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## Correlates of Active and Passive Smoking in the California Teachers Study Cohort

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### ABSTRACT

**Objectives:** These analyses were designed to describe characteristics associated with active and passive smoking in a large cohort of women in order to identify possible confounders of the relationship between smoking exposures and breast cancer risk.

**Methods:** Analyses were based on 1995 data collected from the California Teachers Study (CTS) and were restricted to those with complete and usable tobacco data ( $n = 128,174$ ). Age-adjusted and race-adjusted odds ratios (OR) were generated by unconditional logistic regression.

**Results:** Compared with never smokers, both current and former smokers experienced menarche at an earlier age. Current and former smokers also were more likely than their never smoking counterparts to be nulliparous. Among parous women, current, but not former smokers were less likely than never smokers to have had their first child at an older age. Similarly, among never smokers, those exposed to household passive smoking experienced menarche at an earlier age, were more likely to be nulliparous, and among parous women, were less likely to have had their first child at an older age than never smokers not exposed to passive smoking. Greater alcohol consumption was strongly associated with both active and passive smoking exposures. Compared with never smokers, current smokers were less likely to take antioxidant supplements, whereas former smokers were more likely to take antioxidant supplements. Among never smokers, antioxidant use did not differ depending on passive smoking exposure. A number of other dietary correlates of active and passive smoking were identified.

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**Conclusions:** We identified a number of reproductive and dietary correlates to smoking exposures that underscore the need to adjust for such factors in an analysis of smoking and breast cancer and potentially other disease entities. Furthermore, these findings may suggest potential mechanisms underlying an association between breast cancer and smoking.

## INTRODUCTION

THE POTENTIAL RELATIONSHIP between active and passive smoking and breast cancer risk has been of great interest.<sup>1-4</sup> Breast cancer is the most common cancer in women,<sup>5,6</sup> and most of the known risk factors (e.g., family history of breast cancer, age at menarche) do not offer modifiable avenues for prevention.<sup>7</sup> There is a great deal of literature on smoking and breast cancer dating back over 30 years.<sup>3,4</sup> Earlier studies, paradoxically, seemed to suggest an association with passive but not active smoking,<sup>8,9</sup> whereas more recent, larger, and better designed studies have started to suggest an association for active smoking.<sup>4,10-14</sup> To date, very little has been published with respect to how smoking exposures may relate to other risk factors for breast cancer. Our study provides a detailed description of how both active and passive smoking exposures correlate with established risk factors for breast cancer. Understanding such relationships is fundamental to interpreting previous studies that often did not account for such factors as covariates in their analysis and to the design of future studies investigating the relationship between tobacco-related exposures and breast cancer. Indeed, the identification of such relationships may inform not only investigations of breast cancer but also of other hormonally mediated cancers in which tobacco smoke may play a similar etiological role.

Most studies on passive smoking have been limited to correlates of spousal exposures<sup>15,16</sup> and, thus, have not been able to examine correlates of exposures earlier in life, which may be more relevant to breast cancer risk. Most studies have focused on factors potentially important in cardiovascular disease (CVD)<sup>16-28</sup> and on dietary factors. Such studies have not fully explored reproductive and hormonal factors, which may be more relevant to breast cancer and other hormonally mediated cancers.<sup>19,23,29,30</sup> The objective of our study was to identify correlates of both active and passive smoking in a large prospective cohort specifically designed to examine cancer risk factors, with a particular focus on breast cancer.

## MATERIALS AND METHODS

### *Data*

Our data come from the California Teachers Study (CTS), a cohort established from respondents to a 1995 mailing to all active and retired female enrollees in the California State Teachers Retirement System (STRS). A total of 133,479 women (approximately 40% of those approached) returned the baseline questionnaire and chose to join the cohort. The cohort is followed annually for cancer incidence through linkage with California's statewide cancer registry and is mailed a follow-up questionnaire biennially to update risk factor information and collect information on factors of emerging interest. Use of the study data was reviewed and approved by the California Health and Human Services Agency Committee for the Protection of Human Subjects. A full description of the CTS is available elsewhere.<sup>31</sup>

We based our analysis on information collected from the 1995 CTS baseline questionnaire. Because the CTS focuses primarily on breast cancer, the baseline questionnaire collected extensive information on hormonal, reproductive, life-style, dietary, and health factors hypothesized to be related to breast cancer etiology. For our analysis, we extracted baseline questionnaire information on personal and life-style characteristics highlighted in current literature as important breast cancer risk predictors. Unfortunately, we could not include data for a few established breast cancer risk factors (e.g., hormone therapy, age at menopause, medical radiation) because this information was not then available for analysis. Although the baseline questionnaire collected no individual-level socioeconomic status (SES) data, we linked the participants' residential street addresses to U.S. census data to derive neighborhood (census block group) SES estimates. We created a summary SES metric based on quartiles of a composite score of occupation, education, and income information for the statewide population.

