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PELVIC INFLAMMATORY DISEASE AND EPITHELIAL OVARIAN TUMORS

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Abstract
Background Epithelial ovarian cancer and borderline ovarian tumors consist of several histotypes in which high-grade serous carcinoma is the most common. The majority of epithelial ovarian tumors are considered to originate in the fimbriated end of the fallopian tubes. What initiates these tumors is far from completely understood. Pelvic inflammatory disease has been proposed as a modifiable risk factor for epithelial ovarian tumors. A major cause of pelvic inflammatory disease is *Chlamydia trachomatis* which has been shown to have cancer-causing potential. The overall purpose of this thesis was to study associations of pelvic inflammatory disease and *C. trachomatis* with risk of epithelial ovarian tumors. **Methods** In a cross-sectional study (Paper I) we collected ovarian tissue and corresponding blood samples from 69 women undergoing surgery due to suspected ovarian pathology. *C. trachomatis* specific protein (immunohistochemistry) and *C. trachomatis* DNA (qPCR) from ovarian tissue were analyzed (Paper I). In a nested case-control study (Paper II) prospective blood samples from 92 women diagnosed with high-grade serous ovarian cancer were matched to four controls each for age and date of plasma sampling. *C. trachomatis* specific plasma antibodies were analyzed by commercial ELISA and MIF-test (Paper I and Paper II). We performed a nationwide register-based case-control study where we included 15 072 women diagnosed with epithelial ovarian cancer (Paper III), 4 782 women diagnosed with borderline ovarian tumors (Paper IV), and ten controls each matched for age and residential district. Using national Swedish registers, we retrieved data on history of pelvic inflammatory disease and the potential confounding factors parity, educational level, previous gynecological surgery, and hormonal therapy. **Results** We found *C. trachomatis* DNA in ovarian tissue of eight women with ovarian carcinoma, but not in ovarian tissue from women with borderline ovarian tumors or benign disease. The prevalence of the *C. trachomatis* specific protein did not differ in benign and malignant tissue. Prevalence of *C. trachomatis* specific plasma antibodies was similar in cases and controls at diagnosis and prospectively. A history of clinically verified pelvic inflammatory disease was associated with an increased risk of epithelial ovarian cancer and borderline ovarian tumors. Histotype-specific analyses showed an increased risk of serous carcinoma, high-grade serous carcinoma, clear cell carcinoma, and serous borderline ovarian tumors but not significantly with other histotypes. A dose-response relationship was seen between an increased number of pelvic inflammatory disease episodes and epithelial ovarian cancer, as well as borderline ovarian tumors. **Conclusions** This thesis contributes to an improved understanding of the association between pelvic inflammatory disease and epithelial ovarian tumors. The results regarding *C. trachomatis* are inconclusive and suggests that the association of pelvic inflammatory disease with epithelial ovarian tumors acts through mechanisms other than *Chlamydia* alone.

Keywords
Epithelial ovarian tumors, EOC, Borderline ovarian tumors, *Chlamydia trachomatis*, Pelvic inflammatory disease.

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