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The Gene Encoding Human Vimentin Is Located on the Short Arm of Chromosome 10

SERGIO FERRARI,* LINDA A. CANNIZZARO,‡ RENATA BATTINI,*
KAY HUEBNER,† AND RENATO BASERGA*

*Department of Pathology, Temple University Medical School, and †The Wistar Institute of Anatomy and Biology, Philadelphia; and ‡The Genetics Center, Southwest Biomedical Research Institute, Scottsdale, AZ

SUMMARY

The gene for vimentin, an intermediate-filament protein, is growth regulated. We used Southern blot analysis and in situ chromosome hybridization to determine the location of the human vimentin gene. Our results show that there is only one copy of the vimentin gene and that it is located on the short arm of chromosome 10 (10pter-10q23) close to the interleukin-2 receptor gene, which is also growth regulated. In situ hybridization studies suggest that the most likely location of the vimentin gene is 10p13. Sequence similarities and homologies of human vimentin to other genes are presented.

INTRODUCTION

Vimentin is a protein belonging to the class of intermediate filaments of the cell, a class that includes other proteins such as keratins, desmin, neurofilaments, and nuclear lamins (for a review, see Lazarides 1982). As an intermediate-filament protein, vimentin is part of the cytoskeleton, which is thought to play a major role in the regulation of cell growth. Indeed, the steady-state levels of vimentin mRNA are increased by growth factors (Ferrari et al. 1986). The amount of vimentin mRNA is also increased in peripheral blood mononuclear cells stimulated by phytohemagglutinin (Kaczmarek et al. 1985*b*). The amount of vimentin itself has been reported to be increased in rapidly proliferating cells

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Address for correspondence and reprints: R. Baserga, Department of Pathology, Temple University Medical School, Philadelphia, PA 19140.

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(Connell and Rheinwald 1983; Ben-Zeev 1984) and in cells from human non-Hodgkin lymphomas (Giorno and Sciotto 1985).

The entire coding sequences of the Syrian-hamster (Quax-Jeukin et al. 1983) and human (Ferrari et al. 1986) vimentin genes have been published. Quax et al. (1985) also reported that the human vimentin gene localizes to chromosome 10. The authors themselves, though, expressed some reservations on their findings because they had used a Syrian-hamster probe. In the present paper, we confirm, using a human vimentin probe, that (1) there is only one copy of the human vimentin gene and (2) that it does localize to chromosome 10, more specifically to chromosome region 10p13. In addition, we present a summary of the sequence similarities and homologies of human vimentin with other proteins of intermediate filaments, similarities that indicate the possibility of cross-reactions.

MATERIAL AND METHODS

Molecular Probes

The vimentin (4F1) cDNA insert in the Okayama-Berg vector (Ferrari et al. 1986) was used as probe for in situ chromosomal hybridization and Southern blot analysis of the hybrid panel. A human interleukin-2 receptor (IL2R) gene probe in pBR322 (Leonard et al. 1984) was used as a probe for the short arm of chromosome 10 (Leonard et al. 1985), and a human deoxynucleotidyltransferase (TdT) gene cDNA in pBR322 (Peterson et al. 1984) was used as a marker for the long arm of chromosome 10 (Isobe et al. 1985).

DNA probes were radiolabeled with α-^{32}P dNTPs (for Southern analysis) and α-^{3}H dNTPs (for chromosomal in situ hybridization) to a specific activity of 1×10^8 cpm/0.1 μg DNA or 4×10^7 cpm/μg DNA, respectively.

Cells

Isolation, propagation, and characterization of parental cells and somatic-cell hybrids used in the present study have been described elsewhere (Dalla Favera et al. 1982; Huebner et al. 1985, 1986*a*, 1986*b*, 1986*c*; Isobe et al. 1985; Nagarajan et al. 1985). Hybrids were characterized for expression of enzyme markers assigned to each human chromosome (Dalla Favera et al. 1982). Some hybrid clones were karyotyped by means of trypsin/Giemsa and/or G-11-banding methods (Dalla Favera et al. 1982); in addition, the presence of specific human chromosomes in many of the rodent-human hybrids has been confirmed by means of DNA hybridization using probes for genes assigned to specific human chromosomes (Dalla Favera et al. 1982; Huebner et al. 1985, 1986*a*, 1986*b*, 1986*c*; Isobe et al. 1985; Nagarajan et al. 1985).

