Atp11p and Atp12p were first described as proteins required for assembly of the F1 component of the mitochondrial ATP synthase in *Saccharomyces cerevisiae* (Ackerman, S. H., and Tzagoloff, A. (1990) Proc. Natl. Acad. Sci. U. S. A. 87, 4986–4990). Here we report the isolation of the cDNAs and the characterization of the human genes for Atp11p and Atp12p and show that the human proteins function like their yeast counterparts. 

Human *ATP11* spans 24 kilobase pairs in 9 exons and maps to 1p32.3-p33, while *ATP12* contains ≈8 exons and localizes to 17p11.2. Both genes are broadly conserved in eukaryotes and are expressed in a wide range of tissues, which suggests that Atp11p and Atp12p are essential housekeeping proteins of human cells. The information reported herein will be useful in the evaluation of patients with ascertained deficiencies in the ATP synthase, in which the underlying biochemical defect is unknown and may reside in a protein that influences the assembly of the enzyme.

The energy needs of aerobic organisms are met principally through the process of oxidative phosphorylation in which ATP production by the ATP synthase is coupled to a transmembrane proton gradient (1). The ATP synthase is comprised of an integral membrane component called F0 and a peripheral moiety called F1. Different organisms show variation with respect to the subunit composition of the F0 sector, while the αβγδε oligomeric structure of the catalytic F1 unit has been highly conserved (1). All five F1 subunits and most F0 subunits of the mammalian ATP synthase are nuclear gene products; only two F0 proteins (subunits 6 and 8) are coded for in the mitochondrial DNA (2). As obligate aerobes, mammals are completely depleting the enzyme completely, are those that affect proteins involved in its biogenesis. Genetic screens of respiratory-defective mutants of *Saccharomyces cerevisiae* have identified several nuclear genes (*ATP10, ATP11, ATP12*) whose products are required for ATP synthase assembly (5, 6). Notably, none of these “assembly factors” are structural components of the yeast ATP synthase, which is now reported to contain at least 17 different types of subunits (7, 8). Instead, Atp10p is an assembly factor that is required for proper formation of the F0 component (5), and Atp11p and Atp12p are assembly factors for the F1 oligomer (6). Work in our laboratory has provided evidence that Atp11p interacts specifically with the F1 β subunit (9), while Atp12p binds selectively to the F1 α subunit (10). It is through this binding mechanism that Atp11p and Atp12p are proposed to protect the F1 subunits from forming nonproductive (α)n and (β)n complexes during assembly of the enzyme oligomer (10).

To facilitate the analysis of F1 assembly in human cells, efforts were made to determine whether functional human orthologs of the yeast Atp11p and Atp12p assembly factors exist. To this end, we have isolated a partial human cDNA for Atp11p and have shown that the encoded product interacts with the F1 β subunit. A full-length human cDNA for Atp12p was also isolated, and we report here that human Atp12p interacts with the F1 α subunit in a two-hybrid screen and complements a yeast atp12 mutant. Genomic analysis provides evidence that supports the chromosomal assignment of human *ATP11* to 1p32.3-p33 and of human *ATP12* to 17p11.2.

**EXPERIMENTAL PROCEDURES**

Cloning Human cDNAs for Atp11p and for Atp12p—Reverse transcription of cDNA from human fetal liver mRNA (CLONTECH, Palo Alto, CA) and reactions to rapidly extend the cDNA end (RACE) at the 5′ terminus followed procedures from Life Technologies, Inc. manual number 18374-058. The primers used for first-strand cDNA synthesis to clone *ATP11* were designed to be complementary to nucleotides 3′ to the coding region, using a contig sequence assembled from 70 expressed sequence tags (ESTs) belonging to the UniGene Cluster Hs.10964. The cDNA was dC-tailed at the 5′ end, which enabled Life Technologies’ Abridged Anchor Primer to be annealed at this terminus for 5′ RACE reactions; 3′ primers for such reactions employed sequences complementary to portions of one of the longer ATP11 ESTs (GenBank™ accession number AA150795). To clone the cDNA for human ATP12, a dC-tailed first-strand cDNA was synthesized from human fetal liver mRNA using a poly(dT) primer. This product was then amplified using the 5′ RACE Abridged Anchor Primer (Life Technologies, Inc.) and a 3′ primer that is complementary to nucleotides +943 to +959 in human DNA.

