IL-1β was inversely correlated with disease activity (Rs. 0.216, p=0.013) and lowest in SLEDAI-2K<3 (p=0.001). IL-1β was a moderate driver of cytokine variance for healthy controls, but became more dominant across SLEDAI-2K<3 and SLEDAI-2K≥3, i.e. PC1 (0.59 vs 0.976 vs 0.985).

Conclusions Increased BAFF levels were not a direct agitator of cytokine variation in SLE, suggesting a contribution to disease activity through other pathways. In contrast, the reduction of IL-1β had a dominant effect on cytokine variance in SLE (PC 1). Principal component analysis is a useful asset for cytokine profiling in SLE.
Conclusions Hospitalisation rates for SLE in WA have not decreased over 25 years. Once hospital-based management for SLE was required, the risk of all-cause mortality doubled compared to age and gender-matched controls. This risk was greatest in Medicare reliant or male SLE patients and not due to increased cancer risk.

Background and aims Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that runs an unpredictable disease course. We aimed to understand the characteristics and outcomes of incident hospitalisation for conditions other than the underlying disease in SLE patients.

Methods Using whole-population data linkage of hospital admissions and death records in Western Australia (WA) between 1980 and 2015, we performed a retrospective analysis for patients where SLE (ICD-9-CM 695.4, 710.0 and ICD-10-AM L93.0 and M32) was a co-existing discharge diagnosis. All SLE patients were age- and gender-matched with hospital controls free of rheumatic disease. We investigated the rate and characteristics of the index hospitalisation for comorbidity and the risk of subsequent death by Kaplan-Meier survival and Cox regression.

Results Hospitalisation rates for comorbidity were 13.9/million/year in SLE patients. SLE patients were similar to controls for age and gender, but more likely to be Indigenous, have renal failure, cardiovascular and thrombotic conditions (Table 1). Independent predictors of mortality risk following hospitalisation for a comorbid condition included: SLE diagnosis (OR 1.6, CI: 1.3–1.9, p<0.001) (Figure 1), cerebrovascular events (OR 2.0, CI: 1.2–3.7, p<0.001), renal disease (OR 1.75, CI: 1.4–2.3 p<0.001), thrombotic events (OR 1.8 CI: 1.1–2.8, p=0.001) and reliance on Medicare (OR 1.5, CI: 1.3–1.8, p<0.001) (Table 2).

Conclusions SLE patients were more frequently hospitalised than controls for cardiovascular or renal conditions and this increased their mortality risk. These results strengthen the need for close monitoring and interventions to prevent such comorbidity in all SLE patients.