small amounts of calcium and manganese (*17, 19*); it thus has a composition similar to that of the breunnerite in the Calrissian IDP (Table 1).

Carbonates in carbonaceous chondrites are believed to have crystallized during aqueous alteration of their parent bodies (*18–20*). However, Macdougall and coworkers (*22*) recently showed, on the basis of strontium isotopic measurements, that the carbonates in Orgueil were probably produced much earlier (possibly billions of years earlier) than the period of extensive aqueous activity during which sulfate veins were produced. Thus, they suggested that carbonate formation may have occurred at the time of parent body formation.

Alternatively, the carbonates in the Orgueil meteorite and the Calrissian IDP may be primary components that formed before or during accumulation of solar nebula condensates and interstellar dust to planetary bodies. Lancet and Anders (*23*) suggested that the carbonates in the carbonaceous chondrites could have formed in the solar nebula at approximately 400 K and 10^{-4} atm through catalysts like hydrated silicates and magnetite. Recent TEM studies of IDP's provide evidence that a low-temperature, catalytic synthesis was probably involved in the formation of another mineral in IDP's (*15, 24*).

Studies of interstellar dust have largely been based on theoretical and spectroscopic methods. Now infrared, isotopic, and mineralogical studies of IDP's suggest that they may contain primitive solar materials, some of which may be residues of the protostellar clouds. Detailed investigation of IDP's, therefore, offers potentially important insights into the nature and history of interstellar dust.

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A New HTLV-III/LAV Protein Encoded by a Gene Found in Cytopathic Retroviruses

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The DNA of the HTLV-III/LAV group of retroviruses contains certain additional open reading frames that are not found in typical avian or mammalian retroviruses. The role of these sequences in encoding for gene products that may be related to pathogenesis remains to be resolved. An open reading frame whose 5′ end overlaps with the *pol* gene, but is unrelated to the *env* gene, has been observed in HTLV-III/LAV and visna virus, both cytopathic mammalian retroviruses. Evidence presented here shows that this open reading frame is a bona fide coding sequence of HTLV-III/LAV and that its product, a protein with a molecular weight of 23,000, induces antibody production in the natural course of infection.

THE THIRD MEMBER OF THE RECENTly identified family of human T-lymphotropic viruses, HTLV-III/LAV [also designated ARV (*1–7*)] is now generally accepted as necessary, if not sufficient, for the development of the acquired immune deficiency syndrome (AIDS). In addition to the *gag*, *pol*, and *env* genes that are present in all replication-competent animal retroviruses, HTLV-III/LAV also contains in its genome several other open-reading frames (orf's) (*8–11*). Recently, two lines of evidence have suggested that at least two of the HTLV-III/LAV orf's are indeed functional genes. First, in the process of searching for regions that mediate the observed transactivation, a new gene, *tat*, unrelated to *gag*, *pol*, and *env*, was identified (*12, 13*). Second, an antigen with a molecular weight of 27,000 (p27) was shown to be encoded

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Table 1. Deduced protein sequence for CNBr cleaved sor product of HTLV-III/LAV, based on the nucleotide sequence of Ratner *et al.* (*8*).

Expected CNBr peptides of p23	Number of amino acids in each CNBr peptide	Deduced NH_2-terminal protein sequence of each CNBr peptide*
Peptide 1	7	NH_2-M E N R W Q V M
Peptide 2	8	I V W Q V D R M
Peptide 3	13	R I R T W K S L V K H H M
Peptide 4	160	Y V S G K A R G W F Y R H H Y E—M
Peptide 5	3	N G H—COOH

*The amino acids identified by the radiosequence analysis are underlined.

by one of the HTLV-III/LAV orf's, variably designated as 3′-*orf* (*8*), F (*9*), orf-2 (*10*), E′ (*11*), thus providing direct evidence for the presence of a fifth gene in HTLV-III/LAV (*14*).

