

BMJ Open Change in prevalence of chronic kidney disease in England over time: comparison of nationally representative cross-sectional surveys from 2003 to 2010

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ABSTRACT

Objectives: To determine whether the prevalence of chronic kidney disease (CKD) in England has changed over time.

Design: Cross-sectional analysis of nationally representative Health Survey for England (HSE) random samples.

Setting: England 2003 and 2009/2010.

Survey participants: 13 896 adults aged 16+ participating in HSE, adjusted for sampling and non-response, 2009/2010 surveys combined.

Main outcome measure: Change in prevalence of estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m² (as proxy for stage 3–5 CKD), from 2003 to 2009/2010 based on a single serum creatinine measure using an isotope dilution mass spectrometry traceable enzymatic assay in a single laboratory; eGFR derived using Modified Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKDEPI) eGFR formulae.

Analysis: Multivariate logistic regression modelling to adjust time changes for sociodemographic and clinical factors (body mass index, hypertension, diabetes, lipids). A correction factor was applied to the 2003 HSE serum creatinine to account for a storage effect.

Results: National prevalence of low eGFR (<60) decreased within each age and gender group for both formulae except in men aged 65–74. Prevalence of obesity and diabetes increased in this period, while there was a decrease in hypertension. Adjustment for demographic and clinical factors led to a significant decrease in CKD between the surveyed periods. The fully adjusted OR for eGFR <60 mL/min/1.73 m² was 0.75 (0.61 to 0.92) comparing 2009/2010 with 2003 using the MDRD equation, and was similar using the CKDEPI equation 0.73 (0.57 to 0.93).

Conclusions: The prevalence of a low eGFR indicative of CKD in England appeared to decrease over this 7-year period, despite the rising prevalence of obesity and diabetes, two key causes of CKD. Hypertension prevalence declined and blood pressure control improved but this did not appear to explain the fall.

Strengths and limitations of this study

- This study used nationally representative samples, with later Health Surveys for England (HSEs) pooled over 2 years to increase the numbers and precision of estimates. The surveys used standardised protocols for measurement by trained interviewers and nurses, with all samples tested in the same laboratory with standardised assays.
- The analyses enable a longitudinal comparison of chronic kidney disease (CKD) estimates across different age–sex groupings and the application and comparison of the two equations to derive the estimated glomerular filtration rate (eGFR). Weighting was introduced for both surveyed periods to reduce response bias and account for missing data. A correction factor was applied to the 2003 HSE data to adjust for the shift in measured creatinine due to sample storage.
- The study was limited by the cross-sectional nature of the HSE, which restricts the ability to infer causal relationships from the associations identified. The study was also limited by the use of a single sample to test for serum creatinine in each survey; therefore, the persistence of reduced eGFR levels to confirm chronicity cannot be shown.
- The prevalence of stage 4/5 CKD is likely to be underestimated as the HSE may not fully account for some people in whom more severe CKD (stage 4/5) will be more common.
- The absence of albuminuria data in the 2003 HSE is another major limitation, given its strong independent association with adverse outcomes and its use to stratify risk for prevention and management (eg, use of renin-angiotensin system inhibition).

Periodic assessment of eGFR and albuminuria in future HSEs is needed to evaluate trends in CKD.

INTRODUCTION

Chronic kidney disease (CKD) is recognised as a global public health problem.¹ CKD is defined and staged using the estimated glomerular filtration rate (eGFR) and markers of kidney damage, mainly albuminuria.² Both eGFR and albuminuria are strong independent risk factors for all-cause and cardiovascular disease (CVD) mortality, and progression to end-stage renal disease (ESRD), which may require renal replacement therapy (RRT) by dialysis or transplantation.³ In 2010 in England, the prevalence of RRT was 832 per million population, a 3% increase from 2009; the National Health Service (NHS) costs of RRT were estimated at £780 million for 2009/2010, and the total cost at £1.45 billion, a nearly threefold increase on estimated costs for 2002.^{4 5}

The population prevalence of CKD in England was reported for the first time using data on eGFR and albuminuria in the nationally representative Health Surveys for England (HSEs) 2009 and 2010, though there had previously been estimates based on routine testing using primary care data.^{6 7} In the combined 2009/2010 HSEs, 6% of men and 7% of women had eGFR <60 mL/min/1.73 m² (equivalent to CKD stage 3–5 if chronic) with a strong age gradient.⁸ The prevalence of low eGFR increased in the USA, based on National Health and Nutrition Examination (NHANES) surveys between 1988 and 2004, even after adjusting for adverse trends in risk factors (obesity, diabetes, hypertension), but little is known about CKD prevalence trends in England.^{9–11}

Information on prevalence change is needed to assess the impact of trends in underlying determinants, and of strategies to prevent and manage CKD. Several policy initiatives have been introduced in England that have had an impact on the prevention, detection and management of CKD. The National Service Framework for Renal Services 2004/2005 led to the national reporting of eGFR by clinical biochemistry laboratories from 2006,¹² the General Practice pay for performance Quality Outcomes Framework (QOF) included targets for CKD management from 2006/2007,¹³ and the NHS Vascular Checks Programme, introduced in 2009, includes screening for CKD (stage 3–5) in people aged 35–74 with newly identified type 2 diabetes or hypertension.¹⁴ This study therefore aimed to compare the prevalence of CKD in HSE 2003 with the combined 2009/2010 HSEs and to relate this to any changes in prevalence of risk factors for CKD, particularly obesity, diabetes and hypertension, over this period.

METHODS

Full details of the conduct of the HSE, and the measurement of non-CKD variables and response rates, are shown in the 2003 and 2009 Health Survey for England reports.^{15 16} Survey participants within private households were selected using a multistage stratified random probability sample. Household response rates were 73%

in HSE 2003 and 68%/66% in HSE 2009/2010. In co-operating households, 90% and 89%/86% of adults completed an interview questionnaire while 70% and 62%/57% consented to a nurse visit, of whom 74–76% provided a blood test. The HSE 2003 contained 18 533 individuals, and data from HSE 2009 and HSE 2010 were combined to provide a sample size of 13 065 individuals. This totalled 31 598 individuals for the combined 2003, 2009 and 2010 HSEs. Eligible participants were individuals aged 16 and older who had a valid serum creatinine value. This left 7850 individuals from the 2003 HSE and 6046 individuals from the combined 2009/2010 HSEs, a total of 13 896 individuals for analysis.

