

Death from cervical cancer is preventable by early screening, and equal access to care ensures equal survival irrespective of race (Farley et al., *Cancer* 91, p869; 2001). Cancer of the cervix is the most common diagnosis among all the malignancies treated at University Hospital in Newark / Essex County, NJ; 90% of patients seen by the Gynecologic Oncology Service live in Newark and Essex County. In 2000, 60% of newly diagnosed cervical cancer cases were African-American and 24% Hispanic, commonly presenting at advanced stages. During the early eighties, funding cuts in citywide preventive health initiatives had markedly reduced Pap screening. Subsequently, cervical cancer cases more than doubled among African-American women, with twice the rate of invasive disease of age-matched Caucasians (Holland et al., *Am J Pub Health* 83, p45; 1992). Also in Essex County, the incidence per 100,000 Caucasian females increased from 8.7 in 1988 to 13.9 in 1992. At the time of detection, confined local disease is distinctly less common in Essex County than at the state or the national level (43% vs. 47% and 52% of all cases, respectively). Conversely, for the period 1992-96 the age-adjusted mortality rate from cervical cancer in Essex County was markedly above state and national averages: 4.8 vs. 2.8 and 2.7, respectively, per 100,000. These findings reflect racial inequities that also register state- and nationwide. During 1990-96 in New Jersey, 72% of cervical cancers in Caucasians were diagnosed while still in situ, compared with only 61% of cervical cancers in African-American and 67% in Hispanic women; the mortality rate per 100,000 was 2.3 in Caucasians, but 6.3 in African-American and 4.1 in Hispanic women. Between 1974 and 1997 in the United States, the disparity in the relative 5-year survival rate of African-American and Caucasian patients has grown more than twofold, increasing in a strictly linear manner with an R2 of 0.97, and is projected to exceed 18% in late 2002. In fact, the relative 5-year survival rate of African-American women actually decreased, from 64% in 1974 to 58% in 1997. The development of these racial disparities in cervical cancer is alarming, and requires targeted interventions by local, state, and federal authorities.

#C213 **Colon cancer: Gender, racial, and socioeconomic differences.** Stephen John Morcwitz, *Stephen J. Morcwitz, Ph.D. & Associates, San Francisco, CA.*

Introduction: Researchers have found gender, racial, and socioeconomic differences in cancer prevalence and cancer survival. One study found that men have a higher prevalence of colon polyps and tumors than women. Another report found that African-Americans have the highest colon cancer incidence among ethnic groups and have poorer 5-year survival compared with Whites. Various investigations attribute some of these differences to socioeconomic status and access to health care. However, more research is needed to quantify the risk factors for colon cancer. **Hypothesis:** The present study tests the following null hypothesis: There are no gender, racial, and socioeconomic differences in colon cancer. **Methods:** Data from the population-based 1998 National Health Interview Survey (N=30,534 adults) were analyzed. Descriptive and correlational procedures assessed possible differences in gender, race, and socioeconomic status in colon cancer. **Results and Conclusion:** The null hypothesis was rejected. Among adult males with family incomes below \$20,000, being African-American was positively associated with having colon cancer ($r = +.266, p < .000, N = 190$) while for adult males with family incomes at or above \$20,000, there was no association between race and having colon cancer. Among adult females with incomes below \$20,000, there was no correlation between race and colon cancer. However, there was a positive association between being African American and having colon cancer among adult females with incomes at or above \$20,000, but it was very low ($r = +.071, p < .05, N = 774$). These differences remained significant after controlling for age and other predictor variables. Colon detection and prevention programs should target African-Americans, especially low-income African-American males and other high-risk groups, in order to reduce colon cancer mortality.

POSTER SESSION C

EPIDEMIOLOGY: Radiation Exposure and Cancer Risk

#C214 **The effects of γ -radiation and cytokines on human peripheral blood mononuclear cells.** Ju Young Kim, Eun Shil Kim, Young Yul Lee and Young Kook Kim. *Seoul National University, Seoul, Korea; Han Yang University Hospital, Seoul, Korea; Seoul National University College of Medicine, Seoul, Korea.*

γ -radiation-induced cell death has been studied extensively in a wide variety of cell types and cell lines. Human peripheral blood lymphocytes irradiated in vitro, die by an apoptotic process. Various cytokines-mediated restoration of hematopoiesis after γ -radiation has been reported. In this study, we investigated in vitro effects of γ -radiation and cytokines on cell proliferation, cell cycle and apoptosis on human PBMCs. Effects of γ -radiation on cell proliferation, cell cycle and apoptosis were investigated by the microtetrazolium (MTT) assay, flow cytometry (FACS) analysis and a Western blot assay for 0, 24, 48, 72 h after irradiation.