We measured usual dietary intake during the previous year using an early version of the 1995 Health Habits and History Questionnaire

(HHHQ)<sup>32</sup> and included questions on the consumption frequency and portion size of 103 food and beverage items/groups. We updated the original Block nutrient database using data from various sources.<sup>33–35</sup>

To ascertain active smoking status, the baseline questionnaire asked respondents if they had ever smoked at least 100 cigarettes in their lifetime and if they currently smoke. Respondents answering “yes” to the former question were considered active smokers. We further classified respondents as current smokers, former smokers, or never smokers. Never smokers were separated into two passive smoking exposure categories (ever exposed and never exposed), with passive smoking exposure defined as living with a parent or adult who smoked in the home.

We limited our analysis to respondents with valid tobacco data reported on the baseline questionnaire ( $n = 128,174$ ). We further limited dietary analyses to women whose food consumption self-report we judged to be reasonable (i.e., resulting in a daily caloric intake of between 600 and 5000 calories) ( $n = 122,544$ ).

### Analyses

After reviewing initial cross-tabulations of tobacco exposure distributions, we calculated

prevalence odds ratios (OR) using logistic regression models, adjusting for age and race. When considering dietary characteristics, we further adjusted our models for total caloric intake. We considered micronutrient intake for dietary sources only (i.e., excluding intake from supplements). Patterns of supplement use were considered as a separate variable. To examine correlates of active smoking, we constructed separate models comparing current smokers to never smokers and former smokers to never smokers. Where appropriate, we performed tests for linear trend on the regression coefficients.

## RESULTS

Overall, the prevalence of active smoking in this cohort of women was very low (Table 1). Of the 128,174 women included in our analysis, nearly two thirds were lifetime nonsmokers, and only 5% were current smokers. Among the approximately 85,000 never smoking women, 71% reported having lived with a smoker at some point in their lives.

The distributions of active and passive tobacco exposures varied substantially by age and race/ethnicity (Table 1). Never smokers were more than twice as likely as either current or for-

TABLE 1. DISTRIBUTION OF TOBACCO EXPOSURE BY AGE AND RACE AMONG CALIFORNIA TEACHERS STUDY COHORT MEMBERS WITH VALID TOBACCO DATA ( $n = 128,174$ )

	Total cohort		Active smokers				Never smokers					
			Current smokers		Former smokers		All never smokers		Passive smoking exposure		No passive smoking exposure	
	n	%	n	%	n	%	n	%	n	%	n	%
All women	128,174	100.0	6,487	100.0	36,573	100.0	85,114	100.0	60,343	100.0	24,771	100.0
Age group (years)												
21–30	5,435	4.2	139	2.1	441	1.2	4,855	5.7	2,387	4.0	2,468	10.0
31–39	16,082	12.6	557	8.6	2,507	6.8	13,018	15.3	7,814	12.9	5,204	21.0
40–49	32,379	25.3	1,439	22.2	7,976	21.8	22,964	27.0	16,669	27.6	6,295	25.4
50–59	30,635	23.9	1,895	29.2	10,715	29.3	18,025	21.2	13,829	22.9	4,196	16.9
60–69	22,133	17.3	1,476	22.7	8,273	22.6	12,384	14.5	9,408	15.6	2,976	12.0
70–79	15,096	11.8	804	12.4	4,976	13.6	9,316	10.9	7,011	11.6	2,305	9.3
80+	6,414	5.0	177	2.7	1,685	4.6	4,552	5.3	3,225	5.3	1,327	5.4
Race/ethnicity												
Non-Hispanic white	111,415	86.9	5,664	87.3	33,068	90.4	72,683	85.4	51,764	85.8	20,919	84.4
Black	3,318	2.6	266	4.1	872	2.4	2,180	2.6	1,678	2.8	502	2.0
Hispanic	5,173	4.0	193	3.0	898	2.5	4,082	4.8	2,617	4.3	1,465	5.9
Asian/Pacific Islander	4,394	3.4	133	2.0	758	2.1	3,503	4.1	2,384	3.9	1,119	4.5
Other	3,874	3.0	231	3.6	977	2.7	2,666	3.1	1,900	3.1	766	3.1

mer smokers to be age <40 or > age 79. Compared with current and former smokers, never smokers were more likely to be Hispanic or Asian/Pacific Islander. Passive smoking exposure distributions tended to mirror active tobacco exposure distributions.

