The vibration was also observed with normal-length axonemes with sperm heads attached; in this case, microbeads were not needed for the measurement, because the bright-head image could be directly used to analyse the movement. Gibbons and Gibbons reported[14] that demembranated sperm becomes quiescent in a reactivation solution with an elevated concentration of calcium ions. They suggested that this quiescent state represents an active state in which many dynein crossbridges remain potentiated to produce sliding force. We found that the heads of such quiescent sperm also vibrated at a high frequency: 290 ± 17 Hz at an ATP concentration of 1 mM (average of nine measurements). This raises the possibility that the axonemes are vibrating at a high frequency while dynein arms are generating sliding force.

We were unable to observe the high-frequency vibration in axonemes from which a bundle of outer-doublet microtubules had been extruded after perfusion with elastase and ATP[15,16]. This indicates that either an intact axonemal structure or the presence of inter-doublet links is necessary for the vibration, as it has been suggested that elastase (a protease) preferentially disrupts the inter-doublet links[15]. The intact axonemal structure or inter-doublet links may be required to provide antagonistic forces needed for the vibration[17]; such antagonistic forces may be maintained only in cylindrically closed systems, because dynein arms exert unidirectional force on adjacent microtubules[18].

The axonemal ATP hydrolysis rate under the same conditions as used above was $\sim 2 \times 10^7$ ATP molecules per mg axoneme per min, which, if we assume that the outer and inner arms are identical, corresponds to an average turnover rate of ~20 ATP molecules per s for a single dynein arm (ATP concentration of 1 mM). This result is in agreement with previous data[1,19]. However, only some of the dynein arms might have undergone ATPase activation in the above experiments. In fact, dynein arms crosslinked to microtubules have recently been reported to have an ATP turnover rate as high as 150 molecules per s (T. Shimizu, S. P. Marchese-Ragona and K. A. Johnson, personal communication). Hence, the maximal rate of ATP hydrolysis by a single dynein can be close to, or even identical with, the vibration frequency of ~300 Hz. Thus the high-frequency vibration may be tightly coupled with the ATPase cycle. The observation that orthovanadate reduced the vibration amplitude but not its frequency also supports the idea that the vibration frequency reflects the turnover of individual activated dynein, because orthovanadate is known to reduce the number of active dynein arms by bringing them to a kinetic dead end[20]. We cannot, however, exclude the possibilities that the vibration is caused by cooperative movements of many arms, or by multiple steps in a crossbridge cycle.

We have demonstrated here the feasibility of measuring a sub-nanometre-scale movement (see also ref. 21) and the presence of a high-frequency vibration in a dynein–microtubule system. Vibration at such a high frequency has not been observed in other eukaryotic motility systems except that of insect flight muscle[22]. Because nanometre-scale molecular movement may occur in various cellular mechanisms, it will be interesting to apply our method of study to other biological systems. □

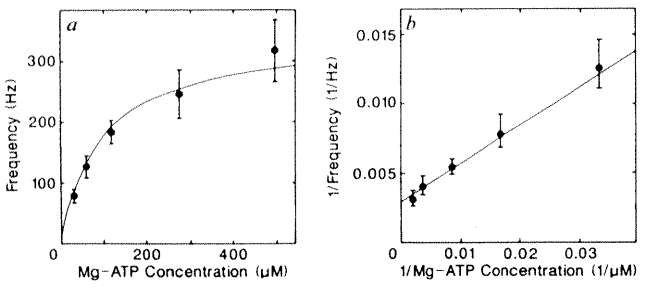

FIG. 4 Dependence of the high-frequency vibration on MgATP concentration. *a*, Mean frequencies and standard deviations (vertical bars) obtained from data with different axonemes. Temperature, 21 ± 1 °C. For each data point, 11–79 samples were measured. *b*, Double reciprocal plot of the data in *a*. This plot yields an apparent K_m of 94 μM MgATP and a maximal frequency of 340 Hz.

