have argued that it could represent a distinct clinical entity. The present study undertook in depth phenotyping along with assessment of cortical function to further explore disease pathophysiology in MND with malignancy (MND-M) patients.

**Methods** Clinical features along with assessment of peripheral and cortical function was undertaken in 13 MND-M and results were compared to sporadic and familial MND cohorts.

**Results** From a cohort 13 patients (10 males; aged 65.2±2.0 years), 30.8% were diagnosed with a haematological malignancy. The lower motor neuron phenotype predominated in the in the MND-M patients (χ²=10.8, P<0.01), with the upper motor neuron (UMN) score being significantly reduced in MND-M patients compared to sporadic and familial MND cohorts (χ²=6.84, P<0.01). The neurological deficits did not respond to treatment of the underlying malignancy in the majority of MND-M (92%) patients, and as such there were no significant differences in survival between the cohorts. Despite a paucity of UMN signs, cortical hyperexcitability was evident in MND-M patients, as indicated by reduction in short interval intracortical inhibition (P<0.01) and increase in motor evoked potential amplitude (P<0.01), that were similar to findings in sporadic and familial MND cohorts.

**Conclusions** The present study suggests that MND-M falls within the spectrum of MND. A co- incidental association between MND and malignancy is underscored by cortical dysfunction and clinical findings which seem within the spectrum of abnormality evident in classical MND phenotypes.
impending synucleinopathy with over 90% developing either Parkinson’s disease (PD), Dementia with Lewy Bodies (DBL) or Multiple System Atrophy (MSA) after 15 years. This finding has stimulated efforts to actively register and track progression of such patients. Here we present experience of a biobanking program established with the aim of identifying prodromal synucleinopathies to facilitate recruitment to neuroprotective trials as they become available.

Methods Patients with iRBD were prospectively and sequentially recruited. Cross-sectional comparator groups consisting of healthy controls, idiopathic PD (within 5 years of diagnosis) and DBL were also recruited. Patients underwent a standardized assessment protocol including clinical phenotyping, neuropsychometric testing, multimodal MRI, polysomnography, quantitative electroencephalography, chronobiology (melatonin and clock gene expression profiling) and gait testing. Subjects were invited for annual and biennial review.

Results 102 patients have been recruited into the study since July 2016, including 35 patients with iRBD, 26 DLB, 19 early PD and 16 controls. 15 patients have returned for follow-up with 3 converting to a synucleinopathy (2 DLB, 1 PD). 75% of participants were able to complete all elements of assessment protocol. Preliminary evaluation of iRBD participants reveals early changes in clock gene expression (BMAL1) and subtle changes in patterns of gait compared to older controls.

Conclusions Our preliminary findings demonstrate utility and feasibility of a prodromal biobanking program within the Australian context aimed at identifying prodromal synucleinopathies. Similar models can be applied to other centers to improve access and create an extended national collaborative network.