Age was grouped into five categories: 16–34, 35–54, 55–64, 65–74 and 75+. There were four separate ethnic groupings: White, South Asian, Black and Other. Socioeconomic factors included: (1) occupation—National Statistics Socio-Economic Classification (NS-SEC, divided into three categories: managerial and professional occupations, intermediate occupations and routine and manual occupations); (2) qualifications grouped as: degree or equivalent; below degree (other qualification) and none (no qualification); (3) household tenure (own vs renting) and (4) access to motor vehicle (none vs any).

Smoking status was defined as current, ex-smoker or never smoked. Hypertension was defined as doctor-diagnosed (pre-existing diagnosis), survey-defined (identified as having high blood pressure (BP, systolic ≥ 140 mm Hg and/or diastolic ≥ 90 mm Hg and/or taking medication for hypertension) at the survey examination), and 'total' (doctor+survey diagnosed). Survey-defined diabetes was glycated haemoglobin (HbA1c) $\geq 6.5\%$ at nurse visit. HbA1c data are presented for those with and without diagnosed diabetes. Body mass index (BMI) was defined as normal (<25 kg/m²), overweight (≥ 25 and <30 kg/m²) and obese (≥ 30 kg/m²). Waist circumference was classified as: <94, 94–102 (high) and >102 cm (very high) for men, and <80, 80–88 (high) and >88 cm (very high) for women. For South Asian men, the waist circumference was classified as: <90, 90–102 (high) and >102 cm (very high). High-density lipoprotein (HDL) cholesterol and total cholesterol were treated as continuous variables.

To investigate medication use, we examined the use of diuretics, β -blockers, renin-angiotensin system (RAS) inhibitors (ACE inhibitors and angiotensin receptor blockers (ARBs)), calcium-channel blockers, other anti-hypertensives in those with doctor-diagnosed hypertension, doctor-diagnosed diabetes and eGFR <60 mL/min/1.73 m², and use of lipid lowering drugs (the majority of which are statins) in the whole population. In 2003, 47% of respondents answered yes to whether they were taking any prescribed medication, and 50% in 2009/2010.

Serum creatinine was assayed using an isotope dilution mass spectrometry (IDMS) traceable enzymatic assay in

a single laboratory (Clinical Biochemistry Department at the Royal Victoria Infirmary, Newcastle Upon Tyne). Both the Modified Diet in Renal Disease (MDRD) equation (in routine use in the UK) and the newer Chronic Kidney Disease Epidemiology Collaboration (CKDEPI) equation (which provides better risk prediction and is recommended for use in international guidelines) were used to define CKD.^{2 17} eGFR values were derived using the standard equations.^{18 19}

Details of laboratory analysis, internal quality control and external quality assurance are provided in the HSE 2009/2010 documentation, with these methods replicated in analysis of the 2003 HSE samples.⁸

Samples were assayed for serum creatinine over a 19-month period with two different batches of the tri-level Internal Quality Control (IQC) material. HSE 2009 and 2010 samples were analysed with Batch 1 or Batch 2 IQC, while HSE 2003 samples were analysed with Batch 2 IQC. The creatinine assay was stable over time with IQC results very close to the expected target values. Batch 1 IQC gave mean (SD) creatinine concentrations of 56 (0.6), 167 (1.3) and 586 (4.9) $\mu\text{mol/L}$ for levels 1, 2 and 3, respectively, compared with target means of 56, 167 and 588 $\mu\text{mol/L}$. Batch 2 material gave mean (SD) creatinine concentrations of 51 (1.1), 175 (2.2) and 597 (5.6) $\mu\text{mol/L}$ for levels 1, 2 and 3, respectively, compared with target means of 51, 175 and 599 $\mu\text{mol/L}$.

The HSE 2003 samples had been stored, frozen at -40°C , and then thawed for measurement in 2010. Although such freezing is not thought to affect creatinine levels,²⁰ we undertook a reanalysis in 2014 of a random sample of 500 serum creatinine samples taken from the 2009 HSE and subsequently frozen and stored under the same conditions as the HSE 2003 samples, stratified by quintile, to determine if there was a shift in measured creatinine on storage. We found that mean serum creatinine increased on storage and was best predicted by a regression equation where the original 2009 serum creatinine value without storage equalled 0.303 plus 0.94 multiplied by the stored serum creatinine value. We assumed that the same effect applied to the 2003 serum creatinine data which were analysed in 2009–2010, and we applied the same adjustment. This decreased the 2003 serum creatinine values. eGFR was classified as below 60 mL/min/1.73 m² or equal to or greater than 60 mL/min/1.73 m². We compared the change in mean serum creatinine in people aged 20–39 without any diabetes or any hypertension as per Coresh *et al.*⁹

STATISTICS

Patient characteristics were compared between the 2003 and 2009/2010 HSEs using χ^2 tests for categorical variables and Mann-Whitney U tests for non-normally distributed continuous variables. eGFR <60 mL/min/1.73 m² prevalence in 2003 and 2009/2010 was compared across age and sex groupings. BP levels were compared in all participants, in those with diagnosed

hypertension and in those with eGFR <60 mL/min/1.73 m²; HbA1c was compared in all participants and in those with doctor-diagnosed diabetes. Binary logistic regression models were used to examine the relationships between eGFR <60 mL/min/1.73 m² and age, sex and socioeconomic and clinical factors to determine if there were significant differences between the two survey periods. The dependent variables were CKDEPI and the MDRD equation eGFR <60 mL/min/1.73 m² (indicative of stage 3–5 CKD). Four models were produced for each: (1) age–sex adjusted; (2) model 1 plus socioeconomic status and ethnicity; (3) model 2 plus behavioural, lipid levels (HDL and total cholesterol) and clinical variables except hypertension and (4) model 3 plus doctor-diagnosed hypertension. Interactions between period and both diabetes and hypertension were tested.