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Homology of 54K protein of signal-recognition particle, docking protein and two *E. coli* proteins with putative GTP-binding domains

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MOST proteins exported from mammalian cells contain a signal sequence which mediates targeting to and insertion into the membrane of the endoplasmic reticulum (ER)[1,2]. Involved in this process are the signal-recognition particle (SRP) and docking protein (DP), the receptor for SRP in the ER membrane[1]. SRP interacts with the signal sequence on nascent polypeptide chains and retards their further elongation[1], which resumes only after interaction of the arrested ribosomal complex with the docking protein[3,4]. SRP is a ribonucleoprotein particle comprising a 7S RNA and six polypeptides with relative molecular masses (M_r) of 9,000 (9K) 14K, 19K, 54K, 68K and 72K (ref. 1). The 9K and 14K proteins are essential for elongation arrest and the 68K–72K heterodimer is required for docking to the ER membrane[5]. The 54K protein binds to the signal sequence when it emerges from the ribosome[6,7]. Docking protein consists of two polypeptides, a 72K α-subunit

(DPα) and a 30K β-subunit (DPβ)[8]. No components structurally homologous to SRP and docking protein have yet been found in yeast or *Escherichia coli*. To understand the molecular nature of the interaction between the signal sequence and its receptor(s) we have characterized a complementary DNA coding for the 54K protein of SRP. Significant sequence homology was found to part of DPα and two *E. coli* proteins of unknown function. The homologous region includes a putative GTP-binding domain.

An expression cDNA library[9] from cytoplasmic poly(A)$^+$ RNA of MDCK (Madin Darby canine kidney) cells was screened with a monoclonal antibody against the 54K subunit of SRP (SRP54)[10]. One positive clone was identified. The cDNA insert in this clone was 1.4 kilobases (kb) in length. Using northern blot analysis on messenger RNA of MDCK cells, we estimated the size of the full-length mRNA for SRP54 to be ~2.5 kb (Fig. 1*a*). After rescreening the library, a 2.4 kb cDNA clone was selected and analysed by *in vitro* transcription and translation (Fig. 1*b*, *c*). The translation product is a 54K protein which co-migrates with SRP54 isolated from canine pancreas and can be immunoprecipitated with an anti-SRP54 antibody (Fig. 1*b*).

The SRP54 cDNA was sequenced. It is 2,321 base pairs (bp) in length and has a single large open reading frame encoding 504 amino acids (Fig. 2); the calculated M_r of the encoded protein is 55.7K. Amino-acid sequences obtained from the N terminus and from tryptic peptides of SRP54 match the deduced amino-acid sequence (Fig. 2). The ATG initiation codon is preceded by 376 nucleotides which contain two ATG codons and two in-frame stop codons (Fig. 2, boxed). The first ATG is directly followed by a stop codon; the second ATG is followed by an open reading frame of 16 codons. Translation in eukaryotes usually starts at the first ATG[11]. It has been shown, however, that ATG/stop codon combinations preceding the initiation site can reduce translational efficiency[11]. We therefore deleted most of the 5′ flanking region, including the first two ATG codons from pSRP54 (Fig. 1*c*). Messenger RNA derived from the resulting plasmid (pSRP54-1, Fig. 1*c*) is translated more efficiently than that from pSRP54 (data not shown).

SRP can be reconstituted from isolated 7S RNA and SRP proteins[12,13]. We tested binding of the cDNA encoded 54K protein to an SRP19/7S RNA complex formed *in vitro*. Association of SRP54 with the complex was observed only in the presence of both 7S RNA and the 19K subunit of SRP (SRP19) (Fig. 3). Thus the components synthesized *in vitro* can interact with each other specifically and form an SRP subparticle.

The SRP54 amino-acid sequence was compared with the Swissprot, NBRF, EMBL and Brookhaven data libraries using the FASTA program of Pearson and Lipman[14]. We found a striking similarity to three proteins: the DPα; a 48K *E. coli* protein with unknown function[15]; and the *E. coli* FTS Y protein which is encoded by a gene of a cell division operon[16]. Regions of homology are outlined in Fig. 4*a* and an alignment of the sequences is shown in Fig. 4*b*. Identical amino acids are shaded in grey. The region between amino acids 100 and 300 of SRP54 is most conserved among all four proteins (Fig. 4). Comparison of the consensus sequence derived from the alignment with the data libraries revealed a weak similarity of the domain corresponding to amino acids 166–227 of SRP54 with amino acids 312–376 of the 70K heat-shock proteins (HSP70). HSP70 proteins have an affinity for hydrophobic surfaces in unfolded proteins[17], which might resemble the interaction of SRP54 with the signal sequence.

