health record (EHR) is a powerful tool to capture coded diagnoses at a population level, accurately identifying SLE births is challenging. Our objective was to develop and externally validate algorithms for identifying births to SLE patients.

**Methods** We used two EHR-based datasets: Vanderbilts Synthetic Derivative and Dukes Clarity. Potential cases had at least 1 SLE code (ICD-9: 710.0 or ICD-10:M32.1*, M32.8, M32.9) and at least 1 ICD-9 or ICD-10 code for pregnancy-related diagnoses. At Vanderbilt, 100 potential cases were randomly selected for chart review and each classified as a case if SLE was diagnosed by a rheumatologist, nephrologist, or dermatologist. Using this dataset, positive predictive values (PPVs) and sensitivity were calculated for combinations of counts of SLE ICD-9 or ICD-10 codes provided by any clinician and by a rheumatologist (rheumatology coded), antimalarial use, positive ANA, and checked lupus labs (dsDNA, C3 or C4). F-score measured the performance of each algorithm. At Duke, potential cases were compared with the Duke Autoimmunity in Pregnancy Registry; cases outside of this registry underwent chart review. Vanderbilt served as a training set; Duke served as validation.

**Results** From Vanderbilts 2.8 million subject records, we identified 433 potential cases. Of the 100 cases randomly selected for chart review, 39 had confirmed SLE and a history of a birth. Of Dukes 659 potential cases, 545 were included in a validation set of which 208 had confirmed SLE. In the training set, algorithms with ICD-10 codes had higher PPVs than algorithms with ICD-9 codes (table 1). The algorithm with the highest F-score of 88% was 4 counts of ICD-9 or ICD-10 codes and checked lupus labs. Algorithms validated well in the Duke dataset. In the validation set, 1 ICD-9 or ICD-10 code (by a rheumatologist) performed best (F-score: 82%).

**Conclusions** We have developed and validated algorithms to detect SLE patients with births in the EHR. The highest performing algorithms use SLE ICD-9 or ICD-10 codes and clinical parameters or ICD-10 codes alone. Algorithms using more SLE coded visits have greater PPVs at a cost to sensitivity. While the PPV and sensitivity nears 90%, EHR cohorts remain complementary to prospective cohorts. However, in the era of big data, developing methods to identify SLE births accurately is critical to examine adverse outcomes such as preterm births.

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## 52 CONTACT DYNAMICS BETWEEN MESENCHYMAL STEM CELLS AND T CELLS IN LUPUS-PRONE MRL/LPR MOUSE MODEL

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**Background** Systemic lupus erythematosus (SLE) is a multiorgan autoimmune disease characterized by autoantibody production and mesenchymal stem cells (MSCs) have emerged as a promising new therapy for the treatment of SLE. MSCs are adult stem cells isolated from various human tissues including bone marrow, adipose tissue, umbilical cord blood, and skeletal muscle; MSCs can differentiate into various cell types and can potentially replace damaged cells *in vivo*. MSCs suppress T cell proliferation and cytokine production, reduce B cell proliferation and antibody secretion, decrease the generation and function of dendritic cells, and reduce the activity of natural killer cells. MSCs also enhance the activity of regulatory T (Treg) cells. MSCs are thought to inhibit T cell functions by two different mechanisms: by producing soluble mediators and by direct cellcell contacts. The soluble immunosuppressive factors produced by MSCs include IL-10, nitric oxide (NO), tumor growth factor (TGF)-, prostaglandin E2 (PGE2), and indoleamine 2,3-dioxygenase (IDO), all of which can inhibit the functions of major immune cells. Yet, much remains to be learned about the contact-dependent T cell inhibition by MSCs.

**Methods** We examined the *in vivo* efficacy of MSCs in lupus-prone MRL/lpr mouse model and examined how MSCs inhibit MRL/lpr T cells by using time-lapse imaging at the single level.

**Results** In this study, we show that transfer of human MSCs increased MRL/lpr mouse survival, decreased T cell infiltration in the kidneys, and reduced T cell cytokine expression. *In vitro*, allogeneic mouse MSCs inhibited MRL/lpr T cell proliferation and cytokine production. Time-lapse imaging revealed that MSCs recruited MRL/lpr T cells establishing long-lasting cellular contacts by enhancing T cell VCAM-1 expression in a CCL2-dependent manner. In contrast, CCL2 deficient MSCs did not induce T cell migration and VCAM-1 expression, resulting in insufficient cell-cell contact. Consequently, CCL2 deficient MSCs did not inhibit IFN- production by T cells and upon transfer no longer prolonged survival of MRL/lpr mice.

**Conclusions** Taken together, our imaging study demonstrates that CCL2 enables the prolonged MSC-T cell interactions needed for sufficient suppression of autoreactive T cells and helps to understand how MSCs ameliorate symptoms in lupus-prone MRL/lpr mice.

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## 53 HEALTH-RELATED QUALITY OF LIFE IN TUNISIAN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOUS

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**Background** Systemic lupus erythematosus (SLE) is a chronic inflamatory disease witch can affect different aspects of the patients life, leading to an impairment of health-related quality of life (HRQOL).

The aim of our study was to investigate the role of demographic, clinical, immunological and psychological aspects in influencing the HRQOL of Tunisian patients with SLE and to compare the efficiency of both generic and specific questionnaires of QOL.