Subject: Schlüssel zum PFS?
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A pharmacogenetic survey of androgen receptor (CAG)n and (GGN)n polymorphisms in patients experiencing long term side effects after finasteride discontinuation.
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Finasteride is a steroid 5-alpha-reductase inhibitor, approved for the treatment of androgenetic alopecia (AGA) and benign prostate hyperplasia. In some patients the treatment is associated with adverse side effects that could become persistent after therapy discontinuation, resulting in the so-called post-finasteride syndrome (PFS). A pharmacogenetic component in the response to finasteride treatment was previously demonstrated. Two polymorphisms (CAG) rs4045402 and (GGN) rs3138869 in the gene encoding for the androgen receptor (AR) have been hypothesized to play a role in finasteride sensitivity. We aimed to compare the rs4045402 and rs3138869 polymorphisms prevalence in a group of 69 selected subjects (AGA+PFS) that used finasteride to treat alopecia and developed persistent side effects, with that in a group of 91 untreated subjects with AGA (AGA), and a group of 76 untreated subjects without AGA (NO-AGA). The rs4045402 and rs3138869 polymorphisms extreme-lengths alleles were more frequent among AGA+PFS (odds ratio, 5.88; 95% CI, 1.87-18.52) and AGA subjects (odds ratio, 3.55; 95% CI, 1.13-11.21) than among NO-AGA subjects, probably reflecting the genetic predisposing factors for AGA development. In conclusion, we described a predictive effect of the less common repeats' length CAG-rs4045402 and GGN-rs3138869 on AGA development. Prospective trials are required to confirm our findings also in other ethnicities, and to highlight possible further pharmacogenetic predictive markers of susceptibility to adverse effects.