### Correlates of active smoking

After adjusting for age and race, active smokers differed from never smokers on a number of potentially important breast cancer risk factors (Table 2). Compared with never smokers, both current and former smokers were more likely to be nulliparous. However, among parous women,

current smokers tended to bear their first child at a younger age than never smokers. This was not true of former smokers. Former smokers were also more likely to report a personal, but not family, history of breast cancer. Current smokers tended to be thinner and exercise slightly less than never smokers, whereas former smokers were similar in body weight to never smokers but tended to exercise more. Current smokers were less likely to receive clinical cancer screening but slightly more likely to perform a monthly breast self-examination. The converse was true for former smokers, who were slightly less likely to perform monthly breast self-examinations but more likely to receive cancer screening. Menarcheal age

TABLE 2. CHARACTERISTICS ASSOCIATED WITH ACTIVE SMOKING STATUS: AGE AND RACE ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION MODELS

Characteristic	Current smoker vs. never smoker		Former smoker vs. never smoker	
	Adjusted OR <sup>a</sup>	95% CI	Adjusted OR <sup>a</sup>	95% CI
Family history of breast cancer	0.99	0.91–1.07	1.02	0.99–1.06
Personal history of breast cancer	0.92	0.81–1.03	1.16	1.09–1.22
Menarcheal age (years)				
≤11	0.93	0.84–1.03	0.95	0.90–0.99
12–14	0.90	0.82–1.00	0.97	0.93–1.02
>14	1.00 (referent)	—	1.00 (referent)	—
Parity				
Parous	1.00 (referent)	—	1.00 (referent)	—
Nulliparous	1.55	1.46–1.64	1.15	1.12–1.19
Age at first full-term pregnancy <sup>b</sup> (years)				
<20	1.00 (referent)	—	1.00 (referent)	—
20–29	0.66	0.59–0.74	0.90	0.84–0.96
≥30	0.52	0.45–0.59	1.02	0.95–1.09
Performs monthly breast self-examination	1.06	1.00–1.12	0.93	0.90–0.95
Screening				
Ever had a Pap smear	0.78	0.65–0.95	1.21	1.09–1.35
Ever had clinical breast examination	0.62	0.53–0.73	1.11	1.01–1.22
Ever had a mammogram <sup>c</sup>	0.54	0.49–0.61	1.20	1.11–1.29
Current physical activity <sup>d</sup> (hours/week)				
Low (≤2.01)	1.00 (referent)	—	1.00 (referent)	—
Medium (>2.01 and <4.92)	0.77	0.72–0.83	1.11	1.07–1.15
High (≥4.92)	0.77	0.72–0.82	1.21	1.21–1.29
Body mass index (kg/m <sup>2</sup> )				
<25	1.00 (referent)	—	1.00 (referent)	—
25–29	0.95	0.89–1.01	1.01	0.98–1.04
≥30	0.76	0.70–0.83	1.00	0.97–1.04
Socioeconomic status (summary metric)				
Quartile 1	1.00 (referent)	—	1.00 (referent)	—
Quartile 2	0.96	0.79–1.17	1.19	1.06–1.33
Quartile 3	0.91	0.75–1.10	1.33	1.19–1.49
Quartile 4	0.79	0.65–0.96	1.46	1.31–1.64

<sup>a</sup>Adjusted for age and race.

<sup>b</sup>Among parous women.

<sup>c</sup>Among women ≥40 years of age.

<sup>d</sup>Based on average strenuous + moderate activity hours/week for the 3 years previous to baseline.

did not differ by active smoking status. Current smokers were least likely, and former smokers were most likely, to be in the highest neighborhood SES quartile. Further adjustment of neighborhood SES models did not substantially change the estimated ORs for the other correlates examined (data not shown).