Sensitivity analyses were performed by replacing doctor-diagnosed diabetes with HbA1c, replacing doctor-diagnosed hypertension with diastolic and systolic BP and adjusting for lipid lowering agents in the full model. Non-response and blood sample weights were used in all analyses to address issues with missing individuals who did not have a blood sample taken and sent to the laboratory for analysis to determine the serum creatinine value. Full details on how the weights were obtained are provided in the final volume of the HSE report each year. The age, education and smoking status of those interviewed, having a nurse visit and having a blood test is similar once non-response is taken into account (data not shown). All analyses were performed using IBM SPSS Statistics V.20.

RESULTS

The final sample for the study comprised 13 896 individuals aged 16+ who had a valid serum creatinine value. Comparing the characteristics of these participants between the 2003 and 2009/2010 surveys, the age structure, gender, NS-SEC and car ownership were similar while the educational level improved and there was an increase in rented tenure (table 1). The prevalence of diabetes however classified increased, as did obesity. In contrast, smoking and hypertension prevalence decreased.

There were significant increases in BMI, waist circumference and HbA1c in the population, although there was no change in HbA1c in those with diagnosed diabetes (table 2). Median BP levels (systolic and diastolic) fell in all groups including those with diagnosed hypertension, doctor-diagnosed diabetes and eGFR <60 mL/min/1.73 m². Median total and HDL cholesterol fell in men and women.

The distribution of serum creatinine is similar for 2003 and 2009/2010 (figure 1). Median serum creatinine increased slightly, leading to a very small non-significant decrease in median eGFR using MDRD and CKDEPI formulae (table 2). Mean serum creatinine for

Table 1 Comparison of prevalence of categorical measures in 2003 and 2009/2010*

Variable	Category	2003		2009–2010		χ^2 Test p Value
		Number	Per cent	Number	Per cent	
All	Aged 16+	7850†	100.0	6046†	100.0	–
Age	16–34	2425	31.0	1847	30.6	0.44
	34–54	2790	35.7	2129	35.3	
	55–64	1126	14.4	886	14.7	
	65–74	813	10.4	639	10.6	
	75+	662	8.5	539	8.9	
Ethnicity	Missing	0	–	0	–	<0.001
	White	7226	92.5	5244	90.7	
	South Asian	332	4.3	243	4.2	
	Black	144	1.8	154	2.7	
	Other	108	1.4	139	2.4	
	Missing	0	–	0	–	
Sex	Male	3795	48.6	2961	49.0	0.80
	Female	4020	51.4	3080	51.0	
	Missing	0	–	0	–	
Qualification	Degree	1375	17.6	1295	22.5	<0.001
	Below degree	4551	58.3	3296	57.0	
	None	1874	24.0	1191	20.6	
	Missing	11	–	3	–	
NS-SEC	Highest	2514	33.7	1894	34.8	0.43
	Middle	1674	22.4	1203	22.1	
	Lowest	3273	43.9	2343	43.1	
	Missing	350	–	345	–	
Car ownership	Yes	6460	82.7	4728	81.7	0.17
	No	1348	17.3	1056	18.3	
	Missing	2	–	1	–	
Tenure	Own	5878	75.4	3955	68.5	<0.001
	Rent	1914	24.6	1817	31.5	
	Missing	11	–	13	–	
Smoking	Current	1960	25.2	1210	21.0	<0.001
	Ex	1877	24.1	1429	24.8	
	Never	3951	50.7	3126	54.2	
	Missing	22	–	20	–	
Body mass index	Normal/underweight (<25 kg/m ²)	2867	39.2	1956	36.8	<0.001
	Overweight (25–30 kg/m ²)	2868	39.2	2047	38.5	
	Obese (>30 kg/m ²)	1587	21.7	1314	24.7	
	Missing	489	–	469	–	
Waist circumference	Low (<94 cm male, <80 cm female)	3060	39.8	2120	37.1	<0.001
	High (94–102 cm male, 80–88 cm female)	1929	25.1	1347	23.6	
	Very High (>102 cm male, >88 cm female)	2703	35.1	2242	39.3	
	Missing	118	–	77	–	
Doctor-diagnosed diabetes	Yes	305	3.9	322	5.3	<0.001
	No	7504	96.1	5715	94.7	
	Missing	6	–	2	–	
Survey-diagnosed diabetes	Yes (HBA1c ≥6.5%)	296	3.8	316	5.5	<0.001
	No (HBA1c <6.5%)	7401	96.2	5417	94.5	
	Missing	113	–	52	–	
Total diabetes	Yes	406	5.2	446	7.4	<0.001
	No	7405	94.8	5585	92.6	
	Missing	0	–	0	–	
Doctor-diagnosed hypertension	Yes	2118	27.2	1501	25.0	0.003
	No	5662	72.8	4527	75.0	
	Missing	36	–	10	–	
Survey-diagnosed hypertension	Yes	2065	31.5	1545	29.2	0.02
	No	4499	68.5	3744	70.8	
	Missing	1246	–	496	–	

Continued

Table 1 Continued

Variable	Category	2003		2009–2010		χ^2 Test p Value
		Number	Per cent	Number	Per cent	
Total hypertension	Yes	2866	36.7	2062	34.2	0.004
	No	4933	63.3	3968	65.8	
	Missing	12	—	0	—	
eGFR CKDEPI	<45 (mL/min/1.73 m ²)	142	1.8	81	1.4	0.07
	<60 (mL/min/1.73 m ²)	444	5.7	303	5.2	0.26
	Missing	0	—	0	—	
eGFR MDRD	<45 (mL/min/1.73 m ²)	146	1.9	80	1.4	0.03
	<60 (mL/min/1.73 m ²)	521	6.7	349	6.0	0.13
	Missing	0	—	0	—	

Bold typeface indicates significance at $p < 0.05$.

*Weighted for non-response (unless stated otherwise).

†Not weighted.

