Advanced prostate cancer risk, selenium, and oxidative stress: the role of genetic variation and environment

STELLINGEN

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Maastricht, 29 oktober 2014

A low selenium status is a risk factor for advanced prostate cancer (this thesis).

Common genetic variation in the selenoprotein genes *SEPP1* and *GPX1* is associated with advanced prostate cancer risk, but does not modify the association between toenail selenium levels and advanced prostate cancer risk (this thesis).

A higher intake of flavonoids is associated with a decreased risk of advanced prostate cancer (this thesis).

Gene–environment interactions in the oxidative stress pathway are involved in the etiology of advanced prostate cancer (this thesis).

When studying the etiology of prostate cancer one should always study disease subgroups based on clinicopathological parameters.

It is pointless to compare the findings from different studies on overall prostate cancer risk if the studies were based on populations with a very different frequency of Prostate-Specific Antigen (PSA) testing in asymptomatic men.

In hypothesis-based gene–environment interaction research it is better to allow some false positive results rather than to guard against any false positives.

Gene–environment interaction studies offer no immediate opportunities for knowledge valorization.

The best way to have a good idea is to have a lot of ideas (Linus Pauling).