For regional localization of the vimentin gene, five hybrid-cell lines retaining partial chromosomes 10 were used. These hybrids were characterized for presence of the IL2R gene (short arm of 10) and TdT gene (long arm of 10) and then were tested for retention of the vimentin gene to determine whether this gene segregated with IL2R or TdT.

Southern Blot Analysis

Two methods were used. In the first method, chromosomal DNA was extracted from frozen human spleen and mouse liver according to a method described by Gross-Bellard et al. (1973), with minor modifications. DNA (20 μg) was digested with the restriction enzymes *Eco*RI, *Bam*HI, *Hin*dIII, *Pst*I, and *Pvu*II under standard conditions. Digested DNA fragments were separated according to size by means of electrophoresis on 1% agarose gels. After alkali treatment, the denatured DNA was transferred to Zeta Bind membrane according to the method of Southern (1975). The *Xho*I fragment containing the human vimentin cDNA was obtained from the recombinant plasmid pL_3A_7A described elsewhere (Ferrari et al. 1986). The insert was separated by means of electrophoresis through a 4% polyacrylamide gel, recovered by electroelution, and labeled by means of the oligonucleotide-labeling method with ^{32}P dCTP to a specific activity of 2×10^9 cpm/μg according to a method described by Feinberg and Vogelstein (1983). The Zeta Bind membrane was prewashed for 1 h in 0.1 × SSC (1 × SSC = 0.15 M NaCl, 0.015 M sodium citrate, pH 7.0) and 0.5% sodium dodecyl sulfate (SDS) at 65 C. Prehybridization was carried out in 50% formamide, 5% dextran sulphate, 100 μg sonicated and denatured salmon-testis DNA/ml, 0.1% Na pyrophosphate, 50 mM Tris-Cl, pH 7.5, 10 × Denhardt's, and 5 × SSC at 42 C overnight. The hybridization was carried out in the same buffer with 10% dextran sulfate and 5×10^6 cpm denatured probe at 42 C for 36 h. After hybridization, filters were washed in 0.1 × SSC and 0.1% SDS at 65 C and exposed to Kodak XAR-5 film with Quanta III intensifying screen overnight. According to Howley et al. (1979) the stringency conditions are compatible with 10% mismatching.

In the second method, DNAs from human cell lines, mouse cell lines, and rodent-human hybrid-cell lines were extracted by means of cell lysis, proteinase K digestion, phenol extraction, and ethanol precipitation. Cellular DNAs were digested with an excess of restriction enzyme (*Hin*dIII), sized in 0.8% agarose gels, and transferred to nitrocellulose or nylon filters according to a method described by Southern (1975). Hybridization was carried out in 50% formamide, 4 × NaCl/cit (1 × NaCl/cit = 0.15 M NaCl/0.015 M sodium citrate, PH 7.0), 0.2 mg sonicated salmon-sperm DNA/ml, and 1 × Denhardt's solution (0.02% bovine serum albumin/0.02% Ficoll/0.02% polyvinylpyrrolidone) at 42 C for 15 h. Some hybridizations were performed at 68 C without formamide. After hybridization, filters were washed and exposed to Kodak XAR-5 film with intensifying screens.

Sequence Similarities and Homologies

Determination of sequence similarities and homologies was carried out using the data available in the Genebank, Palo Alto, CA.

Chromosomal In Situ Hybridization

Metaphase chromosome preparations were obtained by culturing peripheral blood lymphocytes from a normal male subject (46,XY) for 96 h in RPMI

medium supplemented with 15% fetal bovine serum. Cultures were harvested by means of standard procedure.

Probes were radiolabeled with tritium [^{3}H] to a specific activity of 4×10^7 cpm/μg DNA. In situ hybridization was performed using a modification of the standard protocol (Harper and Saunders 1981; Cannizzaro and Emannuel 1984). Slides were aged 10–14 days at 4 C and treated with ribonuclease A (Sigma) for 1 h at 37 C. Chromosomal DNA was denatured at 70 C for 2 min in a 70% formamide:2 × SSC mixture (pH 7.0). Probe DNA was denatured in a hybridization mixture of 25% formamide, 2 × SSC, and 10% dextran sulfate (pH 7.0). The final concentration of probe DNA placed on each slide was 0.035 μg DNA/ml. Hybridization was carried out at 37 C for 18 h. Slides were subsequently rinsed at 39 C, dehydrated in an alcohol series, and air-dried. Slides were then dipped in nuclear-track emulsion (Kodak NTB-2), fan-dried, and stored in light-tight boxes at 4 C. At different time intervals, slides were developed and fixed at 15 C, air-dried, and then stained with a modified Wright-Giemsa staining protocol (Cannizzaro and Emannuel 1984) in which each slide was dipped for 10–16 s in a pH 9.2 borate buffer (50 mM $NaSO_4$, 2.5 mM $Na_2B_4O_7$) at 35 C and then stained for 4–5 min in a staining dish containing 2 parts pH 9.2 buffer:1 part Wright-Giemsa stain (J. T. Baker & Co.). Grains situated on nonoverlapping chromosome regions were counted and scored.