Over its entire length SRP54 is very similar to a 48K *E. coli* protein[15]. This protein has been identified as an open reading frame upstream of the *trm*D operon at 56 min on the *E. coli* chromosome[15]. The N-terminal 60% of SRP54 is homologous to the C-terminal half of DPα and of the FTS Y protein. Proteolytic cleavage experiments suggested that the N-terminal 151 amino acids of DPα are required for association with the ER membrane and that mixed-charge amino-acid clusters surrounding the elastase cleavage site (see arrow Fig. 4*a*) are supposedly involved in interaction with SRP[18]. The FTS Y protein is encoded by a gene in the filamentation temperature sensitive (*fts*) operon at 76 min on the *E. coli* genetic map. Mutations in this operon affect essential cellular functions including heat-shock response and cell division[16]. The function of the FTS Y protein is not known.

Components involved in protein secretion in *E. coli* have been characterized using either genetic or biochemical techniques[2]. Several groups have described partial purification of cytoplasmic factors and oligomeric protein complexes which stimulate protein translocation across membranes in *E. coli* and in yeast systems *in vitro*. None of these proteins seems to be identical with either the 48 K or the FTS Y protein.

The region of strongest similarity between the four proteins (amino acids 100–300 of SRP54) contains three highly conserved sequence elements indicative of guanine-nucleotide-binding sites (Fig. 5). By X-ray crystallography of the elongation factor EF-Tu (ref. 19) and the Ha-*ras* product p21 (ref. 20) it has been shown that these sites are in contact with the GTP and correspond to loops at the ends of adjacent parallel β-strands[19,20].

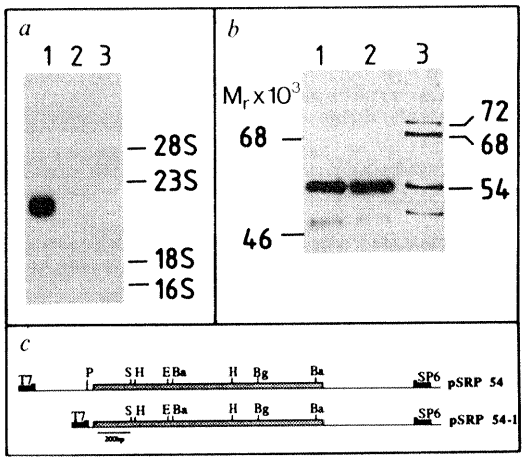


FIG. 1 *a*, Northern blot analysis of poly(A)$^+$ RNA from MDCK cells (lane 1). Control, total RNA from canine pancreas (lane 2) and *E. coli* (lane 3), using pSRP54TEx cDNA as probe. *b*, Translation products of mRNA obtained by *in vitro* transcription from pSRP54. Lane 1, total translation; lane 2, immunoprecipitation with the monoclonal anti-SRP54 antibody[10]; lane 3, 72K, 68K and 54K SRP proteins. *c*, Schematic representation of SRP54 specific cDNA clones. T7, SP6, promoter of bacteriophage T7 and SP6 respectively; restriction-enzyme cleavage sites: P, *Pst*I; S, *Sph*I; H, *Hin*dIII; E, *Eco*RI; Ba, *Bam*HI; Bg, *Bgl*II. The black boxes indicate multiple restriction sites and the shaded areas SRP54 coding regions.

