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16. Subjects were restrained with ketamine (5 to 10 mg per kilogram of body weight), anesthetized with sodium pentobarbital (32.5 mg per kilogram) administered intravenously, heparinized (400 units per kilogram), and given 2 ml of 1 percent sodium nitrite intravenously. The animals were perfused with 4 liters of cold saline solution (0.9 percent) prepared in 0.1*M* phosphate buffer (*p*H 7.4) and then with 12 liters of cold paraformaldehyde (4 percent) and glutaraldehyde (0.1 to 0.2 percent) in 0.1*M* phosphate buffer (*p*H 7.4). A 26-year-old macaque was restrained with ketamine and suffered a cardiac arrest. After cardiopulmonary resuscitation, the animal was heparinized and perfused via the left ventricle with 4 liters of cold saline (0.9 percent) for 5 to 10 minutes, then with 10 liters of acetate-buffered Formalin (10 percent, *p*H 6.5) for 30 minutes, and finally with 7 liters of acetate-buffered Formalin and glutaraldehyde (10 and 0.16 percent, respectively) for 15 minutes.
17. After perfusion, the brains were blocked into coronal slabs (2 to 5 mm). Some tissue sections were cut (50 to 100 μm) on a Vibratome (Lancer) and collected in cold 0.1*M* tris-buffered saline (TBS, *p*H 7.6); remaining tissues from these monkeys were stored in 30 percent sucrose in 0.1*M* phosphate buffer (*p*H 7.4) overnight, frozen on dry ice the next day, cut at 40 μm on a sliding freezing microtome (American Optical), and collected into cold TBS.
18. For immunocytochemistry, sections were incubated and treated with solutions as follows. (i) Triton X-100 (0.4 percent, Sigma) in 0.1*M* TBS (*p*H 7.6) for 20 minutes (20°C); (ii) normal goat serum (3 percent, Dako) in Triton X-100 (0.1 percent) and 0.1*M* TBS for 30 to 60 minutes (20°C); (iii) antibody to ChAT (1:500 to 1:2000) (*14*) in the same solution containing 1 percent normal goat serum for 24 to 48 hours (4°C) with three subsequent rinses (5 minutes each) in cold TBS; (iv) goat antibody to mouse immunoglobulin (1:100, Cappell) in Triton X-100 (0.1 percent) and 0.1*M* TBS containing normal goat serum (1 percent) for 1 hour (4°C) with three subsequent rinses (5 minutes each) in cold TBS; (v) a complex of mouse peroxidase and monoclonal antibody to peroxidase (peroxidase-antiperoxidase; 1:100, Sternberger and Meyer) in 0.1*M* TBS containing normal goat serum (1 percent) for 1 hour (4°C) with three subsequent rinses (5 minutes each) in cold TBS; (vi) a fresh solution of goat antibody to mouse immunoglobulin (1:100) without Triton X-100 for 1 hour (4°C) and three subsequent rinses (5 minutes each) in cold TBS; (vii) a fresh solution of mouse peroxidase-antiperoxidase (1:100) for 1 hour (4°C) with three subsequent rinses (5 minutes each) in cold TBS; (viii) 3,3′-diaminobenzidine tetrahydrochloride (0.05 percent, Aldrich) and H_2O_2 (0.01 percent, Mallinckrodt) in 0.1*M* TBS for 5 to 15 minutes (20°C); (ix) as a control, rat immunoglobulin (1:500 to 1:2000, Sigma) from a stock solution (1 mg of protein per 1 ml of TBS) replaced the antibody to ChAT. Sections were washed in TBS and mounted out of acetate buffer (*p*H 6.0) onto slides that had been coated with chrome-alum. All sections were dehydrated through alcohols and xylenes and mounted with glass cover slips and Permount. Selected sections were treated with OsO_4 (0.05 percent) in H_2O for 0.5 to 1.0 minute and mounted. Finally, selected sections were counterstained with cresyl violet to visualize nuclear groupings.
19. Some slides were stained with thioflavin T (Roboz) as described [P.S. Vassar and C. F. A. Culling, *Arch. Pathol.* **68**, 487 (1959)].
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Common Region on Chromosome 14 in T-Cell Leukemia and Lymphoma

