Original Article

The influence of a cooked-meat meal on estimated glomerular filtration rate

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Abstract

Background Chronic kidney disease (CKD) is an important but under-recognized condition. Recent national guidelines have recommended that biochemistry laboratories report estimated GFR (eGFR) to improve diagnosis of CKD and facilitate disease staging and management. Previous reports have suggested that intake of large amounts of cooked meat can lead to a significant increase in serum creatinine concentration.

Methods Participants ($n = 32$), consisting of 17 healthy volunteers and 15 outpatients, were recruited. Measurement of serum creatinine (kinetic Jaffe method, enzymatic, isotope-dilution mass spectrometry [IDMS]) and cystatin C, and calculation of eGFR were carried out before (i) and after a meal containing cooked meat (ii) and a meat-free meal (iii).

Results Following intake of cooked meat, median serum creatinine concentration (kinetic Jaffe) increased from 80.5 μmol/L preprandially to 101.0 μmol/L 1–2 h postprandially ($P < 0.0001$), and 99.0 μmol/L 3–4 h postprandially ($P < 0.0001$). Median eGFR decreased from 84.0 mL/min/1.73 m^2 preprandially to 59.5 mL/min/1.73 m^2 1–2 h postprandially ($P < 0.0001$), and 64.0 mL/min/1.73m^2 3–4 h postprandially ($P < 0.0001$).Consumption of non-meat-containing meals had little impact on serum creatinine (kinetic Jaffe) and eGFR. Changes in serum creatinine were similar using all three methods, and cystatin C concentration was generally uninfluenced by food intake.

Conclusions Intake of cooked meat has a significant effect on serum creatinine concentration and eGFR. Misclassification of CKD is possible if measurements are made after meals containing cooked meat. Clinicians should ensure that CKD classification is based on samples taken in the appropriate conditions: either fasting or after avoidance of cooked meat on the day of sampling. National guidelines which overlook this factor should be revisited.

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Introduction

Glomerular filtration rate (GFR) is recognized as the best available measure of kidney function. Various methods of measurement exist, including clearance of exogenous markers (e.g. inulin and iothalamate), endogenous markers (creatinine and cystatin C) and calculation of estimated GFR (eGFR) from serum creatinine concentration and patient demographics. In England, the Department of Health has identified reporting of eGFR as a marker of good practice for the identification, assessment and management of chronic kidney disease (CKD),[1] and it is now being routinely reported by many diagnostic NHS laboratories. This has generated considerable interest and debate. The estimation of GFR, using an equation derived from the Modification of Diet in Renal Disease (MDRD) Study data,[2] has also been recommended by the National Kidney Foundation,[3] the American Society of Nephrology, the National Kidney Disease Education Programme,[4] Kidney Health Australia[5] and Kidney Disease: Improving Global Outcomes (KDIGO).[6] The advantages of eGFR over serum creatinine or creatinine clearance estimations include earlier recognition of reduced kidney function, an

understandable referral and management protocol based on an internationally agreed staging system of CKD and the avoidance of the less practical, imprecise, less accurate and more expensive creatinine clearance estimations.[2,7]

When interpreting a creatinine result, clinicians should take into account various extra-renal factors which are known to affect creatinine production and, therefore, serum creatinine concentrations. These include age, gender, ethnicity, body habitus (muscle mass in particular), diet and chronic illness. Of these, only diet may have a rapid and transient effect on creatinine concentration, but this is a factor that national and international organizations have not highlighted in published recommendations.[1,5,6]

It is important to note that the original MDRD equations were derived from blood samples taken during the baseline period of the MDRD study,[8] and that most samples were taken in the morning following a period of fasting and water loading (Levey AS, personal communication). However, in clinical practice, they are generally used in situations where the patient's recent dietary intake is not considered. The cooking process is known to convert a fraction of creatine in the meat to creatinine.[9] Around 20–30 years ago, some small studies demonstrated significant rises in serum creatinine concentration following ingestion of quite large (225–500 g) standardized cooked-meat meals.[10–13] This finding has not been confirmed following normal meal helpings. We, therefore, set up a study to answer the following questions:

(1) Does a normal helping of food, with or without cooked meat, affect serum creatinine (Jaffe method) and eGFR?
(2) If so, is this due to true changes in serum creatinine concentration, or due to non-specific ('pseudo-chromogen') effects on the kinetic Jaffe method?
(3) Is there another endogenous marker of GFR that is not influenced by a cooked meat meal?

