Inflammatory F2-isoprostane, prostaglandin F2α, pentraxin 3 levels and breast cancer risk: The Swedish Mammography Cohort

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A B S T R A C T

Introduction: Breast cancer is a common cancer among women. Identifying cellular participation of F2-isoprostane, prostaglandin F2α (PGF2α) and pentraxin 3 (PTX3) in cancer we evaluated whether their prediagnostic systemic levels that originate from different inflammatory pathways were associated with breast cancer risk.

Methods: Seventy-eight breast cancer cases diagnosed after blood collection and 797 controls from the Swedish Mammography Cohort were analysed for urinary F2-isoprostane, PGF2α and plasma PTX3 levels.

Results: None of the biomarkers investigated were significantly associated with breast cancer risk. However, there was the suggestion of an inverse association with PTX3 with multivariable adjusted ORs (95% CI) of 0.56 (95% CI=0.29–1.06) and 0.67 (95% CI=0.35–1.28) for the second and third tertiles, respectively (p trend =0.20). No associations were observed between F2-isoprostane (OR=0.87; 95% CI=0.48–1.57; p trend =0.67) and PGF2α metabolite (OR=1.03; 95% CI=0.56–1.88; p trend =0.91) comparing the top to bottom tertiles.

Conclusions: The systemic levels of F2-isoprostane, PGF2α and PTX3 witnessed in women who later developed breast cancer may not provide prognostic information regarding tumor development in spite of their known involvement in situ cellular context. These observations may indicate that other mechanisms exist in controlling cellular formation of F2-isoprostane, PGF2α and PTX3 and their systemic availability in breast cancer patients.

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1. Introduction

Breast cancer is the most common cancer and one of the leading causes of cancer death among women [1–4]. Eicosanoids, especially prostaglandins and isoprostanes are related to the inflammatory and oxidative responses in various diseases including breast cancer [5–7]. F2-isoprostanes are a family of prostaglandin derivatives generated in vivo by free radical-induced peroxidation of arachidonic acid, and a major F2-isoprostane, bioactive 8-iso-prostaglandin F2α (8-iso-PGF2α) is increased in several syndromes and diseases associated with oxidative stress [6,8]. Currently, F2-isoprostanes are regarded as the most reliable biomarker of oxidative stress. Cyclooxygenase (COX)-2 has been shown to be expressed in various cells including tumor cells after stimulation by pro-inflammatory compounds, such as growth factors and cytokines leading to the release of primary prostaglandins [9]. Prostaglandin F2α (PGF2α), a potent prostaglandin that regulates several female hormones is associated with breast
2. Materials and methods

2.1. Study population

2.1.1. Recruitment of study population and sample collection

This study included participants from a case-control study nested within the Swedish Mammography Cohort (SMC), which is a population-based prospective cohort study established from 1987 to 1990. Recruitment and characteristics of this cohort have been previously described [28]. In brief, all women born between 1914 and 1948 from Västmanland and Uppsala counties, Sweden received a questionnaire regarding diet and lifestyle factors (74% response rate). In 1997 a second questionnaire was sent to participants who were still alive and residing in the study area (70% response rate). Completion and return of the self-administered questionnaire was treated as informed consent of study participants. The study was approved by the ethics committee at the Karolinska Institutet, Stockholm.

The participants in the nested case-control study came from the SMC-Clinical, a subcohort of 5022 women under the age of 85 and living in the city of Uppsala, that was established between 2003 and 2009. Blood samples were collected at an in-person health examination and participants completed a questionnaire on diet and lifestyle factors. Venous blood samples were collected after a 12-hour overnight fast. Biological samples were immediately centrifuged and separated in a dark room, and stored at −80 °C. In addition, urine samples were also collected from these subjects and kept frozen at −80 °C until analysis.

2.1.2. Selection of cases and controls

From the SMC-Clinical, 78 cases and 797 controls were included in the study.

We defined our cases as participants who provided a blood and urine samples and had a histologically confirmed incident invasive breast cancer diagnosis after blood collection and before December 31, 2011. Cases were ascertained by linkage of the study cohort with Swedish Cancer Registers. These registers have been estimated to provide almost 100% complete case ascertainment (3). Controls were randomly selected from women without a breast cancer diagnosis who had provided a blood and urine sample.

2.2. Laboratory measurements

Urinary samples were analysed for 8-iso-PGF2α as an index of oxidative injury by a validated radioimmunoassay [29]. Urinary samples were also analysed for 15-keto-dihydro PGF2α, major metabolite of PGF2α, as an index of inflammatory response by a validated radioimmunoassay [30].

Pentraxin 3/TSG-14 (PTX3) was determined by a sandwich ELISA kit (DY1826 from R&D Systems (Minneapolis, MN, USA)). The immunoassays were calibrated against highly purified recombinant PTX3 and had a total coefficient of variation of approximately 6%.

2.3. Statistical analysis

Tertiles of 8-iso-PGF2α, PGF2α metabolite, and PTX3 were determined by the distribution among the controls. Logistic regression was used to estimate OR and 95% confidence intervals (95% CI) for the association between tertiles of each biomarker and breast cancer. In the covariate-adjusted model we adjusted for the following a priori potential confounders: age at blood sampling (continuous), date at blood sampling (continuous), oral contraceptive use (ever, never), hormone replacement therapy use (ever, never), age at menarche (<13, 13, ≥13 years), age at menopause (<51, ≥51 years), parity/age at first birth (nulliparous, age at first birth <26/1–2 children, age at first birth 26–30 years/1–2 children, age at first birth ≥31/1–2 children, age at first birth <26/ ≥3 children, age at first birth ≥26 years/≥3 children), family history of breast cancer (yes/no), history of benign breast disease (yes/no), body-mass index (BMI) at blood sampling (continuous), height (continuous), highest education level (elementary, high school, college or greater), physical activity (<38.8, 38.8–<42.3, 42.3–<45.9, ≥45.9 MET-hours/day), and alcohol intake (continuous). Tests for trend were performed using tertile medians. We also conducted analyses excluding cases diagnosed within one
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