in the BXH (12 strains) and CXB (7 strains) series of recombinant inbred strains. Furthermore, the two *H-8* congeneic mouse strains, B6.C-*H-8*^{c}/By and B10.D2 (57N)/Sn, contain the BALB/c and DBA/2 alleles, respectively, at both the *Np-2* and α-chain gene loci. We propose *Tcra* as a symbol for the T-cell receptor α-chain gene locus.

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The human T-cell receptor α-chain gene maps to chromosome 14

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The T-cell receptor for antigen has been identified as a disulphide-linked heterodimeric glycoprotein of relative molecular mass (M_r) 90,000 comprising an α- and a β-chain[1-3]. The availability of complementary DNA clones encoding mouse[4] and human[5] β-chains has allowed a detailed characterization of the genomic organization of the β-chain gene family and has revealed that functional β-chain genes in T cells are generated from recombination events involving variable (*V*), diversity (*D*), joining (*J*) and constant (*C*) gene segments[6,7]. Recently, cDNA clones encoding mouse[8,9] and human[10] α-chains have been described; the sequences of these clones have indicated that functional α-chain genes are also generated from multiple gene segments. It is possible that chromosomal translocations involving T-cell receptor α- and β-chain genes have a role in T-cell neoplasms in much the same way as translocations involving immunoglobulin genes are associated with oncogenic transformation in B cells[11]. In the latter case, the chromosomal localization of the immunoglobulin genes provided one of the first indications of the involvement of such translocations in oncogenic transformation. The chromosomal assignment of the α- and β-chain genes may, therefore, provide equally important clues for T-cell neoplastic transformation. The chromosomal location of the mouse and human β-chain gene family has been determined: the murine gene lies on chromosome 6 (refs 12, 13) whereas the human gene is located on chromosome 7 (refs 13, 14). Here we use a cDNA clone encoding the human α-chain to map the corresponding gene to chromosome 14.

The α-chain cDNA clone used in this study was isolated from a $\phi\lambda$gt10 library, constructed from messenger RNA isolated from the human T-cell line Jurkat[15], by cross-hybridization with a homologous mouse probe[8]. The sequence of the insert corresponds exactly to that of a previously published α-chain cDNA clone isolated from HPB-MLT[10] and comprises about three-quarters of the constant region plus almost the entire 3'-untranslated region of the human α-chain (M.K.L.C., M.J.O., G.T. and S.T., manuscript in preparation).

The insert (~900 base pairs, bp) from the $\phi\lambda$gt10 clone was subcloned into pBR322 and the resulting plasmid (pJ6α2) used to screen human and mouse DNA, and a panel of mouse/human somatic cell hybrid DNAs using Southern hybridization analysis. Human DNA digested with *Hin*dIII and hybridized with the pJ6α2 insert shows two bands of 4.2 and 2.9 kilobases (kb)

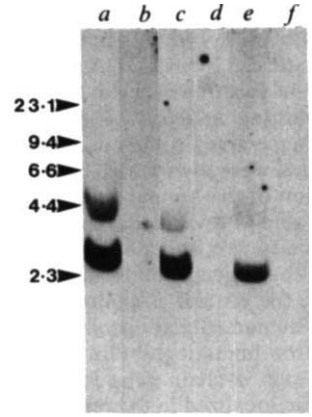

Fig. 1 Southern hybridization analysis of mouse/human somatic cell hybrid DNA. DNA of high M_r was prepared according to Maniatis *et al.*[34] and 20 μg was digested with *Hin*dIII and electrophoresed through 0.8% agarose, blotted onto nitrocellulose and hybridized with oligo-labelled[35] insert from pJ6α2. Hybridization was carried out at 65 °C in 3×SSC. The filters were washed at 65 °C in 0.2×SSC. The sizes of the DNA fragments were determined by reference to the mobilities of $\phi\lambda$*Hin*dIII markers. *a*, Human fibroblast (Bu) DNA; *b*, mouse L-cell (1R) DNA; *c*, hybrid F4SC13 C19 DNA; *d*, hybrid HORL411B6P DNA; *e*, hybrid FIR5R3 DNA; *f*, hybrid DT1.2R DNA.

