malignancies were classified using the ICD-O (2nd edition) and specified NHL morphologies. The codes queried were only for PCNSL. Data were stratified to those 20–49 years of age and those over 50 years of age. A subgroup analysis reflected the racial groups recognized by SEER. Because minority representation was limited in the initial SEER data from 1973 to 1992, only data for the expanded 13 SEER sites (available beginning in 1992) were used. The age-adjusted incidence rates were calculated using the SEER*Stat program. Rates were age-adjusted to the 2000 U.S. standard million population and reported per 100,000 person-years. Ninety-five percent confidence intervals (CIs) were calculated based on properties of the Poisson distribution. All analyses were restricted to persons age >20 years. RESULTS. Whites had a PCNSL incidence rate of 0.94 per 100,000 per year (95% CI, 0.90–0.98); among Blacks, the incidence was 1.10 per 100,000 per year (95% CI, 0.98–1.22; Table 1). The AI/AN and A/PI subgroups had a much lower PCNSL incidence rate of 0.51 (95% CI, 0.28–0.74) and 0.64 (95% CI, 0.56–0.72), respectively. A subanalysis of patients aged 20–49 years at diagnosis demonstrated that the PCNSL incidence in Blacks (IR=1.43 [95% CI, 1.27–1.59]) was twice that of similarly aged Whites (IR=0.72 [95% CI, 0.68–0.76]). The AI/AN and A/PI subgroups had rates of 0.58 (95% CI, 0.30–0.86) and 0.21 (95% CI, 0.15–0.27), respectively. For those above the age of 50 years, the incidence ratio between the two racial groups was reversed; the incidence of PCNSL was 1.30 (95% CI, 1.22–1.38) in Whites and 0.56 (95% CI, 0.40–0.72) among Blacks. PCNSL incidence was 0.34 (95% CI, 0–0.70) in AI/AN and 1.31 (95% CI, 1.00–1.53) in A/PI. Survival for all races and all ages at 12 months, 2 years, and 5 years was 33%, 25%, and 16% respectively. Among all persons 20 years and older, survival at 12 months, 24 months, and 60 months among Whites was significantly higher than in Blacks. When stratified into groups of those age 20 to 49 years and those age 50 years or greater, only 12-month survival and 5-year survival were statistically different in the two racial groups ($P=0.03$ and $P=0.05$, respectively). CONCLUSIONS. The data presented here are suggestive of a racial influence on incidence and mortality of PCNSL. Implications for research and future directions will be discussed.

EP-02. A COLLABORATIVE STUDY OF THE EPIDEMIOLOGY OF OLIGODENDROGLIAL TUMORS

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BACKGROUND: Oligodendroglial tumors are unique subtypes of brain tumors often combined with other miscellaneous glial tumors during data analysis because of the small numbers in any one study. However, individuals with oligodendroglial tumors have different demographic, clinical, and survival characteristics than individuals with other glial histologies, making it important to evaluate risk factors separately. MATERIALS AND METHODS: We collected data on oligodendroglial tumor cases (oligodendrogliomas, anaplastic oligodendrogliomas, and oligoastrocytomas) from five case-control studies of adult gliomas. Two controls per case were frequency-matched on race, gender, age ± 5 years, and study site. We reviewed the questionnaires from each study, and we determined the variables that were common between studies or variables that could be used to create new common variables. We calculated the univariate statistics, and we estimated the odds ratios (ORadj) and 95% confidence intervals (CI) adjusted for age group, gender, race, study site, and year of interview using logistic regression analysis. RESULTS: Overall, 556 cases, including 298 oligodendrogliomas, 130 anaplastic oligodendrogliomas, and 128 oligoastrocytomas (mixed gliomas), and 1,128 controls were compiled. Fifty-three percent of the study participants were male, 90% were Caucasian, and the average age at diagnosis/interview was 44 years. The gender and race frequencies and mean age at diagnosis/interview were similar between cases and controls. In a preliminary data analysis, anaplastic oligodendroglioma cases were more likely than controls to have reported a family history of brain tumors (ORadj = 3.1 [95% CI, 1.4–6.9]). Cases with anaplastic oligodendrogliomas or oligoastrocytomas were less likely than controls to report a history of asthma or allergies combined (ORadj = 0.4 [95% CI, 0.2–0.6] and 0.3 [95% CI, 0.2–0.7], respectively). An inverse relationship was also found between a history of chicken pox and oligodendroglioma (ORadj = 0.7 [95% CI, 0.4–1.0]) or anaplastic oligodendroglioma (ORadj = 0.5 [95% CI, 0.3–0.9]). Oligodendroglioma cases were less likely than controls to have used bottled water or "other" water source compared to a public water source (ORadj = 0.4 [95% CI, 0.2–1.0] and 0.3 [95% CI, 0.1–0.7], respectively). Epilepsy and/or seizures were associated with an increased risk in all three histology groups, but these may be symptoms of a preclinical disease rather than a risk factor. Results were similar when the data were restricted to cases that underwent a pathology review. CONCLUSIONS: Pooling data from several sources has provided an opportunity to investigate etiologic factors in a rare brain tumor subtype. While several factors associated with oligodendroglial tumors are similar to those identified for the broad glioma grouping, some differences in the relationships by histology subtype were apparent. Additional analyses will be presented.