Abstract. *Chromosome 14 breakpoints in malignant human lymphocytes cluster on the long (q) arm near bands q11 and q32. An inversion of chromosome 14 due to breaks in q11.2 and q32.3 has now been found in a newly established childhood T-cell lymphoma cell line and confirmed in T-cell chronic lymphocytic leukemia. A translocation was also found between chromosomes 10 and 14 with a breakpoint at 14q11.2 in another T-cell lymphoma cell line. It is proposed that a proximal region on chromosome 14 in or near sub-band q11.2 is related to T-cell function. Rearrangements in this region may affect the growth of T lymphocytes and be involved in the development of T-cell malignancies.*

Nonrandom chromosome abnormalities mark certain forms of human cancer (*1*). In T-cell chronic lymphocytic leukemia (CLL), an inversion of chromosome 14 due to a break proximally in band q11 and a break distally in band q32 has been reported (*2, 3*). We have observed this chromosome 14 inversion in an established T-cell lymphoma cell line and confirm its occurrence in T-cell CLL. However, the change in chromosome 14 in T-cell malignancies can also be a translocation. The common feature appears to be rearrangement of the proximal region on 14q.

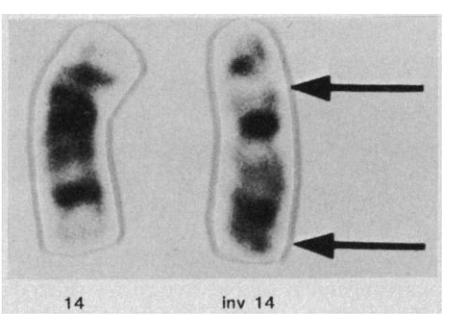

Fig. 1. High-resolution G banding of normal chromosome 14 (left) and inverted chromosome 14 (right) with arrows pointing to breakpoints in q11.2 (above) and q32.3 (below) in T-cell lymphoma cell line SUP-T1.

A T-cell lymphoma cell line (SUP-T1) (*4*) was established from a pleural effusion in a 6-year-old boy who was diagnosed 15 months earlier as having lymphoblastic lymphoma. There was no involvement of bone marrow or central nervous system. The pleural effusion cells, which appeared uniformly malignant, were cultured (*5*). These cells were E-rosette negative at 4° and 37°C and negative for monoclonal antibodies to Leu 5, OKT9, CALLA (common acute lymphocytic leukemia antigen), Ia (I region–associated antigens), and SIg (surface immunoglobulin) but were positive for antibodies to Leu 1, 2a, 3a, 4, 6, and 9 and OKT10. This antigen profile is consistent with T-cells of mid-thymocyte type. Chromosome preparations were made for high-resolution G banding. The karyotype was 46,XY with an inversion of chromosome 14: inv(14)(q11.2q32.3) (Figs. 1 and 2).

The patient with T-cell CLL was a 59-

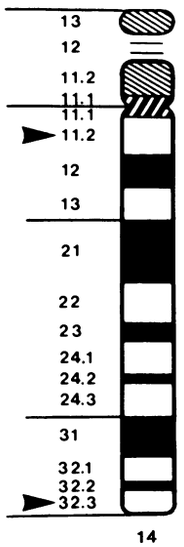

Fig. 2. High-resolution banding diagram of normal chromosome 14 with arrows pointing to the breakpoints in the inversion.

year-old man with a high peripheral blood lymphocyte count and massive hepatosplenomegaly. The lymphocytes appeared mature, averaged 12 μm in diameter, and had a ratio of nucleus to cytoplasm of 5:1. No human T-cell leukemia virus antibodies were detected in serum (*6*). Blood lymphocytes were cultured for 3 and 5 days with phytohemagglutinin, *Escherichia coli* lipopolysaccharide, and concanavalin A as mitogens. High-resolution banding disclosed the same inversion of chromosome 14: inv(14)(q11.2q32.3).