METHODS. A cDNA library was prepared from cytoplasmic poly(A)$^+$ RNA, isolated from MDCK cells essentially as described by Haymerle *et al.*[9] in the bacterial expression vector pTEx (Herz *et al.*, in preparation). 2×10^5 colonies were screened using a monoclonal antibody against SRP54[10]. One positive clone was found (pSRP54TEx). Cytoplasmic poly(A)$^+$ RNA of MDCK cells and total cytoplasmic RNA of canine pancreas and *E. coli* were separated on a 1% agarose-formaldehyde gel[13] and transferred to gene screen plus membrane (NEN). A fragment comprising the whole pSRP54TEx cDNA (1.4kb) insert was labelled by random priming and hybridized according to ref. 26. Filters were washed at 65 °C in 1 mM EDTA, 40 mM $NaHPO_4$ (pH 7.2), 1% SDS. Ribosomal RNA from dog pancreas and *E. coli* were used to calculate the approximate size of SRP54 mRNA. The MDCK cDNA library was rescreened for full-length clones with pSRP54TEx. Twenty-nine additional positive clones were found and their insert sizes tested. For *in vitro* expression, one of the longest cDNA clones was subcloned into the *Sma*I site of pGem-2 (Promega) (*c*, pSRP54). Plasmids were then transcribed with either T7 or SP6 polymerase[27] and the transcripts translated in the wheat germ cell-free system. Protein products were characterized by SDS-PAGE and fluorography (*b*). SRP proteins (*b*, lane 3) were visualized by silver staining[28]. To generate pSRP54-1, the 5′ proximal region of psRP54 was removed with *Pst*I (see Fig. 3).

The presence of the GTP consensus elements in the region of strongest similarity would suggest that all four proteins are GTP-binding proteins. This has recently been shown for the α-subunit of the docking protein for which GTP was found to be required for the docking-protein-dependent release of SRP from the ribosomal complex[21]. Known GTP-binding proteins constitute a family of proteins that use GTP hydrolysis to drive the sequential binding of biological molecules to a series of reaction partners[22]. GTP binding and hydrolysis are thought to produce two different protein conformations with different affinities for an effector molecule. GTP-binding proteins include elongation and initiation factors, and also proteins which mediate the vectorial transport of secretory vesicles. In the latter process GTP is thought to mediate vesicle targeting and vesicle fusion[22].