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§The abbreviations used are: RACE, rapid amplification of cDNA ends; PCR, polymerase chain reaction; EST, expressed sequence tag; SAGE, serial analysis of gene expression; contig, group of overlapping clones; RH, radiation hybrid; X-gal, 5-bromo-4-chloro-3-indolyl β-D-galactopyranoside; BD, binding domain; AD, activation domain.
mRNA 24418\(^\text{5}\) (GenBank\(^\text{TM}\) accession number AF052185, see text for details). This fragment served as the template for a second PCR with an upstream primer (5′-CCAGAATTCGCTCTCGCATC-3′) corresponding to nucleotides –41 to –22 of mRNA 24418, except for two nucleotides (underlined) altered to create an EcoRI site, and a downstream primer (5′-TGCGCTGACGCTGCTGTGAT-3′) complementary to nucleotides +885 to +905 of mRNA 24418 except for two nucleotides (underlined) altered to create an XhoI site. This product was ligated to the EcoRI and XhoI sites of pCUP2A/CEN316 (11) to create pCUPHUATP12/CEN316.

**Plasmids**—The plasmids used in this work are named according to the codons that are included in the constructs, with the initiator ATG taken as codon 1. Codon numbering for each of the sequences is based on the following data base entries: human ATP11, clone HRC06325\(^\text{3}\) (GenBank\(^\text{TM}\) accession number AK026004); human ATP12, mRNA 24418 (GenBank\(^\text{TM}\) accession number AF052185), human F1, \(\beta\) subunit (GenBank\(^\text{TM}\) accession number NM_001686 \(\text{13}\)). All inserts were made by PCR using forward and reverse primers based on the sequence upstream primer (5′-GAAGATCTGTGCTAAGCTG-3′) and amplified a human mRNA 24418 except for two nucleotides (underlined) altered to create a HindIII site of pCUP2A/CEN316 to create pCUPHUATP11/H11032 (16). The yeast two-hybrid screen (21) employed yeast vectors pACT2 and pAS2-1 and host strain Y190 (described above) as supplied in the MATCHMAKER Two-Hybrid System 2 from CLONTECH. Yeast were grown in SD medium omitted for uracil and histidine. Qualitative assessment of expression from the lacZ reporter gene was made using 5-bromo-4-chloro-3-indolyl-\(\beta\)-galactopyranoside (X-gal) as a chromogenic substrate for \(\beta\)-galactosidase in a colony-lift filter assay, as described in the CLONTECH manual. Standard techniques were used for restriction endonuclease analysis of DNA, Southern blotting and hybridization of DNA fragments, and transformations of and recovery of plasmid DNA from *Escherichia coli* (22). Yeast transformations employed the LiAc procedure (23). The preparation of yeast mitochondria and assay of oligomycin-sensitive ATPase activity were done as described previously (24). Protein concentrations were estimated by the method of Lowry et al. (25).

**RESULTS**

Cloning of a Human cDNA for ATP11p—At the time this work was initiated, a TBLASTN search of the human EST database identified the 444-base pair sequence AA150795, which upon translation in the 5′ – 3′ direction in frame 1 yields a putative peptide that shares significant sequence homology with yeast ATP11p (BLAST score: \(p = 3 \times 10^{-5}\)). AA150795 is a member of UniGene Cluster Hs.10964. A contig for human ATP11 assembled with ESTs from this cluster includes a termination codon, but is incomplete at the 5′ terminus. We isolated a human ATP11 cDNA clone bearing more sequence at the 5′ end than any of the 5′-read ESTs from RACE reactions that employed human fetal liver mRNA as the template (see “Experimental Procedures”). However, despite numerous attempts, the best product achieved was found to harbor 270 amino acids, P6. In comparison, the human protein shows 46% similarity and 21% identity with Atp11p of *S. cerevisiae* and 22.3% identity with Atp12p of *H. sapiens*. In physico-chemically similar amino acids) and 21% identity. In comparison, the human protein shows 46% similarity and 22.3% identity with Atp11p of *Schizosaccharomyces pombe* and 58.6% similarity and 31% identity with Atp11p of *Drosophila yakuba* (11). The black bar in Fig. 1 highlights a stretch of nine amino acids, PFXXXLPR (termed the “flipper sequence”), which can be considered a signature sequence for ATP proteins (see Ref. 11). The arrow indicates the first amino acid (Lys-83) that is encoded by the partial human cDNA for ATP11p isolated in our laboratory. The primary translation product of yeast ATP11 includes a leader sequence at the amino terminus that targets the protein to mitochondria (16); the first amino acid of the mature protein is predicted to be Glu-40 (27) (boxed

