Abstract

There is currently no curative treatment for the metastatic stage of prostate cancer and effective treatments are therefore urgently needed. Inducing apoptotic cell death in prostate cancer cells is one possible approach and increased calcium levels have been shown to induce cell death in a number of prostate cancer cell lines. Expression of calcium channels in cancer cell lines is one approach by which intracellular calcium can be increased to induce cell death.

hTRP3 (the human homologue of TRP3 a member of the TRP calcium channel family) was therefore inducibly expressed in LNCaP prostate cancer cells and its effects on calcium inflow and cell death examined.

Western blot analysis and fluorescence revealed that both EGFP and hTRP3 were inducibly expressed in LNCaP cells. Calcium measurements in hTRP3 transfected LNCaP cells suggested that hTRP3 was activated by thapsigargin induced store depletion and OAG, but was not constitutively active. Transient transfection of hTRP3 into LNCaP cells also did not induce apoptosis, which was confirmed by Hoechst 33258 staining. This may have been due to low levels of hTRP3 expression, which resulted in only small increases in stimulated calcium inflow. To determine if hTRP3 can be used to induce cell death in LNCaP cells further experiments optimising expression and activation of hTRP3 need to be carried out.