SRP and docking-protein mediate signal-sequence targeting

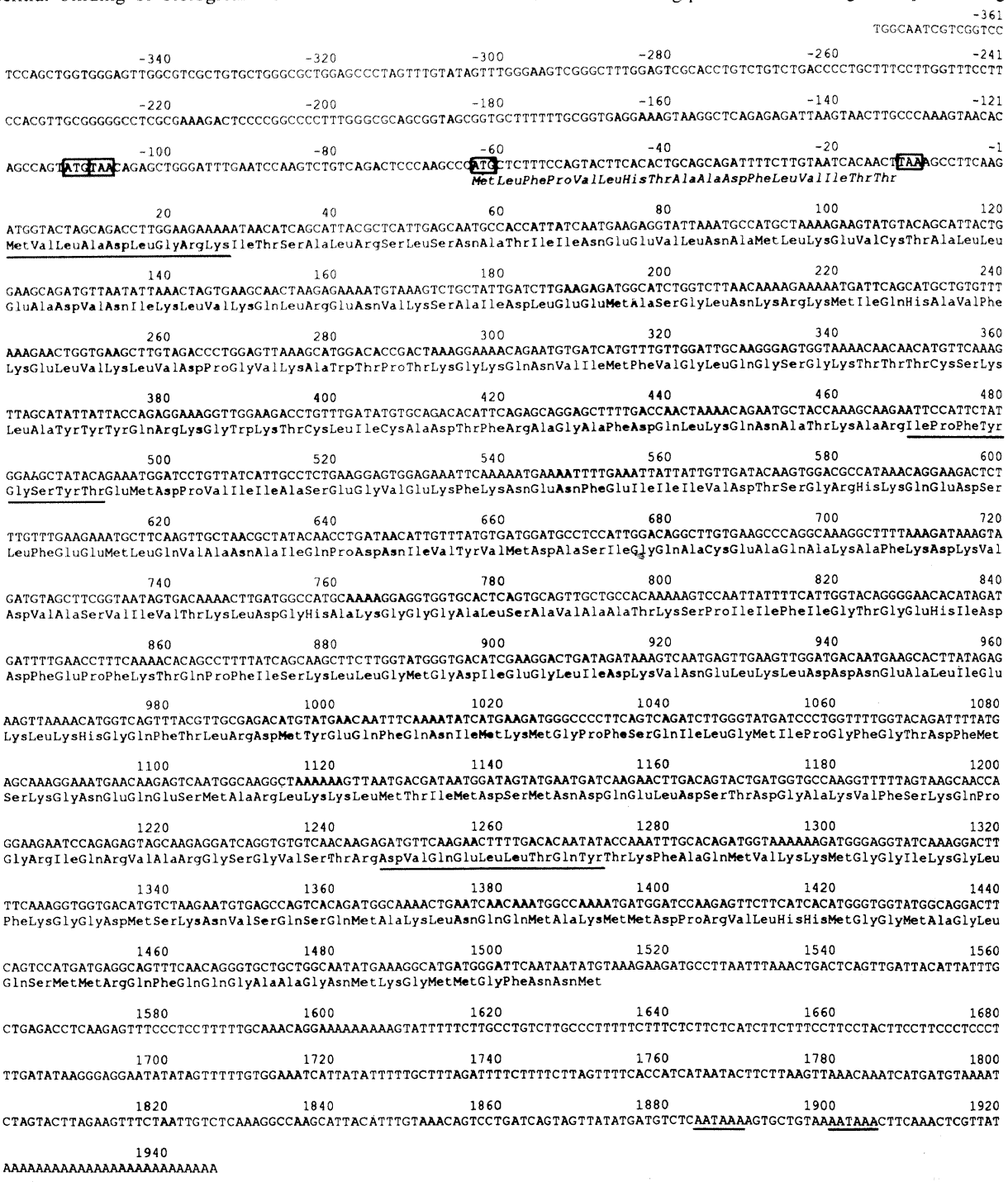


FIG. 2 Sequence of the SRP54 cDNA and the deduced amino-acid sequence of SRP54. The initiating methionine was identified by comparison with the protein sequence derived from the N-terminus of SRP54. Sequences obtained by sequencing tryptic peptides of SRP54 were found to be identical with the amino-acid sequence deduced from pSRP54. These sequences are underlined. The positions of two ATG and in-frame stop codons in the 5'untranslated region of pSRP54 are indicated by boxes. Polyadenylation signals (AATAAA) in the 3'untranslated region are underlined.

METHODS. Restriction fragments of pSRP54 (digested at restriction sites indicated in Fig. 1*c*) were subcloned into M13 vectors[29]. Subclones were sequenced by the dideoxy-chain-termination method using Sequenase (USB) and gaps or ambiguities were resolved by using internal oligodeoxynucleotide primers. The N-terminal 8 amino acids of SRP54 were determined by the method of Matsudaira[30] after separation of SRP proteins by SDS-PAGE and blotting onto polyvinylidene-difluoride membranes. Internal amino-acid sequences were obtained after tryptic digestion of SRP54 protein (blotted onto nitrocellulose)[31] followed by selection of peptides by HPLC.

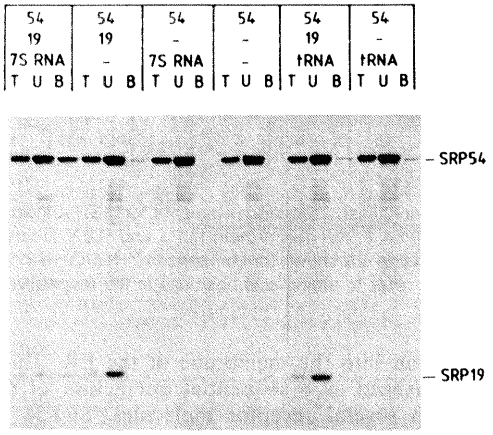
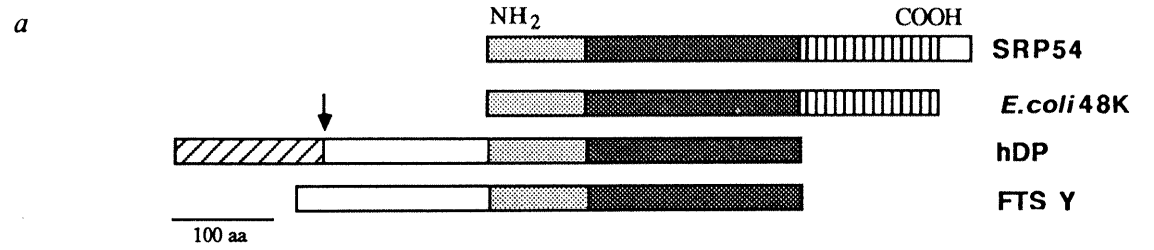


