PET9, AAC1, and AAC3 are members of a gene family encoding adenine nucleotide translocators of the mitochondrial inner membrane. The primary role of the translocators is to pump ADP into the mitochondrion and ATP out of the organelle. However, under certain conditions, such as during exponential growth on glucose under aerobic conditions, they act in the opposite direction, importing ATP into mitochondria. Competition experiments suggest that they also import heme into mitochondria, and all three proteins bind to heme. Genetic evidence indicates a possible role of the ADP/ATP carriers in apoptosis. Pet9p physically interacts with various complexes of the mitochondrial inner membrane, including the TIM23 complex and the cytochrome c oxidase - ubiquinol-cytochrome c reductase supercomplex, and these interactions are dependent on the presence of the phospholipid cardiolipin.PET9 encodes the major isoform of the translocator. A pet9 null mutant strain, despite having wild-type copies of AAC1 and AAC3, is unable to respire, and additionally displays a \"petite-negative\" phenotype, meaning that it cannot survive the loss of the mitochondrial genome. AAC1 is expressed at a very low level compared to PET9, and can complement the pet9 mutant phenotypes only if the gene, with its native promoter, is present in multiple copies. AAC3 is expressed primarily under anaerobic conditions, and is also capable of complementing the pet9 mutant phenotypes if overexpressed. The pet9 aac1 aac3 triple null mutant is viable under standard conditions, but is inviable under anaerobic conditions.All three paralogs are similar to adenine nucleotide translocators in higher organisms. Mutations in the human ANT1 gene, encoding the heart and skeletal muscle-specific isoform, are associated with serious diseases. Dominant missense ANT1 mutations are found in patients with progressive external ophthalmoplegia. The equivalent mutations in S. cerevisiae PET9 cause reduction or loss of function, and may lead to the formation of an unregulated channel in the mitochondrial membrane. A recessive ANT1 mutation, whose yeast equivalent in PET9 causes a complete loss of function, is associated with hypertrophic cardiomyopathy.