Another T-cell lymphoma cell line (SUP-T4) (*4*) has been established (*5*) from a patient in relapse. This cell line did not contain the inversion of chromosome 14. Instead, it had a translocation between chromosomes 10 and 14: t(10;14)(q23;q11.2) (Fig. 3). The inversion of chromosome 14 was thus found both in SUP-T1, a childhood T-cell lymphoma cell line, and in adult T-cell CLL. This inversion has been reported in a lymphosarcoma cell line (*7*) and has been described in direct chromosome studies of an adult T-cell lymphoma (*8*). The inversion of chromosome 14 also occurs in T-cell CLL (*2, 3*). Contrary to one interpretation (*3*), we believe that the chromosome 14 inversion is not a specific marker for T-cell CLL but rather is a marker for a number of T-cell malignancies.

To evaluate the functional significance of the chromosome 14 inversion, we determined the nucleoside phosphorylase (NP) activity (band 14q13) and immunoglobulin heavy-chain gene configuration (band 14q32) in the lymphoma cell line SUP-T1 (*9*). The NP activity in SUP-T1 was low (0.18 ± 0.02 enzyme units per milligram of protein) compared to normal lymphocytes (0.36 ± 0.05 enzyme units per milligram of protein, mean ± standard deviation) (*10*). However, since normal thymocytes have low NP activity, the activity in SUP-T1 appears appropriate for T cells of the mid-thymocyte stage (*11*). In a Southern blot analysis with a heavy-chain joining region (J_H) probe, the SUP-T1 cells showed a germ line heavy-chain configuration (*12*). In a case of T-cell CLL with the same inversion of chromosome 14, there was no evidence of heavy-chain rearrangement when J_H and constant region probes were used (*13*). While only a few T-cell tumors have been described with rearranged immunoglobulin genes, almost all B-cell tumors (with or without 14q32 changes) have heavy-chain rearrangements (*14*). Thus, no specific changes resulting from the chromosome inversion were identified. Aberrations of band 14q32 do not mandate heavy-chain gene rearrangements.

In 1975 we reported a translocation between both number 14 chromosomes in T-cell CLL in a patient with ataxia telangiectasia (*15*). To date, there are four reports of ataxia telangiectasia patients with leukemia who have had chromosome analysis. All had T-cell subacute lymphocytic leukemia (one case) or T-cell CLL (three cases) with a translocation t(14;14)(q11 → 12;q32) (*16*). This translocation between both number 14 chromosomes is analogous to the inversion of chromosome 14. It juxtaposes the proximal and distal regions of the long (q) arm of chromosome 14: bands 14q11 → 13 and 14q32. All rearrangements juxtaposing these two regions of 14q in malignant cells have, to our knowledge, been in T-cell malignancies. Other chromosome 14 rearrangements are found in T-cell malignancies (*8*). These rearrangements include, for example, a translocation between chromosomes 11 and 14 in T-cell acute lymphoblastic leukemia (*17*) and the translocation between chromosomes 10 and 14 in T-cell lymphoma.

The common denominator in all these chromosome 14 rearrangements in T-cell malignancies is a break in the proximal portion of 14q. This break is variously reported as being in bands 14q11 → 13 (*2, 3, 7, 8, 15–17*). On the basis of chromosome studies of ataxia telangiectasia, we suggested in 1975 that "structural rearrangement of 14q is directly related to abnormal growth of lymphocytes" as a "step in the development of lymphoid malignancies" (*15*). It now appears that chromosome 14 contains two (or more) regions relating to lymphocytes: a distal region, 14q32, containing genes pertinent to B cells, the immunoglobulin heavy-chain genes (*18*); and a proximal region, which we propose concerns T-cells. This T-cell region would seem by high-resolution chromosome banding to be in or near sub-band 14q11.2.