CKDEPI, Chronic Kidney Disease Epidemiology Collaboration; eGFR, estimated glomerular filtration rate; HBA1c, glycated haemoglobin; MDRD, Modified Diet in Renal Disease; NS-SEC, National Statistics Socio-Economic Classification.

those aged 20–39 without doctor-diagnosed hypertension or diabetes increased slightly from 70.6 $\mu\text{mol/L}$ ($\text{SD} \pm 13.6$) in 2003 to 71.4 $\mu\text{mol/L}$ ($\text{SD} \pm 14.3$) in 2009/2010 ($p = 0.09$).

The proportion of individuals with MDRD eGFR <60 mL/min/1.73 m² decreased from 6.7% in 2003 to 6% in 2009/2010 ($p = 0.13$) and in those with

eGFR <45 mL/min/1.73 m² from 1.9% to 1.4% ($p = 0.03$). The corresponding figures for CKDEPI were 5.7% and 5.2% ($p = 0.26$) and 1.8% and 1.4% ($p = 0.07$), respectively. The prevalence of low eGFR fell in all age and gender groups and with either CKDEPI or MDRD equations, except in men aged 65–74 in whom there was a slight increase (figure 2).

Table 2 Weighed comparison of continuous measures in 2003 and 2009–2010

Variable	Category	2003 Median value (IQR)	2009–2010 Median value (IQR)	Mann-Whitney U test p Value
Serum creatinine ($\mu\text{mol/L}$)	Median value	71.7 (62.3–82.1)	72.0 (62.0–83.0)	0.66
eGFR (mL/min/1.73 m ²)	MDRD	90.5 (77.2–105.4)	90.3 (77.1–104.7)	0.62
	CKDEPI	99.3 (84.1–113.9)	98.6 (84.0–112.5)	0.11
	All	26.2 (23.3–29.4)	26.6 (23.5–30.0)	<0.001
BMI (kg/m ²)	Male	26.6 (24.0–29.4)	27.0 (24.2–29.9)	0.001
	Female	25.7 (22.7–29.5)	26.1 (23.1–30.0)	<0.001
	All	90.6 (81.1–100.0)	92.0 (81.6–101.7)	<0.001
Waist circumference (cm)	Male	95.8 (88.0–104.0)	96.7 (88.2–105.0)	0.05
	Female	84.6 (76.4–94.0)	86.3 (77.3–96.7)	<0.001
	All	125.5 (115.5–138.0)	124.5 (114.0–136.0)	<0.001
Systolic BP (mm Hg)	Doctor-diagnosed HT	135.5 (124.0–149.5)	134.0 (122.2–145.5)	<0.001
	Doctor-diagnosed DM	134.5 (122.5–148.0)	131.8 (120.0–143.5)	<0.001
	eGFR <60 (CKDEPI)	140.2 (126.0–156.0)	131.8 (119.0–143.5)	<0.001
	eGFR <60 (MDRD)	137.5 (124.0–153.7)	129.2 (118.0–142.5)	<0.001
	All	73.0 (65.5–80.5)	72.5 (65.5–80.5)	<0.001
	Doctor-diagnosed HT	77.5 (70.0–85.5)	76.0 (68.0–83.5)	<0.001
Diastolic BP (mm Hg)	Doctor-diagnosed DM	72.0 (64.5–80.5)	71.50 (64.5–78.5)	<0.001
	eGFR <60 (CKDEPI)	71.5 (62.9–80.5)	68.5 (60.5–76.0)	<0.001
	eGFR <60 (MDRD)	72.5 (64.0–81.5)	69.0 (61.5–76.8)	<0.001
	All	5.20 (5.00–5.50)	5.30 (5.10–5.70)	<0.001
	Doctor-diagnosed DM	6.90 (5.90–8.20)	6.90 (5.90–8.30)	0.85
	All	1.50 (1.20–1.70)	1.40 (1.20–1.70)	<0.001
HDL cholesterol (mmol/L)	Male	1.30 (1.20–1.60)	1.30 (1.10–1.50)	<0.001
	Female	1.60 (1.40–1.90)	1.60 (1.30–1.90)	0.046
	All	5.40 (4.70–6.20)	5.20 (4.40–5.90)	<0.001
Total cholesterol (mmol/L)	Male	5.40 (4.70–6.20)	5.10 (4.30–5.90)	<0.001
	Female	5.40 (4.70–6.20)	5.20 (4.50–6.00)	0.001

Bold typeface indicates significance at $p < 0.05$.

BP, blood pressure; Hb, haemoglobin; CKDEPI, Chronic Kidney Disease Epidemiology Collaboration; DM, diabetes mellitus; eGFR estimated glomerular filtration rate; HDL, high-density lipoprotein; HT, hypertension; IQR, interquartile range; MDRD, Modified Diet in Renal Disease.

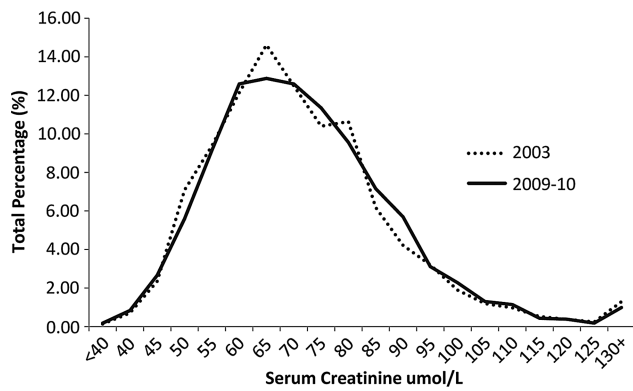


Figure 1 Distribution of serum creatinine ($\mu\text{mol/L}$) for the 2003 and 2009/2010 survey data. Serum creatinine categories are grouped in bands of $5 \mu\text{mol/L}$ from $40 \mu\text{mol/L}$ to $130 \mu\text{mol/L}$. Serum creatinine values $<40 \mu\text{mol/L}$ and those $>130 \mu\text{mol/L}$ are grouped together.

