Weighing the data on diet and prostate cancer

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n 2001, my colleagues and I asked whether the link between nutrition and prostate cancer was evidence-based or merely a suspicion.¹

The descriptive epidemiology of prostate cancer suggests that it is indeed a preventable disease. The evidence and scientific data of a variety of prevention strategies include, among many others, dietary fat reduction.²

Omega-3 and omega-6 polyunsaturated fatty acids are essential fatty acids. The Western diet contains a disproportionally high amount of omega-6 and a low amount of omega-3 polyunsaturated fatty acids, and the resulting high omega-6/omega-3 ratio is thought to contribute to cardiovascular disease, inflammation and cancer. Studies in human populations have linked high consumption of fish or fish oil to a reduced risk of colon, prostate and breast cancer, although other studies have failed to find a significant association. Nonetheless, the available epidemiological evidence, combined with the demonstrated effects of omega-3 polyunsaturated fatty acids on cancer in animal and cell culture models, has motivated the development of clinical interventions using omega-3 polyunsaturated fatty acids in the prevention of cancer.³

In this issue of *CUAJ*, Amin and colleagues⁴ have addressed an interesting issue, namely, the impact of nutritional factors on prostate cancer development. They looked at a database of 917 patients planned to have transrectal ultrasonography–guided biopsies based on an elevated serum prostate-specific antigen (PSA) level, a rising serum PSA level or an abnormal digital rectal examination. They also performed univariable and multivariable logistic regression analysis for prediction of cancer and its aggressiveness using a self-administered food frequency questionnaire given before the biopsies.

The authors found that on multivariable analyses, a meat diet including red meat, ham and sausages was associated with an increased risk of prostate cancer, and a fish diet was associated with less prostate cancer. None of the tested dietary components were associated with prostate cancer aggressiveness.

The authors have nicely pointed out the strengths and weaknesses of their study, including its limitations. By definition, case–control studies are prone to certain types of unavoidable biases. However, the strength of the paper lies in its multivariable analysis and the detailed questionnaire that patients completed before undergoing the prostate biopsy. Noteworthy, red meat, ham and sausages as well as fish clearly had unfavourable and favourable impacts, respectively. Potatoes, interestingly enough, had a protective odds ratio close to statistical significance although it is not clear whether this has something to do with potatoes themselves or whether patients who eat a lot of potatoes actually have other types of dietary habits that protect them.

With respect to the incidence of aggressive prostate cancer and its relation to diet, it is not clear whether the absence of an association was due to the low number of cancers found in that group or whether increasing the number of these patients would have potentially brought positive results. For instance, with respect to pulses, such as beans and peas, the odds ratio for a Gleason score above 7 and number of cancer cores above 3 was, respectively, 1.86 and 2.89. Most odds ratios close to 3 would probably be significant on a large patient population.

Recently, the association between prostate cancer risk and fresh and preserved fish consumption among participants of a populationbased case-control study (1534 cases, 1607 controls) was investigated in Canada.⁵ Fish intake was measured using a dietary questionnaire that collected frequency of consumption of a given portion size of both fresh and preserved fish. Logistic regression analysis demonstrated an inverse association between preserved fish and prostate cancer risk for all levels of consumption, but reductions only reached statistical significance for the category of 1 to 3 servings of preserved fish per month (odds ratio 0.78, confidence interval 0.64–0.95). Consumption of any fat or energy from preserved fish was also associated with reduced risk. There was no suggestion of reduced prostate cancer risk with consumption of fresh and canned fish. These results suggest that consumption of preserved fish may reduce the risk of developing prostate cancer. It is not crystal clear why the difference between preserved and fresh fish is so important, but it warrants further study. These sometimes contradictory results highlight our

limitations in the understanding and effectiveness of prostate cancer preventive means and the numerous grey areas that need additional investigations.

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Competing interests: None declared.

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