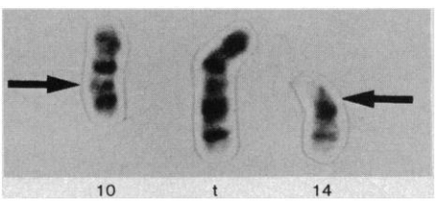

Fig. 3. Translocation between chromosomes 10 and 14 in T-cell lymphoma cell line SUP-T4 with arrows pointing to breakpoints in q23 and q11.2, respectively.

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5. Cells were cultured in petri dishes with two layers of agar: a feeder layer of normal peripheral blood leukocytes (10^6) and erythrocytes (10^6) in 0.5 percent agar, and an overlayer of test cells (5×10^5) in 0.3 percent agar. The cultures were incubated at 37°C in a hypoxic (7 percent O_2, 10 percent CO_2, 83 percent N) environment for 18 days. Single colonies were selected, passaged in agar with a fresh feeder layer, and transferred into liquid culture media. Details of this culture system have been described for acute lymphocytic leukemia [S. D. Smith, E. M. Uyeki, J. T. Lowman, *Blood* **52**, 712 (1978)] and for non-Hodgkin's lymphoma [S. D. Smith, G. W. Wood, P. Fried, J. T. Lowman, *Cancer* **48**, 2612 (1981)].
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Different Red Light Requirements for Phytochrome-Induced Accumulation of *cab* RNA and *rbcS* RNA

Abstract. *For several species of plants the abundance of those transcripts encoding the chlorophyll a/b binding protein (*cab *RNA) and the small subunit of ribulose-1,5-biphosphate carboxylase-oxygenase (*rbcS *RNA) has been established as being under the control of phytochrome. However, this conclusion does not take into account the various types of phytochrome control based on both the fluence of red light necessary to induce the response and the ability of far red light either to induce or to reverse the response. The fluence of red light necessary to induce the accumulation of* rbcS *RNA was found to be 10,000 times greater than that necessary to induce the accumulation of* cab *RNA. Furthermore, far red light alone was capable of inducing the accumulation of* cab *RNA. It is possible, therefore, that developing pea buds accumulate* cab *RNA before* rbcS *and that* cab *RNA is not subject to the normal end-of-day signals affecting many phytochrome responses.*

The abundance of those transcripts encoding the light-harvesting chlorophyll a/b protein complex (*cab* RNA) and the small subunit of ribulose-1,5-bisphosphate carboxylase-oxygenase (*rbcS* RNA) is regulated by phytochrome in pea (*1*, *2*), mung bean (*1*), *Lemna* (*3*), and barley (*4*). Excitation of the phytochrome system similarly affects the abundance of several other nuclear- and chloroplast-encoded transcripts: phytochrome in oats (*5*), reduced nictotinamide adenine dinucleotide phosphate: protochlorophyllide oxidoreductase in barley (*6*), the large subunit of *rbc* protein in pea (*1*) and mung bean (*1*), a 17-kilodalton chloroplast polypeptide in pea (*7*), photogene in pea (*1*) and mustard (*8*), and approximately ten other nuclear-encoded transcripts in pea (*1*) and five others in mung bean (*1*).

In several of these studies, multiple pulses of red light or dark-adapted rather than dark-grown plants were used. In all the studies pulses of red light having extremely large fluences were used to elicit the phytochrome response. Furthermore, time-course studies (*1*, *2*) were designed on the basis of these large fluences, and a relation was suggested between them and the apparent development of the plant. Because land plant seedlings are normally exposed to a logarithmic gradient of red light as they grow up through the soil (*9*), knowing the fluence of red light at which the transcripts of phytochrome-induced genes begin to show altered abundance would presumably be useful in studies of normal seedling development in the soil.