The unusually large number of genes identified in HTLV-III/LAV is not the only feature that distinguishes this class of viruses from other replication-competent retroviruses. In most replication-competent retroviruses, the 5′ end of the *env* gene overlaps with the 3′ end of the *pol* gene. In HTLV-III/LAV, however, the *env* gene is located about 1.1 kb downstream from the *pol* gene and there is another orf whose 5′ end overlaps with the 3′ end of the *pol* gene. This orf, known as *sor* (*8*), orf Q (*9*), orf-1 (*10*), and P′ (*11*), encodes an unusually large number of tryptophan residues. These features of HTLV-III/LAV are also found in visna virus, a cytopathic retrovirus of sheep that is distantly related to HTLV-III/LAV (*9, 15*). The question of whether sor is a bona fide coding sequence of HTLV-III/LAV is addressed in the present study. We describe here the identification of a new HTLV-III/LAV antigen with an approximate molecular weight of 23,000 (p23). Amino acid sequence analysis of p23 has allowed us to map its coding origin to *sor*.

Figure 1 shows the detection of p23 in an HTLV-III/LAV producer cell line, Molt-3/HTLV-III (*16*), by the techniques of radioimmunoprecipitation and sodium dodecyl sulfate–polyacrylamide gel electrophoresis (RIP/SDS-PAGE) (*17, 18*). Three of the five representative sera from patients with AIDS or AIDS-related complex (ARC) and from asymptomatic homosexual males reacted to p23 (Fig. 1). Serologically, p23 appears to be unrelated to *env* encoded gp160/120 (*19–22*), *gag* encoded p55, p24 (*19–21*), 3′-*orf* encoded p27 (*14*) and a 25K antigen that shares a common epitope with p24 (*23*). There appears to be no correlation between the detection of p23 and other antigens (Fig. 1A). The virus specificity of p23 is further demonstrated by the absence of p23 in the HTLV-III uninfected parental cell line, Molt-3 (Fig. 1B), and by the inability of 20 HTLV-III/LAV seronegative laboratory workers to recognize p23.

To determine whether the coding origin of p23 is *sor*, we analyzed the NH_2-terminal protein sequence by radiosequence analysis (*17, 18, 24*). Radiolabeled p23 was isolated from samples of the Molt-3/HTLV-III cell line metabolically labeled with [^{3}H]valine by RIP/SDS-PAGE. Automated Edman degradation analysis of radiolabeled p23 eluted from the gel did not yield any radioactive peak in the first 20 degradation cycles. This could result from the absence of valine in the NH_2-terminal region of p23, in which case

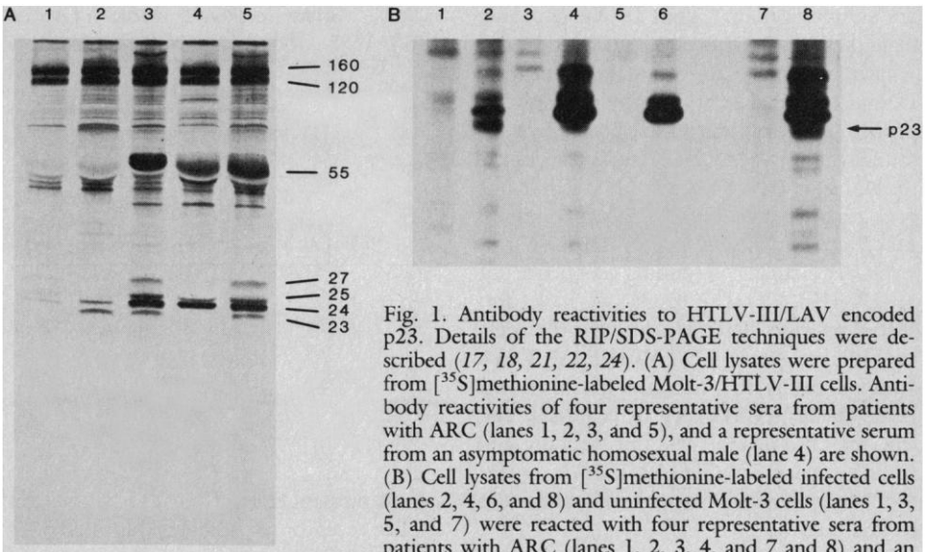
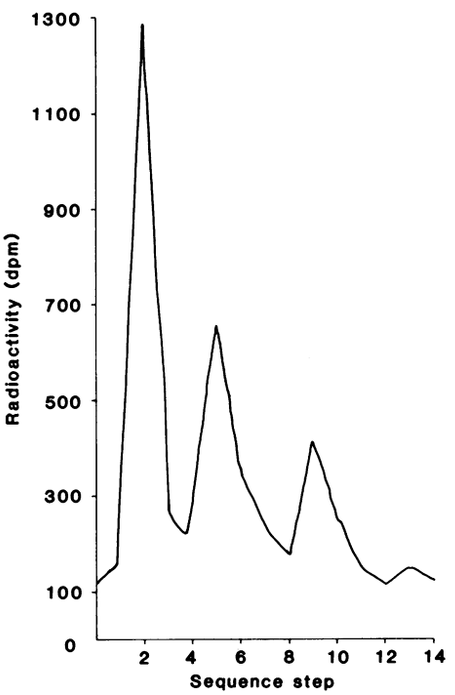