Methods

Participants ($n = 32$, 15 men and 17 women, median age 54.5 years, age range 18–86 years), consisting of 17 laboratory volunteers and 15 outpatients attending the Care of the Elderly Day Hospital at Wishaw General Hospital, were recruited to the study from June to August 2006. All were Caucasian. Participants who agreed to take part were recruited sequentially from the Day Hospital without review of medical notes. Laboratory staff were volunteers.

Exclusion criteria included vegetarianism or any other reasons for not taking a meat diet, renal dialysis therapy, previous renal transplantation and age under 18 years. Informed consent was taken at the first visit. At the second and third visits, blood sampling was undertaken before and after a meal containing cooked meat (32 subjects) and no cooked meat (23 subjects). The first sample was taken 4 h after a normal breakfast, which contained no cooked meat – the preprandial sample. Two samples were taken in the postprandial period, the first after 1–2 h and the second 3–4 h after lunch. Laboratory volunteers' meals were supplied by the hospital canteen and Care of the Elderly Day Hospital participants' meals by the same provider, Serco®. Fluid intake was not restricted. Samples were separated and analysed for creatinine using a kinetic Jaffe method. Aliquots were frozen at −40°C for subsequent analysis (see below). Estimated GFR was calculated using the adjusted isotope-dilution mass spectrometry (IDMS) traceable version of the MDRD equation,[14] with assay-specific adjustment factors, namely:

$$\begin{aligned}&\text{eGFR}\,(\text{mL/min/1.73m}^2)\\&\quad = 175 \times [(\text{serum creatinine}\,(\mu\text{mol/L}) - \text{intercept})\\&\quad \times 0.011312/\text{slope}]^{-1\times154} \times [age]^{-0\times203} \times 1.212\\&\quad (\text{if black}) \times 0.742\,(\text{if female})\end{aligned}$$

The intercept and slope are Beckman reagent user-specific factors provided by the United Kingdom National External Quality Assessment Scheme (UKNEQAS) to correct for between-method creatinine biases and to ensure IDMS traceability. The National Kidney Foundation classification of CKD[3] was used for staging to allow comparison of pre- and postprandial results. Aliquots of serum were also analysed for creatinine by IDMS (Guy's Hospital, London) and by an enzymatic method (Kent and Canterbury Hospital). Cystatin C was measured on further serum aliquots (Kent and Canterbury Hospital). The meat-containing lunch options included lamb hot-pot, beef curry, roast beef sandwiches, roast beef, steak mince, pasta bolognaise, lamb curry, braised steak, beef ghoulash, steak pie, roast lamb, chicken chasseur and chicken curry. Non-meat-containing meals included soup with bread and salad, potato scones, sandwiches with fillings, vegetable lasagne, pasta and others. The significance of any changes in creatinine concentrations, eGFR and cystatin C concentrations was analysed by the Wilcoxon signed rank test. Ethical approval for the study was obtained from the Lanarkshire Ethics Committee.

Analytical techniques

(1) *ID-MS creatinine*: A modified liquid chromatographic ID-MS method. Serum was diluted with deionized water containing d3-creatinine (CDN Isotopes, Qmx Laboratories, Thaxted, UK) and

proteins precipitated with acetonitrile (Rathburn Chemicals Ltd, Walkerburn, UK). Following mixing and centrifugation, supernatants were transferred to a 96-deep-well plate and 1 μL automatically injected into a mobile phase stream of acetonitrile:water. Chromatography was performed on a Chirobiotic T column (Advanced Separation Technologies, Congleton, UK) and precursor/product ion pairs (*m/z* 114.2/44.2, 117.2/47.2) were acquired in positive ion multiple reaction monitoring mode using a Sciex API4000 (Applied Biosystems, Warrington, UK). The concentration of the stock creatinine standard used has been confirmed using National Institute of Standards and Technology (NIST) standard reference material, creatinine 914a (Laboratory of the Government Chemist, Teddington, UK), and pooled and spiked quality control samples with assigned values determined using EU Community Bureau of Reference certified reference material sera (Report EUR 17115EN) CRM 573 (68.7 μmol/L), 574 (105.0 μmol/L) and 575 (404.1 μmol/L) (Laboratory of the Government Chemist, Teddington, UK) were included with each assay. Between-day coefficients of variation in control sera were $< 3\%$ ($n = 56$) at concentrations of 64.8, 183.8 and 495.5 μmol/L.

