

Aspirin and Prostate Cancer Incidence and Mortality—Letter

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In a recent publication, Hurwitz and colleagues (1) focused on the associations of aspirin and non-aspirin nonsteroidal anti-inflammatory drugs (NA)-(NSAID) use with prostate cancer incidence, mortality (death from prostate cancer among men without a cancer history), and case fatality (death from prostate cancer among men with prostate cancer) in a population-based cohort of white and black men (1). The authors examined 5,060 white men and 1,534 black men from the Atherosclerosis Risk in Communities study (1). They found that aspirin use was not connected with prostate cancer incidence (1). They observed that aspirin use was inversely linked to prostate cancer mortality and case-fatality among white and black men on daily aspirin therapy and/or for cardiovascular disease prevention (1). The study demonstrated that NA-NSAIDs use did not relate to these endpoints (1). The authors conclude that benefits of aspirin for preventing prostate cancer mortality may require to be factored into risk-benefit calculations of men in view of an aspirin regimen (1). NSAIDs have been suggested to moderately decrease the risk of prostate cancer (1). However, it still remains unclear whether NSAIDs protect against prostate cancer mortality and case-fatality (1). Large-scale next-generation genetic analyses of

prostate cancer have frequently detected the importance of focal genomic deletions inactivating PTEN (2). Loss of PTEN in radical prostatectomy samples often coexists with genomic rearrangements involving the ETS family transcription factors (2). PTEN loss has been reported to be reproducibly linked to adverse oncologic outcomes and help to differentiate indolent tumors from those likely to progress (2). In this light, PTEN has been proposed as a useful prognostic biomarker to distinguish potentially aggressive grade group 1 or 2 tumors, which might make patients poor candidates for active surveillance programs (2). The PTEN signaling pathway has been suggested to be a molecular mechanism underlying aspirin-mediated cellular changes (3). Aspirin has been documented to significantly increase the expression of PTEN when compared with that in the control group (3). Conversely, NA-NSAID use has been statistically significantly connected with PTEN loss (4). PTEN deficiency has also been shown to promote pathologic vascular remodeling of human coronary arteries (5). Taken together, I suppose that the potential benefits of aspirin against prostate cancer may be mediated by PTEN upregulation unlike NA-NSAIDs that appear to down-regulate PTEN. Further research examining the association between aspirin use and decrease in prostate cancer risk by up-regulation of PTEN is warranted among men with and without preexisting cardiovascular risk factors.

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