FIG. 3 Binding of *in vitro* synthesized SRP54 to SRP19–7S RNA. The binding assays contained SRP 7S RNA (7S RNA), transfer RNA (tRNA), SRP54 (54) and SRP19 (19) in the combinations indicated on top of the figure. Total translations (T), complexes bound to DEAE Sepharose (B) and unbound (U) material were characterized by SDS PAGE and fluorography.

METHODS. Formation of an RNP particle was tested by a modification of a previously published method[13] *In vitro* transcripts from pSRP19[13] and from pSRP54-1 (Fig. 1*c*) were translated for 30 min at 25 °C in a wheat germ cell free system. EDTA was added to 5 mM final concentration, to release nascent chains from ribosomes and tRNA, and incubation was continued for 15 min. The translation reactions were then adjusted to 5mM magnesium acetate and 500 mM potassium acetate. SRP54 translation reaction (5 μl) SRP19 translation reaction and (10 μl) and 7S RNA or tRNA (1 μg) were mixed in appropriate combinations and incubated at 25 °C for 30 min. The volume was adjusted to 200 μl with 500 mM potassium acetate, 5 mM magnesium acetate, 50 mM Tris, pH 7.4, wash buffer. One-fifth of the reaction volume was removed and trichloroacetic acid (TCA) precipitated to monitor for total protein synthesis (T). DEAE-Sepharose CL-6B (Pharmacia) (30 μl) equilibrated in wash buffer was added to the remainder and incubated at 4 °C for 15 min. After centrifugation the unbound supernatant fraction (U) was TCA precipitated. The DEAE-Sepharose beads were washed twice with wash buffer and DEAE-bound material (B) released at 4 °C with 200 μl the same buffer adjusted to 2 M KCl. Eluted protein was TCA precipitated and all samples analysed on 10–15% Laemmli-type SDS-polyacrylamide gels[32] and subjected to flourography.



b

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  1  MVLADLGRKITSALRSL-SNATII--NEEVLNAMLKEVCTALLEADVNIKLVKQLRENVKSAIDLEEMA  SRP54
  1   MFDNLTDRLSRTLRNI-SGRGRL--TEDNVKDTLREVRMALLEADVALPVVREFINRVKEKAVGHEVN  E.coli48
312  ATKGTLGGMF-GMLKGL-VGSKSL--SREDMESVLDKMRDHLIAKNVAADIAVQLCESVANKLEGKVMG  hDP
194  GFFARLKRSLLKTKENLGSGFISLFRGKKIDDDLFEELEEQLLIADVGVETTRKIITNLTEGASRKQLR  FTSY

 67  SGLNKRKMIQHAVFKELVKLVDPGVKAWTPT--------KGKQNVIMFVGLQGSGKTTTCSKLAYYYQRK  SRP54
 66  KSLTPGQEFVKIVRNELVAAMGEENQTLNLA--------AQPPAVVLMAGLQGAGKTTSVGKLGKFLREK  E.coli48
377  TFSTVTSTVKQALQESLVQILQPQRRVDMLRDIMDAQRRQRPYVVTFCGVNGVGKSTNLAKISFWLLEN  hDP
263  DAE--------ALYGLLKEEMGEILAKVDEPLNVE---GKAPFVILMVGVNGVGKTTTIGKLARQFEQQ  FTSY

129  GWKTCLI-CADTFRAGAFDQLKQNATKAR-------------IPFYGSYTEMDPVIIASEGVEKFKNEN  SRP54
128  HKKKVLVVSADVYRPAAIKQLETLAEQVG-------------VDFFPSDVGQKPVDIVNAALKEAKLKF  E.coli48
446  GF-SVLIAACDTFRAGAVEQLRTHTRRLSALHPPEKHGGRTMVQLFEKGYGKDAAGIAMEAIAFARNQG  hDP
321  G-KSVMLAAGDTFRAAAVEQLQV-------------WGQRNNIPVIAQHTGADSASVIFDAIQAAKARN  FTSY