The effects of interleukin-3 (IL-3), granulocyte macrophage-colony-stimulating factor (GM-CSF), stem cell factor (SCF), and erythropoietin (EPO) irradiated PBMCs with a fixed dose (2Gy) were determined by clonogenic (colony-forming) assays after 14 days incubation. γ -radiation inhibited the cell proliferation. γ -radiation also induced apoptosis as evidenced by FACS detection of sub-G1 DNA content and annexin V binding assay and caused a loss of mitochondrial membrane potential ($\Delta\psi(m)$), suggesting that apoptosis was triggered by the mitochondrial pathway. Western blot analysis demonstrated that the activation of caspase-3 shown by the cleavage of the proform to the active subunit p17 and the cleavage of poly(ADP-ribose) polymerase (PARP). We also found the increase of Bax, decrease of Bcl-2, activation of Caspase-9 at the protein level in irradiated PBMCs. Following γ -radiation, the number of granulocyte/macrophage colony-forming units (CFU-GM) and colony forming units/erythroblasts (CFU-E) from PBMCs in the presence of IL-3 (10ng/ml) + SCF (50ng/ml) were significantly increased compared to control group. These findings suggest that γ -radiation inhibited cell proliferation and induced apoptosis by the mitochondrial pathway in PBMCs. Treatment of human PBMC with IL-3 + SCF after γ -radiation was synergistic radioprotective effect on hematopoietic cells.

POSTER SESSION C

EPIDEMIOLOGY: Other Lifestyle Factors

#C215 **Reproductive factors and risk of glioma and meningioma.** Elizabeth Hatch, Martha Linet and Peter Inskip. *Boston University School of Public Health, Boston, MA; Division of Cancer Epidemiology and Genetics, National Cancer Institute, Bethesda, MD.*

The etiology of brain tumors is largely unknown and the only confirmed risk factors, high-dose ionizing radiation and certain rare genetic conditions, explain only a small fraction of the incidence. Several lines of evidence, including sex differences in the incidence of glioma and meningioma, the presence of sex steroid receptors in brain tumor tissue, and reports of rapid growth of meningioma during pregnancy, point towards a possible role of reproductive hormones in brain tumor etiology. Whereas the incidence of glioma is higher among males than females, the reverse is true for meningioma. Epidemiologic studies have been sparse and inconsistent, but several have reported a reduced risk of glioma among parous women. Here, we report associations between reproductive factors and the incidence of glioma and meningioma in females, based on a large, hospital-based case-control study conducted between 1994 and 1998 in three U.S. hospitals. A total of 212 cases of intracranial glioma and 151 cases of meningioma were enrolled. Cases were histologically confirmed and interviewed by research nurses in the hospital within 8 weeks of first microscopic diagnosis. The control series (n=436) consisted of female patients hospitalized with a variety of other conditions. Controls were frequency-matched to cases by age, ethnicity, and distance of residence from the hospital. Odds ratios (OR) and 95% confidence intervals (CI) were computed to assess the associations between tumor incidence and age at menarche and menopause, gravidity, parity and age at first birth. Glioma risk increased with older age at menarche (p for trend=0.06). Compared to women who had begun menstruating at age 11 or younger, women whose menarche occurred at age 14 or older had an OR of 1.92 (95%CI=1.08-3.39) of developing glioma. In contrast, there did not appear to be a relationship between age at menarche and risk of meningioma. Age at menopause was unrelated to risk of glioma, but there was a suggestion of increasing risk of meningioma with increasing age at menopause. Risk of glioma was not significantly associated with gravidity or parity, although the associations were in the same direction as reported previously in the literature (OR for ever parous vs. nulliparous=0.85 (95%CI=0.54-1.35). Early age at first birth appeared to be protective (OR=0.43 (95%CI=0.23-0.83) for a first birth <20 vs. being nulliparous), but there was no clear trend in risk for age at first birth greater than twenty. Meningioma cases tended to have more pregnancies and births than controls, but the associations were not significant (OR for ever parous vs. nulliparous=1.33 (95%CI=0.75-2.37). Age at first birth was not related to meningioma risk. Overall, there were no clear associations between reproductive factors and risk of meningioma, but there was a suggestion of reduced risk of glioma associated with early menarche or early age at first birth.