The dietary habits of current and former smokers compared with never smokers, adjusted for age, race, and caloric intake, are summarized in Table 3. The dietary characteristics of current smokers were substantially different from those

of the never smokers and generally reflected a less healthy diet among current smokers. Compared with lifelong never smokers, current smokers were less likely to consume three or more daily servings of fruit or vegetables and consumed more fat and less fiber, dietary vitamin C, vitamin E, beta-carotene, and phytoestrogens. Furthermore, current smokers were less likely than never smokers to use antioxidant dietary supplements. In comparing the dietary habits of former smokers with never smokers, the differences were smaller and the patterns less consis-

TABLE 3. DIETARY CHARACTERISTICS ASSOCIATED WITH ACTIVE SMOKING STATUS: ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION MODELS

Characteristic	Current smoker vs. never smoker			Former smoker vs. never smoker		
	Adjusted OR <sup>a</sup>	95% CI	Trend p value	Adjusted OR <sup>a</sup>	95% CI	Trend p value
Total energy intake (kcal/day) <sup>b</sup>						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	0.92	0.85–1.00				
Quartile 3	0.91	0.84–0.99				
Quartile 4	0.92	0.85–1.00				
Quartile 5	1.11	1.02–1.20				
Fruit (3 + servings/day vs. ≤2)	0.38	0.34–0.42	n/a	0.85	0.82–0.89	n/a
Vegetables (3 + servings/day vs. ≤2)	0.59	0.53–0.65	n/a	0.96	0.92–1.00	n/a
Fat intake (g/day)						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	1.24	1.13–1.36				
Quartile 3	1.49	1.35–1.64				
Quartile 4	2.04	1.84–2.26				
Quartile 5	3.06	2.69–3.48				
Fiber intake (g/day)						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	0.50	0.46–0.53				
Quartile 3	0.36	0.33–0.39				
Quartile 4	0.25	0.23–0.27				
Quartile 5	0.16	0.14–0.18				
Supplement use <sup>c</sup>						
Nonusers	1.00 (referent)	—	} n/a	1.00 (referent)	—	} n/a
Frequent user of most common antioxidants	0.72	0.68–0.76				
Infrequent user of most common antioxidants	0.89	0.80–0.98				
User of other supplements	0.98	0.89–1.08				
Dietary vitamin C (mg/day)						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	0.63	0.58–0.68				
Quartile 3	0.48	0.44–0.52				
Quartile 4	0.40	0.37–0.43				
Quartile 5	0.31	0.28–0.34				
Dietary vitamin E (mg/day)						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} 0.05
Quartile 2	0.78	0.72–0.85				
Quartile 3	0.71	0.65–0.77				
Quartile 4	0.69	0.63–0.76				
Quartile 5	0.64	0.57–0.71				

TABLE 3. (CONT'D) DIETARY CHARACTERISTICS ASSOCIATED WITH ACTIVE SMOKING STATUS: ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION MODELS

Characteristic	Current smoker vs. never smoker			Former smoker vs. never smoker		
	Adjusted OR <sup>a</sup>	95% CI	Trend p value	Adjusted OR <sup>a</sup>	95% CI	Trend p value
Dietary beta-carotene ( $\mu\text{g}/\text{day}$ )						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	0.73	0.68–0.79				
Quartile 3	0.58	0.54–0.63				
Quartile 4	0.49	0.45–0.54				
Quartile 5	0.37	0.33–0.40				
Phytoestrogens ( $\mu\text{g}/\text{day}$ ) <sup>d</sup>						
Quartile 1	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
Quartile 2	0.99	0.91–1.07				
Quartile 3	0.96	0.88–1.04				
Quartile 4	0.92	0.84–1.01				
Quartile 5	0.84	0.76–0.92				
Alcohol (g/day)						
<5	1.00 (referent)	—	} <0.001	1.00 (referent)	—	} <0.001
5–14.99	1.53	1.44–1.64				
>15	3.65	3.42–3.89				

<sup>a</sup>Adjusted for age, race, and total caloric intake.

<sup>b</sup>ORs not adjusted for total caloric intake.

<sup>c</sup>Supplement user definitions: Nonuser, no regular use (never or less than once a week); Frequent user of most common antioxidant, takes vitamin A/beta-carotene, vitamin C, or vitamin E at least 4 times/week or takes multivitamin at least 4 times/week plus takes vitamin A/beta-carotene, vitamin C, or vitamin E 1–3 times/week; Infrequent user of most common antioxidants, takes vitamin A/beta-carotene, vitamin C, or vitamin E 1–3 times/week; User of other supplements, takes supplements at least once a week but does not take multivitamins, vitamin A/beta-carotene, vitamin C, or vitamin E at all.