(2) *Enzymatic creatinine*: An enzymatic wet chemistry method was used on an Integra 800 analyser (Roche Diagnostics Ltd, Burgess Hill, UK) utilizing a creatininase/creatinase/sarcosine oxidase system. The between-day coefficients of variation were $< 2.5\%$ at concentrations of 109 and 371 μmol/L.

(3) *Kinetic Jaffe creatinine*: a kinetic Jaffe method was used on a Beckman Coulter LX20 Analyser (Beckman Coulter, High Wycombe, UK) at Wishaw General Hospital. The between-day coefficients of variation were $< 2.8\%$ at concentrations of 99 and 204 μmol/L.

(4) *Cystatin C*: measured by a particle-enhanced nephelometric immunoassay on a BN Prospec analyser (Dade Behring Ltd, Milton Keynes, UK). The laboratory reference range was 0.54–1.06 mg/L, and between-day imprecision was 3.5% at a concentration of 2.3 mg/L.

Results

Results were obtained for 32 participants following the cooked-meat meal and for 23 of these participants following the meat-free meal. A table of all results from samples taken before and after a meal containing cooked meat is available in the Appendix.

Does diet affect eGFR?

Creatinine (kinetic Jaffe) and eGFR following a cooked-meat meal: The changes in serum creatinine concentration and eGFR are summarized in Table 1. Serum creatinine concentration increased, from the baseline preprandial

Table 1 *Comparison of median eGFR, serum creatinine concentration (using 3 different analytical methods) and cystatin C concentration*

	Time	eGFR from kinetic Jaffe (mL/min/1.73 m^2)	Creatinine (kinetic Jaffe) (μmol/L)	Creatinine (IDMS) (μmol/L)	Creatinine (enzymatic) (μmol/L)	Cystatin C (mg/L)
Meat meal	Preprandial	84.0	80.5	77.8	76.9	0.87
(*n*=32)	(95% CI)	(70.0–98.0)	(71.0–94.0)	(64.1–89.3)	(62.0–86.0)	(0.75–1.13)
	1–2 h postprandial	59.5	101.0	100.1	95.9	0.87
	(95% CI)	(59.0–80.0)	(89.0–110.0)	(79.8–107.0)	(79.1–105.0)	(0.76–1.11)
	P value	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.45
	3–4 h postprandial	64.0	99.0	93.5	93.1	0.83
	(95% CI)	(54.0–79.0)	(87.0–114.0)	(85.4–108.0)	(86.4–106.0)	(0.74–1.11)
	P value	< 0.0001	< 0.0001	< 0.0001	< 0.0001	0.06
No meat meal	Preprandial	76.5	89.5	81.2	81.3	1.13
n=23	(95% CI)	(64.0–105.0)	(70.0–96.0)	(65.4–92.9)	(62.0–90.8)	(0.81–1.22)
	1–2 h postprandial	77.5	88.5	82.9	81.3	1.06
	(95% CI)	(65.0–105.0)	(71.0–97.0)	(61.2–90.0)	(62.2–90.0)	(0.79–1.19)
	P value	0.009	0.05	0.008	0.07	0.006
	3–4 h postprandial	80.0	86.5	82.4	82.0	1.10
	(95% CI)	(64.0–105.0)	(66.0–95.0)	(62.9–91.0)	(62.0–89.3)	(0.78–1.20)
	P value	0.006	0.06	0.003	0.11	0.129

All *P* values compare preprandial and postprandial results. CI, confidence interval