RESULTS

The Vimentin Gene Is a Single-Copy Gene

Figure 1 shows Southern blots of human and mouse genomic DNAs hybridized to a human vimentin cDNA probe. When we applied a final wash of 0.1 × SSC, 0.1% SDS at 65 C after hybridization, the human vimentin probe hybridized to a single restriction fragment (see the pattern shown with *Eco*RI and *Hin*dIII) of human DNA, indicating that human vimentin is encoded by a single-copy gene. The detection of weakly hybridizing restriction fragments is the result of cross-hybridization of human vimentin with the human desmin and/or other intermediate-filament genes (Quax et al. 1985). (For the similarities and homologies with other intermediate filaments, see table 1.) The restriction pattern of the vimentin gene in human genomic DNA is also strikingly different from the restriction pattern of mouse genomic DNA. Having confirmed that there is only one gene copy of vimentin, we then proceeded to determine its localization on human chromosomes.

The Vimentin Gene Maps to Human Chromosome Region 10pter-10q23

To determine the chromosomal localization of the human vimentin gene, *Hin*dIII-digested DNAs from a panel of rodent-human hybrid cells retaining overlapping subsets of human chromosomes were tested for the presence of the human vimentin gene by means of hybridization of the vimentin cDNA probe, 4F1, to Southern blots containing control and hybrid DNAs. Figure 2 depicts a representative Southern blot of *Hin*dIII-digested mouse (fig. 2, lane 1), human (fig. 2, lane 2), and hybrid (fig. 2, lanes 3–16) DNAs. Presence of the 4.0-kb

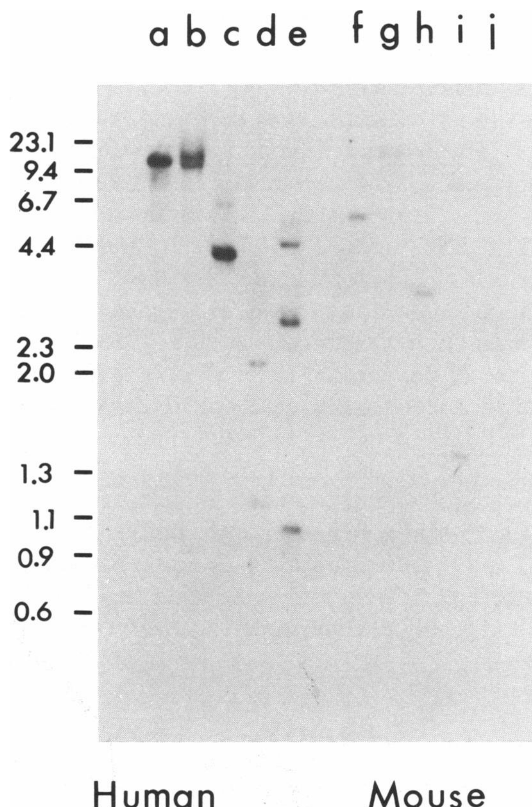

FIG. 1.—Southern blot analysis of human and mouse genomic DNA after digestion and hybridization to a probe for human vimentin. The procedures are described in Material and Methods. The vimentin probe has been described in the paper by Ferrari et al. (1986). Human-DNA lanes and restriction enzymes are as follows: a, *Eco*RI; b, *Bam*HI; c, *Hind*III; d, *Pst*I; and e, *Pvu*II. The same order of restriction enzymes obtains for the mouse-DNA lanes (f–j).

