

Milk and risk of renal cell cancer: Genetic research sheds new light

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While previous research had suggested that drinking milk was related to factors that may increase the risk of renal cell cancer, results of a recent study exploiting the genetic contribution to variation in milk consumption suggest that this may not be the case.

"The data in this study provide no concrete evidence of a need to alter [milk](#) drinking in any way," said lead researcher Nicholas Timpson, Ph.D., lecturer in genetic epidemiology at the MRC CAiTE Center in the department of social medicine at the University of Bristol, United Kingdom. "If anything, the failure of genetic findings to replicate the association between milk and renal cell [cancer](#) suggests that fears that milk consumption might elevate [cancer risk](#) are likely to be unfounded."

These study results are published in the May issue of *Cancer Epidemiology, Biomarkers & Prevention*, a journal of the American Association for Cancer Research.

Previously reported studies suggested a connection between milk intake and renal cell carcinoma risk, and whether this represents a causal association or is the result of bias is currently unclear. Timpson and colleagues used a genetic marker to try to help untangle this observation.

From 1999 through 2003 the researchers conducted a large, hospital-based, case-control study from four central and eastern European countries.

Using observational, genetic and phenotypic data, they determined whether the genetic variant at the gene MCM6 — known to be associated with lactose tolerance — may be used as a non-confounded and unbiased marker for milk consumption's link to cancer risk.

For adult milk drinkers vs. non-milk drinkers in this study, the difference in the odds of renal cell carcinoma was approximately 35 percent. However, when assessing the relationship in a more direct way by using genetic data there was no association between the two.

"We found evidence for the often-questioned relationship between milk consumption and cancer, yet when we used genotypes to verify this relationship, there was no corroboratory evidence," Timpson said. "This does suggest that the basic findings may be subject to the kinds of biases and inaccuracies that often upset epidemiological research, but that this study would need to be undertaken on a much larger scale in order to verify these initial findings."

Johanna Lampe, Ph.D., an editorial board member of *Cancer Epidemiology, Biomarkers & Prevention* who is not associated with this study, said this study demonstrates the complexities of evaluating dietary exposures and cancer risk.

"These results are a reminder to proceed with caution when interpreting data that suggest an association between intake of specific foods and risk of a particular cancer. Human diet is complex and typically involves adherence to certain dietary patterns that are also tied to other lifestyle behaviors," said Lampe, full member and nutrition scientist in the division of public health sciences at Fred Hutchinson Cancer Research Center, Seattle, Wash.

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