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Omega-3 polyunsaturated fatty acid intake through fish consumption and prostate specific antigen level: Results from the 2003 to 2010 national health and examination survey

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ABSTRACT

The etiology of prostate cancer is uncertain, but intake of omega-3 polyunsaturated fatty acids (n-3 PUFAs) may play a role. We evaluated prostate specific antigen (PSA) levels with fish consumption (the primary source of n-3 PUFAs) and calculated PUFA intake. Serum PSA concentrations were available from 6018 men who participated in the 2003–2010 National Health and Nutrition Examination Survey (NHANES). Fish consumption was calculated via 30-day Food Frequency Questionnaire data, whereas n-3 PUFA intake was calculated from 24-h dietary recalls. We employed multivariable logistic and linear regression models to evaluate the association of these exposure variables with PSA levels while controlling for relevant covariates. PSA levels were lower in men who ate more breaded fish, but no other types of fish consumption or n-3 PUFA intake were associated with PSA levels. Our findings provide little evidence for a role of fish or n-3 PUFA consumption in influencing PSA levels.

1. Introduction

Prostate cancer is the most common type of cancer among males in the United States. An estimated 238,590 American males have been diagnosed with prostate cancer in 2013 [1] while roughly 1 in 6 American men will receive a positive diagnosis for this cancer in their lifetime [2]. The underlying mechanisms for prostate cancer are unclear but are thought to involve specific eicosanoids, such as prostaglandins, which play a key role in the development of prostate by upregulating prostate cancer progression [3].

Environmental and dietary factors that modify the regulation of these eicosanoids may influence risk of prostate cancer risk, such as the nutritional intake of omega-3 (n-3) polyunsaturated fatty acids (PUFAs). The n-3 PUFAs, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are known to inhibit cyclooxygenase and lipooxygenase pathways which metabolize arachidonic acids to eicosanoid prostaglandins [4]. Prostaglandins, synthesized from PUFAs, are known to modulate tumor metastasis, immunoregulation, tumor promotion, and cell proliferation [5]. This mechanism suggests that an increase in EPA and DHA intake would likely decrease the risk of developing prostate cancer by inhibiting prostaglandin development.

The primary source of exposure to EPA and DHA is through the consumption of fish [6]. Previous studies have examined the relationship between total fish consumption and incidence of prostate cancer, but results have been inconclusive [7]. Other studies attempted to find the association between calculated PUFA concentrations, found from the dietary intakes of participants, and incidence of prostate cancer. The results of some of these studies showed a decreased incidence among those with higher PUFA concentrations, while others showed adverse associations with increased PUFA concentrations [8].

Prostate specific antigen (PSA) has been used as a screening test for prostate cancer in older men [9,10], with a higher PSA level indicating higher likelihood of the participant having prostate cancer. Generally, screening recommendations have used a cutoff of 4 ng/mL PSA concentration level or above to indicate prostate cancer in an individual or for further diagnostic testing [11].

Using National Health and Nutrition Examination Survey (NHANES) data from 2003 to 2010, we evaluated the association between self-reported 30-day fish consumption (with specific fish types), estimated 24-h EPA and DHA nutrient intake, and prostate-specific antigen (PSA) levels. We hypothesized that an increase in fish consumption and estimated PUFA intake is associated with a lower PSA level.

2. Patients and methods

2.1. Study population

NHANES is a national survey designed to assess the health and nutritional status of the United States population. Details of the

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NHANES probability sampling strategy and data collection procedures have been previously described [12,13]. Briefly, NHANES is designed to be a representative sample of the overall US population when appropriate weights are considered. Participants are recruited and evaluated in two-year cycles starting in 1999. Mobile examination centers (MECs) are parked in selected counties for about six weeks. Selected NHANES participants from those counties are asked to visit one of the MECs for physical examinations, medical testing, and interviews by a trained professional. In this study, four (2-year) cycles were combined to include national data from 2003 to 2010, based on available data for our analyses.

We analyzed NHANES data on males aged 40 or above, the age cut off for eligibility for PSA testing. From 2003 to 2010 there were 6856 males 40 years of age and older that were examined at MECs around the United States. Of these, 6018 (87.8%) have complete data for total PSA level. For each of our analyses we restricted our sample size to those males who had data on the exposure of interest. A total of 5770 (84.2%) participants with data on DHA and EPA intake were used in our PUFA analyses. For the specific fish consumption analyses, 4525 participants (66%) were used after excluding those with missing fish consumption data.

2.2. Assessment of PSA level

Serum levels of total PSA were measured in eligible men after excluding those with current infection or inflammation of the prostate gland and history of prostate cancer. Also excluded were those men who had a rectal examination in the past week, prostate biopsy within the past month, or cystoscopy within the past month. Additionally excluded from our analyses were those with missing data on any of the above criteria [14].