*Hind*III fragment of the human vimentin gene is observed in only one hybrid-cell line, BD3 (fig. 2, lane 15), which retains an intact human chromosome 10 (as described in the legend to fig. 2). Two other hybrid DNAs (fig. 2, lanes 5, 6), which previously had been shown to retain the human TdT gene at 10q23-10q25 (Isobe et al. 1985), did not retain the human vimentin gene, suggesting discordance between the human vimentin gene and chromosome region 10q23-10q25. Thus, additional hybrid DNAs were tested for the presence of the human vimentin gene, and all hybrids were tested for presence of both the human TdT gene and the human IL2R gene, which has been mapped to the short arm of chromosome 10 (Leonard et al. 1985). Figure 3 summarizes the results obtained using a panel of 28 rodent-human hybrids. As shown in figure 3, the pattern of retention of the human vimentin gene in the rodent-human hybrid panel is concordant with the pattern of retention of the IL2R gene in the same hybrids and is discordant with the pattern of retention of all other human chromosome regions. Therefore, we confirm the assignment of the human vimentin gene to

TABLE 1

Rank, Sequence (Length[a])	% Similarity or Homology
1, Human vimentin (1,401)	100
2, Hamster vimentin (1,398)	91.3
3, Hamster desmin (897)[b]	68.6
4, Mouse glial fibrillary acidic protein (1,212)[c] ..	61.2
5, Chicken desmin (297)[b]	57.5
6, Mouse epidermal keratin type 1 subunit 1 (1,713)	35.6
7, Human 50-kd type 1 epidermal keratin (1,419)..	35.1
8, Human epidermal keratintype II (1,689)	34.4
9, Mouse epidermal keratin subunit II (1,662) ...	34.3
10, Mouse 68-kd neurofilament protein (911)[b]	31.0
11, Human tubulin (1,356)	25.1
12, Mouse endo B cytokeratin (1,272)	22.4
13, Human lamin C (1,719)	16.2
14, Human lamin A (2,109)	16.1

[a] In base pairs.
[b] Not full-length sequence.
[c] Homologies with chicken vimentin and mouse endocytokeratin A also exist, but they been have omitted because the known sequences are too short for a stringent comparison.

chromosome 10 (Quax et al. 1983) and regionally localize the vimentin gene to chromosome region 10pter-10q23.

The Vimentin Gene Maps to the Short Arm of Chromosome 10

To confirm and refine the localization of the vimentin gene on chromosome 10, in situ hybridization of the vimentin cDNA probe to normal human metaphase chromosomes was performed.

A total of 284 metaphases were analyzed, and 906 grains were counted. The

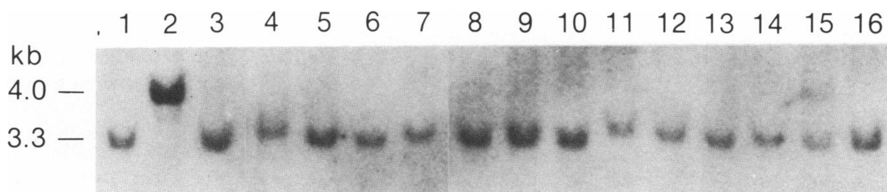

FIG. 2.—Segregation of the human vimentin gene in mouse-human hybrids. Lanes 1 and 2 represent, respectively, DNA (10 μg) from the mouse cell line and the human cell line. DNA (15 μg) from mouse-human hybrids is as follows: lane 3, M442S retaining 4p, 8q24–8qter, partial 12, and 14-pter-14q32; lane 4, c121 retaining 7; lane 5, 77-31 retaining 1, 3–9, partial 10q, 13, 14, 17, 18, 20, 22, and X; lane 6, 77-30 retaining 3–5, 9, partial 10q, 13, 14, 17, 20, 22, and X; lane 7, c12 retaining 3–5, 14, 17, 20, 22, X, and Y; lane 8, 3c retaining 4q, 6, 9, 12, 14, 17, 21, and 22; lane 9, B2 retaining 4p, 6, 12, 20, and X; lane 10, c11 retaining 4p, 6, and X; lane 11, PB-5 retaining 2p, 3p, and 17q; lane 12, GL-3 retaining 3, 4, 6, 7, partial 12, 14, 15, and 17–20; lane 13, S5b retaining 3–7, 9, 15, 17, 18, and X; lane 14, GB-31 retaining 17; lane 15, BD3 retaining 2, 3, 5–8, 10–12, 14, 16, 18–21, and X; and lane 16, AA3 retaining 4, 18, and X, cleaved with an excess of restriction endonuclease *Hind*III, fractionated on an agarose gel, transferred to a nitrocellulose filter, and hybridized to ^{32}P-labeled vimentin cDNA plasmid. Molecular weights of the mouse and human vimentin *Hind*III fragments are shown on the left of the figure.

