Table 2.16. Case–control studies of second-hand tobacco smoke and cancer of the ovary

Reference, study location and period	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	(Case/Control) Relative risk (95% CI)*	Adjustment for potential confounders	Comments
Goodman and Tung (2003) USA	558 cases out of 972 eligible (431 invasive malignancies and 127 borderline or low malignant potential tumours); 1993–1999; ≤ 18 yrs; Residency in Hawaii or Los Angeles County for at least 1 yr; No history of cancer of the ovary	607 women; Hawaiian controls randomly selected from lists of female Oahu residents interviewed by Health Surveillance Program of Hawaii Dept of Health; LA controls selected based on neighbourhood walk procedure via computer algorithm on case address; Matched on ethnicity, age, study location, at least one intact ovary	Structure in person interview Hawaii Tumor Registry Los Angeles County Cancer Surveillance Program	Among never smokers with gestational and childhood tobacco smoke exposure *Gestational* No Yes Unknown *Childhood* No Yes *One smoker* Mother Father	 (288/303) 1.0 (ref) (40/39) 1.27 (0.78–2.06) (23/25) 1.08 (0.59–1.98) (130/128) 1.0 (ref.) (221/239) 0.98 (0.72–1.35) (22/20) 1.19 (0.60–2.33) (129/133) 0.99 (0.70–1.41)	Age, ethnicity, education, study site, use of oral contraceptive pill, parity, and tubal ligation	No association of gestational or childhood exposure to second-hand tobacco smoke with risk of invasive or borderline cancer of the ovary among never smokers

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Baker *et al*. (2006) USA (NY) Active and passive smoking and risk of ovarian cancer	434 women with primary epithelian ovarian, peritoneal, or fallopian cancers	868 age and region matched hospital controls with non-neoplastic conditions	Questionnaire	*Smoking status* Never smoked, no ETS exp Never smoked, daily ETS exp *Usual daily ETS exp (h) among never smokers* 0 (reference) < 2 2–8 > 8	*Overall* (127/170) 0.68 (0.47–0.99) (119/300) 0.77 (0.53–1.10) *All ovarian cancers* (n=434) 0.68 (0.47–0.99) *Serous* (*n* = 267) 0.60 (0.38–0.94) *Mucinous* (*n* = 34) 0.71 (0.22–2.32) *Endometrioid* (*n* = 54) 1.02 (0.44–2.39) *Clear cell* (*n* = 28) 0.50 (0.16–1.59) *Borderline* (*n* = 28) 0.92 (0.29–2.96) (127/170) ref. (44/101) 0.68 (0.42–1.10) (49/131) 0.54 (0.29–1.01) (26/68) 0.39 (0.10–1.48) *P* for trend = 0.04	Smoking status adjusted: age, resident in western NY, income, usual BMI, history of vaginal infection, yr of study participation, lifetime duration of breastfeeding, usual daily exposure to second-hand smoke (ETS), and multiplicative interaction between income and ETS exposure Usual daily ETS, Adjusted: age, residence in western NY, income, usual BMI, history of vaginal infection, year of study participation, and lifetime duration of breastfeeding	Similar protective effect noted for smokers with moderate or high exp based on smoking intensity, duration, and cumulative exposure, as well as for never smokers exposed to ETS. Results did not differ substantially by histologic subtype. Although prevailing theories of ovarian cancer etiology implicate incessant ovulation, characteristics of the study population suggest that anovulation was not the protective mechanism in this study. Immunosuppression by nicotine or upregulation of enzymes that metabolize carcinogens may be responsible for the effects observed. Int J Gynecol Cancer 2006;16(Suppl. 1), 211–218