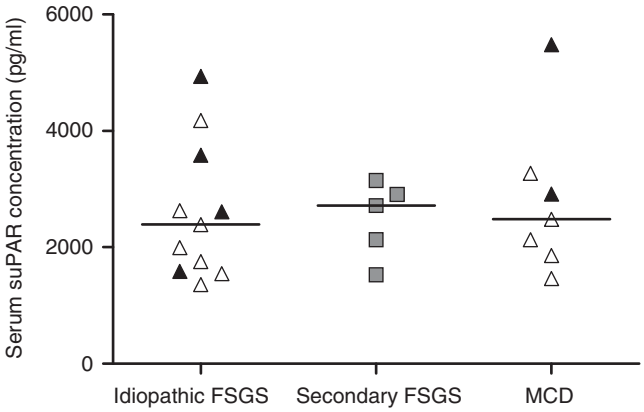

Figure 1 | Serum suPAR concentrations in patients with idiopathic focal segmental glomerulosclerosis (FSGS), minimal change disease (MCD) and secondary FSGS. Median suPAR concentration is indicated by the horizontal line. △, steroid-sensitive (complete remission); ▲, steroid-resistant (no remission); , no immunosuppressive therapy.

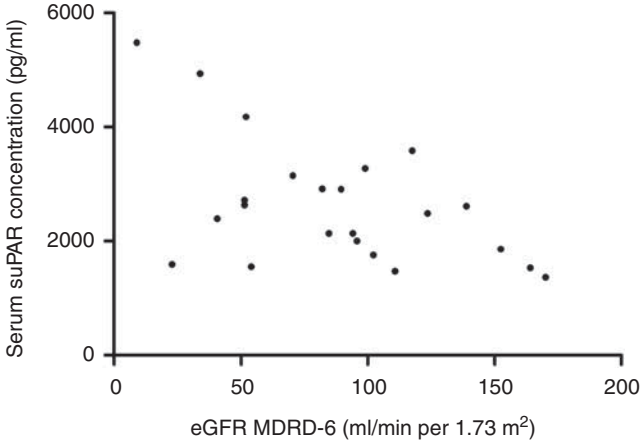

Figure 2 | Correlation analysis of suPAR with estimated glomerular filtration rate (eGFR) MDRD-6. Spearman's rho −0.46, $P = 0.03$.

focal segmental glomerulosclerosis (FSGS) and not in patients with other glomerular diseases. The authors proposed that high serum suPAR (⩾3000 pg/ml) is specific for primary FSGS. However, in many of their patients with suspected primary FSGS, serum albumin was normal or unknown. The absence of hypoalbuminemia despite heavy proteinuria suggests that FSGS is due to hyperfiltration rather than primary FSGS.[2] It is also unknown whether suPAR is associated with response to corticosteroids. To determine the value of suPAR in routine clinical practice we measured suPAR concentration in sera of adult patients with idiopathic (primary) FSGS ($n = 11$), secondary FSGS (hyperfiltration $n = 3$, familial $n = 2$), and minimal change disease (MCD; $n = 7$) with the Quantikine Human suPAR Immunoassay (R&D Systems, Minneapolis, MN). Median serum suPAR concentration was not different between idiopathic FSGS (2392 pg/ml), secondary FSGS (2716 pg/ml), and MCD (2482 pg/ml), nor did it predict steroid responsiveness in patients with idiopathic FSGS/MCD (Figure 1). We found a negative correlation between suPAR and estimated glomerular filtration rate Modification of Diet in Renal Disease (MDRD)-6 (Figure 2). In conclusion, suPAR concentration is not a specific marker for idiopathic FSGS in this study and it does not reliably predict response to treatment. Although our data indicate that for the individual patient suPAR is not a reliable marker, the small size of our study does not allow definite conclusions on the role of suPAR in FSGS. A larger study is needed to clarify this issue.

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The contribution of chronic kidney disease to the global burden of major noncommunicable diseases

To the Editor: We have read the report of the ISN Global Outreach Programme,[1] which confirms that, for the less developed countries compared with the more developed ones, chronic glomerulonephritis (GN) and not diabetic nephropathy (DiabNx) is still the leading cause of end-stage renal failure (ESRF).

The more developed Asian countries including Singapore, Malaysia, India, and China are now faced with the burden of diseases associated with lifestyle changes, such as diabetes mellitus and obesity. Despite the decreasing prevalence of chronic kidney disease-associated proteinuria due to chronic GN and hypertensive nephrosclerosis, as a result of programs to control and retard progression in these two diseases, the dilemma of the epidemic proportion of DiabNx is far from being contained. In Singapore, the incidence of DiabNx as a cause of ESRF in 1983 was 17%, in 2004 it was 58%, and in 2008 it further rose to 63% in patients initiating dialysis.[2] However, the incidence of ESRF has stabilized over the past 3 years. In 1975, it was 90 per million population (p.m.p.), with a progressive increase to peak at 264 p.m.p. in 2007. Over the past 3 years (2006–2008), it has remained at 238, 264, and 245 p.m.p. (ever start dialysis) or 207, 212, and 211 p.m.p. (survive 90 days of dialysis).[2]

Angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEIs/ARBs) have been around for years,

but the incidence of ESRF due to DiabNx has not stabilized.[3] There is also the growing concern that long-term use of ACEIs/ARBs could promote renal fibrosis.[4] This may be one explanation of why the incidence of ESRF due to DiabNx has not plateaued despite the stabilization of the incidence of ESRF. Perhaps the time has come for ISN experts to focus on the burden of ESRF due to DiabNx in these countries.

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