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residue in S. cerevisiae Atp11p sequence, Fig. 1). As mitochondrial leader sequences are known to be rich in basic and hydroxylated residues and deficient in acidic amino acids (28), we predict that the amino terminus of the mature human Atp11p protein begins in the vicinity of Glu-57 (boxed residue in H. sapiens Atp11p sequence, Fig. 1). By this analysis our cDNA clone encodes a human Atp11p protein that lacks ~25 amino acids from the mature amino terminus.

**Functional Studies with Truncated Human Atp11p Proteins**—Previous work has indicated that yeast Atp11p retains function following the deletion of either 72 amino acids from the amino terminus or 135 amino acids from the carboxyl terminus of the mature protein (24). Hence, it was deemed worthwhile to pursue functional studies with incomplete cDNAs coding for partial human Atp11p products. In one set of studies, the yeast two-hybrid screen was used to test the putative human Atp11p assembly factor for binding interactions with the human F1 subunit, as yeast ATP11p (9). Plasmids coding for the mature portions of the human F1 sequence for mature human Atp11p (Lys-83 through Glu-300, plasmid pAS2-1/HuATP11(83–300)) and of the human F 1–553) and of the human F1 subunit did not stain blue with X-gal. By this analysis our cDNA clone encodes a human Atp11p protein that lacks ~25 amino acids from the mature amino terminus.

**Cloning of a Human cDNA for Atp12p and Alignment of Its Product with Other Homologous Proteins**—**The TBLASTN program** identified the human mRNA 24418 (GenBank™ accession number AF502185) of UniGene cluster Hs.13434 as the estimated start site of the mature protein. The arrow indicates a highly conserved and functionally relevant acidic amino acid (see text for details).

**Discussion**

Cloning of a Human cDNA for Atp12p and Alignment of Its Product with Other Homologous Proteins—The TBLASTN program identified the human mRNA 24418 (GenBank™ accession number AF502185) of UniGene cluster Hs.13434 as the estimated start site of the mature protein. The arrow indicates a highly conserved and functionally relevant acidic amino acid (see text for details).
number AL121764, clone SPAC9, \(^7\) \(p = 3 \times 10^{-32}\). A multiple alignment of these four sequences, along with Atp12p from \(S.\) \(\text{cerevisiae}\) and \(H.\) \(\text{sapiens}\), is given in Fig. 2. Overall, the extent of similarity (12.7%) and identity (4.6%) between all six Atp12p sequences is low. However, pairwise analysis yields similarity values higher than 40% and identities of at least 20% in most cases (Table I). The mitochondrial leader peptide of \(S.\) \(\text{cerevisiae}\) Atp12p is estimated to span the first 30 amino acids such that mature protein starts with Gly-31 (14) (boxed residue in \(S.\) \(\text{cerevisiae}\) Atp12p sequence, Fig. 2). The other four eukaryotic Atp12p proteins are likewise predicted to harbor a mitochondrial targeting sequence, while the protein of the photosynthetic bacterium should not and is accordingly much shorter at the amino terminus. The fact that Glu-289 of \(S.\) \(\text{cerevisiae}\) Atp12p aligns with an acidic amino acid in the other five sequences (see arrow, Fig. 2) is of particular significance, since work in yeast has shown that an acidic side chain is required in this position for maximal Atp12p activity (14).

