

ABATEMENT OF TYPE 1 DIABETES AS A RESULT OF POLYCHLORINATED BIPHENYL (PCB)-153 EXPOSURE IN THE NON-OBESE DIABETIC (NOD) MOUSE MODEL

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Type 1 diabetes (T1D) is an autoimmune disorder characterized by the T-cell mediated destruction of insulin-producing β -cells in the islets of Langerhans in the pancreas. The steady increase in prevalence and incidence of T1D across the globe suggests disease onset may be contributed by various factors, other than genetics. One such factor is environmental pollutants. Given that polychlorinated biphenyl (PCB)-153 is a highly abundant pollutant in both the environment and mammalian tissues, there is reason to believe the compound may be an environmental factor influencing disease susceptibility and onset. Non-obese diabetic (NOD) mice were exposed to PCB-153 in a 10-day acute (50mg/Kg or 0.5mg/Kg) or 16-week chronic (12.5mg/Kg or 0.125mg/Kg) fashion, by intraperitoneal injections. Analysis of various immune parameters, including: immunophenotyping, T-cell proliferative response, and cytokine analysis revealed that both acute and chronic exposure to PCB-153 caused significant immunosuppression in all PCB-153 exposed mice. Based on the significant decreases in CD4⁺ T-helper cells observed in immunophenotyping and interleukin (IL)-2 during cytokine analysis, it is plausible to believe that T-helper 1 (T_{H1}) cells are the most susceptible cell population to PCB-153 exposure. This is further supported by the decrease in T1D incidence observed in mice chronically exposed to either dose of PCB-153. These findings give important implication on the protective effects of PCB-153 exposure on T1D, primarily through immunosuppression of pathogenic cell subsets.