DETECTION OF CHROMOSOMAL TRANSLOCATION IN PROSTATE CANCER AND BENIGN PROSTATIC HYPERPLASIA BY FLUORESCENCE IN SITU HYBRIDIZATION (FISH)

Cebulska-Wasilewska A1,2, Miszczyk J1, Dobrowolska B4, Dobrowolski Z4
1Environmental and Radiation Biology Department, The H. Niewodniczanski Institute of Nuclear Physics PAN, Cracow, Poland
2Epidemiology and Preventive Medicine Department CM UJ, Cracow, Poland
3Environmental and Radiation Biology Department, The H. Niewodniczanski Institute of Nuclear Physics PAN, Cracow, Poland
4Urology Department and Clinic CM UJ, Cracow, Poland

Key words: FISH, prostate cancer (PC), benign prostatic hyperplasia (BPH)

Prostate cancer is one of the most common males’ cancers worldwide and it ranks third in Poland. It is believed that prostate cancer is the result of an accumulation of mutations. Genetics predisposition (polymorphisms) and alterations on multiple chromosomes, in particular on chromosome 1, are considered as possible causes of increased risk of diseases. In addition to this prostate cancer risk might be strongly influenced by familial history and environmental factors for example: tobacco smoking, marital status, diet, exposure to cadmium, zinc, selenium. In this study we have chosen chromosome 1 to compare the chromosomal translocation frequencies detected in lymphocytes of prostate cancer (PC), benign prostatic hyperplasia patients (BPH) and healthy donors. We have examined cells in metaphase using Fluorescence In Situ Hybridization (FISH) as a powerful tool for detection of numerical chromosome translocation. Our results showed significant higher frequencies of translocations in patients with prostatic cancer than with the benign prostatic hyperplasia. We
have also studied correlation between occurrence of cancers in family and level of translocation frequency. We have found that prostate cancer patients who had reported other cancers in family had statistically higher frequency of translocations than other patients. Prostate cancer patients expressed disease significantly earlier than BPH. Our results confirm that association exists between predisposition to genetic instability chromosome 1.