Functional Studies with Human Atp12p—The product encoded by the putative human cDNA for Atp12p was evaluated for function in yeast two-hybrid and genetic complementation assays. The two-hybrid screen employed plasmid pAS2-1/HUATP12(27–289), which codes for the Gal4p DNA binding domain fused to what is approximated to be the mature portion of the human Atp12p protein (Met-27 through Glu-289), and pACT2 plasmids bearing fusions between the Gal4p transcription activation domain and the sequences for the human F1, \(\alpha\) and \(\beta\) subunits (see above). Yeast cells, which co-produced the Gal4p fusion proteins that paired human Atp12p with the human \(\alpha\) subunit stained dark blue with X-gal, while there was only a weak blue signal for cells in which Gal4pBD-human Atp12p was produced in combination with the Gal4pAD-human \(\beta\) subunit. Such results are in accord with the fact that yeast Atp12p shows high selectivity for the yeast \(\alpha\) subunit over the yeast \(\beta\) subunit in a two-hybrid screen (10).

The cDNA cloned for human Atp12p is anticipated to encode a precursor protein that is complete with respect to mitochondrial targeting function. Hence, for complementation studies, the entire cDNA was subcloned into a multicopy yeast vector 3' of the yeast \(\text{CUP}1\) promoter (plasmid pCUPHUATP12/YEp), and this plasmid was used to transform the respiratory-deficient \(\alpha\)W303\(\Delta\)ATP12 strain aW303\(\Delta\)ATP12. Such transformants failed to grow on ethanol-glycerol plates. On the premise that the human targeting peptide might not function correctly in yeast, a plasmid was constructed to produce a protein chimera in which the yeast Atp11p leader is fused to an approximated form of the mature human Atp12p protein (Ile-33 through Glu-289, plasmid pG13L/HUATP12(33–289)). The yeast \(\alpha\)tp12 mutant harboring this plasmid grows moderately well on ethanol-glycerol plates (Fig. 3). The level of oligomycin-sensitive mitochondrial ATPase activity measured for this transformant is 2.49 units/mg, which can be compared with 0.12 unit/mg measured for the aW303\(\Delta\)ATP12 mutant and 4.71 units/mg measured for the respiratory competent parent, W303-1A. In contrast, pG13L/HUATP12(33–289) does not confer respiratory competence to the \(\alpha\)tp11 mutant, aW303\(\Delta\)G13.

Genomic Characterization of Human ATP11 and ATP12—The ESTs belonging to the \(\text{ATP11}\) UniGene cluster \(\text{Hu}.\)\(10964\) (176 homologous sequences) and those of the \(\text{ATP12}\) UniGene cluster \(\text{Hu}.\)\(13434\) (76 homologous sequences) were derived from a wide distribution of tissues, including brain, breast, colon, heart, kidney, lung, muscle, stomach, and whole embryo. SAGE data also indicate a wide tissue distribution for these genes, although a light tag frequency within most tissues (less than 62 tags/million tags surveyed for each gene) suggests low to moderate abundance of each transcript. Several other mammalian ESTs show strong homology to \(\text{ATP11}\) (14 mouse, 3 rat, and 1 Xenopus) and 1 chicken), which suggests that Atp11p and Atp12p orthologs are present also in these species.

The \(\text{ATP11}\) cDNA sequence is collectively contained within three draft human genomic sequences (AL357392, AL136373, and AC013595). Alignment of the cDNA and genomic sequences yields nine regions of homology, which suggests a genomic structure of 9 exons. The precise exon/intron boundaries of all 9 putative exons, each of which contains consensus \(\text{gt/ag}\) splice donor/acceptor sequences, and the length of all but one intron, could be inferred from the alignment (Table II). Atp11p is predicted to span a minimum of 32.5 kilobase pairs of

| H. sapiens | D. melanogaster | R. capsulatus | S. pombe | S. cerevisiae | C. elegans |
|-----------|----------------|--------------|---------|--------------|-----------|
| 56.1      | 52.1           | 44.6         | 46.5    |              |           |
| 49.0      | 47.7           | 33.3         |         |              |           |
| 48.2      | 44.8           | 43.4         |         |              |           |
| 43.2      | 44.8           | 19.2         |         |              |           |
Human Atp11p and Atp12p Proteins

**DISCUSSION**

Proteins involved in the assembly pathway for the ATP synthase are prime suspects for harboring genetic lesions responsible for deficiencies in this enzyme. This article presents the first report of assembly proteins that have been identified for the human ATP synthase. Atp11p and Atp12p were recognized originally as proteins required for correct assembly of the F1 component of the ATP synthase in S. cerevisiae (6). Several lines of evidence indicate that we have assigned correctly the human mRNAs HRC06325 (GenBank™ accession number AK026681) and 24418 (GenBank™ accession number AF052185) as ATP11 and ATP12, respectively. First, the results of amino acid sequence alignments are excellent in both cases, with regions of homology that extend throughout the protein sequences (Figs. 1 and 2 and Table I). Of added note are specific sequence elements, such as the PXFXXXXLPR consensus sequence in human Atp11p and a highly conserved acidic residue near the carboxyl terminus of human Atp12p, which strengthen the interpretation of the alignment data.