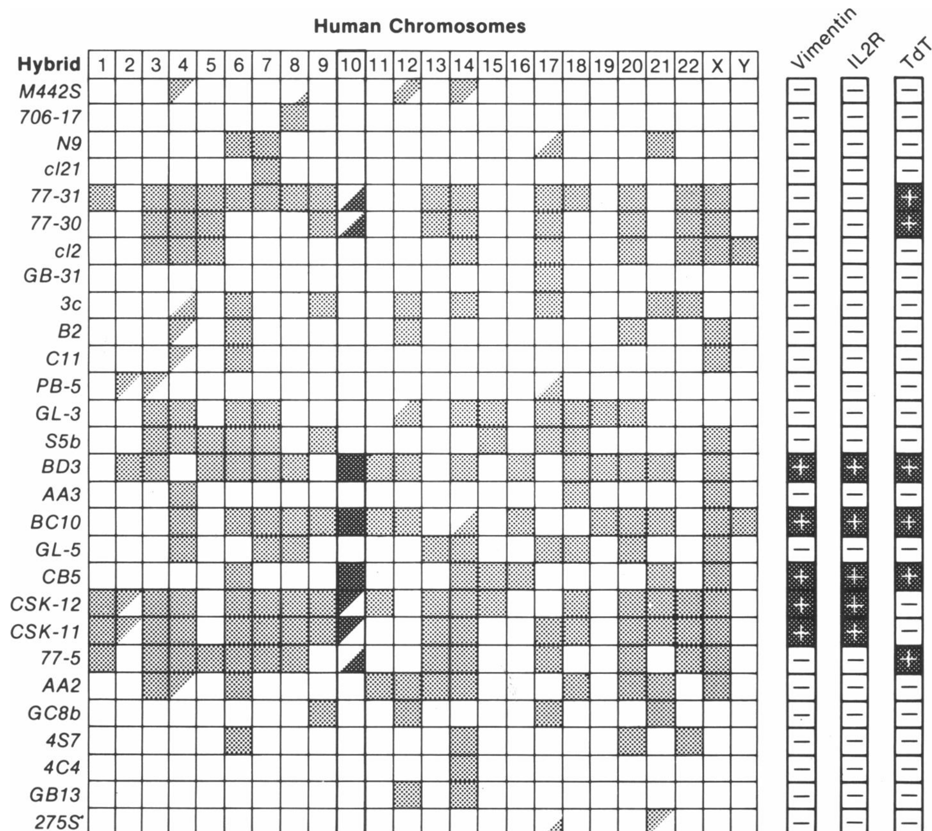

FIG. 3.—Cosegregation of the human vimentin gene with the IL2R gene on chromosome 10 in 28 rodent-human hybrids. ■ Indicates that the hybrid names in the left column contain the chromosome indicated in the upper row; ◪ indicates presence of the long arm (or, in some cases, a part of the long arm, as indicated by a smaller fraction of stippling) of the chromosome shown above the column; ◩ indicates presence of the short arm of the chromosome listed above the column; and □ indicates absence of the chromosome listed above the column. The column for chromosome 10 is boldly outlined and stippled to emphasize correlation of presence of this chromosome (or region of this chromosome) with presence of the vimentin sequence. The pattern of retention of the vimentin gene in the panel is compared with the pattern of segregation of the IL2R and TdT genes in the three columns to the right of the figure, where presence of the sequences in the hybrid is indicated by a stippled box with a plus sign and absence of the gene is indicated by an open box enclosing a minus sign.

primary site of hybridization was the short arm of chromosome 10 (10p), with 11% of the total grains hybridized to this region. Some grains were also found on the arm of chromosome 10. Grains were therefore located between the p-terminal portion of the short arm extending to the q24 region of the long arm. The number of grains hybridized to both the short and long arms of chromosome 10 consisted of 17.5% of grains counted over all chromosomes. Figure 4 illustrates the grain distribution of vimentin cDNA on all chromosomes.

