemia cells (hybrid number 515) (Table 1). In T cells from ALL patients with a t(11;14) translocation, the breakpoint on chromosome 14 has been previously assigned to band 14q13 (*5*), but karyologic analysis of our ALL hybrids indicates that the breakpoint is probably closer to the centromere and involves band 14q11 (Fig. 1), in agreement with other reports (*2*). The breakpoint on chromosome 11 may also be more proximal than the p13 location previously reported (*5*).

The presence of the relevant chromosomes in the hybrid clones is summarized in Table 1. Southern blot analysis of DNA from the hybrids (Fig. 2) indi-

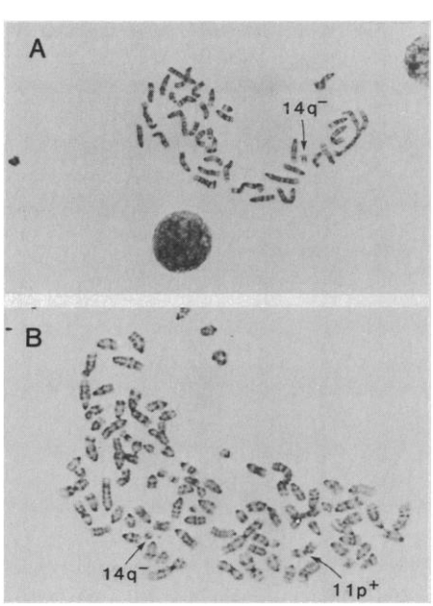
cates that those hybrids with only the $14q^-$ chromosome and no other relevant chromosome, contain segments encoding the variable (V_α) but not the constant (C_α), region of the α-chain (Table 1 and Fig. 1). However, hybrids carrying the $14q^-$ chromosome and the $11p^+$ chromosome, but not the other relevant chromosomes, contain both the V_α and C_α segments (Table 1 and Fig. 1). Hybrid 517 B-D3-D2A, which has the $11p^+$ and no other relevant chromosome, has only Cα sequences (Table 1). We conclude that the chromosome breakpoints in both cases of ALL directly involve the locus for the α chain of the T-cell receptor and that the V_α segments are proximal to the C_α segment at band 14q11.

We have also examined these hybrids for the expression of lactic dehydrogenase-A (LDH-A) and nucleoside phosphorylase, which have been previously mapped to 11p (*8*) and 14q (*9*), respectively; for the presence of the Harvey *ras* oncogene (c-H-*ras*) and β-globin, that have been assigned to 11p (*10*); and for the *bcl*-1 locus, that has been assigned to

Table 1. Presence of the V_α and C_α genes in hybrids between mouse BW5147 cells and human ALL cells.

Hybrids	Locus		Human markers*					Human chromosomes†			
	V_α	C_α	NP	LDH-A	c-H-*ras*	β-globin	*bcl*-1	14	$14q^-$	11	$11p^+$
517 A-A3	+	+	+	+	+	+	−	++	+++	−	−
517 B-D3	+	+	+	+	+	+	+	−	+	−	+
517 B-B1	−	−	−	+	+	+	+	−	−	+	−
517 B-A3	+	−	+	+	+	+	−	−	+	−	−
517 B-D3-G8	+	−	+	+	+	+	−	−	++	−	−
517 B-D3-G9	+	+	+	+	+	+	+	−	++	−	+++
517 B-D3-D2A	−	+	−	−	−	−	+	−	−	−	++
517 A-A3-A10	+	+	+	+	+	+	−	+	++	−	−
517 A-A3-G7	+	−	+	+	+	+	−	−	+++	−	−
515 BD2-CF3	+	−	+	+	+	+	−	−	+	−	−
515 BD2-CF6	+	−	+	+	+	+	−	−	+++	−	−

* NP, nucleoside phosphorylase; LDH-A, lactic dehydrogenase A. †Frequency of metaphases with relevant chromosomes: −, none; +, 10–30 percent; ++, 30–50 percent; +++, >50 percent. At least 25 metaphases were examined for each hybrid after trypsin-Giemsa staining (*3*). Selected metaphases were studied by the G11 technique to confirm the human origin of relevant chromosomes (*3*).



