In this work, we studied two yeast ABC transporters, Pdr10p and Pdr15p. At the time of assignment of this thesis, it was believed that these proteins contribute to the yeast MDR phenotype (PDR) on the grounds of their high homology to another yeast MDR protein, Pdr5p. In order to study these pumps, two sets of isogenic null-mutant strains were prepared with all possible combinations of gene deletions. We report that both of the studied proteins are very important in sustaining the normal plasma membrane microenvironment for the most abundant, and essential, yeast plasma membrane protein, H^+-ATPase and so influence the membrane potential. Pdr10p and Pdr15p thus play an as yet unknown role in regulation of the activity of this enzyme. Furthermore, we report that deletion of the genes coding for these proteins severely reduces the ability of the H^+-ATPase to be activated by the protonophore CCCP which is a weak acid. Studies performed with immunosuppressant FK506 further show that this compound reduces the viability of *S. cerevisiae* mutant strain PLY643 lacking genes coding for Pdr5p, Snq2p and Yor1p. Further deletion of Pdr10p and Pdr15p does not increase the lethality of this compound. Neither CCCP nor FK506 are substrates of the studied pumps.