Table 1 | Common patient characteristics quoted from the reports by Goraya *et al.*[1] and Mahajan *et al.*[2]

	Goraya *et al.*, CKD2 control (*n*=40)	Mahajan *et al.*, NaCl (*n*=40)	Goraya *et al.*, CKD2 and HCO_3 (*n*=40)	Mahajan *et al.*, $NaHCO_3$ (*n*=40)	Goraya *et al.*, CKD2 and F+V (*n*=40)	Mahajan *et al.*, placebo (*n*=40)
Males (%)	47.5	48	47.5	48	47.5	48
Black/white/Hispanic (%)	62.5/22.5/15.0	63/23/15	62.5/20.0/17.5	63/20/18	62.5/25.0/12.5	63/25/13
Age (years)	51.5 ± 8.3	51.5 ± 8.3	51.2 ± 8.2	51.2 ± 8.2	51.3 ± 8.5	51.3 ± 8.5
Systolic BP (mm Hg)	134.3 ± 8.3	152.6 ± 14.7	134.1 ± 5.8	155.3 ± 12.6	133.7 ± 8.6	155.2 ± 12.9
eGFR (ml/min)	75.6 ± 6.5	75.6 ± 6.5	75.3 ± 6.1	75.3 ± 6.1	75.6 ± 6.2	75.6 ± 6.2
Plasma total CO_2 (mmol/l)	26.0 ± 0.8	26.4 ± 0.8	25.9 ± 0.6	26.2 ± 0.7	25.9 ± 0.8	26.0 ± 0.9
PRAL (mmol/day)	59.3 ± 21.1	59.3 ± 21.1	64.3 ± 17.7	64.3 ± 17.7	60.4 ± 19.4	60.4 ± 19.4
8-h NAE (mEq)	24.6 ± 5.7	24.8 ± 6.4	24.8 ± 5.6	24.8 ± 5.6	24.6 ± 5.0	24.0 ± 5.6
Ualb (mg/g Cr)	413.6 ± 147.9	413.6 ± 147.9	419.3 ± 150.8	419.3 ± 150.8	422.2 ± 151.6	422.2 ± 151.6
UNAG (U/g Cr)	2.7 ± 0.4	2.6 ± 0.5	2.7 ± 0.4	2.7 ± 0.4	2.7 ± 0.7	2.7 ± 0.7
UET-1 (ng/g Cr)	5.5 ± 1.1	5.7 ± 0.8	5.7 ± 1.0	5.7 ± 1.0	5.5 ± 1.2	5.5 ± 1.2
Urine Na^+ excretion (mmol/g Cr)	71.6 ± 7.9	70.6 ± 10.2	70.9 ± 10.2	70.9 ± 10.2	73.0 ± 9.5	73.0 ± 9.5
Urine K^+ excretion (mmol/g Cr)	38.6 ± 5.5	38.2 ± 6.2	41.1 ± 6.1	41.1 ± 6.1	39.5 ± 6.6	39.5 ± 6.6

Abbreviations: BP, blood pressure; CKD, chronic kidney disease; Cr, creatinine; eGFR, estimated glomerular filtration rate; F+V, fruits and vegetables; NAE, net acid excretion; PRAL, potential renal acid load; Ualb, urine albumin-to-creatinine ratio; UET-1, urine endothein-1-to-creatinine ratio; UNAG, urine *N*-acetyl-β-D-glucosaminidase-to-creatinine ratio. Values are expressed as number, percentage, or mean ± SD as appropriate.

description, the 'NaCl' groups should not be considered as the control, and 'F + V' seems like placebo with 5-year observation.

The long-term follow-up of the MDRD study revealed the risk of the low-protein diet,[3] which has been believed to be good for CKD by both patients and health professionals for a long time. The interesting hypothesis of Goraya *et al.* could have been an alternative dietary intervention for CKD patients if proven appropriately. Disappointingly, the serious suspicion of faking research has made a mess of the series of their RCTs.

1. Goraya N, Simoni J, Jo C *et al.* Dietary acid reduction with fruits and vegetables or bicarbonate attenuates kidney injury in patients with a moderately reduced glomerular filtration rate due to hypertensive nephropathy. *Kidney Int* 2012; **81**: 86–93.
2. Mahajan A, Simoni J, Sheather SJ *et al.* Daily oral sodium bicarbonate preserves glomerular filtration rate by slowing its decline in early hypertensive nephropathy. *Kidney Int* 2010; **78**: 303–309.
3. Menon V, Kopple JD, Wang X *et al.* Effect of a very low-protein diet on outcomes: long-term follow-up of the Modification of Diet in Renal Disease (MDRD) Study. *Am J Kidney Dis* 2009; **53**: 208–217.

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The Authors Reply: We thank Dr Obi *et al.* for their comment[1] regarding similarities in subject groups in our two *Kidney International* publications[2,3] and their question of whether the two studies are truly randomized controlled studies given that some groups across the studies are very similar. Since 1999 we serially recruited subjects with hypertensive nephropathy and various estimated glomerular filtration rate (eGFR) levels to test the hypothesis that dietary acid reduction reduces acid retention,[4] reduces kidney injury,[2] and slows eGFR decline.[3] Subjects were recruited and serially entered into protocols designed to test these hypotheses. The first protocol randomized three sets of subjects with stage 2 eGFR, each of whom underwent three serial stages of studies to assess the effect of dietary acid reduction on acid retention (baseline and after 30 days), kidney injury (before and after 30 days), and, after a 30-day washout, the effect on eGFR after 5 years. Each subject was followed up in excess of 5 years, but in each case the follow-up began at recruitment and study entry. Specifically, they were not recruited as a group and then followed as a group at the same time. We initially submitted one manuscript to *Kidney International* that included the described three serial stages of study done on each subject to test all the three hypotheses. The Editors asked that we not test all the three hypotheses in a single manuscript, but to do so in separate manuscripts. We chose first to submit a manuscript detailing the main, randomized protocol in these stage 2 subjects that showed long-term (5-year) effects on eGFR[3] followed by separate submissions of the shorter-term portions of this protocol in many of the same subjects that examined the effects on acid retention[4] and, most recently, kidney injury.[2] Consequently, some subject groups that were randomized for the 5-year protocol to examine the effects of the interventions on eGFR were the same groups used to test the two remaining hypotheses as described but were not further randomized.

1. Obi Y, Hama H, Suzuki Y *et al.* Implausible similarities in patient characteristics between two randomized controlled studies: a coincidence is unlikely. *Kidney Int* 2012; **82**: 115–116.

2. Goraya N, Simoni J, Jo C-H *et al.* Dietary acid reduction with fruits and vegetables or sodium bicarbonate reduces kidney injury in subjects with moderately reduced GFR due to hypertensive nephropathy. *Kidney Int* 2012; **81**: 86–93.
3. Mahajan A, Simoni J, Sheather S *et al.* Daily oral sodium bicarbonate preserves glomerular filtration rate by slowing its decline in early hypertensive nephropathy. *Kidney Int* 2010; **78**: 303–309.
4. Wesson DE, Simoni J, Broglio K *et al.* Acid retention accompanies reduced GFR in humans and increases plasma levels of endothelin and aldosterone. *Am J Physiol Renal Physiol* 2011; **300**: F830–F837.

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