The cytochrome bc1 complexis a highly conserved enzyme of the mitochondrial respiratory chain. In S. cerevisiae it consists of three catalytic subunits, Cobp, Rip1p, and Cyt1p, plus seven additional subunits: Cor1p, Qcr2p, Qcr6p, Qcr7p, Qcr8p, Qcr9p, and Qcr10p. The crystal structure of the complex shows that two functional units, each containing these ten subunits, associate with each other in the mitochondrial inner membrane. Assembly of a functional complex requires two proteins, Cbp3p and Cbp4p, that are not components of the complex but may associate with it during assembly. It also requires Bcs1p, an AAA-family ATPase that interacts with a precursor of the complex to mediate incorporation of the Rip1p and Qcr10p subunits. The mechanism of energy transfer by the complex, known as the protonmotive Q cycle, has been studied in detail. The net result of the Q cycle is the stepwise transfer of an electron through the complex from ubiquinol to cytochrome c, coupled with the translocation of a proton across the mitochondrial inner membrane. The function of the cytochrome bc1 complex is essential to the energy-generating process of oxidative phosphorylation, which is carried out by the enzyme complexes of the mitochondrial respiratory chain.Qcr7p, known as the ubiquinone-binding subunit, is essential for assembly and activity of the cytochrome bc1 complex. Qcr7p, Cor1p, and Qcr2p comprise a large domain of the complex that extends into the mitochondrial matrix. Qcr7p may be involved in electron transport through the region of the complex known as center o, and genetic evidence also suggests a role in proton uptake by the complex. Unlike most mitochondrially imported proteins, Qcr7p does not have an N-terminal cleavable presequence that directs import; however, mutations in the N-terminal region cause instability of the cytochrome bc1 complex, and levels of Rip1p and Qcr8p are decreased, indicating that they are not fully assembled into the complex. QCR7 is conserved across eukaryotes, although the potato and human orthologs fail to functionally complement the qcr7 null mutation.