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Alcohol and prostate cancer risk

In a large follow-up study, we found that heavy regular alcohol consumption and binge drinking during midlife were associated with a significantly increased risk of prostate cancer risk. Specifically, men who were heavy drinkers (>14 drinks/week) were at a 46% higher risk of prostate cancer compared to those who were light drinkers (≤ 3 drinks/week). Among current drinkers, binge drinkers – defined as consuming 5 or more drinks in one sitting at least once per month – were at a 28% increased risk of prostate cancer compared to non-binge drinkers. Interestingly, alcohol abstinence was associated with an increased risk of prostate cancer-specific mortality compared to light alcohol consumption.

This study was conducted in a large population of Finnish twins with 30 years of follow-up data. This unique population of men included a wide range of alcohol consumption, and a high prevalence of heavy drinking and reported binge drinking.

This twin population also allowed for the application of powerful analytic methods to control for potential confounding by genetics and shared early environment.

In this setting, we examined the association between alcohol and prostate cancer in two ways: (1) individual-based analyses of the entire cohort, and (2) analyses within twin pairs discordant for alcohol consumption and prostate cancer outcome (i.e. twin 1 develops prostate cancer and twin 2 does not, and both differ in their alcohol intake).

If the association between alcohol and prostate cancer is rooted in familial environmental factors, one would expect to find an association *between* twin pairs, but not *within* discordant twin pairs raised in the same household. If the association between alcohol and prostate cancer is rooted in genetic factors, one would expect to find an association within dizygotic (DZ) twin pairs (who share on average 50% of their segregating genes), but not within monozygotic (MZ) twin pairs (who share 100% of their genome sequence).

Lastly, if the association is rooted in non-familial environmental factors (independent of shared early environment and genetic factors), then one would expect to find an association within both DZ and MZ twin pairs. Non-familial environmental factors include all environmental factors unique to twin individuals as well as direct causal associations between alcohol consumption and prostate cancer.

Our co-twin analyses suggested that alcohol consumption may be associated with prostate cancer risk independent of early environmental and genetic factors.

The biologic mechanisms potentially linking alcohol and prostate cancer are complex. Further study is needed to explore how alcohol intake may impact different stages of prostate carcinogenesis and grades of disease.

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