There was an increase in the mean number of antihypertensive agents taken in individuals with: doctor-diagnosed hypertension (1.19 in 2003 to 2.01 in 2009/2010), doctor-diagnosed hypertension and doctor-diagnosed diabetes (1.47–2.57); MDRD $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ (1.30–1.77); and CKDEPI $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$

(1.35–1.93). The proportion taking RAS inhibitors in individuals with doctor-diagnosed diabetes, doctor-diagnosed hypertension, MDRD $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ or CKDEPI $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ also increased, as did overall lipid lowering agent use (online supplementary appendix 1).

The age–sex adjusted OR of having low eGFR (MDRD $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$) in 2009/2010 compared with 2003 was 0.84 (95% CI 0.72 to 0.98) and fully adjusted was 0.75 (0.61 to 0.92; [table 3](#)). The corresponding ORs for CKDEPI were 0.85 (0.72 to 1.00) and 0.73 (0.57 to 0.93; [table 4](#)).

Sensitivity analyses replacing doctor-diagnosed diabetes with HbA1c and doctor-diagnosed hypertension with diastolic and systolic BP made little difference to the adjusted ORs, as did the inclusion of lipid lowering agents. No interactions between period and diabetes or hypertension were identified.

DISCUSSION

These analyses show that CKD prevalence in England estimated by serum creatinine-based equations in England appeared to decrease from 2003 to 2009/2010. This decrease was seen across all age groupings (except in men

Figure 2 Comparison of low eGFR ($< 60 \text{ mL/min/1.73 m}^2$) prevalence difference for MDRD and CKDEPI equations between the 2003 and 2009/2010 HSEs for each age group by gender. CKD, chronic kidney disease; CKDEPI, Chronic Kidney Disease Epidemiology Collaboration; eGFR estimated glomerular filtration rate; HSE, Health Survey for England; MDRD, Modified Diet in Renal Disease.

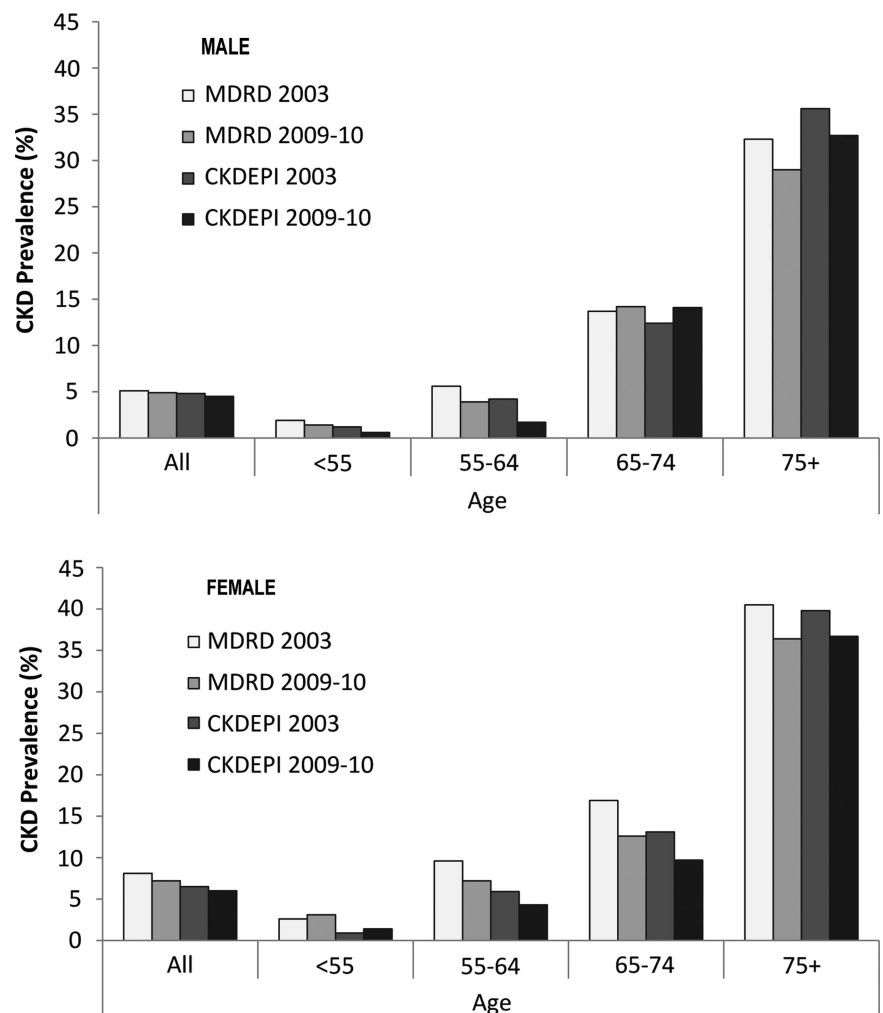


Table 3 Prevalence and associations of low eGFR (<60 mL/min/1.73 m²) by MDRD equation with adjustment for sociodemographic and clinical factors

