Pesticide Use and Prostate Cancer Incidence in a Prospective Cohort Study

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Introduction:

Prostate cancer has been observed to occur in excess among farmers and other agricultural workers in a number of countries around the world.

Methods:

A prospective cohort of 89,658 pesticide applicators and their spouses were followed for cancer incidence for an average of 8.5 years. Members of the cohort completed questionnaires about pesticide use, occupations, lifestyle, and medical history during enrollment (1993–1997) and at 2 subsequent periods of time. A total of 1289 incident prostate cancers cases were identified among the 57,311 male pesticide applicators through December 2003 through linkage with population-based cancer registries. Two exposure metrics were used to estimate exposure. One was an estimate of total days of a specific pesticide use in a life-time; the second, total days of exposure weighted by a published estimate of exposure intensity.

Results:

Male cohort members experienced a small, but significant, elevated risk of prostate cancer compared with the general populations of the study states (SIR = 1.24; 95% CI, 1.18–1.33). A family history of prostate cancer among first-degree relatives of male cohort members conferred a twofold excess risk of prostate cancer. Significant linear exposure-response trends ($P \leq 0.05$) were observed for 6 specific pesticides (fonofos, phorate, coumaphos and chlorpyrifos, which are organothiophosphates; butylate, an herbicide; and permethrin, an insecticide), but only among those with a family history of prostate cancer, confirming a preliminary published report from this study.

Discusion/Conclusions:

The consistency of our findings with 2 different exposure metrics, in both study sites and with preliminary published results, suggests that genetic susceptibility among pesticide applicators may play an important role in prostate cancer etiology. Shared environmental risk factors for prostate cancer among family members, however, may also explain these results. Planned molecular studies nested within the same cohort to assess genetic and epigenetic modes of action might clarify and explain these associations.