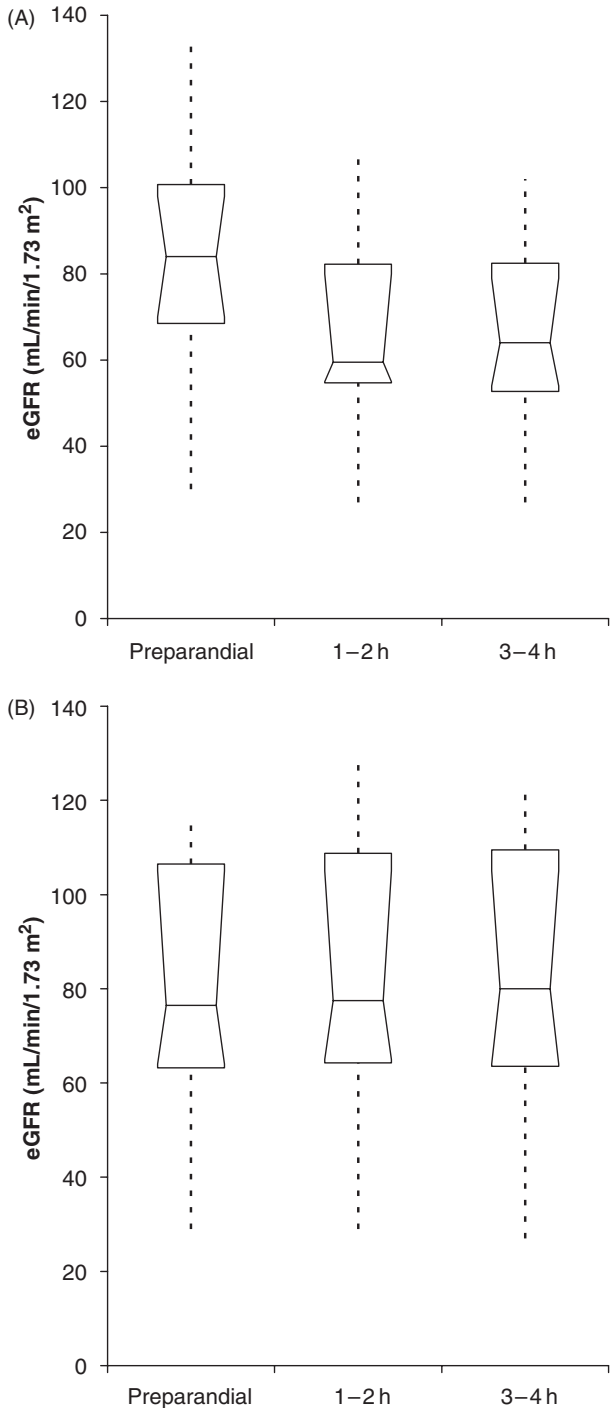

Figure 1 *eGFR before and after meals containing cooked meat (A) and no meat (B). Range, median and interquartile range are displayed*

sample, by 20.5 μmol/L at 1–2 h postprandially and 18.5 μmol/L at 3–4 h postprandially. eGFR decreased from the baseline preprandial sample by 24.5 mL/min/1.73 m^2 at 1–2 h postprandially and 20 mL/min/1.73 m^2 at 3–4 h postprandially. Figure 1 is a plot of median preprandial eGFRs and median postprandial eGFRs 1–2 h and 3–4 h after a meal containing either cooked meat or no meat.

Maximal postprandial serum creatinine concentrations were reached by 18 participants at the 1–2 h mark, and by 12 participants at the 3–4 h mark.

Creatinine (kinetic Jaffe) and eGFR following a meat-free meal: The changes in serum creatinine concentration and eGFR are summarized in Table 1. The eGFR rose by a modest, though statistically significant, level after a meat-free meal. The eGFR increased, from the baseline preprandial sample, by 1.0 mL/min/1.73 m^2 after 1–2 h, and by 3.5 mL/min/1.73 m^2 after 3–4 h. The decrease in serum creatinine of 1.0 μmol/L at 1–2 h, and 3.0 μmol/L 3–4 h after a meat-free meal, did not reach significance.

Is this due to true changes in serum creatinine concentration or due to interference with the kinetic Jaffe method?

To investigate possible interference, serum creatinine was analysed by three different methods; namely the kinetic Jaffe (as above), IDMS and enzymatic methods.

Increases in serum creatinine measured by the three methods were similar after a cooked meat meal (see Table 1) at both time intervals. There is, therefore, no evidence of significant assay interference affecting the kinetic Jaffe method.

Is there another marker of GFR not influenced by a cooked-meat meal?