Variable	MDRD				
	Prevalence of CKD (%)†	OR (95% CI)‡	OR (95% CI)§	OR (95% CI)¶	OR (95% CI)††
HSE year					
2003	6.7	1	1	1	1
2009–2010	6.0	0.84 (0.72 to 0.98)*	0.84 (0.73 to 0.99)*	0.75 (0.61 to 0.92)**	0.75 (0.61 to 0.92)**
Age					
16–34	0.2	1	1	1	1
35–54	2.1	10.8 (5.3 to 22.0)**	11.1 (5.5 to 22.8)**	10.8 (5.0 to 23.4)**	10.7 (4.9 to 23.2)**
55–64	6.8	37 (18 to 75)**	36 (18 to 73)**	33 (15 to 72)**	32 (14 to 69)**
65–74	14.5	87 (43 to 175)**	82 (40 to 167)**	65 (30 to 143)**	62 (28 to 135)**
75+	35.6	276 (138 to 555)**	247 (122 to 501)**	216 (99 to 470)**	202 (93 to 440)**
Sex					
Male	5.0	1	1	1	1
Female	7.7	1.42 (1.22 to 1.65)**	1.37 (1.17 to 1.60)**	1.69 (1.36 to 2.10)**	1.66 (1.34 to 2.06)**
Ethnic					
White	6.8	–	1	1	1
South Asian	1.7	–	0.83 (0.43 to 1.59)	0.73 (0.33 to 1.60)	0.71 (0.32 to 1.56)
Black	1.7	–	0.38 (0.15 to 1.00)	0.33 (0.09 to 1.19)	0.32 (0.09 to 1.16)
Other	1.6	–	0.81 (0.29 to 2.30)	0.64 (0.17 to 2.46)	0.64 (0.17 to 2.44)
Tenure					
Own	6.3	–	1	1	1
Rent	6.6	–	1.34 (1.11 to 1.60)**	1.23 (0.97 to 1.57)	1.23 (0.96 to 1.56)
Education					
Degree level	2.4	–	1	1	1
Below degree	4.4	–	1.31 (0.99 to 1.74)	1.15 (0.83 to 1.60)	1.20 (0.84 to 1.70)
None	14.9	–	1.52 (1.13 to 2.04)**	1.20 (0.84 to 1.70)	1.23 (0.96 to 1.57)
Smoking					
Never	6.1	–	–	1	1
Ex-smoker	10.1	–	–	1.17 (0.91 to 1.49)	1.15 (0.85 to 1.54)
Current smoker	3.2	–	–	1.02 (0.75 to 1.38)	1.04 (0.80 to 1.40)
BMI (kg/m ²)					
Normal (<25)	3.4	–	–	1	1
Overweight (25–30)	6.6	–	–	1.16 (0.91 to 1.49)	1.15 (0.90 to 1.47)
Obese (>30)	8.4	–	–	1.31 (0.99 to 1.71)	1.26 (0.96 to 1.65)
HDL cholesterol					
Continuous	–	–	–	0.51 (0.38 to 0.67)**	0.51 (0.38 to 0.68)**
Total cholesterol					
Continuous	–	–	–	0.91 (0.84 to 1.00)	0.92 (0.84 to 1.00)
Doctor-diagnosed diabetes					
No	5.9	–	–	1	1
Yes	17.3	–	–	1.42 (0.92 to 2.22)	1.36 (0.87 to 2.11)
Doctor-diagnosed hypertension					
No	4.0	–	–	–	1
Yes	13.3	–	–	–	1.27 (1.03 to 1.55)*

*p<0.05; **p<0.01.

†Prevalence for the combined 2003 and 2009–2010 HSEs.

‡Adjusted for age and sex.

§Adjusted for age, sex, ethnicity, tenure and education.

¶Adjusted for age, sex, ethnicity, tenure, education, smoking, BMI, HDL cholesterol, total cholesterol and doctor-diagnosed diabetes.

††Adjusted for age, sex, ethnicity, tenure, education, smoking, BMI, HDL cholesterol, total cholesterol, doctor-diagnosed diabetes and doctor-diagnosed hypertension.

BMI, body mass index; CKD, chronic kidney disease; eGFR estimated glomerular filtration rate; HDL, high-density lipoprotein; HSE, Health Survey for England; MDRD, Modified Diet in Renal Disease.

aged 65–74), for CKD defined by MDRD and CKDEPI eGFR equations was more pronounced for the MDRD equation and occurred despite the increased prevalence of diabetes and obesity.²¹ Using the CKDEPI equation in

place of MDRD to define CKD resulted in a lower prevalence of CKD. While it reduced overall prevalence, the CKDEPI equation identified more individuals aged 75+ with CKD compared with the MDRD equation.^{22 23}

Table 4 Prevalence and associations of low eGFR (<60) by CKDEPI equation with adjustment for sociodemographic and clinical factors

Variable	CKDEPI				
	Prevalence of CKD (%)†	OR (95% CI)‡	OR (95% CI)§	OR (95% CI)¶	OR (95% CI)††
HSE year					
2003	5.7	1	1	1	1
2009–2010	5.2	0.85 (0.72 to 1.00)	0.86 (0.72 to 1.01)	0.73 (0.57 to 0.93)*	0.73 (0.57 to 0.93)*
Age					
16–34	0.1	1	1	1	1
35–54	1.0	15.1 (4.5 to 51.4)**	16.1 (4.7 to 55.0)**	13.8 (4.0 to 47.2)**	13.5 (3.9 to 46.5)**
55–64	4.2	67 (20 to 225)**	67 (20 to 227)**	55 (16 to 190)**	52 (15 to 177)**
65–74	12.5	219 (66 to 725)**	219 (65 to 731)**	162 (47 to 553)**	151 (44 to 517)**
75+	36.8	890 (269 to 2938)**	844 (253 to 2808)**	754 (222 to 2559)**	693 (203 to 2355)**
Sex					
Male	4.6	1	1	1	1
Female	6.3	1.15 (0.97 to 1.36)	1.11 (0.93 to 1.31)	1.31 (1.01 to 1.68)*	1.28 (1.00 to 1.65)*
Ethnic					
White	5.8	–	1	1	1
South Asian	1.4	–	0.93 (0.43 to 2.00)	0.89 (0.35 to 2.42)	0.85 (0.34 to 2.16)
Black	2.0	–	0.56 (0.23 to 1.39)	0.55 (0.16 to 1.82)	0.53 (0.16 to 1.77)
Other	1.6	–	1.19 (0.40 to 3.56)	1.13 (0.29 to 4.41)	1.13 (0.29 to 4.41)
Tenure					
Own	5.3	–	1	1	1
Rent	6.0	–	1.44 (1.19 to 1.75)**	1.29 (0.98 to 1.69)	1.28 (0.97 to 1.69)
Education					
Degree level	1.8	–	1	1	1
Below degree	3.6	–	1.36 (0.97 to 1.90)	1.05 (0.69 to 1.58)	1.04 (0.69 to 1.58)
None	13.6	–	1.51 (1.08 to 2.13)*	1.12 (0.76 to 1.66)	1.11 (0.75 to 1.65)
Smoking					
Never	5.2	–	–	1	1
Ex-smoker	9.0	–	–	1.09 (0.77 to 1.55)	1.07 (0.75 to 1.52)
Current smoker	2.6	–	–	0.80 (0.70 to 1.41)	0.79 (0.54 to 1.14)
BMI (kg/m ²)					
Normal (<25)	2.7	–	–	1	1
Overweight (25–30)	5.5	–	–	1.14 (0.86 to 1.51)	1.12 (0.85 to 1.49)
Obese (>30)	7.2	–	–	1.31 (0.96 to 1.80)	1.25 (0.91 to 1.72)
HDL cholesterol					
Continuous	–	–	–	0.40 (0.29 to 0.56)**	0.40 (0.29 to 0.57)**
Total cholesterol					
Continuous	–	–	–	0.93 (0.84 to 1.04)	0.94 (0.86 to 1.04)
Doctor-diagnosed diabetes					
No	5.0	–	–	1	1
Yes	16.3	–	–	1.55 (0.96 to 2.48)	1.46 (0.91 to 2.35)
Doctor-diagnosed hypertension					
No	3.1	–	–	–	1
Yes	12.3	–	–	–	1.33 (1.05 to 1.67)**