Fig. 1. Antibody reactivities to HTLV-III/LAV encoded p23. Details of the RIP/SDS-PAGE techniques were described (*17, 18, 21, 22, 24*). (A) Cell lysates were prepared from [^{35}S]methionine-labeled Molt-3/HTLV-III cells. Antibody reactivities of four representative sera from patients with ARC (lanes 1, 2, 3, and 5), and a representative serum from an asymptomatic homosexual male (lane 4) are shown. (B) Cell lysates from [^{35}S]methionine-labeled infected cells (lanes 2, 4, 6, and 8) and uninfected Molt-3 cells (lanes 1, 3, 5, and 7) were reacted with four representative sera from patients with ARC (lanes 1, 2, 3, 4, and 7 and 8) and an asymptomatic homosexual male (lanes 5 and 6). Sera in lanes 1 and 2 were the same as that in lane 2 of (A). Sera in lanes 3 and 4 were the same as that in lane 3 of (A). Sera in lanes 5 and 6 were the same as that in lane 4 of (A). Sera in lanes 7 and 8 were the same as that in lane 5 of (A).

p23 would be unlikely to be a *sor* product, or from the NH_2-terminal residue of p23 being inaccessible to Edman degradation. To distinguish between these possibilities, we treated the [^{3}H]valine-labeled p23 with cyanogen bromide (CNBr), which is known to cleave at the COOH side of methionine residues, and subjected the unsorted CNBr fragments of p23 to automated Edman degradation. Valine residues were found in degradation cycles 2, 5, and 9 (Fig. 2). Valines at position 2 and 5 match with the deduced protein sequence of peptide 2 and valine at position 9 matches with peptide 3 (Table 1). The disproportionately high yield at position 2 could have resulted from the presence of peptide 4 in the unfractionated CNBr fragments. This result suggested the likelihood that p23 is encoded by *sor* and the NH_2-terminal residue of p23 is inaccessible to Edman degradation.



To verify that the observed match with the unfractionated CNBr fragments was not due to chance alone, we separated the CNBr fragments of [^{3}H]valine- and [^{35}S]cysteine-labeled p23 by Sephacryl S-200 chromatography and then subjected the fragments to automated Edman degradation (Fig. 3). Comparison between NH_2-terminal protein sequences of CNBr fragments recovered in the three different pooled fractions with the deduced protein sequences presented in Table 1 revealed that pool I contained peptide 4, pool II contained peptide 3, and pool III contained peptide 2. Pool II appeared to contain small amounts of peptide 1 and peptide 2 also, as indicated by the presence of valine residues at positions 2, 5, and 6. The same analyses carried out with [^{3}H]arginine- or [^{3}H]tryptophan-labeled p23 gave similar results (Fig. 4). All arginine or tryptophan residues identified in pools IV to X

Fig. 2. Radiosequence analysis of unfractionated CNBr peptides of p23. Radiolabeled p23 was isolated from 50×10^6 Molt-3/HTLV-III cells metabolically labeled with 10 mCi of [^{3}H]valine (New England Nuclear, NEN), specific activity 55 mCi/mmol). The serum used for immunoprecipitation is the same as the one shown in lane 5 of Fig. 1A. Procedures for preparation of radiolabeled peptides and CNBr cleavage were the same as described previously (*17, 18*). Automated Edman degradation of radiolabeled p23 was performed as described (*24*).

are summarized in Table 1. We conclude from these results that p23 is encoded by *sor*.

