

(ORs) and 95% confidence interval (CIs) were calculated to estimate colon cancer risk by anatomic subsites, using multiple logistic regression models. Interaction between PA and BMI was tested by adding an interaction term in the model.

RESULTS: Colon cancer risk increased significantly with usual BMI among men, with OR = 1.7 (CI = 1.1-2.4) among those in the highest quintile of BMI. The corresponding OR for women was 1.4 (CI = 1.0-2.1), with excess risk largely confined to pre-menopausal women. Risk was inversely associated with PA, regardless of the type of activities. Compared to those with high levels of TPA and OPA, persons with low levels of both activities had substantially elevated risk of colon cancer (OR = 2.8, CI = 1.3-4.1 for men, and OR = 3.9, CI = 1.6-5.1 for women). This association appeared to be more prominent for distal than proximal cancer. In both men and women, risk increased with low PA at each level of BMI, and with high BMI at each level of PA. Compared to those with lowest quintile of BMI and high PA, risk increased to nearly six fold in men (OR = 5.9, CI = 1.9-6.6) and threefold in women (OR = 2.8, CI = 1.6-5.1) with highest quintile of BMI and low PA.

CONCLUSION: High BMI and low PA increased the risk of colon cancer even in the relatively low-risk Chinese population. Risk was further increased with significant effect modification between these two risk factors.

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P007
USING KNOWLEDGE OF DEVELOPMENTAL BIOLOGY TO MINIMIZE CONFOUNDING IN THE STUDY OF MENOPAUSE AND MENINGIOMAS: A POPULATION-BASED STUDY

JF Grutsch, FG Davis, J Propp, B Mccarthy, CG Lis, Office of Research, Cancer Treatment Centers of America® at Midwestern Regional Medical Center, Zion, IL, and Department of Epidemiology and Biostatistics, University of Illinois School of Public Health, Chicago, IL

PURPOSE: To investigate the relationship between menopause and meningioma risk, we utilized knowledge from developmental biology to stratify risk by anatomical location. Results from the Mifepriestine clinical trial question the validity of the hypothesis that ovarian hormones promote the development of meningiomas in women. Moreover, recent discoveries in embryology suggest that the meningeal membranes in the calvarium and skull base are regulated by independent developmental pathways.

METHODS: We evaluated the correlation between age at diagnosis and incidence of meningiomas, using population-based data from the Central Brain Tumor Registry of the United States (CBTRUS). We stratified our results by anatomical location to reflect different developmental pathways regulating embryogenesis, thus minimizing confounding.

RESULTS: Using Linear Regression models and Joint-Point analysis, we found that expected menopausal age is protective for tumors arising in the cerebral hemispheres (the second-degree polynomial T ratio was statistically significantly better fit than the linear model, Wald test $p = 0.0006$), but not in the skullbase (there was no difference between the linear, first and second degree polynomial, Wald Test $p > 0.05$). Paradoxically, female preponderance is much greater in the skullbase.

CONCLUSION: The lack of a protective effect of menopause in skullbase tumors suggests another gender-dependent mechanism of action. This result indicates the plausibility of unrelated oncogenic pathways leading to the development of meningiomas. The traditional ovarian hormonal hypothesis must be carefully reevaluated in future research, however our analysis supports this hypothesis only for calvarium meningiomas. We conclude that the etiology of meningiomas is contingent upon tumor location. Future studies investigating the molecular and cellular mechanisms of meningioma development must take these factors into consideration.

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P008
MENOPAUSAL ESTROGEN THERAPY AND ENDOMETRIAL CANCER IN A US COHORT: RECENCY AND POTENTIAL INTERACTIONS WITH OTHER RISK FACTORS

JV Lacey Jr, LA Brinton, ME Sherman, A Schatzkin, C Schairer, Division of Cancer Epidemiology and Genetics, National Cancer Institute, NIH

PURPOSE: Menopausal estrogen therapy (ET) increases endometrial cancer risk, but certain key aspects of this association are unknown, including whether risk disappears after ET cessation and whether risk is higher in women with lower body mass index (BMI) or in smokers.

METHODS: The NCI's Breast Cancer Detection Demonstration Project Follow-up study included 61,431 women. To analyze data for the 30,509 women who reported a natural menopause and were at risk of endometrial cancer, we excluded women who reported menopause due to surgery, radiation, or unknown reasons, and women who developed endometrial cancer, had a hysterectomy, or died before baseline. We collected ET and other data at up to 6 telephone interviews and 3 mailed questionnaires between 1979 and 1998. Poisson regression with time-dependent variables for ET and smoking produced rate ratios (RRs) with 95 per cent confidence intervals (CIs), adjusted for other known endometrial cancer risk factors.

RESULTS: We identified 541 endometrial cancers but limited this analysis to person-years and cancers among users of no hormone therapy (168 cancers) or only ET (167 cancers). Risk increased with increasing ET duration, and RRs decreased with increasing time since last use, from 10.5 (95% CI, 7.5-14.9) for current use to 1.6 (95% CI, 1.1-2.3) for last use 10 or more years ago. Elevated RRs appeared for long-term ET, regardless of recency, and for current ET, regardless of duration. The RRs for 5 or more years of ET declined across increasing quartiles of BMI, from 13.0 among ≤ 21.3 kg/m², to 16.0 among 21.3-23.2 kg/m², to 7.4 among 23.2-25.8 kg/m², to 2.4 among ≥ 25.8 kg/m². Current smokers had a significantly reduced RR, but RRs for 5 or more years of ET among current, former, and never smokers were 28.5, 4.3, and 6.8, respectively; all CIs excluded 1.0.

CONCLUSION: Endometrial cancer risk decreased with increasing time since last ET use, but remained significantly elevated even 10 years after last use. Lower BMI and current smoking may exacerbate risk associated with ET, but significantly increased risks were not limited to these groups.

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