The results of cystatin C analyses are contained in Table 1. There was a marginal fall in the median serum cystatin C in the case of the 1–2 h samples after the non-meat-containing meal, but all other changes were not statistically significant.

Discussion

Our study highlights the impact of a cooked meat meal on serum creatinine concentration and eGFR, with a potentially significant impact on the diagnosis and staging of CKD. This is due to an increase in serum creatinine *in vivo* as shown by the similar increases in results using three different methods.

Although the increase in serum creatinine concentration after the consumption of cooked meat was first reported in 1933 with sporadic reports thereafter,[9–13,15–17] this factor appears to have been overlooked in recent national and international CKD guidelines. This may be because there is a perception that the effect is limited to ingestion of large amounts of meat, using laboratory methods for creatinine which are outdated.

Widespread introduction of MDRD-based eGFR reporting by laboratories has highlighted the relatively high prevalence of CKD, a condition that has previously been poorly recognized and under-diagnosed. The Third National Health and Nutrition Examination Survey (NHANES III),[18] where specimens were taken from mainly fasting subjects[19] in a North American population, estimated stages 3–5 CKD at 4.7%. In a study of 41,051 adult patients in primary care practices in England, using non-fasting specimens for creatinine analysis and eGFR calculated using a creatinine method calibrated to the MDRD laboratory, the prevalence of CKD stages 3–5 was 9.7%.[20] It is likely that some of the difference in the prevalence of low eGFR in these studies is due to differences between the populations studied, but an underestimation of eGFR when sampling non-fasted subjects may also be making a significant contribution. Other studies seeking to evaluate the performance of MDRD-based eGFR have been undertaken, but have not specified whether participants had fasted or avoided cooked meat for a suitable time before sampling.[21–26] It is, therefore, not clear whether the results may have been affected by dietary intake.

From April 2006, general practitioners in the UK have been encouraged, through the Quality and Outcomes Framework, to maintain databases on patients with CKD stage 3 or worse, and laboratories are advised to report eGFR using the MDRD equation. As a result of the exponential nature of the MDRD-based eGFR calculation, a fixed increase in serum creatinine concentration will produce a greater reduction in eGFR in earlier stages of CKD. Depending on the results of other investigations, 12 of our 32 participants (seven volunteers and five Day Hospital patients) could have been allocated an incorrect CKD stage. Eleven of these changed from a preprandial eGFR of > 59 mL/min/1.73 m^2 to a postprandial eGFR of < 60 mL/min/1.73 m^2, thus potentially misclassifying them as CKD stage 3. This may result in additional investigations and referral of unsuitable patients to specialist renal clinics. Guidelines published by the Joint Specialty Committee on Renal Medicine[27] recommend a range of further laboratory and clinical investigations in patients identified as having CKD. The cost implications of mis-identification over a large population may be significant.

Cystatin C is a cysteine protease inhibitor of low molecular weight freely filtered at the glomerulus and catabolized in the renal tubules. It is a more sensitive and specific marker of renal dysfunction than serum creatinine.[28] Our study shows that serum cystatin C concentrations appear to be robust to the effects of both meat and non-meat-containing meals. The slight decrease in cystatin C following both types of meals is probably consistent with the known postprandial increase of true GFR as measured by inulin clearance.[16] This data further strengthen the case for the use of serum cystatin C as a screening test for CKD which could be reliably used in non-specialist settings.

The strengths of our study include the performance of creatinine measurement by two widely used modern laboratory methods, comparison to the reference method (IDMS), the inclusion of a wide age range of subjects of both genders, and the preparation of normal helpings of a range of meat-containing meals. Although conducted on small numbers of subjects, it was adequately powered to demonstrate a clear outcome and was performed with minimal inconvenience to participants.

A weakness of our study is that we did not extend the sampling period beyond 4 h after cooked meat intake. However, past studies have indicated that serum creatinine concentration may increase for up to 10 h.[12] Although we included a wide range of meat-containing meals, and compared the results with meals which clearly did not contain meat, we did not quantify the amount of meat in each meal, nor did we attempt to evaluate the complete range of dietary constituents. Also, classification of CKD using eGFR should not be based on a single result, but on results taken more than three months apart.

