Atx1p is an 8 kDa cytosolic mettallochaperone required for copper-dependent iron absorption. Atx1p transports copperionsfrom Ctr1p on the plasma membrane to Ccc2p on a post-Golgi vesicle for eventual targeting to the cell-surface high-affinity iron uptake protein Fet3p. atx1 deletion mutants are deficient in iron absorption, but this deficiency is suppressed by adding copper. A second, endocytosis-dependent pathway is capable of delivering copper to Ccc2p. ATX1 was identified as a multicopy suppressor of the superoxidesensitivity phenotype of superoxide dismutasedeficient cells. A weak intrinsic antioxidant activity of Atx1p stoichiometrically compensates for SOD deficiency when ATX1 is overexpressed. ATX1 expression is induced by oxygen, but not by copper. It is also induced by iron through the action of Aft1p, but is not strictly dependent on Aft1p.The human homolog, called Atox1 or HAH1, complements deletion of ATX1, as do Arabidopsis thaliana CCHand C. elegans CUC-1. Atox1 is a candidate gene for the 5q deletion syndrome.