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**Letter to Editor**

# A biomarker for screening early chronic kidney disease

The number of patients with chronic renal failure appears to be increasing and we have a limited and largely ineffective preventive strategy for early detection and intervention. Present tests, such as measuring changes in serum creatinine and creatinine clearance, are insensitive and changes in the serum creatinine are not apparent until there is a 50 percent loss of renal function. Creatinine clearance is influenced by the phenomenon of hyperfiltration which can mask the problem and looses time when preventive strategies might still be able to halt progression.

The fractional excretion of magnesium (FE Mg) has been proposed as an early marker to screen for renal functional impairment [1]. FE Mg correlates well with the magnitude of tubulointerstitial fibrosis. FE Mg reflects the tubular cell's ability to reabsorb the glomerular filtrate of magnesium and retain the intracellular second most abundant cation magnesium.Therefore, a low value of FE Mg reflects an intact tubulointerstitial structure, whereas a high value of FE Mg indicates kidney disease associated with tubulointerstitial fibrosis.This has been demonstrated as useful in screening diabetic nephropathy patients [2, 3]. It is more sensitive than using microalbuminuria. FE Mg has also been useful in the screening for kidney diseases associated with intact tubulointerstitial structures and differentiates it from diseases associated with tubulointerstitial fibrosis [5-7].

The efficacy of therapy is greater, when treatment is initiated early in the course of renal impairment, and when there is still an adequate functional reserve, as observed in normoalbuminuric type 2 diabetes or in a variety of chronic kidney diseases, associated with minimally impaired renal function. By utilizing FE Mg as a sensitive marker for screening renal disease severity we, as well as others, have been able to improve renal function in normoalbuminuric type 2 diabetic nephropathy and nephrosis, associated with focal segmental glomerulosclerosis. This experience contrasts with therapy which simply slows progression but is unable to significantly improve and retain function [4, 7-10].

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