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DIFFERENTIAL RISK OF MALIGNANT MELANOMA BY SUNBED EXPOSURE TYPE. *MA Papas, AH Chappelle, (Chappelle Toxicology Consulting, Chadds Ford, PA), and WB Grant (Sunlight, Nutrition and Health Research Center, San Francisco, CA)

Background: A 2006 International Agency for Research on Cancer (IARC) meta-analysis reported a weak positive association between sunbed use and cutaneous malignant melanoma (meta-odds ratio = 1.2, 95% confidence interval: 1.0, 1.3). The lack of detailed measurement of sunbed usage is a key limitation of this meta-analysis. Distinct differences exist between unsupervised use of home sunbeds, regulated usage of professional salon sunbeds, and sunbeds used by doctors as medical devices. The resulting misclassification may bias reported estimates for overall sunbed exposure and risk of melanoma. Methods: We abstracted additional data from the 19 studies identified by the IARC meta-analysis. Sunbed exposure was classified with three alternative categories not considered by the published meta-analysis: home, professional salon, and medical office. Summary odds ratios (OR) and 95% confidence intervals (CI) were computed by pooled analysis.Results: Five studies had data available on the type and setting of tanning bed usage most commonly reported. The pooled OR for ever-use of home sunbeds was 1.4 (95% CI: 1.2, 1.7); while that for ever-use of sunbeds in a professional salon was 1.1 (95% CI: 0.9, 1.2); and that for medical office sunbeds was 2.0 (95% CI: 0.9, 4.3).Conclusion: Detailed exposure information is a critical limitation for observational studies of sunbed usage. The reported association between sunbed usage and risk of melanoma may be biased by exposure misclassification. When professional sunbed usage is considered independent of home and medical exposures there is no association with melanoma.

1004-S

COMPARISON OF SELF AND REGISTRY-REPORTED RACE AND ETHNICITY AMONG CHILDREN WITH LEUKEMIA. *Karen Bartley[1], Monique Does[1], Steve Selvin[1], Peggy Reynolds[2], Patricia Buffler[1] ([1]University of California, Berkeley, School of Public Health, Berkeley, CA; [2]Cancer Prevention Institute of California, Berkeley, CA)

Background: Childhood leukemia incidence, derived from cancer registry, differs up to 4-fold by race/ethnicity. Registry-based ethnicity is classified using medical records which may differ from self-report. This study compares self-report race/ethnicity to registry classification to determine whether observed higher childhood leukemia in some ethnic groups can be explained by misclassification. To date, no similar study has been reported for any pediatric cancers.Methods: We compared parent-reported race/ethnicity of 972 case participants of the California Childhood Leukemia Study, to that reported in the California Cancer Registry (CCR). Concordance was assessed using sensitivity, specificity, positive predicted value, and kappa coefficients ($\kappa$). Predictors of registry misclassification by race/ethnicity were assessed using loss-based cross validation methods. Results: Concordance was high for Hispanics ($\kappa$ = 0.85) and for Asian Pacific Islander (API), Black, or White race ($\kappa$ = 0.76, 0.73, and 0.75 respectively), and low for Native Americans ($\kappa$ = 0.17) and children of mixed race ($\kappa$ = 0.10). For Hispanic, Black and Native American children, misclassification was not predicted by any of the characteristics assessed; among White, API, and children of mixed race, father's race and country of origin were significantly predictive. Conclusions: Race/ethnicity in the CCR reflects self-reported race/ethnicity, with the exception of Native American and children of mixed race, among children with leukemia. Misclassification is unlikely to account for observed differences in incidence by race/ethnicity.

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EXPOSURE TO MULTIPLE PESTICIDES AND RISK OF NON-HODGKIN LYMPHOMA IN MEN FROM SIX CANADIAN PROVINCES. *K Hohenadel, SA Harris, JM McLaughlin, JJ Spinelli, P Pahwa, JA Dosman, PA Demers, AE Blair (Occupational Cancer Research Centre, Toronto)

A number of individual pesticides have been linked to non-Hodgkin lymphoma (NHL) with variable consistency. However, the impact of exposure to multiple pesticides has not been well studied. Data from a six-province Canadian case-control study conducted between 1991 and 1994 were analyzed to investigate the relationship between NHL and exposure to: (a) the total number of insecticides, herbicides, and fungicides used; (b) the number of potentially carcinogenic pesticides used; and (c) commonly used pesticide combinations. Cases (n=513) were identified through provincial cancer registries and controls (n=1506), frequency matched by age and region, were obtained through provincial health records, telephone listings or voter lists. In multiple logistic regression analyses, risk of NHL tended to increase with the number of pesticides used. Participants reporting exposure to a single pesticide were not at increased risk of NHL (odds ratio [OR]=0.80, 95% confidence interval [CI]=0.44–1.47), while those exposed to two to four (OR=1.39, CI=1.02–1.91) or five or more pesticides (OR=1.63, CI=1.20–2.21) were at greater risk. Similar results were obtained in analyses restricted to herbicides and insecticides. Odds ratios increased further when only pesticides designated as potentially carcinogenic by the International Agency for Research on Cancer were considered (OR[1 pesticide]=1.30, CI=0.90–1.88; OR[2 to 4]=1.54, CI=1.11–2.12; OR[5 or more]=1.94, CI=1.17–3.23). Since exposure to multiple pesticides is common among commercial applicators and agricultural workers these results underscore the importance of not restricting our assessment of cancer risk to single exposures.

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ASSOCIATION BETWEEN TIMING OF ADJUVANT TREATMENT AND SURVIVAL: A POPULATION-BASED STUDY OF COLORECTAL CANCER PATIENTS IN ALBERTA. *Lima I., Yasui Y., Scarfe A., Winget M. (University of Alberta, Edmonton, Alberta)

Surgery followed by adjuvant treatment has been the treatment recommendation for stage III colon cancer and stage II/III rectal cancer since 1990. Clinical trials have not assessed the time by which adjuvant treatment should be started relative to surgery for optimal survival benefit. Residents of Alberta diagnosed with stage III colon cancer and stage II/III rectal cancer in 2000-2005 who had surgery were included in the study. Patients were identified from the Alberta Cancer Registry and linked to hospital data and data from the 2001 Canadian Census. Cox proportional hazards models were used to estimate hazard ratios (HR) of death by the timing of adjuvant treatment. A total of 2,332 patients were included in the study. Stage III colon cancer patients who received adjuvant chemotherapy 12-16 weeks after surgery or more than 16 weeks/no treatment had a 43% and 107% higher risk of dying compared to those treated within 8 weeks of surgery (HR=1.43, 95% confidence interval (CI) 0.96-2.13 and HR=2.07, 95%CI 1.56-2.76, respectively). Similarly, stage II/III rectal cancer patients who received adjuvant treatment more than 12 weeks after surgery or did not receive it had a 40% and 60% higher risk of rectal cancer death compared to those who received it within 8 weeks. Analyses were controlled for age, year, and region of residence at diagnosis; sex; neighbourhood-level socioeconomic factors; and number of co-morbidities. The results of this study are consistent with current guideline recommendations in Alberta. Adjuvant treatment for patients with stage III colon cancer and stage II/III rectal cancer should be initiated within 12 weeks after surgery to maximize treatment benefits.