*p<0.05; **p<0.01.

†Prevalence for the combined 2003 and 2009–2010 HSEs.

‡Adjusted for age and sex.

§Adjusted for age, sex, ethnicity, tenure and education.

¶Adjusted for age, sex, ethnicity, tenure, education, smoking, BMI, HDL cholesterol, total cholesterol and doctor-diagnosed diabetes.

††Adjusted for age, sex, ethnicity, tenure, education, smoking, BMI, HDL cholesterol, total cholesterol, doctor-diagnosed diabetes and doctor-diagnosed hypertension.

BMI, body mass index; CKD, chronic kidney disease; CKDEPI, Chronic Kidney Disease Epidemiology Collaboration; eGFR estimated glomerular filtration rate; HDL, high-density lipoprotein; HSE, Health Survey for England.

The 2003, 2009 and 2010 HSEs were nationally representative samples, with the 2009/2010 data pooled over 2 years to increase the numbers and precision of estimates. The age–sex characteristics of the different study

periods sampled were similar. The surveys used standardised protocols for measurement by trained interviewers and nurses. All samples were tested in the same laboratory with standardised assays. The analyses enable a

longitudinal comparison of CKD estimates across different age–sex groupings and the application and comparison of the two equations to derive eGFR. We accounted for the shift in measured creatinine on storage in the 2003 HSE serum creatinine data by introduction of a correction factor derived from analysis of the effect of storage using 2009 data. Non-response weighting was undertaken in the HSE for both surveyed periods to reduce response bias and account for missing data for individuals who did not have blood samples taken and hence had no serum creatinine value. We used the HSE study design and the non-response weights to provide national prevalence estimates at each period and adjusted for a wide range of sociodemographic factors, so residual confounding due to differences in sample characteristics is less likely. Moreover, the distribution of age groupings was similar for both periods; hence, it does not explain the observed eGFR differences. The ethnic composition of the surveys changed over time with a small fall in the white population, but we adjusted for this change in the analysis.

The study was limited by the cross-sectional nature of the HSE, which restricts the ability to infer causal relationships from the associations identified. However, the use of new, cross-sectional samples enables the measurement of general population CKD prevalence at different time points. A single sample was tested for serum creatinine in each survey, and therefore the persistence of reduced eGFR levels to confirm chronicity cannot be shown. This is standard practice in national surveys such as NHANES, whereas studies based on routine testing, such as the QICKD study, can assess chronicity.²⁴ Given the individual variation in kidney function, more extreme values will be averaged out on repeated testing (regression to the mean), reducing the prevalence of low eGFR.²⁵ The results may therefore slightly overestimate the prevalence of CKD. There were very few cases from the key minority ethnic groups to give robust data on ethnic differences in the prevalence of CKD. South Asians and black groups have higher rates of renal replacement but have been found to have a lower prevalence of CKD than Caucasians.^{26 27}

The prevalence of stage 4/5 CKD is likely to be underestimated as, while the HSE is able to adjust for non-response among the general population in private households, it may not fully account for some in whom more severe CKD (stage 4/5) will be more common. This includes people who were not able to give a blood or urine sample because of poor health and those who did not participate due to concurrent illness or hospitalisation, as well as those in residential care.

The absence of albuminuria data in the 2003 HSE is a major limitation, given its strong independent association with adverse outcomes and its use to stratify risk for prevention and management (eg, use of RAS inhibition).³ We have therefore been unable to estimate changes in the prevalence of albuminuria per se, in all CKD (stages 1–5), and fully assess prevention and management.

The fall in low prevalence of eGFR could be due to (1) chance, (2) the artefact of differences in the serum creatinine measurement, (3) changes in serum creatinine production rather than excretion by the kidney, (4) residual confounding by differences in sample characteristics not adjusted for by sample weighting and (5) the true fall in eGFR. The two sets of samples were analysed in multiple analytical runs over a 19-month time period, which could lead to differences in results; however, during this time period, the internal quality control data indicate that the assay was accurate when compared with the assigned target values and stable. We found a storage artefact on serum creatinine measurement and accounted for this by the introduction of a correction factor. A change in serum creatinine over time independent of the kidney function could be due to less muscle mass (leading to lower serum creatinine production); there is no evidence for this and it seems unlikely to have occurred at the population level.

A decline in dietary protein consumption from cooked meat could also lead to a change in serum creatinine. Statistics from the National Diet and Nutrition Survey show that meat consumption increased from 2001–2002 to 2008–2010 while protein intake remained virtually stable over the same period.²⁸ The mean consumption of meat and meat products increased from 154 g/day in 2001–2002 to 194 g/day in 2008–2010; protein intake contributing to food energy for adults aged 19+ increased slightly from 16–17% in 2001–2002 to 17–18% in 2008–2010; meat and meat products contributed to 37–38% of all protein intake for adults aged 19–64, with little change compared to 2008–2010. Cooked meat consumption has been shown to increase serum creatinine in small case studies of volunteers and of patients with diabetic nephropathy, and hence national guidance is to avoid eating cooked meat for 12 h before a blood test for creatinine,²⁹ but this was not done in HSE.