Second, results from yeast two-hybrid screens support the claim that the human cDNAs for ATP11 and ATP12 isolated in our laboratory encode assembly factors for human F1, that are fundamentally similar to their yeast counterparts. The yeast Atp11p and Atp12p proteins are known to be substrate-specific; Atp11p binds to the F1 β subunit (9), and Atp12p binds to the F1 α subunit (10). Comparable observations were made in two-hybrid assays with the human proteins in that human Atp11p showed specificity for binding the human F1 β subunit, and human Atp12p was selective for binding the human F1 α subunit. Finally, the human ATP12 cDNA rescues the respiratory defect of a yeast atp12 mutant (Fig. 3), which is compelling evidence for equivalence of function.

A curious observation made during the genetic complementation experiments was that the atp12 strain is not rescued with the full-length cDNA for human Atp12p, but rather with a genetic hybrid coding for a chimera bearing the mitochondrial targeting sequence of the yeast ATP11 protein fused to the mature portion of the human Atp12p protein. The limitations that prevent the substitution of the complete human ATP12 gene for the yeast gene in our experiments are not known. Nor can we comment with certainty about the fact that the respiratory-deficient phenotype of a yeast atp11 mutant is not rescued by a plasmid that produces most of the mature human Atp11p protein fused to the yeast Atp11p targeting sequence. While it is true that the human protein encoded by our ATP11 cDNA clone is estimated to be deficient for 25–30 amino acids from the mature amino terminus, this was not considered to be of particular consequence, first because yeast Atp11p retains function following the removal of up to 72 amino acids from its mature amino terminus (24), and second because the truncated human Atp11p protein shows evidence of binding to the yeast F1 β subunit in a two-hybrid screen. On this point it is noteworthy that Atp11p of Drosophila binds the yeast F1 β subunit in a two-hybrid assay, yet the foreign protein confers only a minimum of respiratory competence to an atp11 mutant (11). Cumulatively, such results suggest there may be an aspect of Atp11p mechanism, peripheral to F1 β subunit binding, that limits the ability of non-yeast Atp11p proteins to function properly in the context of a yeast cell. For example, it is not yet known if Atp11p is regulated in some way, nor is it known if Atp11p action is dependent on some other mitochondrial protein(s).

In recent years there have been reports of ATP synthase deficiencies that are ascribed to nuclear mutations and which correlate with disease symptoms of severe lactic acidosis and...
cardiomyopathy (30, 31). The amount of ATP synthase is also reported as decreased, relative to the amounts of the respiratory complexes of the oxidation phosphorylation pathway, in samples from patients with Alzheimer’s disease (32). Of further interest is that the chromosomal regions to which human ATP11 (1p32.2-p33) and ATP12 (17p11.2) map have both been implicated in human disease. For example, a putative locus for hereditary congenital ptosis, a muscle-specific disorder that is characterized by unilateral or bilateral drooping of the upper eyelids, has been localized to 1p32-p34.1 (33). Muscle-Eye-Brain disease, which includes severe early-onset muscle weakness, congenital myopia, and mental retardation also maps to this region (1p32-p34) (34). In addition, deletion of 1p32-p33 is frequently observed in a number of malignancies, including meningioma and carcinomas of the lung, colon, and stomach (35, 36). Disease loci that map to 17p11.2 include the neuropathies Charcot-Marie-Tooth type IA and Hereditary Neuropathy with Liability to Pressure Palsies (37, 38), the chromosome microdeletion disorder of the Smith-Magenis syndrome (39), and frequent deletion breakpoints in medulloblastomas (40). The determination of whether Atp11p or Atp12p contribute to any of these disease phenotypes awaits further investigation.

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Atp11p and Atp12p Are Assembly Factors for the F₁-ATPase in Human Mitochondria
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