Background and Aims: Arsenic (As) is a common environmental toxicant to which millions of people are exposed via contaminated drinking water. Arsenic is linked to increased risk of infectious diseases and cancer, but its mechanism of action is uncertain. The immune system, especially T cell function (and specifically T regulatory cells (Treg), which play a critical role in suppressing inflammation), may be an important mediator of As toxicity.

Methods: The study will use prospectively collected data from a New Hampshire cohort of pregnant women and their infants to assess whether in utero As exposure (maternal pregnancy urine As) is associated with: 1) impaired T cell and Treg function in infant cord blood, and 2) decreased immune indicators of placental tolerance. We will 3) conduct exploratory analyses of cord blood T cell and Treg function as predictors of enhanced risk of childhood infections and atopic disorders, and 4) test the feasibility of assessing the relation of As with vaccine response, Treg numbers, and atopy (including IgE levels) in 1 year olds.

Preliminary Results: To date we have 213 cord blood and 162 placenta samples, with the goal of collecting at least 250 of each. The mean pregnancy urinary As concentration (excluding arsenobetaine) is 6.7 (range 0.4 – 288) ug/L. In preliminary analyses of cryopreserved cord blood lymphocytes from 16 newborns, the correlation between log(urine As) and T cell proliferation is 0.63 (p=0.01) and for Treg function -0.33 (p=0.21).

Conclusions: Our preliminary findings support a potential role of in utero As exposure in altering newborn T cell, including Treg, function. With complete data, we will more fully address the impact of As on immune markers in cord blood and placenta to help inform whether As-related changes in these immune markers affect the risk of childhood infectious and allergic diseases.