Null Results in Brief

Cancer Epidemiology, Biomarkers & Prevention

Dietary Benzo[*a*]pyrene Intake from Meat and the Risk of Colorectal Cancer

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Abstract

Introduction: One possible mechanism for the postulated link between high consumption of meat and colorectal cancer (CRC) is the content of benzo[*a*]pyrene (BaP) in meat.

Methods: We investigated this association in a population-based case-control study in Western Australia (567 cases and 713 controls). Participants' self-report of meat consumption and lifestyle was used in conjunction with the CHARRED carcinogen database to estimate their BaP intake.

Results: Dietary exposure to BaP from meat consumption was not associated with the risk of CRC.

Conclusions: Our findings do not support the hypothesis that BaP exposure from meat consumption is a risk factor for CRC.

Impact statement: This large-scale case-control study used a detailed meat questionnaire in conjunction with the CHARRED database in a large population with high meat intake but was unable to find any association between intake of BaP and colorectal cancer. *Cancer Epidemiol Biomarkers Prev; 19(12); 3182–4.* ©2010 AACR.

Introduction

High intake of meat has been linked to the increased risk of colorectal cancer (CRC) (1). A possible mechanism for this link may be the levels of polyaromatic hydrocarbons (PAHs) produced in meat by pyrolysis of meat fats during cooking (2). One of the potentially carcinogenic PAHs identified as occurring in cooked meat is benzo[*a*]pyrene BaP (2). BaP levels are a function of the fat content of meat, cooking method, level of doneness, proximity to and type of heat source, and smoking process.

Previous epidemiological studies of CRC and colorectal adenoma have not given a clear result. One possible reason for the different results is a low population intake of BaP. Given that the Australian population has a much higher average meat intake than most other countries, it may be more likely that any effect could be clearly seen.

Methods

This study was undertaken as part of the Western Australian Bowel Health Study (WABOHS) (3). Cases were residents of Western Australia, aged 40–79 years, and diagnosed with a first incident CRC between July 2005 and February 2007. Frequency sex and 5-year age matched controls were recruited from the Western Australia Electoral Roll. Participants completed a questionnaire on lifestyle and demographic factors and a food frequency questionnaire (FFQ). In addition, the WABOHS meat consumption questionnaire (3) was completed by 575 cases and 709 controls to obtain information on frequency of consumption, cooking techniques, serving size, and levels of doneness of meat 10 years earlier. Values for BaP in nanograms per day were calculated by linking the meat consumption data to the CHARRED carcinogen database(4). CHARRED does not contain BaP values for fish and lamb so we used BaP values for skinless chicken that has similar fat content.

We calculated Odds Ratios and 95% Confidence Intervals for CRC associated with BaP from total meat intake adjusted for sex, age, BMI, smoking, physical activity, and intake of alcohol, multivitamins, fruit and vegetables, cereal, total energy, fat, and fiber. Models were repeated for BaP from red meat and from white meat.

Results

Demographic data on the subjects is available in a previous publication (3). The mean exposure to BaP was almost identical for cases and controls (Table 1). In multivariate models, quartiles of estimated BaP exposure from total meat, red meat, and white meat, were not associated with CRC (Table 2). A nonsignificant increase in the risk of CRC was observed with higher levels of intake of BaP derived from white meat. When fish and lamb were excluded, the results were similar.

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Table 1. BaP exposure estimates (nanograms per day) derived from total, red, and white meat consumption (all meat types included) by CRC case and control status, Western Australia, 2005–2007

	Controls ($n = 714$)					Cases ($n = 577$)					*t* test
	Mean	SD	Median	Minimum	Maximum	Mean	SD	Median	Minimum	Maximum	P value
All meat			(ng/day)					(ng/day)			
BaP	78.60	84.90	54.20	0.05	560.77	79.90	86.30	51.85	0.13	594.90	0.78
Red Meat BaP	65.00	77.20	41.84	0.00	548.47	66.30	80.40	41.52	0.00	587.17	0.77
White meat BaP	13.50	19.60	5.91	0.00	150.25	13.50	17.90	7.11	0.01	147.33	0.99
No fish/no lamb											
BaP	74.30	82.80	50.03	0.00	549.73	75.10	84.50	48.04	0.00	588.87	0.87
Red meat BaP	65.00	77.20	42.05	0.00	548.45	66.50	80.60	41.51	0.00	587.05	0.74
White meat BaP	9.30	19.70	1.17	0.00	175.58	8.60	16.50	1.85	0.00	146.26	0.49

Table 2. Odds ratios[a] and 95% confidence intervals for association between the quartiles of BaP exposure estimates and the risk of CRC, Western Australia, 2005–2007

	All meats included		
	Range	OR	95% CI
BaP	(ng/d)		
Q1	<17.1	1.00	
Q2	17.2–54.3	1.41	1.01–1.95
Q3	54.4–108.9	1.06	0.75–1.48
Q4	108.9	0.96	0.67–1.36
Trend *P* value		0.39	
Red meat BaP			
Q1	<10.4	1.00	
Q2	10.4–42.0	1.23	0.89–1.70
Q3	42.1–86.6	1.05	0.75–1.47
Q4	86.6	0.90	0.64–1.27
Trend *P* value		0.40	
White meat BaP			
Q1	<2.4	1.00	
Q2	2.5–5.9	1.16	0.83–1.62
Q3	6.0–15.2	1.34	0.97–1.87
Q4	15.2	1.23	0.88–1.72
Trend *P* value		0.17	

[a]ORs adjusted for sex, age, BMI, smoking, alcohol, multivitamin, fruit and vegetable consumption, physical activity, cereal, total energy, fat, fiber.

Discussion

In our population-based case-control study, we found that dietary exposure to BaP derived from total meat, red meat, or white meat consumption does not contribute to increased risk of CRC in Western Australians.

Although the PAHs content of food, and in particular BaP, has been extensively studied, only 3 studies have examined the link between dietary exposure to BaP and the risk of CRC. Two of these have found BaP to be a risk factor for colorectal cancer (5, 6) while the third found an interaction between BaP exposure and variants of *UGT1A1* and *UGT1A9* genes (7). The results from our study are in contrast with those findings from these previous studies.

Limitations of our study revolve around the estimation of exposure to BaPs. Use of the American CHARRED database may have overestimated BaP exposure as BaP is formed from fat and Australian meat tends to have lower fat content than the grain-fed meat produced in the United States. Also, we used BaP exposure as a surrogate for exposure to PAHs although BaP comprises only between 1 and 20% of total carcinogenic PAHs (8) and there may be cocarcinogenicity between BaP and other types of PAHs. In addition, we were unable to adjust for other potential sources of exposure to BaP, except for cereal consumption and smoking, although ingestion is the most prominent route of environmental exposure to PAHs (2). Finally, we used skinless chicken data to estimate the BaP exposure from fish and lamb consumption. However, sensitivity analysis showed no significant differences in risk estimates when the fish and lamb data were excluded from analysis.

One of the strengths of this study was higher levels of meat consumption and hence higher levels of exposure to meat-derived compounds in Western Australians. The estimated level of exposure in our study population was almost four times that of previous U.S. studies (5).

Although this study found no association with BaP, as a marker of PAHs, and the risk of CRC, this group of potential carcinogens may still have etiologic relevance for CRC. Better estimation of dietary exposure in conjunction with measurement of biomarkers of long term exposure, and perhaps taking into account non-dietary sources of exposure may help to elucidate the potential link between dietary exposure to PAHs and the risk of CRC.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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