<sup>d</sup>Defined as the sum of average daily consumption of genistein, daidzein, formononetin, biochanin A, coumestrol, matairesinol, and secoisolariciresinol.

tent. When compared with never smokers, former smokers were more likely to use antioxidant dietary supplements and have diets high in phytoestrogens, yet their fruit and vegetable consumption did not differ as substantially. The data also suggest that former smokers were less likely than never smokers to have a high-fiber diet but were more likely to have a diet low in fat. Compared with never smokers, alcohol consumption was considerably higher among active smokers, particularly among those who continued smoking.

#### Correlates of passive smoking exposures

Characteristics associated with passive smoking exposures among never smokers are summarized in Tables 4 and 5. After adjusting for age and race, passive smoking exposures were associated with a number of reproductive factors potentially important in breast cancer etiology. Of particular note, passive smoking-exposed women

were more likely to experience menarche before age 12 and were more likely to bear their first child at an early age. The association of passive smoking exposure with earlier menarche (<12 years of age) was limited to those who had lived with a smoker as a child (OR = 1.19, 95% confidence interval [CI] 1.12–1.27) and was less apparent among those who had lived with a smoker only as an adult (OR = 1.08, 95% CI 0.98–1.18) (data not shown). In contrast to active smoking status, passive smoking exposure did not appear strongly associated with neighborhood SES. The dietary characteristics of passive smoking-exposed women suggest a generally less healthy diet compared with those women who have never lived with a smoker. Although more modest, these differences followed the same general pattern as those observed among current smokers.

Table 6 includes a summary of the correlates identified here and how they might confound the relationship between active and passive smoking

TABLE 4. CHARACTERISTICS ASSOCIATED WITH PASSIVE SMOKING EXPOSURE AMONG LIFETIME NEVER SMOKERS: AGE AND RACE-ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION

Characteristic	Exposed to passive smoking <sup>a</sup>	
	Adjusted OR <sup>b</sup>	95% CI
Menarcheal age (years)		
≤11	1.17	1.10–1.24
12–14	1.04	0.98–1.10
>14	1.00 (referent)	—
Parity		
Parous	1.00 (referent)	—
Nulliparous	0.96	0.93–0.99
Age at first full-term pregnancy <sup>c</sup> (years)		
<20	1.00 (referent)	—
20–29	0.60	0.55–0.67
≥30	0.59	0.53–0.66
Performs monthly breast self-examination	1.02	0.99–1.05
Screening		
Ever had a Pap smear	1.13	1.02–1.27
Ever had a mammogram <sup>d</sup>	1.11	1.02–1.21
Ever had a clinical breast examination	1.25	1.13–1.38
Current physical activity (hours/week) <sup>e</sup>		
Low (≤2.01)	1.00 (referent)	—
Medium (>2.01 and <4.92)	1.04	1.00–1.09
High (≥4.92)	1.06	1.02–1.11
Body mass index (kg/m <sup>2</sup> )		
<25	1.00 (referent)	—
25–29	1.14	1.10–1.19
≥30	1.27	1.21–1.33
Socioeconomic status (summary metric)		
Quartile 1	1.00 (referent)	—
Quartile 2	1.00	0.88–1.13
Quartile 3	0.94	0.83–1.06
Quartile 4	0.90	0.79–1.01

<sup>a</sup>Passive smoking exposure, ever lived with a smoker during either childhood or adulthood.

<sup>b</sup>Adjusted for age and race.

<sup>c</sup>Among parous women.

<sup>d</sup>Among women ≥40 years of age.

<sup>e</sup>Based on average strenuous+moderate activity hours/week for the 3 years previous to baseline.

and breast cancer. Table 6 is predicated on assumptions about the relationship between these factors and breast cancer, assumptions we made based on the body of evidence in the literature. For example, the literature suggests that older age at first full-term pregnancy increases the risk of breast cancer. Our analyses revealed that current smokers were less likely to have a first full-term pregnancy at an older age. Therefore, if the relationship between current smoking and breast cancer is examined without adjustment for age at first full-term pregnancy, the relative risk estimate for current smoking (if there is in fact a risk) would likely be underestimated. The results, summarized in this fashion, demonstrate that the

patterns of correlates for passive smokers are remarkably similar to those of current smokers, whereas former smokers seem to have a different constellation of risk factor distributions.

