One of the pathomechanisms of prostate carcinogenesis is losing the ability of the epithelial cells in the prostate gland to accumulate zinc. Zinc could induce apoptosis in prostate tissue through releasing of cytochrome c and other proapoptotic molecules which is regulated by Bcl-2 family protein. Moreover, cytochrome c release is associated with the transport system in the mitochondrial membrane. However, it is not clear, whether cytochrome c is released through VDACs (voltage dependent anion channels) or by the involvement of the TOMM (translocase of outer mitochondrial membrane) complex protein. Both play a role in molecule transport through the outer mitochondrial membrane. In this study we investigated the levels of mRNA expression of Bcl-2 family (proapoptotic Bax and Bid, antiapoptotic Bcl-2) as well as VDAC isoforms (VDAC1, VDAC2, VDAC3) and TOMM isoforms (TOMM20, TOMM22, TOMM40) genes in 13 paraffin embedded prostate cancer tissue compared to 5 normal prostate tissue using custom designed quantitative PCR array (SAB Biosciences). We found a significant increase of mRNA expression of antiapoptotic Bcl-2 and VDAC1 genes in prostate cancer tissue compared to normal tissue. There is no significant difference in mRNA expression between the proapoptotic Bax and Bid genes, the VDAC2 and the VDAC3 isoforms as well as the three TOMM isoforms in prostate cancer tissue and normal tissue. Our results provide first evidence that both Bcl-2 and VDAC1 play a role for prostate carcinogenesis; however, studies with larger cohorts of patients will help to clarify whether Bcl-2 and VDAC1 could be used as diagnostic biomarkers.