Because there are no complementary DNA clones that hybridize to the *sor* region probe (*11*), and because of the lack of immunoreactivity to *sor*-related peptides expressed in the prokaryotes (*25*), it has not been clear whether *sor*, which occurs only in the cytopathic viruses HTLV-III/LAV and visna, is actually a coding sequence of HTLV-III/LAV. The results we have presented provide direct evidence for the presence of a *sor* gene in HTLV-III/LAV. These findings bring the number of functional genes of HTLV-III/LAV to six (*gag, pol, env, tat, 3'-orf*, and *sor*), thus making the coding capacity of HTLV-III/LAV by far the largest among replication-competent retroviruses.

The inaccessibility of p23 to Edman degradation suggests that some kind of NH_2-terminal modification has occurred. It is not clear if this modification is present on the native p23 or is the result of our isolation procedure. The latter possibility appears to be less likely because identical methods were used to isolate *env* gene encoded antigens of HTLV-I (*18*), HTLV-II (*26*), and HTLV-III/LAV (*22*).

It has been speculated that, if *sor* is a coding sequence, its product can be translated either from a transcript that can make a *gag-pol-sor* read-through product or from a spliced messenger RNA (mRNA) that uses a splice acceptor (or acceptors) located 5' of the *sor* coding sequence and does not contain *gag* and most of the *pol* coding sequences (*11*). Because no *gag-pol-sor* read-through product has been detected, it is likely that *sor* is translated from a spliced mRNA. A recently identified mRNA of approximately 5.5 kb, which was detected by a synthetic oligonucleotide probe complementary to a *sor* coding region, could in theory be one of the candidate transcripts from which p23 is translated (*27*).

The recognition of p23 by HTLV-III/LAV seropositive patients demonstrates that, like *env* encoded gp160, gp120, and gp41 (*19–22, 28*), 3'-*orf* encoded p27 (*14*), and *gag* encoded p17 and p24 (*19–21*), *sor* encoded p23 is also involved in antibody induction during the natural course of infection. Detection of circulating antibody to *sor* encoded p23 does not necessarily correlate with the detection of circulating antibody to *gag* encoded p24 and 3'-*orf* encoded p27 (Fig. 1 and Table 2). On the basis of experiments with more than 60 human sera, five serological profiles can be identified when antibody responses to *env* encoded gp160 and gp120, *gag* encoded p24, 3'-*orf* encoded p27, and *sor* encoded p23 are considered. It is possible that some of these profiles are correlated with clinical manifestations of disease or with virus replication status and may prove useful in differential diagnosis and prognosis.

The function of p23 is not known. Because *sor* transcripts were not detected in an HTLV-III producer line, the *sor* product was recently speculated to be a repressor that would only be expressed in cells that are in a "lysogenic" state (*29*). This hypothesis is unlikely to be true, in view of the detection of *sor* encoded p23 in the virus producer line Molt-3/HTLV-III and the detection of candidate *sor* transcripts in another HTLV-III producer line, H9/HTLV-III (*27*). However, it is interesting to note that *sor* encoded p23 is unusually rich in tryptophan (*8–11*). Since tryptophan residues are rare in most

Table 2. Various serological profiles of serum samples from people with circulating antibody to gp160/gp120 as detected by radioimmunoprecipitation assay.

Serological profiles	Presence (+) or absence (−) of antibody to			
	gp160/120 (*env*)	p24 (*gag*)	p23 (*sor*)	p27 (3'-*orf*)
1	+	−	−	−
2	+	+	−	−
3	+	+	−	+
4	+	+	+	±*
5	+	+	+	+

*One example of this profile is shown in lane 2 of Fig. 1A, where p27 is marginally detectable.