The extent of misclassification of CKD in clinical practice due to the use of MDRD-based eGFR calculations on blood samples taken after a meat-containing meal, if any, is currently unknown. We suggest that this would be a good topic for further research and audit. However, based on our results, we recommend that measurement of serum creatinine and calculation of eGFR for the purposes of diagnosis of CKD should be carried out when a patient has fasted or specifically avoided a cooked meat meal on the day of blood sampling. We would recommend that national practice guidelines, including forthcoming recommendations from the Scottish Intercollegiate Guidelines Network and National Institute of Health and Clinical Excellence,[29] should incorporate this recommendation.

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Additional Information

Ethical approval

Ethical approval was obtained from the Lanarkshire Ethics Committee on 28 June 2006 (REC reference number 06/S1001/49).

Competing interests

All authors declare that they have no conflict of interests relating to the publication of this article and therefore have nothing to declare.

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Appendix

Subject demographics and individual results of serum creatinine (3 methods), eGFR and cystatin C before and after a cooked-meat meal

No.	Age (years)	Sex	Timing (pp=postprandial)	Meal	Jaffe creatinine (μmol/L)	eGFR (mL/min/ 1 · 73 m^2)	CKD stage	IDMS creatinine (μmol/L)	Cystatin C (mg/L)	Enzymatic creatinine (μmol/L)
1	50	M	Preprandial	Chicken curry	101	72	<3	93	0.98	98
			1–2 h pp		133	52	3	118	1.00	122
			3–4 h pp		129	54	3	122	1.02	122
2	18	F	Preprandial	Steak pie, chips	57	135	<3	56	0.80	56
			1–2 h pp		77	92	<3	77	0.78	77
			3–4 h pp		74	97	<3	72	0.78	72
3	22	F	Preprandial	Steak pie, chips	64	112	<3	59	0.65	54
			1–2 h pp		80	84	<3	80	0.66	76
			3–4 h pp		81	83	<3	77	0.67	76
4	44	F	Preprandial	Beef sandwich	62	101	<3	56	0.61	53
			1–2 h pp		67	92	<3	61	0.79	58
			3–4 h pp		64	97	<3	61	0.63	60
5	21	F	Preprandial	Beef sandwich	64	113	<3	61	0.75	60
			1–2 h pp		72	97	<3	70	0.71	65
			3–4 h pp		71	99	<3	65	0.74	61
6	48	M	Preprandial	Beef curry	79	95	<3	77	0.76	79
			1–2 h pp		110	63	<3	106	0.75	105
			3–4 h pp		100	71	<3	103	0.73	101
7	78	F	Preprandial	Steak mince, potato,	118	41	3	111	2.45	111
			1–2 h pp	vegetables	128	37	3	124	2.31	122
			3–4 h pp		139	33	3	132	2.21	130
8	82	F	Preprandial	Steak mince, potato,	96	52	3	88	1.17	94
			1–2 h pp	vegetables	121	39	3	112	1.21	115
			3–4 h pp		130	36	3	124	1.16	124
9	79	F	Preprandial	Steak mince, potato,	58	98	<3	51	0.97	49
			1–2 h pp	vegetables	77	68	<3	66	0.91	66
			3–4 h pp		81	64	<3	74	0.87	77
10	84	F	Preprandial	Steak mince, potato,	73	72	<3	64	1.16	67
			1–2 h pp	vegetables	89	56	3	80	1.20	79
			3–4 h pp		102	48	3	91	1.18	90
11	75	F	Preprandial	Steak mince, potato,	153	30	3	148	1.76	141
			1–2 h pp	vegetables	167	27	4	164	1.82	156
			3–4 h pp		167	27	4	160	1.68	157
12	86	M	Preprandial	Steak mince, potato,	99	66	<3	97	1.36	90
			1–2 h pp	vegetables	108	60	<3	104	1.35	99
			3–4 h pp		108	60	<3	106	1.35	100
13	73	F	Preprandial	Roast beef, potato,	97	52	3	86	1.68	86
			1–2 h pp	soup	110	45	3	101	1.67	98
			3–4 h pp		119	41	3	108	1.77	106
14	77	F	Preprandial	Roast beef, potato,	67	82	<3	60	1.12	58
			1–2 h pp	soup	81	64	<3	75	1.14	72
			3–4 h pp		80	66	<3	76	1.15	70
15	77	F	Preprandial	Braised steak, potato	77	69	<3	65	1.21	70
			1–2 h pp		97	52	3	95	1.11	97
			3–4 h pp		96	52	3	87	1.14	90
16	76	M	Preprandial	Steak pie, vegetables,	100	67	<3	98	1.39	88
			1–2 h pp	soup	119	54	3	113	1.36	106
			3–4 h pp		124	52	3	115	1.34	116

