

Abstract #: 2717

Parental Tobacco Smoking and the Risk of Acute Myeloid Leukemia in Children: the Childhood Leukemia International Consortium (CLIC).

C. Metayer, PhD¹, E. Roman, PhD², E. Petridou, PhD³, J. M. Mejía Aranguré, PhD⁴, J. Schüz, PhD⁵, C. Magnani, PhD⁶, A. M. Mora, MD⁷, B. Mueller, PhD⁸, S. Koifman⁹, J. Dockerty, PhD¹⁰, T. Lightfoot, PhD², E. Hatzipanatelis, MD¹¹, J. Rudant, PhD¹², J. Flores-Lujano, PhD⁴, P. Kaatsch, PhD¹³, L. Miligi, PhD¹⁴, C. Wesseling, PhD⁷, D. R. Doody, MS⁸, M. S. Pombo-de-Oliveira, MD¹⁵, A. Y. Kang, MPH¹, K. McCauley, BS¹ and J. Clavel, PhD¹²

¹University of California, Berkeley, CA, USA, ²University of York, York, United Kingdom, ³National and Kapodistrian University of Athens, Athens, Greece, ⁴Unidad Médica de Alta Especialidad Hospital de Pediatría del Centro Médico Nacional, Mexico City, Mexico, ⁵International Agency for Research on Cancer (IARC), Lyon, France, ⁶Università del Piemonte Orientale, Novara, Italy, ⁷Universidad Nacional, Heredia, Costa Rica, ⁸University of Washington, Seattle, WA, USA, ⁹Environmental and Public Health Program, National School of Public Health, Rio de Janeiro, Brazil, ¹⁰University of Otago, Otago, New Zealand, ¹¹Aristoteles University of Salonica, Salonica, Greece, ¹²Université Paris-Sud, Villejuif, France, ¹³University Mainz, Mainz, Germany, ¹⁴ISPO Cancer Prevention and Research Institute, Florence, Italy, ¹⁵Instituto Nacional de Câncer (INCA), Rio de Janeiro, Brazil

INTRODUCTION: Leukemia is the most common cancer in children <15 years of age. Acute myeloid leukemia (AML) accounts for approximately 17% of childhood leukemia. Tobacco smoke contains carcinogens known to damage somatic and germ cells, and its association with adult AML is well established. However, due to the rarity of AML, few studies have examined the relationship of

tobacco smoking to childhood AML. To overcome this limitation, we pooled data from case-control studies in Oceania, Europe and North, Central, and South Americas participating in the Childhood Leukemia International Consortium (CLIC).

METHODS: Original data on parental cigarette smoking before and after birth, and socio-demographic characteristics were obtained from 13 studies conducted from 1974 to 2012, leading to 1333 AML cases and 13,173 controls. Information on the number of cigarettes smoked/day was available for 84%. We used unconditional logistic regression to estimate odds ratios (OR) and 95% confidence intervals (CI), adjusted for age, sex, ethnicity, parental education, and study center.

RESULTS: Smoking habits were stable over time for fathers and to a lesser extent for mothers. AML was associated with paternal lifetime smoking (OR = 1.35, 95% CI = 1.11–1.63), with a dose-response relationship observed for smoking before conception and during pregnancy (ORs for both periods = 1.12 for every 10 cigarettes/day, *p* for trend = 0.06), but not after birth (OR = 1.04, 95% CI = 0.92–1.18, *p* for trend = 0.12). The ORs for maternal smoking before conception, during pregnancy and after birth were null.

CONCLUSIONS: Our analysis based on the largest study of childhood AML to date supports an association between AML and paternal smoking. The high correlation between pre- and post-natal smoking limits our ability to identify critical windows of exposure; however, the lack of associations with maternal smoking at any time before and after birth, suggests that paternal smoking before conception plays a role in the etiology of childhood AML.