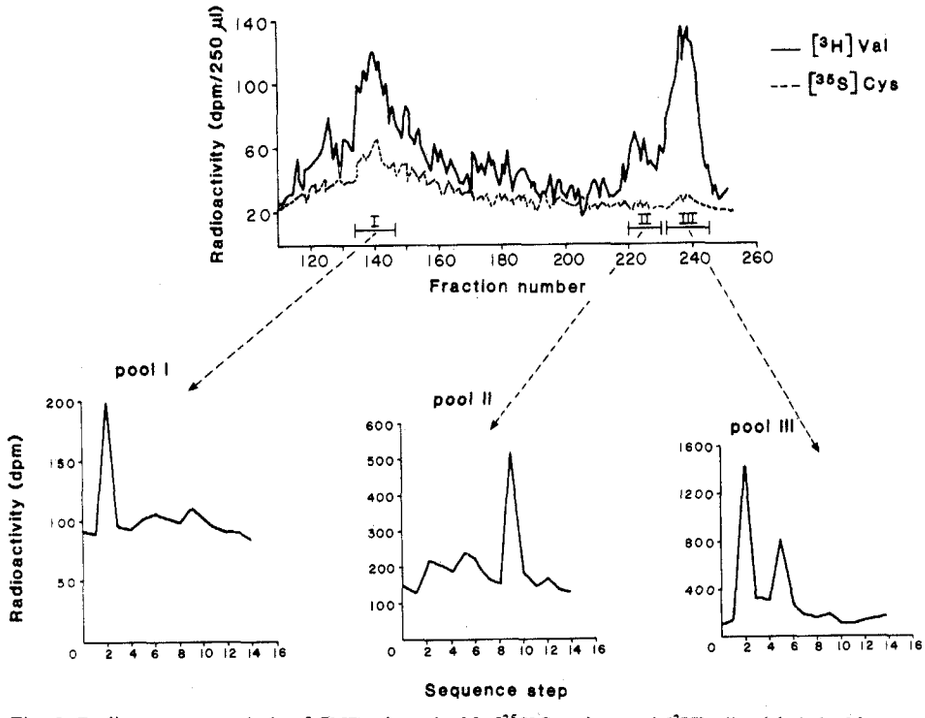


Fig. 3. Radiosequence analysis of CNBr cleaved p23. [^{35}S]Cysteine- and [^{3}H]valine-labeled p23 was isolated by RIP/SDS-PAGE. After CNBr cleavage (*17*), peptides were separated in a 2 by 195 cm column that had been equilibrated in 6*M* guanidine HCl plus 0.5% acetic acid. Fractions of 2.0 ml were collected and pooled as indicated. Each pool was then subjected to automated Edman degradation.

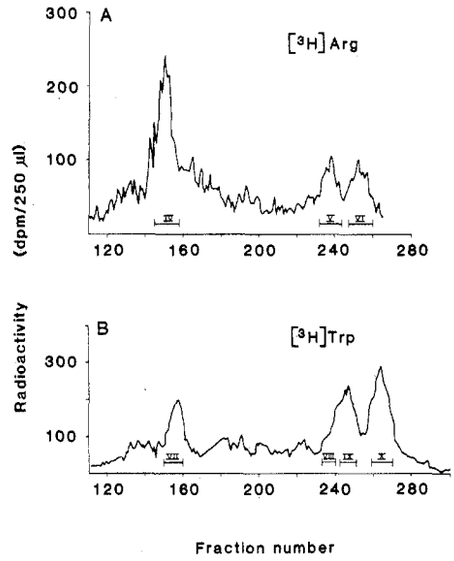


Fig. 4. Sephacryl S-200 chromatography of CNBr peptides of (A) [^{3}H]arginine-labeled p23 or (B) [^{3}H]tryptophan-labeled p23. The amounts of isotope used were 10 mCi of [^{3}H]arginine (NEN, specific activity 30 mCi/mmol) and 10 mCi of [^{3}H]tryptophan (NEN, specific activity 20 mCi/mmol), respectively. Separation procedures were the same as those described in Fig. 3. Results of protein sequence analysis of pooled fractions indicated are summarized in Table 1.

proteins, this may imply that the function of p23 is subject to structural constraints exerted by the tryptophan residues. The observation that an analogous open reading frame of visna, designated orf Q (*29*), is also unusually rich in tryptophan residues, raises the possibility that some functional features common to these two cytopathic viruses are mediated by this region of the virus.

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Replicative and Cytopathic Potential of HTLV-III/LAV with *sor* Gene Deletions

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The genome of the human T-lymphotropic virus type III (HTLV-III/LAV) has the potential to encode at least three polypeptides in addition to those encoded by the *gag*, *pol*, and *env* genes. In this study, the product of the *sor* (short open reading frame) region, which overlaps the 3′ end of the *pol* gene, was found to be a protein with a molecular weight of 23,000. An assay was developed for testing the ability of cloned HTLV-III proviruses to produce viruses cytopathic for T4$^+$ lymphocytes. In the cell line used, C8166, neither the HTLV-III *sor* gene product nor the complete 3′-*orf* gene product were necessary for the replication or cytopathic effects of the HTLV-III.