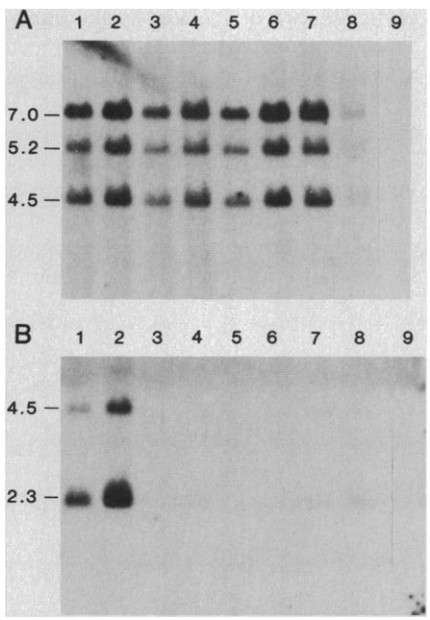


Fig. 1 (left). (A) Partial trypsin–Giemsa–banded metaphase from hybrid subclone 517 AA3-G7 containing the $14q^-$ chromosome (arrow), but not the normal 14, normal 11, or the $11p^+$. (B) Partial trypsin–Giemsa–banded metaphase from hybrid 517 B-D3 containing both the $11p^+$ and $14q^-$ translocation chromosomes (arrows), but not the normal 11 or normal 14.

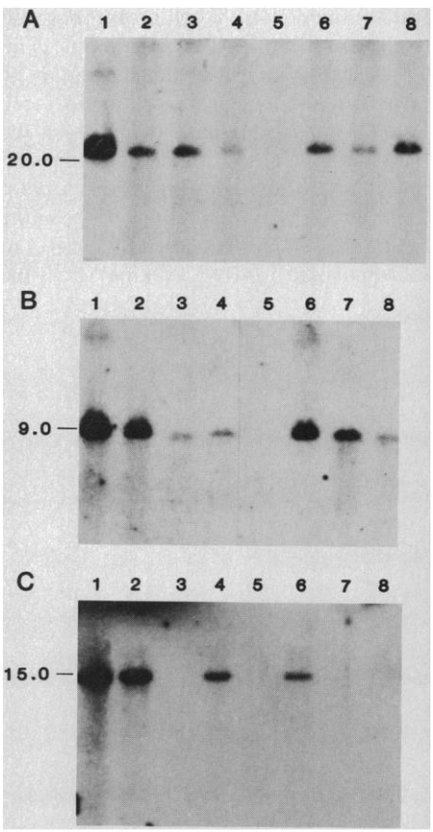


Fig. 2 (center). Southern blot hybridization of Hind III–digested cellular DNA hybridized to a V_α–specific probe (A) and a C_α–specific probe (B). The V_α probe is the Ava I–Pst I fragment isolated from the pHαT3 cDNA clone (*1*). The C_α probe is the cDNA clone pHαT1, which contains part of the constant region and all of the 3′ untranslated region of the α-chain locus of the T-cell receptor (*1*). Lane 1, DNA isolated from hybrid 517 B-D3; lane 2, parental DNA from patient 8511; lane 3, hybrid 517 B-A3; lane 5, hybrid 517 A-A3-G7; lane 6, hybrid 517 B-D3-G8; lane 7, hybrid 515-BD2-CF6; lanes 4 and 8, two additional hybrid DNA's; lane 9, the parental BW5147. Sizes are in kilobases (kb). Fig. 3 (right). Southern blot hybridization of cellular DNA's. Lane 1, patient 8511; lane 2, hybrid 517 B-D3; lane 3, hybrid 517 A-A3-G7; lane 4, hybrid 517 B-B1; lane 5, the mouse thymoma fusion partner BW5147; lane 6, hybrid 517 B-D3-G9; lane 7, hybrid 517 A-A3; lane 8, hybrid 517 B-D3-G8. (A) The DNA was digested with Eco RI and hybridized with a human c-Harvey-*ras* probe (c-*bas*) (*12*). (B) The DNA was digested with Hind III and hybridized to a human β-globin probe, H15 (*12*). This probe contains the Pst I–Pst I fragment that corresponds to nucleotides 9112–13554 in the β-globin map. The differences in the intensities of the bands in lanes 7 and 8 of (A) and (B) are due to differences in amounts of DNA's loaded in these lanes. (C) The DNA was digested with Bcl-1 and hybridized with pRcSMR, a probe specific for 11q13 (*bcl*-1) (*11*). Sizes are in kb.