Two kinds of phytochrome responses are defined by the fluence of red light at which they are initiated and by the abili-

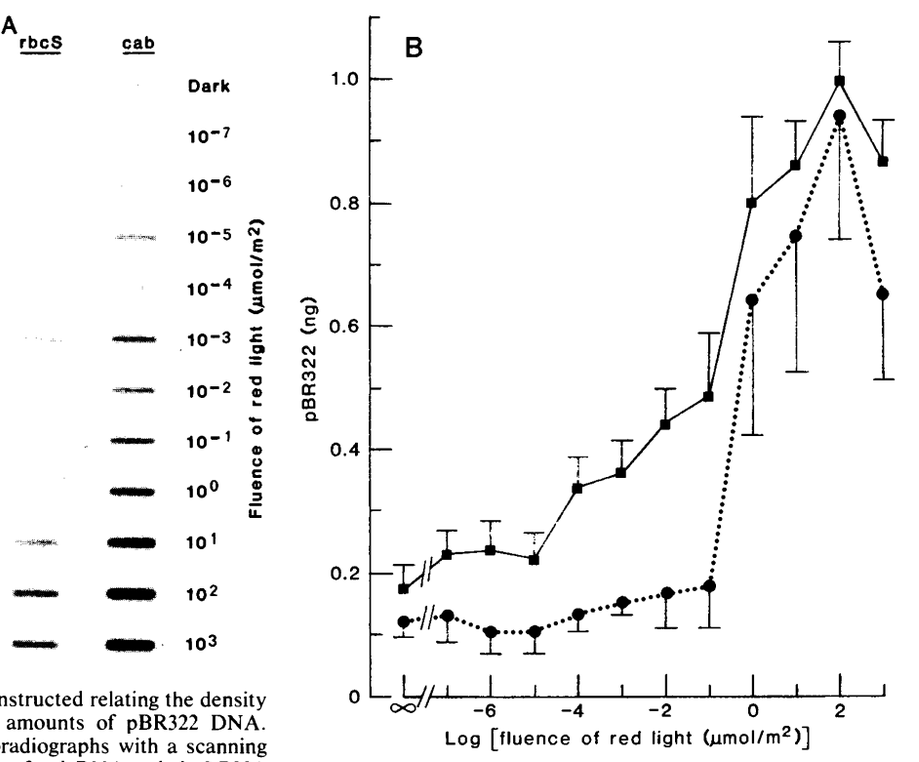

Fig. 1. Red light fluence response of *cab* RNA and *rbcS* RNA. Seeds of *Pisum sativum* var. Alaska were imbibed for 5 hours at 28°C in absolute darkness, planted on water-saturated Kimpac (Kimberly-Clarke), and maintained in absolute darkness at 28°C and 85 percent relative humidity for 6 days. Sets of seedlings were irradiated at each of the fluences indicated. Times for irradiations (μmol/m^2) were as follows: 10^{-7} to 1, 0.1 second; 10, 1 second; 10^2, 10 seconds; 10^3, 100 seconds. The red light source has been described (*12*). All seedlings were returned to darkness for 24 hours. Apical buds were harvested onto ice under dim green light in a 4°C cold room, quickly frozen in liquid nitrogen, and stored at −70°C until the RNA was extracted. Extracted RNA was fixed to nitrocellulose filters. The cDNA probes for *cab* RNA (pAB96) and *rbcS* RNA (pSS15) (*14*) were nick-translated and used to probe the blots. Hybridization conditions have been described (*1*); wash conditions were 0.3 × standard saline citrate at 65°C. A dilution series of the plasmid vector pBR322 DNA was also present on each slot blot. Hybridization of the pBR322 sequences in the probes to the pBR322 DNA on the blot provided a set of concentration standards on each autoradiograph. A standard curve was constructed relating the density of the autoradiograph images to the known amounts of pBR322 DNA. Density was determined by scanning the autoradiographs with a scanning densitometer (Hoeffer). The relative abundance of *cab* RNA and *rbcS* RNA was determined by comparing the density of the experimental slots to the standard curve. Each point represents three to five independent experiments. In each experiment approximately 100 pea buds were used per point. (A) An example of the slot blots used to calculate the fluence response curves shown in (B) for *cab* RNA (■) and *rbcS* RNA (●).