Highlight report: *N*-acetyltransferase 2 and urinary bladder cancer risk

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In recent years, genome-wide association studies have identified and validated a set of genetic variants that are associated with urinary bladder cancer risk (e.g., Figueroa et al. 2016; Rafnar et al. 2014; Selinski 2014a, b; Selinski et al. 2012, 2013; Kiemeny et al. 2010; review: Golka et al. 2011). After identification of the most relevant individual variants, it became possible to identify SNP interactions (Schwender et al. 2015) and to differentiate the contribution of genetic predisposition and occupational exposure to urinary bladder cancer risk (Selinski et al. 2016; Krech et al. 2013, 2016).

Recently, it has become clear that at least one of these genetic variants, NAT2*6A/*6A is not only associated with increased risk to develop bladder cancer, but also with prognosis of bladder cancer patients (Selinski et al. 2017). NAT2*6A/*6A leads to a particularly slow enzymatic activity of *N*-acetyltransferase 2, the so-called ultra-slow acetylator (Selinski et al. 2014; Ruiz et al. 2012; Patillon et al. 2014). In the lately published study with 586 urinary bladder cancer patients, ultra-slow acetylators were shown to have a higher recurrence risk and a shorter recurrence-free time compared to genotypes associated with a more rapid acetylation phenotype (Selinski et al. 2017). This association was particularly strong in smokers. The result is of high interest, since a higher risk of recurrence in NAT2*6A/*6A patients justifies a more intensive monitoring of this subgroup after surgery. Progress in the field of the prognostic relevance of genetic variants on prognosis of

bladder cancer is currently hampered by the lack of large cohorts with available DNA and a carefully established follow-up. Nevertheless, one of the most important steps in future will be to analyze also all other risk variants identified in genome-wide association studies with respect to their prognostic relevance after surgery of bladder cancer.

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