We used the HSE study design and non-response weights and adjusted for a wide range of sociodemographic factors, so residual confounding due to differences in sample characteristics differences is less likely. Moreover, the distribution of age groupings was similar for both periods, so it does not explain the observed eGFR differences.

Key risk groups for developing CKD are people with hypertension and/or diabetes, especially if they have albuminuria. In this study, there was evidence of modest reductions in the prevalence of hypertension, better control of hypertension in key groups, and greater use of RAS inhibitors that have antiproteinuric as well as BP lowering effects, though the period changes in eGFR remained after correction for changes in hypertension prevalence. There is evidence from some studies using HSE, primary care databases and QOF data,^{30–32} though not all,³³ of improved hypertension control in the past decade. However, there are ethnic disparities with poorer control of BP in Black and South Asians who

have a higher risk of progression to need RRT.³⁴ The population salt consumption also fell during the past decade, which is likely to have influenced population BP.^{35–36} CKD prevalence could also fall if those identified with moderate CKD were treated more aggressively, especially those with hypertension and/or albuminuria, leading to increased eGFR in some people to above 60 mL/min/1.73 m². The limited HSE data suggest better BP control and greater use of RAS inhibitors in those with eGFR <60 mL/min/1.73 m². Karunaratne *et al* examined BP control in those with and without CKD in a primary care population in Kent and showed that BP control had improved in patients with CKD over time pre and post the introduction of QOF and that it was greater than in non-CKD patients with hypertension. They also showed increased use of RAS inhibitors and other antihypertensive agents in patients with CKD.³⁷

There was evidence of increased lipid lowering agent use (indicative of increased statin use) and a small fall in population lipid levels. There is some evidence of reno-protective effects of statins in patients with CKD; A lower rate of decline in GFR was found in patients with renal disease who took antilipemic agents.³⁸ In the Heart Protection Study, the use of the hypolipidemic drug simvastatin reduced the rise in slightly elevated creatinine over time in participants with diabetes and non-diabetic CKD.³⁹ In the SHARP trial, allocation of the lipid lowering ezetimibe plus simvastatin in participants not already on dialysis at randomisation reduced the outcome of ESRD or a doubling of creatinine with an OR of 0.93, though this was not statistically significant.⁴⁰ In the GREACE trial, statin treatment prevented a decline in renal function in people with high blood lipids and coronary heart disease; patients not treated with statins showed a 5.2% decrease in creatinine clearance, while patients treated with statins showed a 4.9% increase in creatinine clearance.⁴¹ However, our period changes were not altered by adjusting for statins (lipid lowering drugs) or lipid levels (HDL, total cholesterol).³⁷

There are limited data from other countries with which to compare these findings. Coresh *et al* analysed the US NHANES surveys of 1988–1994 and 1999–2004, both of which collected albuminuria and eGFR data. The prevalence of albuminuria and MDRD eGFR <60 mL/min/1.73 m² increased, the latter from 5.6% to 8.1%.⁸ The albuminuria increase was explained by changes in levels of obesity, diabetes and hypertension, whereas such adjustment only partly explained the fall in eGFR. Changes in population serum creatinine explained most of the remainder of the eGFR changes; this was analysed by comparing the mean serum creatinine in young people aged 20–39 without diabetes or hypertension and this had increased across the surveys.⁹ The authors suggested that this rise in serum creatinine could be either due to residual laboratory assay differences or changes in dietary protein or muscle mass. Grams *et al* showed that prevalence of eGFR <60 mL/min/1.73 m² had also increased using the same

survey data when eGFR was estimated using cystatin C, a marker of kidney function that is independent of muscle mass, and this was not explained by changes in demography, hypertension, diabetes or obesity, suggesting a true increase in low eGFR.⁴¹

We can compare the estimated national CKD prevalence for HSE with QOF returns which record diagnosed CKD in primary care.⁴² Prevalence has been increasing with improvements in detection and recording, and in 2010 it was 4.2%. The figures are not directly comparable as comparing a single screened value versus routine testing with presumed allowance for chronicity, but this may suggest some underdiagnosis of CKD.

If this change in prevalence in England is true, then based on the HSE 2003 age–sex-specific estimates and 2001 and 2011 Census data, the estimated number of CKD cases (for those aged 16 and over) would be 2.62 million based on the MDRD equation, falling by 0.03 million for 2009/2010. Equivalent figures for CKDEPI eGFR <60 mL/min/1.73 m² are 2.23 million and 0.02 million increase, respectively. The impact of such changes would be twofold: a consistent pool of patients at risk of progressing to need RRT; and a contribution to consistent cardiovascular incidence and mortality. The former is supported by stabilised acceptance rates onto RRT in England.⁴

CONCLUSIONS

The prevalence of a low eGFR appears to have decreased in England from 2003 to 2009/2010, despite increases in obesity and diabetes. It is unclear why this has occurred and it is difficult to infer directly that this is due to current policies to improve prevention of CKD and the identification and management of people with CKD. There is a need for repeated national prevalence estimates to further assess CKD patterns over time, including measures of albuminuria and of cystatin C, both of which were available in HSE 2009 and 2010.

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Contributors GRA was involved in the analysis and interpretation of the data. PJR drafted the paper. GRA, PJR, SF and GM made substantial contributions to the study conception and design. JSM co-ordinated the Health Surveys for England. DO provided background information on chronic kidney disease policy. JD conducted the laboratory analyses. All authors critically reviewed the paper and were involved in the drafting and approval of the manuscript. GRA is the guarantor.

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Ethics approval Approval was obtained from the London Multi-Centre Research Ethics Committee for the 2003 survey (HSE 2003 ref MREC/02/2/72) and approval was obtained from the Oxford B Research Ethics Committee for both 2009 and 2010 surveys (HSE 2009 ref 08/H0605/103, HSE 2010 ref 09/H0605/73).

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Data sharing statement The HSE 2003, 2009 and 2010 are archived with the UK Data Service. The Technical Appendix, statistical code and data set are available from the corresponding author Grant Aitken at G.Aitken@soton.ac.uk. Creatinine measurements for the HSE 2003 undertaken for this study will be archived in due course.

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