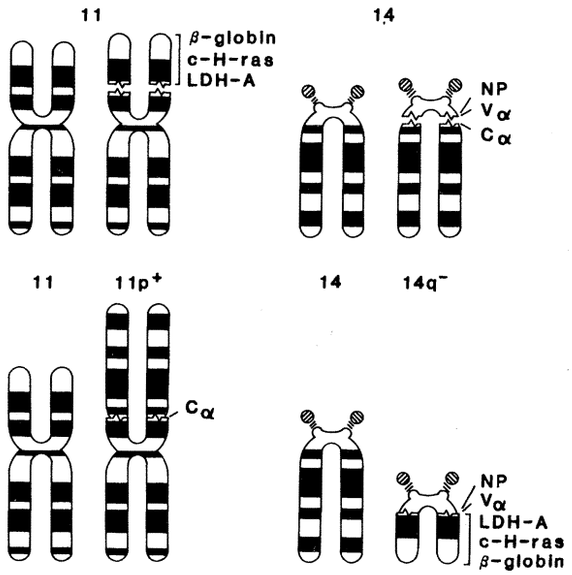


Fig. 4. The t(11;14) (p13;q11) translocation in ALL. The translocation breakpoint on chromosome 14 splits the locus for the α-chain of the T-cell receptor. The V_α genes remain on the $14q^-$ chromosome, while the C_α translocates to the involved chromosome 11 ($11p^+$). The gene for human nucleoside phosphorylase remains on the involved chromosome 14 ($14q^-$). The genes LDH-A, β-globin, and c-H-*ras* translocate to the involved chromosome 14 ($14q^-$).

11q13 (*11*). The genes for the β-globin and LDH-A, as well as c-H-*ras* (*12*), were distal to the breakpoint on 11p, while the gene for nucleoside phosphorylase was proximal to the q11.2 breakpoint on chromosome 14 (Table 1 and Figs. 3 and 4).

As these results indicate, the locus for the α-chain of the T-cell receptor is split by the chromosome 14 breakpoint (Fig. 4). It seems likely that this locus may be involved in oncogene activation in T-cell neoplasms in a manner similar to the involvement of the immunoglobulin loci in Burkitt lymphoma (*3*). Thus, a putative oncogene, for which we propose the name of *tcl*-2, may reside at band p13 of chromosome 11. By "walking" 5′ of the C_α locus on appropriately rearranged chromosomes it should be possible to isolate genes involved in T-cell tumors, as we have done in the case of B-cell lymphomas and leukemias carrying the t(11;14) (q13;q32) and t(14;18) (q32;q21) chromosome translocations (*11, 13*).

While the two leukemias we have investigated are not related to the human T-cell leukemia virus (HTLV-I), a recent report indicates that there are abnormalities (translocations and inversions) of chromosome 14 at band 14q11 in Japanese patients with T-cell leukemias (*14*). This suggests that HTLV-I may not be leukemogenic per se. This virus may play an important role in leukemogenesis, perhaps analogous to the role of Epstein Barr virus in African Burkitt lymphoma (*3*), by expanding the population of lymphocytes at risk of developing specific chromosomal translocations. Thus activation of *tcl*-1 (*1*) and *tcl*-2 in T-cell neoplasms because of their proximity to the locus for the α-chain of the T-cell receptor may be the critical step in the pathogenesis of many T-cell malignancies.

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