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I have recently joined the Renal Scientist's Working Party, as representative for Wales taking over from Professor Nicholas Topley. I graduated from the University of Warwick with a BSc (Hons) in Virology, which included an intercalated year in the Novartis Research Institute in Vienna. My subsequent research experience during my PhD and post-doctoral positions has been in the area of cytokine biology and inflammation at the University of Wales College of Medicine and Cardiff University. I have been working within the laboratories of Professor Nicholas Topley and Dr. Simon Jones at the School of Medicine in Department of Medical Biochemistry and Immunology and Institute of Nephrology since 2003. Their groups form part of a 'Virtual Institute', the Infection, Immunity and Inflammation Interdisciplinary Research Group (I³ IRG). In 2006, I was awarded a Kidney Research UK (KRUK) Career Development Fellowship to begin a project investigating the role of cytokine signalling in the development of peritoneal fibrosis, a frequent peritoneal dialysis (PD)-associated complication and major cause of PD treatment failure.

Continuous ambulatory peritoneal dialysis (CAPD) is used to treat a significant number of patients with end-stage renal disease and the development of fibrosis of the peritoneal membrane is a major cause of treatment failure. Recurrent episodes of bacterial infections, predominantly caused by *Staphylococcus* species, are the main complication of CAPD. These infections are accompanied by inflammation of the peritoneal cavity or peritonitis. Recent data from the Biopsy Registry has identified that those patients with a history of episodic peritonitis have significantly more membrane thickening than those who remain infection free. The frequency, duration and severity of these periodic infections may therefore be important in promoting tissue fibrosis, thus reducing the capacity of the peritoneal membrane to allow adequate dialysis. We have developed and validated a model of recurrent peritoneal inflammation in order to assess the importance of cytokines (soluble protein immune mediators) in the development of peritoneal fibrosis. Recurrent episodes of peritonitis in the model cause alterations in the inflammatory response in terms of leukocyte recruitment, cytokine production and transcription factor activation. The results obtained so far have identified other factors altered downstream of recurrent inflammation, which may be involved in the fibrotic process. These factors are also being examined in clinical PD samples to validate these differences and may represent potential new therapeutic avenues to promote membrane survival in PD patients.

As a relative newcomer to the area of Nephrology, I have been struck by the quality of renal research within the UK, and also by the breadth of research interests. I think the cross over with other research disciplines is an asset to renal research, enabling the interaction of scientists and clinicians from different background specialities, and thereby producing high-quality research. I hope the Renal Association will continue to encourage these interactions and to attract scientists to Nephrology from other specialities.

