

17. Rheumatic and Non-Rheumatic Autoimmune Diseases in SLE Offspring

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Autoimmune diseases (AID) have familial aggregation and frequently share a common genetic predisposition. Only few small uncontrolled studies have evaluated the risk of AID in SLE offspring, with inconsistent results. In a large population-based study, we aimed to determine if children born to mothers with SLE have an increased risk of rheumatic and non-rheumatic AID compared to children born to mothers without SLE.

The "Offspring of SLE mothers Registry (OSLER)" includes all women who had ≥ 1 hospitalization for delivery after SLE diagnosis, identified through Quebec's universal healthcare databases (1989-2009). OSLER also includes a randomly selected control group of women, matched at least 4: 1 for age and year of delivery, who did not have a diagnosis of SLE prior to or at the time of delivery. We identified children born live to SLE mothers and their matched controls, and ascertained rheumatic (i.e. juvenile idiopathic arthritis, SLE, systemic sclerosis, Sjögren's disease, inflammatory myositis, systemic vasculitis) and non-rheumatic (i.e. type 1 diabetes, inflammatory bowel disease, psoriasis, celiac disease, autoimmune thyroid disease, myasthenia gravis, multiple sclerosis) AID based on ≥ 1 hospitalization or ≥ 2 physician visits with a relevant diagnostic code, at least 2 months apart but within 24 months. We performed multivariate analyses to adjust for maternal age, education, and ethnicity, as well as calendar year of birth and sex of the child.

509 women with SLE had 719 children, while 5824 matched controls had 8493 children. Mean follow-up was 9.1 (SD 5.8) years. Compared to controls, children born to mothers with SLE had similar records of rheumatic diagnoses [0.14% (95% CI 0.01, 0.90) vs 0.19% (95% CI 0.11, 0.32)]. However, there was a trend towards more non-rheumatic AID in offspring of mothers with SLE versus controls [1.11% (95% CI 0.52, 2.27) vs 0.48% (95% CI 0.35, 0.66)].

In multivariate analyses, children born to mother with SLE had a substantially increased risk of non-rheumatic AID compared to controls (OR 2.62, 95% CI 1.10, 6.24), while results were inconclusive for the risk of rheumatic AID (OR 0.78, 95% CI 0.10, 5.92).

These novel data suggest that, compared to children from the general population, children born to women with SLE have an increased risk of non-rheumatic AID. Our effect estimate for the risk of rheumatic AID is inconclusive. Further study of these children, throughout late childhood, adolescence, and adulthood, would be additionally enlightening.