

antiproteinuric treatment' [1]. We are pleased to answer both questions.

First, it is believed that we forgot to mention a few papers. Before going into the details of the papers mentioned, we would like to stress the special hallmarks of our patient. She had infantile nephrotic syndrome, caused by nail-patella syndrome, as proven by mutation analysis, was treated exclusively with drugs interfering with the renin-angiotensin-aldosterone system and went into complete remission with a follow-up of more than 2 years. Indeed, we did not mention the patient reported by Sreedharan and Bockenbauer [2]. In fact, this infant, with congenital nephrotic syndrome of unidentified aetiology, did indeed go into complete remission with captopril alone, had a relapse when the drug was withheld and responded again, but the follow-up time was rather short. We did mention the patient described by Guez *et al.* [3], but this again was congenital nephrotic syndrome, this time with the Finnish type as a most likely diagnosis. But the treatment consisted of indomethacin and enalapril, and the remission was incomplete at the time of writing. There are at least two other papers that reported similar patients treated successfully with indomethacin and captopril [4,5]. The paper by Soliman *et al.* was, unfortunately, overlooked for the simple reason that it was not published yet when we wrote our paper [6]. This baby presented with infantile nephrotic syndrome; the biopsy showed focal segmental glomerulosclerosis—which by all means can hardly be considered an aetiological category—but no mutation could be identified in the following genes: NPHS1, NPHS2, WT1 and PLCE1. The patient went into complete remission but with a multidrug regimen consisting of albumin infusions, furosemide, labetalol, amlodipine, vitamin E, captopril and enalapril. The follow-up was >1 year, and renal function was estimated to be within the normal range. Also, we wonder why the title of this paper ends with a question mark, 'is it renin-angiotensin blockade?'

Second, the question was raised why we waited for 2 years before adding losartan to enalapril. From our short Case Report section, it is difficult to explain how well our patient was doing under enalapril therapy. In this early period of life, she was never really nephrotic, had no oedema and normal growth and development. Serum albumin was slightly below the lower limit of normality and serum cholesterol slightly above the normal figures for age. Moreover, she was normotensive with blood pressure figures around the 10th percentile for her age, and therefore it took some time to reach what we consider the optimal dose of enalapril, i.e. 1 mg/kg. In fact, even if the proteinuria remained almost constant when calculated in mg/kg with this treatment, it decreased by the mere fact of growth so that we did not consider the therapy as futile. Of course, no one could foresee how well she would respond to losartan. At the time this drug was added to enalapril, the first data on the combined therapy in childhood were emerging. When we were writing our paper, however, we could find only a handful of paediatric patients in whom this treatment was being used in the English literature. Even today, there is insufficient data available about dosages, efficacy, tolerance, side effects and long-term outcome of paediatric renal patients

treated with the combination of an ACE inhibitor and an angiotensin-receptor antagonist.

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### Low prevalence of chronic kidney disease in Far-East Asian populations: impact of the ethnicity factor?

Sir,

We read with great interest three epidemiological studies recently published in *Nephrology Dialysis and Transplantation*, all studies coming from Far-East Asian countries (two Chinese and one Japanese) [1–3]. It could be of interest to compare prevalence results of Chinese and Japanese data. Of course, such a comparison may be difficult and misleading because the characteristics of the populations are different: type 2 diabetes in the Japanese study [3] and the general population in the Chinese studies [1,2]. However, the prevalence of CKD in diabetic patients was 10.2% in the Chinese population [2] versus 15.3% in the Japanese one [3]. Of course, the differences in ethnicity and lifestyle may explain such discrepancies. However, we were also impressed by the differences in the MDRD study equations used in the Chinese versus Japanese populations. In Table 1, we show the results of a simulation (man of age 60 years old) for the MDRD study equation results applying to different ethnicities. The results are somewhat intriguing (and constant even if age or sex is modified in the simulation). Indeed, for the same creatinine value, the MDRD equation results are logically highest with the