Our initial analyses examined correlates of passive smoking in adulthood and childhood separately. In general, correlation patterns were not distinctly different for childhood and adulthood exposures and, therefore, are not presented here. Two notable exceptions were the association with age at menarche, which was stronger for those exposed only during childhood than for those exposed only during adulthood, and the association with body mass index (BMI), which also was stronger for those exposed only during childhood



TABLE 5. DIETARY CHARACTERISTICS ASSOCIATED WITH PASSIVE SMOKING EXPOSURE AMONG LIFETIME NEVER SMOKERS: ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION

Characteristic	Exposed vs. not exposed to ETS <sup>a</sup>		
	Adjusted OR <sup>b</sup>	95% CI	Trend p value
Total energy intake (kcal/day) <sup>c</sup>			
Quartile 1	1.00 (referent)	—	} <0.001
Quartile 2	1.01	0.96–1.06	
Quartile 3	1.05	1.00–1.10	
Quartile 4	1.05	1.00–1.11	
Quartile 5	1.16	1.11–1.22	
Fruit (3 + servings/day vs. ≤2)	0.84	0.80–0.88	n/a
Vegetables (3 + servings/day vs. ≤2)	0.89	0.84–0.93	n/a
Fat intake (g/day)			
Quartile 1	1.00 (referent)	—	} 0.003
Quartile 2	1.03	0.98–1.08	
Quartile 3	1.04	0.99–1.10	
Quartile 4	1.07	1.00–1.13	
Quartile 5	1.14	1.06–1.24	
Fiber intake (g/day)			
Quartile 1	1.00 (referent)	—	} <0.001
Quartile 2	0.88	0.83–0.92	
Quartile 3	0.80	0.76–0.85	
Quartile 4	0.76	0.72–0.80	
Quartile 5	0.69	0.65–0.73	
Supplement use <sup>d</sup>			
Nonuser	1.00 (referent)	n/a	} n/a
Frequent user of most common antioxidants	0.99	0.95–1.02	
Infrequent user of most common antioxidants	1.00	0.94–1.06	
User of other supplements	0.96	0.90–1.02	
Dietary vitamin C (mg/day)			
Quartile 1	1.00 (referent)	—	} <0.001
Quartile 2	0.88	0.84–0.93	
Quartile 3	0.84	0.80–0.89	
Quartile 4	0.78	0.74–0.82	
Quartile 5	0.72	0.68–0.76	
Dietary vitamin E (mg/day)			
Quartile 1	1.00 (referent)	—	} 0.90
Quartile 2	0.97	0.93–1.02	
Quartile 3	0.96	0.91–1.01	
Quartile 4	0.96	0.91–1.01	
Quartile 5	0.98	0.92–1.05	

*continued*

(OR = 1.22, 95% CI 1.15–1.29) than for those exposed only during adulthood (OR = 1.11, 95% CI 1.03–1.19).

## DISCUSSION

The wealth of conflicting results pertaining to the relationship between smoking and breast cancer<sup>1–4</sup> suggests that such a relationship, if one truly exists, is likely to be fairly complex. The analyses presented here represent the first detailed description of the interrelationships between active and passive smoking exposures and

a comprehensive set of established breast cancer risk factors. Our analyses have identified a number of reproductive, hormonal, and dietary correlates of smoking exposures that underscore the need to adjust for such factors in an analysis of smoking exposures and breast cancer.

The majority of studies published to date on the correlates of smoking have focused on dietary factors.<sup>26,36–61</sup> Our results are generally consistent with the body of literature on such factors. Our analyses of active smokers found that current smokers tended to have less healthy diets consisting of fewer fruit and vegetable servings, lower fiber intake, lower intake of many micronutrients, less frequent use of antioxidant vit-

TABLE 5. (CONT'D) DIETARY CHARACTERISTICS ASSOCIATED WITH PASSIVE SMOKING EXPOSURE AMONG LIFETIME NEVER SMOKERS: ADJUSTED ODDS RATIOS AND 95% CONFIDENCE INTERVALS ESTIMATED FROM LOGISTIC REGRESSION

Characteristic	Exposed vs. not exposed to ETS <sup>a</sup>		
	Adjusted OR <sup>b</sup>	95% CI	Trend p value
Dietary beta-carotene ( $\mu\text{g}/\text{day}$ )			
Quartile 1	1.00 (referent)	—	} <0.001
Quartile 2	0.91	0.86–0.95	
Quartile 3	0.88	0.84–0.93	
Quartile 4	0.83	0.79–0.87	
Quartile 5	0.78	0.74–0.82	
Phytoestrogens ( $\mu\text{g}/\text{day}$ ) <sup>c</sup>			
Quartile 1	1.00 (referent)	—	} 0.05
Quartile 2	0.97	0.93–1.02	
Quartile 3	0.99	0.94–1.04	
Quartile 4	0.98	0.93–1.03	
Quartile 5	0.94	0.89–0.99	
Alcohol (g/day)			
<5	1.00 (referent)	—	} <0.001
5–14.99	1.52	1.46–1.57	
$\geq 15$	1.70	1.61–1.80	

<sup>a</sup>Passive smoking exposure, ever lived with a smoker during either childhood or adulthood.

