Expression and Function of ETS Genes in Prostate Cancer

Stellingen

1. In clinical prostate cancer, full-length ETV1 can be overexpressed by translocation of the complete gene to novel genomic regions (this thesis).

2. Truncated ETV1, lacking the N-terminal transactivation domain, produced by fusion genes in prostate cancer, has not the same molecular and biological properties as full length ETV1 (this thesis).

3. Prostate cancer patients with high expression levels of TMPRSS2-ERG fusion transcripts starting at an alternative upstream first exon (exon 0) have a longer PSA-recurrence free survival (this thesis).

4. In ERG-positive prostate cancer a sub-group of patients with rapid progression can be identified (this thesis).

5. Genes involved in cell-cell communication, TGF-β signalling and bone remodelling are the major contributors to the gene-classifier of cancer progression in ERG-positive prostate cancers (this thesis).


7. Mouse studies support the assumption that ERG overexpression is not sufficient for prostate cancer development; additional genetic and/or epigenetic alterations are essential. (Zong et al, Proc Natl Acad Sci USA 2009;106:12465-70; Klezovitch et al, Proc Natl Acad Sci USA 2008;105:2105-10).

8. Paulo et al. (Neoplasia 2012;14:600-11) indicate that overexpression of ERG and ETV1 in clinical prostate tumor samples partially affects the same genes but this finding is not sufficiently supported by the experimental data.

9. Although prostate cancer is polyclonal with genetic heterogeneity between the different tumour foci, metastases in a patient (Liu et al, Nat Med 2009;15:559-65) seem to have a monoclonal origin.

10. “The last function of reason is to recognize that there are an infinity of things which surpass it” Blaise Pascal

11. “When nothing goes right…go left” Anonymous

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