**Table 1.** Results of MDRD study equations for a 60 years old man with different ethnicity factors

Creatinine (mg/dl)	MDRD	MDRD	MDRD	MDRD
	Caucasian = $175 \times$ $SCr^{-1.154} \times$ $Age^{-0.203}$	Black American = $175 \times$ $SCr^{-1.154} \times$ $Age^{-0.203} \times 1.21$	Japanese = $194 \times$ $SCr^{-1.094} \times$ $Age^{-0.287}$	Chinese = $175 \times$ $SCr^{-1.234} \times$ $Age^{-0.179}$
0.5	170	205	128	198
1	76	92	60	84
1.5	48	58	38	51
2	34	41	28	36
2.5	26	32	22	27
3	21	26	18	22

Black American correction factor. This is classically explained by the higher muscular mass of Black American peoples compared to Caucasians [4]. The results are more astonishing in the comparison of the two Asian equations, just as they are used in the epidemiological studies. In fact, for the same creatinine value, the Chinese results are systematically higher than the Japanese ones. If the Japanese results are lower than the Caucasian results (which may perhaps be viewed as logical regarding muscular mass differences between these two ethnicities), the Chinese results are systematically higher (and very close to Caucasians). The differences in the MDRD study equation results that are linked to the ethnicity factor would be higher between Japanese and Chinese patients on one hand than those between Chinese and Caucasian (or even Black) patients on the other hand. Differences between Japanese and Chinese MDRD study equations are questionable, strong and will have a profound impact on the CKD prevalence. We do not have all data at hand to decide which of the Japanese or the Chinese equations are the best ones. From our point of view, the way the Asian equations have been built is very different (regarding creatinine recalibration, GFR reference method and statistics used). However, neither the Chinese nor the Japanese equations are totally free from criticism. Whatever, the Chinese MDRD results seem systematically higher than Japanese and Caucasian results that could explain, at least in part, the lower prevalence of CKD observed in the Chinese population, notably compared to American NHANES data. Lastly, it should not be concluded from our demonstration that the Chinese equation is not accurate. Indeed, we, and others, have shown that the MDRD study equation underestimates GFR in a healthy population [5,6] and thus overestimates the CKD prevalence in epidemiological studies [5]. So, the Chinese MDRD study equation and the Chinese epidemiological data could be closer to the 'truth' than the American and Japanese ones.

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## Reply

Sir,

Dr Delanaye and his colleagues compared three epidemiological studies [1–3], two from China [1,2] and one from Japan [3]. They found that the prevalence of chronic kidney disease (CKD) was 10.2% in the Chinese diabetic population, and 15.3% in the Japanese population. We agree with the postulation that differences in ethnicity and lifestyle might be one of the explanations of this discrepancy, but the vintage of diabetes might add more impact on the observed prevalence. Patients with longer diabetes vintage were more prone to have CKD.

The simulation of Dr Delanaye and his colleagues was very impressive. According to the simulation, for three 60-year-old Caucasian, Chinese and Japanese males, if each of their plasma creatinine was measured in a local laboratory and each of the measured values was 1 mg/dL, the estimated glomerular filtration rate (eGFR) would be 84, 76 and 60 mL/min/1.73 m<sup>2</sup> calculated from equations for their own race. We should pay attention to the creatinine value, 1 mg/dL, used in the simulation. If the three plasma samples were measured simultaneously in one single lab, using the 'gold' creatinine measurement method, they might deviate from 1 mg/dL and be different from each other, due to inter-laboratory variance. Usually, clinicians do not order 'gold' creatinine assays, so the systemic difference between clinically used creatinine measurement methods and 'gold' methods was built in each 'race-specific' equation.

The modification of diet in renal disease (MDRD) study [4] used renal clearance of <sup>125</sup>I-iothalamate, the Chinese study [5] used plasma clearance of <sup>99m</sup>Tc-DTPA and the Japanese study [6] used renal clearance of inulin as reference GFR. If there were a 'gold' standard of GFR, each of the three reference GFR methods might deviate from the 'gold' standard and be different from each other, due to