Most grains on the short arm of chromosome 10 were within the region

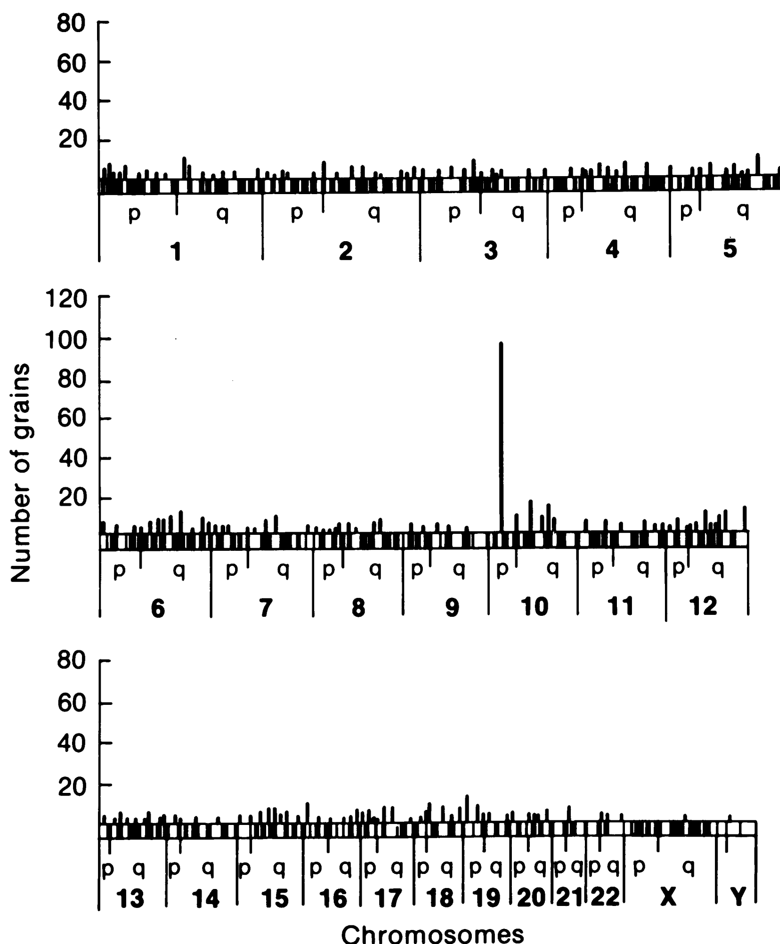

FIG. 4.—Histogram of grain distribution to metaphase chromosomes after hybridization with a human vimentin probe. Some 284 metaphases were analyzed, and 906 total grains were counted; 159 (18%) of these 906 grains were counted on 10p + 10q, and 101 (64%) of these 159 grains on chromosome 10 were on 10p only. According to results of in situ chromosome hybridization, the human vimentin gene is located on chromosome 10p, probably 10p13.

10p13, and these results suggest that this is the most likely location of the vimentin gene.

Sequence Similarities and Homologies of the Human Vimentin Gene

Sequence similarities and homologies of the human vimentin gene are summarized in table 1. The coding sequence of human vimentin is 91% homologous to the coding sequence of Syrian-hamster vimentin, the only other vimentin gene that has been fully sequenced. The other genes fall into clearly defined groups. Desmin and glial fibrillary acidic protein have substantial (57%–68%) sequence similarities to vimentin. A second group (sequence similarity ~30%)

includes keratins and a neurofilament protein, whereas nuclear lamins belong to a third group, one that shows modest (16%) sequence similarity. Interestingly, tubulin (not an intermediate filament) displays a 25% sequence similarity to human vimentin.

DISCUSSION

The human vimentin gene was previously localized to chromosome 10 by Quax et al. (1985). However, these investigators themselves expressed some reservations because they had used a Syrian-hamster probe and because the vimentin sequence shows considerable homologies to the sequences of other intermediate-filament genes, such as desmin, the keratins, and the lamins of the nuclear envelope (Geisler and Weber 1981; Quax et al. 1983; Marchuk et al. 1984; Johnson et al. 1985; McKeon et al. 1986; Singer et al. 1986; and see table 1). Our data confirm and extend the results of Quax et al. (1985): the human vimentin gene is located on chromosome 10 and, more precisely, on the short arm of chromosome 10, at 10p13.

This location is particularly interesting in view of the fact that the vimentin gene is growth regulated (Ferrari et al. 1986). The vimentin gene maps near another growth-regulated gene, the IL2R gene (Leonard et al. 1984, 1985). The steady-state levels of vimentin mRNA increase very rapidly after peripheral blood mononuclear cells are stimulated by mitogens, preceding the appearance of c-myc mRNA (Kaczmarek et al. 1985*b*). Similarly, the steady-state levels of the mRNA for the IL2R increase in phytohemagglutinin-stimulated peripheral blood mononuclear cells (Kaczmarek et al. 1985*b*). However, in purified T-lymphocytes, the induction of both vimentin and IL2R mRNAs necessitates the addition of IL-2 (Kaczmarek et al. 1985*a*). It is conceivable that the two genes are coordinately regulated in human lymphocytes.

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