<sup>b</sup>Adjusted for age, race, and caloric intake.

<sup>c</sup>ORs not adjusted for total caloric intake.

<sup>d</sup>Supplement user definitions: Nonuser, no regular use (never or less than once a week); Frequent user of most common antioxidants, takes vitamin A/beta-carotene, vitamin C, or vitamin E at least 4 times/week or takes multivitamin at least 4 times/week plus takes vitamin A/beta-carotene, vitamin C, or vitamin E 1–3 times/week; Infrequent user of most common antioxidants, takes vitamin a/beta-carotene, vitamin C, or vitamin E 1–3 times/week; User of other supplements, takes supplements at least once a week but does not take multivitamins, vitamin A/beta-carotene, vitamin C, or vitamin E at all.

<sup>e</sup>Defined as the sum of average daily consumption of genistein, daidzein, formononetin, biochanin A, coumestrol, matairesinol, and secoisolariciresinol.

amin supplements, and greater intake of dietary fat and alcohol. This is consistent with a large body of evidence showing that current smokers generally consume less healthy diets than non-smokers.<sup>21,26,36–60</sup> However, most of the previous studies did not separately consider former and never smokers. Of the few studies evaluating dietary characteristics of former smokers, most reported that their habits fell somewhere between those of current and never smokers but were more similar to never smokers.<sup>39,51,59</sup> Two studies found higher fruit and vegetable intake among former smokers, suggesting that smoking cessation may accompany the adoption of other healthy lifestyle changes.<sup>46,61</sup> Our analyses also suggest that respondents who quit smoking may have made some targeted changes toward more healthful diets. Our finding that physical activity levels were highest among former smokers and lowest among current smokers agrees with most previous reports<sup>42,62,63</sup> and is consistent with the

adoption of healthier lifestyle characteristics among former smokers.

Among lifetime never smokers in this cohort, those exposed to passive smoking in the home generally reported less healthy diets. These findings are consistent with the literature.<sup>19–28,30</sup> Heavier body weight associated with spousal passive smoking exposures is well documented, and our findings confirm this relationship.<sup>19,21,24,25,27,30</sup> Our data showed no correlation between passive smoking exposure and any measure of supplement use, in contrast to two previous studies that noted modestly lower prevalences of supplement use among non-smokers exposed to passive smoking compared with those not exposed.<sup>15,23</sup> Our finding that among never smokers, passive smokers reported more cancer screening than their nonexposed counterparts seems inconsistent with the generally less healthy diets observed among passive smokers in this cohort and with previous stud-

TABLE 6. SUMMARY OF POTENTIAL CONFOUNDING EFFECTS OF FACTORS ON RELATIVE RISK ESTIMATES FOR TOBACCO EXPOSURES AND BREAST CANCER

<i>Tobacco exposure</i>	<i>Factors for which failure to adjust could attenuate estimate of tobacco risk</i>	<i>Factors for which failure to adjust could inflate estimate of tobacco risk</i>
Current smoking	Age at first full-term pregnancy Body mass index Socioeconomic status	Fruit and vegetable consumption Dietary fat and fiber intake Vitamin C, E, and beta-carotene intake Use of antioxidant supplements Alcohol consumption Parity
Former smoking	Use of antioxidant supplements Dietary fat intake Dietary phytoestrogen intake Physical activity	Parity Socioeconomic status Alcohol consumption
Passive smoking	Age at first full-term pregnancy	Fruit and vegetable consumption Dietary fat and fiber intake Vitamin C and beta-carotene intake Alcohol consumption Age at menarche Body mass index Parity

ies that have reported that nonsmokers exposed to passive smoking are less likely to participate in preventive health screening.<sup>21,25,64</sup> SES status strongly influences the prevalence of screening. However, there is no evidence of area SES differences between these groups of nonsmoking women in our data.