THE HUMAN T-LYMPHOTROPIC VIRUS type III (HTLV-III/LAV) is the primary etiological agent of the acquired immune deficiency syndrome (AIDS) and associated disorders (*1*). Infection with the virus can result in a depletion of the T4$^+$ subset of lymphocytes in patients, which results in the immune suppression characteristic of AIDS (*2*). HTLV-III can exert cytopathic effects on cultured peripheral blood lymphocytes as well as on established lymphocyte lines, mimicking the effects on the host cell in vivo (*1, 3*).

The complete nucleotide sequence of several independent isolates of the virus has been determined (*4*). In addition to the genes that encode structural components of the virus (the *gag* and *env* genes) and a gene that encodes functions required for replication (the *pol* gene) (*5*), the genome of all strains of this virus has the potential to encode at least three additional polypeptides. The tat_{III} gene, which includes two coding exons, one located just 5′ to *env* and a second located within an alternative reading frame of *env*, encodes the 14K transactivator protein (molecular weight 14,000) (*6, 7*). The product of tat_{III} induces high levels of expression of genes under the control of the HTLV-III long terminal repeat (LTR) via interaction with specific target sequences (*8*). Expression of tat_{III} in infected cells greatly stimulates the rate of virus protein expression and thereby contributes to efficient replication of the virus (*9*). A 27K protein of unknown function is encoded by the 3′-*orf* reading frame, located between the *env* gene and the 3′ LTR (*10*).

A third potential coding region in the HTLV-III genome, designated *sor* for short open reading frame, overlaps the 3′ end of the *pol* gene (*4*). Studies of virus-specific messenger RNA (mRNA) species present in infected cells revealed two spliced polyadenylated RNA species, of 5.5 and 5.0 kilobases

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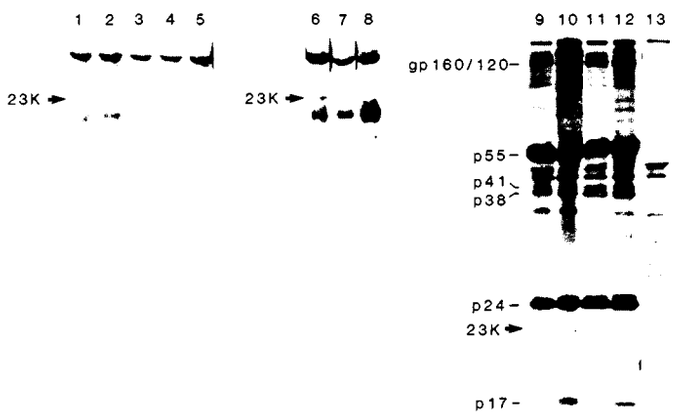

Fig. 1. Immunoprecipitation of extracts from HTLV-III–infected cells with antiserum to oligopeptide S. Cell lines were labeled overnight with [^{35}S]methionine at 100 μCi per milliliter and prepared for immunoprecipitation with rabbit antiserum to oligopeptide S (lanes 1 to 8) or sera from HTLV-III–infected individuals (lanes 9 to 13) as described (*17*). Lanes 1 and 2: uninfected H9 cells and H9 cells infected with HTLV-III (H9/IIIB), respectively, precipitated with the antiserum to oligopeptide S. Lanes 3, 4, and 5: immunocompetition of H9/IIIB cell lysates for the antiserum to oligopeptide S with increasing amounts (5, 10, and 20 μg, respectively) of oligopeptide S present in the reaction. Lanes 6, 10, and 12: Jurkat-tat_{III} cells transfected with pHXBc2. Lanes 7, 9, and 11: Jurkat-tat_{III} cells transfected with the pDsor1. Lanes 8 and 13: untransfected Jurkat-tat_{III} cells. Cells were labeled 5 to 6 days after transfection by the DEAE-dextran technique (*18*) with 15 μg of plasmid DNA. Antiserum to oligopeptide S was used to precipitate cell extracts in lanes 6 to 8, patient antiserum T.W. in lanes 9 and 10, and patient antiserum 4-3 in lanes 11 to 13.