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Maternal Epidemiology of Brachial Plexus Birth Injuries in California: 1996-2012

Mary Claire Manske¹, Machelle D. Wilson², Barton L. Wise³, Joy Melnikow⁴, Herman L. Hedriana⁵, Michelle A. James^{6,7} and Daniel J. Tancredi⁸

¹University of California Davis; ²Principal Biostatistician, Clinical and Translational Science Center, Department of Public Health Sciences, Division of Biostatistics, University of California Davis, Sacramento, California, United States; ³Professor, Department of Internal Medicine, University of California Davis, Sacramento, California, United States; ⁴Professor Emeritus, Department of Family and Community Medicine, University of California Davis, Sacramento, California, United States; ⁵Professor and Chief, Department of Obstetrics and Gynecology, Division of Maternal-Fetal Medicine University of California Davis, Sacramento, California, United States; ⁶Professor, Department of Orthopaedic Surgery, University of California Davis, Sacramento, California, United States; ⁷Department of Orthopaedic Surgery, Shriners Hospitals for Children Northern California, Sacramento, California, United States and ⁸Professor in Residence, Department of Pediatrics, University of California Davis, Sacramento, California, **United States**

OBJECTIVES/GOALS: To evaluate the incidence of brachial plexus birth injury (BPBI) and its associations with maternal demographic factors. Additionally, we sought to determine whether longitudinal changes in BPBI incidence differed by maternal demographics. METHODS/STUDY POPULATION: We conducted a retrospective cohort study of over 8 million maternal-infant pairs using California's Office of Statewide Health Planning and Development Linked Birth Files from 1991-2012. Descriptive statistics were used to determine BPBI incidence and the prevalence of maternal demographic factors (race, ethnicity, age). Multivariable logistic regression was used to determine associations of year, maternal race, ethnicity, and age with BPBI. Excess population level risk associated with these characteristics was determined by calculating population attributable fractions. RESULTS/ ANTICIPATED RESULTS: The incidence of BPBI between 1991-2012 was 1.28 per 1000 live births, with peak incidence of 1.84 per 1000 in 1998 and low of 0.9 per 1000 in 2008. Incidence varied by demographic group, with infants of Black (1.78 per 1000) and Hispanic (1.34 per 1000) mothers having the highest incidences. Controlling for relevant covariates, infants of Black (AOR=1.88, 95% CI 1.70, 2.08), Hispanic (AOR=1.25, 95% CI 1.18, 1.32) and advanced-age mothers (AOR=1.16, 95% CI 1.09, 1.25) were at increased risk. Disparities in risk experienced by Black, Hispanic, and advanced-age mothers contributed to a 5%, 10%, and 2% excess risk at the population level, respectively. Longitudinal trends in incidence did not vary among demographic groups. Population-level changes in maternal demographics did not explain changes in incidence over time. DISCUSSION/ SIGNIFICANCE: Although BPBI incidence has decreased in California, demographic disparities exist. Infants of Black, Hispanic, and advanced-age mothers are at increased BPBI risk compared to White, Non-Hispanic, and younger mothers.

Unlocking the Potential of Simalikalactone D as an Anticancer Agent in Ethnically Diverse Breast Cancer Populations

Annelis O. Sanchez-Alvarez¹, Prof. Pablo E. Vivas-Mejia² and Prof. Claudia Ospina³

¹Comprehensive Cancer Center-UPR; ²Medical Science Campus-UPR and ³Inter American University of Puerto Rico, Bayamon

OBJECTIVES/GOALS: This project focuses on investigating the potential of Simalikalactone D (SKD) as an anticancer agent, exploring the mechanisms underlying SKD's induction of cell death, and assessing the impact of SKD on diverse breast cancer cell lines. Also, it Investigates the compound's mechanisms of action beyond caspase 3-dependent pathways. METHODS/STUDY POPULATION: Three breast cancer cell lines were used: SKBR3, MDA-MB-231, and MDA-MB-468. Two triple-negative breast cancer cell lines are included to address cancer disparities across diverse ethnic backgrounds. Viability assays were conducted to determine half-maximal inhibitory concentrations (IC50). Caspase 3 activity assay was performed to evaluate apoptosis as a possible cell death pathway. Wound healing and colony formation assays are used to assess cell migration and clonogenic capacity. Proteomic analysis and phosphoarray analysis are planned for a deeper understanding of SKD's anticancer properties, as well as testing for caspase 3 independent pathways. RESULTS/ANTICIPATED RESULTS: SKD demonstrated substantial cytotoxicity against all three breast cancer cell lines. IC50 values for SKBR3, MDA-MB-231, and MDA-MB-468 were 60.0 nM, 65.0 nM, and 116 nM, respectively. SKD induces cell death via caspase 3-independent pathways. Further experiments are needed to confirm and elucidate the molecular pathways being impacted. SKD inhibited cancer cell migration and clonogenic potential, suggesting it can reduce tumor growth and metastatic tendencies. DISCUSSION/SIGNIFICANCE: The study highlights SKD's cytotoxicity across diverse breast cancer cell lines. It underscores the mechanism of action, a caspase 3 independent pathway. These findings hold promise for the development of innovative anticancer treatments and emphasize the importance of exploring varied cellular responses to mitigate global cancer disparities.

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¹OHSU and ²Portland State University

OBJECTIVES/GOALS: To truly improve health equity and accessibility, we must develop a diverse and inclusive workforce. The BUILD EXITO program developed as a collaboration between a network of undergraduate programs and a CTSA hub and now has become a sustainable resource that will outlive NIH funding. We will disseminate our successful model. METHODS/STUDY POPULATION: The BUILD EXITO program has completed 10 years of NIH funding, a partnership between OCTRI and