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No.	Age (years)	Sex	Timing (pp=postprandial)	Meal	Jaffe creatinine (μmol/L)	eGFR (mL/min/ $1 \cdot 73\,m^2$)	CKD stage	IDMS creatinine (μmol/L)	Cystatin C (mg/L)	Enzymatic creatinine (μmol/L)
17	74	M	Preprandial	Steak mince, potato,	71	103	<3	65	1.07	64
			1–2 h pp	vegetables	69	107	<3	62	1.03	57
			3–4 h pp		72	102	<3	64	1.02	59
18	75	M	Preprandial	Steak mince, potato,	101	67	<3	94	1.20	93
			1–2 h pp	vegetables	116	56	3	107	1.11	105
			3–4 h pp		114	57	3	107	1.13	107
19	75	M	Preprandial	Roast beef, potato,	82	86	<3	78	1.13	75
			1–2 h pp	soup	84	84	<3	91	1.17	78
			3–4 h pp		86	81	<3	85	1.19	84
20	83	F	Preprandial	Chicken chasseur,	88	57	3	80	1.21	81
			1–2 h pp	potato, soup	89	57	3	77	1.11	77
			3–4 h pp		88	57	3	83	1.18	81
21	50	M	Preprandial	Steak mince, potato,	73	108	<3	65	0.93	62
			1–2 h pp	vegetables	90	83	<3	77	0.87	80
			3–4 h pp		94	79	<3	86	0.89	88
22	44	F	Preprandial	Braised steak, potato,	64	97	<3	57	0.67	56
			1–2 h pp	vegetables	101	55	3	99	0.65	93
			3–4 h pp		87	66	<3	85	0.65	86
23	38	F	Preprandial	Braised steak, chips	64	100	<3	60	0.73	60
			1–2 h pp		92	64	<3	91	0.74	84
			3–4 h pp		85	70	<3	89	0.73	89
24	36	F	Preprandial	Braised steak, chips	70	90	<3	64	0.76	61
			1–2 h pp		101	57	3	98	0.78	92
			3–4 h pp		91	65	<3	91	0.76	86
25	49	F	Preprandial	Pasta bolognaise	74	79	<3	71	0.72	68
			1–2 h pp		94	59	3	93	0.76	92
			3–4 h pp		103	53	3	105	0.75	100
26	60	M	Preprandial	Lamb curry, rice	93	77	<3	89	0.75	85
			1–2 h pp		122	55	3	124	0.75	115
			3–4 h pp		114	60	<3	110	0.74	103
27	23	M	Preprandial	Roast lamb, rice,	90	97	<3	93	0.74	83
			1–2 h pp	vegetables	103	83	<3	104	0.76	99
			3–4 h pp		99	87	<3	101	0.73	96
28	59	M	Preprandial	Lamb curry	101	70	<3	89	0.85	92
			1–2 h pp		162	39	3	159	0.81	154
			3–4 h pp		148	44	3	155	0.83	156
29	26	M	Preprandial	Steak, potato,	86	101	<3	81	0.73	84
			1–2 h pp	vegetables	104	80	<3	105	0.78	105
			3–4 h pp		99	85	<3	96	0.70	100
30	47	M	Preprandial	Lamb hotpot, potato,	87	88	<3	81	0.87	79
			1–2 h pp	vegetables	125	56	3	124	0.89	120
			3–4 h pp		123	58	3	116	0.83	119
31	43	M	Preprandial	Pasta bolognaise	94	81	<3	92	0.68	86
			1–2 h pp		109	68	<3	109	0.70	104
			3–4 h pp		114	64	<3	115	0.69	110
32	33	M	Preprandial	Pasta bolognaise	77	110	<3	73	0.72	68
			1–2 h pp		100	80	<3	98	0.69	94
			3–4 h pp		90	91	<3	102	0.70	95