184  FEIIIVDTSGRHKQEDSLFEEMLQVA------NAIQPDNIVYVMDASIGQACEAQAKAFKDKVD-----  SRP54
184  YDVLLVDTAGRLHVDEAMMDEIKQVH------ASINPVETLFVVDAMTGQDAANTAKAFNEALP-----  E.coli48
514  EDVVLVDTAGRMQDNAPLMTALAKLI------TVNTPDLVLFVGEALVGNEAVDQLVKFNRALADHSMA  hDP
376  IDVLIADTAGRLQNKSHLMEELKKIVRVMKKLDVEAPHEVMLTIDASTGQNAVSQAKLFHEAVG-----  FTSY

242  -----VASVIVTKLDG-HAKGGGALSAVAATKSPIIFIGTGEHIDDFEPFKTQPFISKLLGMGDIEGLI  SRP54
242  -----LTGVVLTKVDG-DARGGAALSIRHITGKPIKFLGVGEKTEALEPFHPDRIASRILGMGDVLSLI  E.coli48
577  QTPRLIDGIVLTKFDTIDDKVGAAISMTYITSKPIVFVGTGQTYCDLRSLNAKAVVAALMKA          hDP
440  -----LTGITLTKLDG-TAKGGVIFSVADQFGIPIRYIGVGERIEDLRPFKADDFIEALFARED        FTSY

305  DKVNELKLD--DNEALIEKLKHGQ-FTLRDMYEQFQNIMKMGPFSQILGMIPGFGTDFMSKGNEQESMA  SRP54
305  EDI-ESKVDRAQAEKLASKLKKGDGFDLNDFLEQLRQMKNMGGMASLMGKLPGMG-QIPDNVKSQMDDK  E.coli48

371  RLKKLMTIMDSMNDQELDSTDGAKVFSKQPGRIQRVARGSGVSTRDVQELLTQYTKFAQMVKKMGGIKG  SRP54
372  VLVRMEAIINSMTMKERAKPEIIK-----GSRKRRIAAGCGMQVQDVNRLLKQFDDMQRMMKKMK----  E.coli48

440  LFKGGDMSKNVSQSQMAKLNQQMAKMMDP----RVLHHMGGMAGLQSMMRQFQQGAAGNMKGMMGFNNM  SRP54
432  --KGG----------MAKMMRSMKGMMPPGFPGR                                     E.coli48
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FIG. 4 Alignment of protein sequences homologous between SRP54, an *E. coli* 48K protein, DPα and the *E. coli* FTS Y protein using the multiple sequence alignment algorithm of Vingron and Argos[33]. The dark grey areas in *a* show the region of highest similarity between the four proteins. The light shaded area indicates a segment with weaker but still significant similarity. A region homologous between SRP54 and *E. coli* 48K only is indicated by vertical hatching. Diagnonal hatching at the N-terminus of hDPα indicates the domain with which this protein interacts with the DPβ subunit in the ER membrane[8,23]. The arrow shows the elastase cleavage site[18]. Sequences aligned for homology are shown in *b*[15,16,34]. Identical amino acids are indicated by shading.

Consensus		G	x	x	x	G	K	T/S			D	x	x	G			N	K	x	D
SRP54	108	G	L	Q	G	S	G	K	T	190	D	T	S	G	248	T	K	L	D	
***E.coli* 48K**	107	G	L	Q	G	A	G	K	T	190	D	T	A	G	248	T	K	V	D	
hDP	425	G	V	N	G	V	G	K	S	520	D	T	A	G	588	T	K	F	D	
FTS Y	301	G	V	N	G	V	G	K	T	382	D	T	A	G	446	T	K	L	D	
N-RAS, human	10	G	A	G	G	V	G	K	S	57	D	T	A	G	116	N	K	C	D	
EF-Tu, *E.coli*	18	G	H	V	D	H	G	K	T	80	D	C	P	G	135	N	K	C	D	