Table 1 Segregation of a human α-chain gene in somatic cell hybrids

Hybrid (ref.)	Human chromosomal constitution	Presence of human-specific bands*
DUR4.3 (25)	3, 10, 11, 12, 13, 14, 15, 17, 18, 20, 21, 22, X	+
CTP34B4 (26)	1, 2, 3, 5, 6, 7, 8, 12, 14, 16, 17, 18, X	+
SIR74ii (27)	1, 2, 4, 12, 14, 17, 18, 20, 21, 22, X	+
3W4 C15 (28)	7, 10, 11, 12, 14, 15, 17, 21, X	+
HORP27R C14 (29)	4, 7, 11, 12, 14, 15, 21, X	+
CTP41-2 (30)	2, 7, 8, 14, 16, 17, X	+
F4SC13 C19 (31)	1, 14, X	+
FIR5R3 (32)	14, 18	+
DT1.2R (25)	3, 10, 11, 17, 18, 20, 21, 22	−
HORL411B6P (29)	1, 3, 11, 18, 22, X	−
Cl 21 (33)	7	−
Controls		
1R (28)	Mouse L-cell	−
Bu†	Human fibroblast	+

Note that several of the hybrids have been recloned and recharacterized since their description in the original reference. Human chromosomal constitution was determined by a combination of karyotypic[25], isozyme[36] and antigenic analysis[30]. Batches of cells for DNA production were checked by isozyme analysis.

* The hybridization conditions are given in Fig. 1 legend. Under conditions of high stringency only the 4.2- and 2.9-kb human bands were detected. In all cases where the hybrids were scored positive both bands were visible.

† A human fibroblast line derived from a human ovarian teratoma. This line was obtained from Dr S. Povey (MRC Biochemical Genetics Unit, London).

(Fig. 1*a*). This result is consistent with the nucleotide sequence of the human α-chain (ref. 10 and G.T. and S.T., unpublished observation), in which a *Hin*dIII site is located in the constant region, and suggests that a single constant-region gene segment is present in the human genome. One hybridizing band of 6 kb was detected with *Hin*dIII-digested mouse DNA probed at low stringency (data not shown). As the 4.2- and 2.9-kb bands were apparently specific to human DNA and the mouse-specific bands could be removed by washing at high stringency (Fig. 1*b*), the presence of the 4.2-kb and 2.9-kb fragments in *Hin*dIII-digested mouse/human somatic cell hybrid DNA could be used to determine the chromosomal location of the α-chain gene. Figure 1*c–f* shows Southern blotting analysis of DNA from four such hybrids. For hybrids positive for the human α-chain gene, both human-specific bands were always detected (see, for example, Fig. 1*c*, *e*). Inspection of the human chromosomal content of a panel of hybrids (Table 1) reveals that the human α-chain gene maps to chromosome 14. We, and others, have shown previously that the human β-chain gene family maps to chromosome 7 (refs 13, 14). Thus, the α- and β-chain genes lie on different chromosomes. The human β-chain genes also lie on a different chromosome from the immunoglobulin *Igh*, *Igk* and *Igl* loci. In contrast, the human α-chain gene is syntenic with the *Igh* loci, which have been localized in the region 14q32.33-14qter[16]. The human oncogene c-*fos* has also been localized to chromosome 14 in the region 14q21-q31[17].

A conserved linkage group between human chromosome 14 and mouse chromosome 14 occurs at the nucleoside phosphorylase (*Np*) locus (at 14q13.1 in human; reviewed in ref. 18). In man, nucleoside phosphorylase deficiency has been associated with severe T-cell immunodeficiency[19]. Interestingly, the gene encoding the mouse α-chain has been mapped to chromosome 14, close to the *Np-2* locus[20,21]. This observation suggests that the human α-chain gene may also lie close to the *Np* locus in a conserved linkage group. If this is the case, the human α-chain gene, although on the same chromosome, must reside a considerable distance from the *Igh* locus.

Abnormalities of chromosome 14 have been associated with human B-lymphocyte malignancies. Thus, Burkitt lymphomas are often associated with 8;14 translocations resulting in a juxtaposition of the c-*myc* and *Igh* loci[11]. Inversions of chromosome 14 due to breaks near q11 and q32 are found in T-cell chronic lymphocytic leukaemia and lymphomas, and simple translocations involving chromosome 14 have also been observed in T-cell malignancies[22–24]. For example, a translocation involving chromosomes 10 and 14 with a breakpoint at 14q11.2 has been found in a T-cell lymphoma cell line[24]. The involvement of the 14q11 region in these inversions and translocations suggests that it is related to T-cell function and that rearrangements involving this region may be involved in T-cell malignancies. If the human α-chain gene is located, by analogy with the linkage in the mouse, at or near the *Np* locus, it would be a candidate for such a gene. We are presently performing *in situ* hybridization analysis to test this possibility.

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Stimulation of the Na/H exchanger of sea urchin eggs by phorbol ester

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On fertilization of a sea urchin egg, marked changes occur in the cytoplasmic concentration of calcium and hydrogen ions. These ionic signals represent the necessary and sufficient stimuli for the increased metabolism, protein synthesis and DNA synthesis that constitute egg activation[1–3]. Cytoplasmic alkalinization, the major immediate cause of the increased rate of protein synthesis which