accurately capture cell shape. We postulated that this might be important as T cells adopt different shapes when scanning for antigen and after recognizing MHC class II-restricted peptides.

**Methods** We implemented a deep convolutional neural network (DCNN) that accurately identified both cell position and shape. The DCNN output was then analyzed with a tuned convolutional neural network (TNN) to identify distance and cell shape features that best discriminated between different T cell populations relative to dendritic cells (DCs). We refer to this analysis pipeline as CDM³.

**Results** In mice, CDM³ discriminated between cognate and non-cognate T cell interactions with DCs with a sensitivity and specificity similar to most TPEM measures. In human non-cognate T cell interactions with DCs with a continuous or never users. Discontinue HCQ accumulate damage at a different rate than cohort has changed over time and b) whether patients who previously used HCQ and those who never used, and higher compared to continuous users, suggesting continuous HCQ use may be important for damage prevention in SLE.

**Conclusions** CDM³ provides a novel tool for quantifying in situ adaptive immune cell networks broadly applicable to the study of human diseases including autoimmunity and cancer.

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**TD-05 DYNAMIC CONTRAST ENHANCED MRI (DCE-MRI) DEMONSTRATES HIPPOCAMPUS PERMEABILITY IN SLE**

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**Background** Cross-reactive, anti-dsDNA/N-methyl D-aspartate receptor antibodies (DNRAb) have been associated with cognitive impairment in SLE. The mouse model demonstrates selective effects of DNRAb on hippocampal neurons following blood brain barrier (BBB) breach.1 We previously identified abnormal hippocampal glucose hypermetabolism in SLE patients that correlated with serum DNRAb titers and poor performance on neuropsychological (NP) testing.2 However, little is known about how antibodies access brain in humans. We evaluated BBB permeability (BBBP) in SLE and healthy control (HC) subjects with DCE-MRI and hypothesized that regions with abnormal hypermetabolism would also demonstrate altered BBBP.

**Methods** 6 SLE subjects with no history of NP symptoms and 6 age and gender matched HCs underwent NP testing using the Automated Neuropsychological Assessment Metric (ANAM) computerized battery and DCE-MRI on a 3.0 tesla magnet. MRI sequences were acquired according to standard protocols; permeability imaging used DCE technique with axial 3D-SPGR T1-WI sequences and 80 cine phases using TR=25 ms, TE=3.8 ms, FOV=24 mm, and matrix size of 128 × 256. Magnevist Gadolinium contrast, 0.1 mmol/kg IV, was dosed at 5 cc/sec following a 5 sec delay. Post-processing of images into BBBP parameters of K-trans (ml/100 gm/min) and VE (ml/100 gm) was performed using Olea Sphere 2.2 and 2.3 with the Tofts extended permeability model. This