FIG. 5 The three consensus sequence elements for a GTP-binding domain[35] are present in SRP54, *E. coli* 48K protein, hDPα and FTS Y. N-Ras and EF-Tu GTP- binding domains are shown for comparison[35]. Numbers in front of the sequence motifs refer to amino-acid positions in the respective protein.

to and insertion into the membrane of the ER. This process could be envisaged as a sequential interaction of the signal sequence with several receptor molecules. SRP54 has been shown to interact with the signal sequence. The site of interaction has not yet been detemined. Binding sites of ligand (effector) molecules in Ras proteins and in EF-Tu are thought to comprise a region between the first and the second GTP consensus site[19,20]. The corresponding site in SRP54 might interact with the signal sequence. It is possible that the signal sequence, upon interaction of SRP with the docking protein, is translocated from the GTP-binding domain of SRP54 to the homologous domain of DPα or, with lower efficiency, is directly inserted into the ER membrane after release from SRP[23].

Prokaryotic and eukaryotic signal sequences are very similar[24]. The structural similarity of the signals would suggest that their receptors share structural features as well. This hypothesis is supported by data from experiments which show the functional interaction of bacterial proteins with the eukaryotic secretion machinery[25]. The striking similarity between the SRP54 and the *E. coli* 48K protein over nearly their entire length might point to a related function. DPα and the *E. coli* FTS Y protein lack the C-terminal domain that is homologous between SRP54 and the *E. coli* 48K protein. They both have non-homologous extensions at the N terminus. The presence of the *fts y* gene in a locus affecting cell division might point to a membrane related function of the FTS Y protein. Protein export from *E. coli* is thought to occur near zones of adhesion, the future sites of cell division[2].

The discovery of highly conserved putative GTP-binding domains in SRP54, DPα and two *E. coli* proteins is a further indication of the central role of GTP-binding proteins in intracellular targeting and sorting mechanisms[22]. □

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Model for signal sequence recognition from amino-acid sequence of 54K subunit of signal recognition particle

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PROTEIN targeting to the endoplasmic reticulum in mammalian cells is catalysed by signal recognition particle (SRP)[1,2]. Cross-linking experiments have shown that the subunit of relative molecular mass 54,000 (M_r 54K; SRP54) interacts directly with signal sequences as they emerge from the ribosome[3,4]. Here we present the sequence of a complementary DNA clone of SRP54 which predicts a protein that contains a putative GTP-binding domain and an unusually methionine-rich domain. The properties of this latter domain suggest that it contains the signal sequence binding site. A previously uncharacterized *Escherichia coli* protein has strong homology to both domains. Closely homologous GTP-binding domains are also found in the α-subunit of the SRP receptor (SRα, docking protein) in the endoplasmic reticulum membrane[5-8] and in a second *E. coli* protein, ftsY, which resembles SRα. Recent work has shown that SRα is a GTP-binding protein and that GTP is required for the release of SRP from the signal sequence and the ribosome on targeting to the endoplasmic reticulum membrane[9]. We propose that SRP54 and SRα use GTP in sequential steps of the targeting reaction and that essential features of such a pathway are conserved from bacteria to mammals.

We isolated a cDNA clone encoding SRP54 to understand better the mechanism of signal sequence recognition. SRP was purified to homogeneity from canine pancreas[1] and its protein components separated by preparative SDS-PAGE. SRP54 electroeluted from the gel was sequenced at the N-terminus by Edman degradation. In addition, tryptic fragments were sequenced. From the N-terminal amino-acid sequence, we generated a DNA probe using the polymerase chain reaction[10] (PCR) (Fig. 1A). We used this probe to isolate overlapping cDNA clones from a λ-phage gt10 library (Fig. 1*b*). Figure 1*c* shows the cDNA sequence and deduced primary amino-acid sequence of SRP54. Our available protein sequence information was distributed over the entire predicted coding sequence, thus making the identification of the cDNA unambiguous. The N-terminal sequence data match the predicted amino-acid sequence starting immediately after the first AUG found in the cDNA.

Sequence analysis revealed striking homologies of SRP54 to

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