EP-03. AN EPIDEMIOLOGICAL STUDY OF BRAIN CANCER INCIDENCE AMONG A COHORT OF JET ENGINE MANUFACTURING WORKERS: METHODOLOGICAL ISSUES

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INTRODUCTION: In 2002, the University of Pittsburgh and the University of Illinois at Chicago undertook a 7-year exploratory historical cohort and cancer incidence study to investigate a suspected excess of malignant brain cancers at a jet engine manufacturing plant in North Haven, CT. Our cohort included more than 223,000 former and current employees with work experience since 1952 in one or more than eight manufacturing facilities in CT. We evaluated the incidence of benign and malignant central nervous system (CNS) neoplasms, with emphasis on the brain, from 1976 to 2004 and compared the incidence rates to national and regional incidence rates. Our investigation was complemented by a companion exposure assessment project at the University of IL at Chicago, which will characterize the historical work practices and exposures that occurred in each study plant. Ultimately, we will use the work history and exposure data in the cancer incidence study to examine the relationship between CNS neoplasm incidence and the past working environment of the plants. METHODS: Our cancer incidence tracing protocol entailed matching our cohort with cancer registries from 24 states to find incident CNS cases among our cohort members. This presentation will highlight some of the methodological issues involved with undertaking a cancer incidence study of this scope and magnitude. These issues include differences in the application process, institutional review board procedures, requirements for cohort matching, regulations involving the release of data, data availability, and cost. Specific examples from our study will be given. RESULTS and CONCLUSIONS: The authors will present recommendations for improving cancer incidence tracing in the United States to ultimately allow epidemiologists and clinicians greater, and more simplified, access to valuable cancer incidence data. In a later phase of this study, the results of the cancer incidence tracing for the jet engine manufacturing cohort will be presented.

EP-04. GENDER, BIRTH SEASONALITY, AND BIRTH DEFECTS AS RISK FACTORS FOR PEDIATRIC BRAIN TUMORS: A CALIFORNIA CANCER REGISTRY STUDY

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BACKGROUND: Birth characteristics have been studied as possible risk factors for pediatric brain tumors. Seasonality has also been examined as a proxy for infectious exposure resulting in pediatric brain tumors. In addition, birth defects are thought to be associated with an increased risk for pediatric cancer because of their relationship to genetic syndromes. Studies done in Europe and North America have found that children with birth defects have a 2-fold increase in childhood malignancies, especially in blood and central nervous system (CNS) tumors. Other epidemiologic studies have addressed gender as a risk factor for pediatric brain tumors. However, the majority of these studies have had limited power and did not distinguish between the tumor subtypes. METHODS: We obtained data on 4,070 cases of CNS cancers from the population-based California Cancer Registry. Cases were children aged 0–14 years who were diagnosed between 1988 and 2004 and matched via probabilistic record linkage to a California birth certificate. Four controls were selected for each case from state birth files and matched based on gender and birth year. Cases and controls were screened for noted morphologic defects at birth as recorded on the birth certificates. For the seasonality analyses, we compared the distribution of birth months to the distribution of all births in California in 1998 (n=523, 410 births). RESULTS: Overall, males had more CNS tumors than females (male:female 1.21, $P<0.0001$).When stratified by tumor subtype, males had a higher incidence of germ cell tumors (145 males:69 females 2.1, $P<0.0001$) and medulloblastomas (331 males:215

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