Because much of the literature on the correlates of smoking has arisen from cardiovascular research, not much is known about how smoking exposures may vary with reproductive and hormonal factors. A 1992 study examining tobacco correlates relevant to cervical cancer reported riskier sexual histories, higher parity, and earlier age at first sexual intercourse among smokers compared with nonsmokers.<sup>44</sup> Our finding of earlier age at first full-term pregnancy is consistent with this finding of earlier sexual experiences among smokers. Our finding that both current and former smokers were more likely to be nulliparous conflicts with the findings by Holly et al.<sup>44</sup> but is consistent with other reports that smoking impairs fertility,<sup>65,66</sup> although our data do not allow us to examine this relationship directly. Nonetheless, the higher prevalence of nulliparity among both former and current smokers may put them at a higher risk than never smokers of developing breast cancer.

Compared to the literature on active smoking, even less is known about hormonal and repro-

ductive factors that may correlate with passive smoking exposures. Among our cohort's never smokers, those exposed to passive smoking experienced menarche at an earlier age (a factor associated with higher breast cancer risk) but were more likely to be parous and to have given birth at an earlier age (factors associated with reduced breast cancer risk). Only one study has examined the effect of passive smoking exposure on menarcheal age. Conducted in 1998, a study of Polish schoolgirls reported a lower mean menarcheal age among girls whose mothers smoked compared with daughters of nonsmoking mothers.<sup>67</sup> This, coupled with our results, suggests a pathway by which passive smoking may affect a woman's risk of developing breast cancer. In our study, the earlier menarche association was limited to women with childhood passive smoking exposures and not to active smokers or those exposed to passive smoking later in life. This is consistent with recent findings that smoking may be related to breast cancer risk only among women who begin smoking during adolescence.<sup>68,69</sup> However, the mechanism by which passive smoking exposure could cause earlier menarche is unclear, particularly given that higher endogenous estrogen levels are associated with earlier menarche and that tobacco exposures are thought to act antiestrogenically.

Our finding that passive smoking-exposed

women were more likely to be parous confirms two previous studies reporting increased parity associated with spousal smoking.<sup>15,30</sup> No other investigation has specifically examined age at first full-term pregnancy, although one study of married Chinese women reported that those who married smoking spouses married at an earlier age.<sup>15</sup> Increased parity is associated with a reduced risk of breast cancer.

There are a number of limitations to our study worth noting. Because the CTS cohort is, to some extent, an occupational cohort, CTS study participants are likely to be more homogeneous than the general population. All members of the cohort have at least a college degree, and all have worked in a public school system. Many also now have or in the past had other occupations. The fact that the prevalence of current smoking in the cohort is much lower than in the general population<sup>20</sup> suggests that the results from this cohort may not be generalizable to the statewide population. However, our results on dietary correlates, for which there is a considerable body of literature, are very consistent with results from other studies. This is reassuring and suggests that the interrelationships between smoking exposures and the hormonal and reproductive factors reported here may also reflect such relationships in the general population. Furthermore, the population of women who constitute the CTS cohort represents the women most at risk for breast cancer (i.e., educated, white, middle-aged women).

The large size of the cohort, coupled with the wealth of information available on potential correlates of interest, allowed us to examine many factors with tremendous statistical power. Although this is one of our study's strengths, it can pose interpretational problems. In drawing conclusions, we focused on the size of estimated ORs rather than the statistical significance of our estimates. This is a subjective undertaking, and not all investigators will agree with our conclusions.

Finally, it should be noted that the passive smoking exposures in our analysis are based on household sources only and do not capture exposures outside the home. This limitation is not unique to our study. Most health-related studies of passive smoking historically have relied on household sources to estimate passive smoke exposures.

A number of the correlates identified here have modest associations with tobacco exposures (ORs <2), although they are of the same order of mag-

nitude as many of the direct effects of these factors on breast cancer.<sup>7</sup> Although these factors, if not taken into account, are likely to bias tobacco-related risk estimates, it is unlikely that they are of sufficient size to fully explain many of the tobacco-related risk estimates reported to date.<sup>70</sup>

In summary, the significantly different risk factor profiles we observed among current, former, and never smokers, as well as the differences noted among never smokers with and without passive smoke exposure, emphasize the importance of separating out these exposure groups when evaluating risks associated with active smoking. Our identification of a number of reproductive and hormonal correlates, which are known risk factors for breast cancer, are especially intriguing and warrant further research. In particular, our finding that childhood passive smoking exposures are associated with a younger menarcheal age suggests a potential pathway by which passive smoking may influence breast cancer risk.

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