Total PSA level (ng/mL) was recorded using the Hybritech PSA method on the Beckman Access using serum samples from the participant collected by an NHANES physician. Further laboratory methodology has been previously described [13]. Continuous and dichotomous (above or below 4 ng/mL) total PSA data were used as outcome variables in our analyses.

2.3. Dietary EPA and DHA from NHANES

Dietary information was collected through two different NHANES questionnaires. Specific fish types consumed were found using a 30-day Food Frequency Questionnaire while EPA and DHA estimates were calculated using 24-h dietary recalls. Both methods assess EPA and DHA intake level since fish is the primary dietary source of these PUFAs [6].

2.4. Specific fish consumption

A Food Frequency Questionnaire (FFQ) was used to examine specific foods the participants consumed and number of times each was eaten in the 30 days prior to the interview. Included in this questionnaire was data on specific types of fish and shellfish consumed. Types of fish included: tuna, bass, catfish, cod, flatfish, haddock, mackerel, perch, pike, pollock, porgy, salmon, sardines, sea bass, shark, swordfish, trout, walleye, and an “other fish” category. Types of shellfish assessed included: clams, crabs, crayfish, lobsters, mussels, oysters, scallops, shrimp, and an “other shellfish” category. Cooking method information of fish (whether fish was breaded or not breaded) was also recorded. We categorized the total number of fish meals into 4 fish groups (all fish, breaded fish, non-breaded fish, and shellfish) after obtaining continuous fish meal variables from NHANES. Each group was then organized into 4 categorical levels (none, low, medium, and high) based on tertiles of distribution of non-zero fish consumption values. We also created a dichotomous (none vs. any) consumption variable for all the fish consumption groups.

2.5. Assessment of EPA and DHA intake

A 24-h dietary recall was used to assess nutrients and food components (including EPA and DHA levels). The participants provided specific foods they had eaten in the past 24 h to a trained interviewer and then followed-up with a second 24 h recall 3–10 days later via telephone. Amounts of energy and 64 nutrients/food components (including PUFA intake) from each food recalled in the NHANES questionnaire was then calculated for each 24-h recall using the United States Department of Agriculture's Food and Nutrient Database for Dietary Studies, 5.0 [13]. NHANES reports EPA and DHA measurements in grams. The average of the two 24-h recalls was calculated for both EPA and DHA. The average EPA and DHA concentrations for each participant were then used to calculate a sum value of the two estimates (EPA+DHA) to test for correlation. Since average EPA, DHA, and their summation were highly correlated, only the average EPA+DHA was used in the final analysis. Values for the EPA+DHA variable were energy adjusted using the energy density method (g/1000 kcal) [15]. Continuous values of energy adjusted EPA+DHA were used to create a categorical EPA+DHA variable (none, low, medium, or high) based on tertiles with non-zero values. A dichotomous (none vs. any) variable was also created.

2.6. Covariates

The following covariates were selected to be controlled for in our analysis because they have been reported to be associated with PSA level: age, race (black, white, and other), education (less than high school, high school, and above high school), BMI, and smoking status (100 cigarettes in lifetime vs. less than 100 cigarettes in lifetime) [16–18]. Fish oil supplementation (yes or no) taken in the 30 days prior to the examination was also included as a covariate because of their relationship with individual EPA+DHA intake levels. Methodology for the calculation of fish oil supplementation using available NHANES supplement data was similar to Hoffmire et al. [19].

2.7. Statistical analysis

Descriptive analyses were conducted to describe measures of central tendency and variability for the variables included in our analysis. Adjusted ORs allowed us to determine which covariates were independent predictors of PSA, using PSA dichotomously. All covariates, with the exception of education, proved to be significantly associated with PSA. Subsequently, we performed several regression analyses to estimate the association between fish consumption, estimated PUFA intake, and PSA level using different ways of defining the primary exposure and predictor variables. That is, PSA was examined both as a continuous and a dichotomous (above or below 4 ng/mL) variable, where 4 ng/mL is the current clinical cut-point for a positive screen [20]. For PSA as a continuous variable, we employed linear regression models; whereas, we utilized logistic regression models for PSA as a dichotomous variable. Fish consumption was analyzed both as a categorical variable (none, low, medium, or high) using tertiles of the distribution of exposure among those that ate any fish, and as a dichotomous variable (none vs. any). Similarly, EPA+DHA intake was used as a continuous variable (g/1000 kcal), a categorical variable (none, low, medium, or high) based on tertiles of the exposure distribution among those with any estimated intake, and as a dichotomous variable (none vs. any). All regression models were run with and without controlling for the aforementioned a priori selected covariates to estimate crude and adjusted associations between PSA concentrations and fish or PUFA intake.