PNS 2023, Copenhagen – BPNS Richard Hughes Travel Bursary report

The PNS returned to Europe this year, hosted by the beautiful city of Copenhagen. The four-day meeting brought together plenary lectures, special interest groups, cutting edge scientific and clinical research, hundreds of posters in the enormous viewing hall, and friendships and collaborations from across the globe. Although Professor Reilly was slightly miffed at her room being situated on only the *5th* floor of the 21-floor architectural spectacle that was the AC Bella Sky, the hotel and adjoining conference centre provided an excellent venue for the meeting of worldwide neuropathy experts and enthusiasts.

My non-lecture highlights for included meeting, for the first time, collaborators (whom I'd only previously met my email or on zoom) from Brazil and Australia, building new collaborations with Italian colleagues in the poster viewing hall, and being interviewed by Tanya Stojkovic and her team from the French neuropathy journal (although I think next time probably best to stay behind the camera!). The range of talks and speakers was testament to the current state of neuropathy research, and I particularly enjoyed Marina Kennerson's invited plenary lecture on the 'Dark Genome' and Richard Lewis' Presidential lecture sharing candid anecdotes of his education in neuropathy. I was particularly pleased for my friend and collaborator Natalia Dominik winning the Ricard and Mary Bunge Prize for her study on ARHGAP19 variants causing CMT. Socially – the Tivoli Gardens were a majestic venue for the opening party, re-living childhood exhilaration of rollercoasters and nostalgic décor from the 19th and 20th centuries.

I am indebted to the BPNS awarding me the Richard Hughes Travel Bursary to allow me to travel to such a fantastic conference and present my work on *GJB1 Variant Classification*. It was a pleasure to present my platform presentation in the CMT parallel session and enjoying interactive questioning from Rita Horvath, Andrea Cortese and Kleopas Kleopa. The main message was that in our study of 387 patients with variants in the gene, those with variants of uncertain significance (VUS) clinically progressed identically to those with pathogenic variants. This suggests the VUS are disease causing and with more evidence would be classed as pathogenic. I also used disease specific classification criteria to elevate the number of variants classified as pathogenic, compared with VUS.