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Title

Abstract PO-200: Increased risk of luminal A and HER2-type breast cancer with lifetime cigarette smoking among non-Hispanic Black and White women in the Young Women's Health History Study

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AACR Abstract

TITLE: Increased risk of Luminal A and HER2-type breast cancer with lifetime cigarette smoking among Non-Hispanic Black and White women in the Young Women's Health History Study

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Previous studies have found that tobacco exposure is associated with an increased risk of premenopausal breast cancer (BC) overall and some studies suggest that risk is only increased for Luminal A BC. Few studies have described the association between tobacco exposure and BC risk with characterization of the human epidermal growth factor receptor 2 (HER2) subtype. This analysis explored associations between smoking over the life course and BC risk overall and by BC subtype among a socioeconomically diverse population of young non-Hispanic (NH) Black and White women.

Data were examined from a population-based case-control study in women under 50 years of age, the Young Women's Health History Study. In total, 1,812 women with invasive BC (1,130 NH White, 682 NH Black) and an area-based sample of 1,381 (716 NH White, 665 NH Black) control women, frequency matched to cases by five-year age group, study site and self-reported race were identified from the Los Angeles County and Metropolitan Detroit SEER registry areas. Lifetime smoking histories were collected from in-person interviews. Sample-weighted logistic regression analysis was conducted to estimate the association between lifetime cigarette smoking status (never versus ever smoker) and BC risk adjusted for known BC risk factors and study site. Multinomial logistic regression analysis was conducted for analyses by BC subtype. Heterogeneity in the odds ratio (OR) estimates by BC subtype and cross-product interaction terms of smoking status by race and by household percent poverty (HHP) were evaluated by the Wald test.

In adjusted models, BC risk overall was not significantly associated with ever smoking at least 1 cigarette a day for 6 months or more (OR 1.18; 95% confidence interval (CI) 0.97-1.43). By BC subtype, ever smokers displayed a statistically significant 30% increase in Luminal A BC risk compared to never smokers (OR 1.30; 95% CI 1.03-1.64) and a 90% increased risk of HER2-type BC (OR 1.90; 95% CI 1.17-3.08). Smoking was not associated with risk of Luminal B or Triple Negative BC. Associations between ever smoking and BC risk significantly differed by BC subtype ($P_{heterogeneity}$=0.02).

Statistical interactions by race or by HHP were not observed. However, we noted that in stratified models the association between smoking and risk for HER2-type BC was higher among NH White compared to NH Black women and among women with HHP ≥150% compared to HHP <150%. For Luminal A BC, the association with smoking did not differ by race and risk was higher among women with HHP <150% compared to HHP ≥150%.

Although other studies have identified an association between smoking and Luminal A BC, this may be the first study to identify an association between smoking and hormone receptor negative, HER2-type BC risk. An increased risk for HER2-type BC among NH White women and women with HHP ≥150% was suggested. Research in HER2-type BC is a relatively new and evolving field as HER2 expression was often underreported in the pathology reports of cases diagnosed before 2005.