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Chronic kidney disease, albuminuria and socioeconomic status in the Health Surveys for England 2009 and 2010

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ABSTRACT

Background Renal replacement therapy rates are inversely related to socioeconomic status (SES) in developed countries. The relationship between chronic kidney disease (CKD) and SES is less clear. This study examined the relationships between SES and CKD and albuminuria in England.

Methods Data from the Health Survey for England 2009 and 2010 were combined. The prevalence of CKD 3–5 and albuminuria was calculated, and logistic regression used to determine their association with five individual-level measures and one area-level measure of SES.

Results The prevalence of CKD 3–5 was 5.2% and albuminuria 8.0%. Age–sex-adjusted CKD 3–5 was associated with lack of qualifications [odds ratio (OR) 2.27 (95% confidence interval 1.40–3.69)], low income [OR 1.50 (1.02–2.21)] and renting tenure [OR 1.36 (1.01–1.84)]. Only tenure remained significant in fully adjusted models suggesting that co-variables were on the causal pathway. Albuminuria remained associated with several SES measures on full adjustment: low income [OR 1.55 (1.14–2.11)], no vehicle [OR 1.38 (1.05–1.81)], renting [OR 1.31 [1.03–1.67)] and most deprived area-level quintile [OR 1.55 (1.07–2.25)].

Conclusions CKD 3–5 and albuminuria were associated with low SES using several measures. For albuminuria this was not explained by known measured causal factors.

Keywords chronic kidney disease, epidemiology, socioeconomic factors

Background

Chronic kidney disease (CKD) is a global public health problem with the prevalence of CKD stage 1–5 of 10–16% in adults.[1–4] CKD is defined and staged by level of kidney function (estimated glomerular filtration rate, eGFR) and the presence of markers of kidney damage (most commonly albuminuria).[5] Both eGFR and albuminuria are strong independent risk factors for all-cause and cardiovascular disease (CVD) mortality;[6,7] progression to end-stage renal disease (ESRD);[8] need for renal replacement therapy (RRT, dialysis and renal transplant) and acute kidney injury.[8]

Understanding inequalities in CKD is important in guiding strategies for prevention, detection and intervention. There is an inverse relationship between RRT rates and socioeconomic status (SES) in the UK and in other countries, whether using area-level deprivation measures as a proxy for individual status[9–14] or individual socioeconomic measures.[15] The relationship between CKD and SES is less clear. Nationally representative surveys in some (but not all) developed countries

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(including those with and without national health services) have shown variation in CKD prevalence by SES, both within and between countries.[16] Initial analysis of the 2009 and 2010 Health Surveys for England [HSEs, nationally representative surveys similar to US National Health and Nutrition Examination Surveys (NHANES)] showed mixed evidence for variation of CKD prevalence by area-level deprivation status, depending on severity of CKD.[17,18] Data from NHANES III demonstrated the association between microalbuminuria and poverty,[19] but no evidence is available in the UK on the relationship between albuminuria and SES. The association between low SES and increased risk of CKD diagnosis[20] and increased severity of CKD at presentation to renal services[21] has been demonstrated in the UK. Any observed variations in the CKD prevalence may be explained by differences in lifecourse exposures harmful to the kidney, such as foetal environment, environmental toxins, tobacco, obesity, hypertension and diabetes; and access to and use of health services. However, consideration needs to be given to the different measures of SES used and limitations of area-level proxies. This study aimed to provide detailed analysis of the associations of several socioeconomic factors (using both area-level and individual measures) with CKD stage 3–5, using the Chronic Kidney Disease Epidemiology Collaboration (CKDEPI) equation to estimate GFR, and with albuminuria in the 2009 and 2010 HSEs.[22]

Methods

Full details of HSE methods, measurement of non-CKD variables and response rates are given in the HSE reports.[17,18] A random, nationally representative sample was selected each year using a stratified, two-stage sample of private addresses. Participants completed an interview questionnaire; most consented to a nurse visit. In the 2009 or 2010 HSE, a valid urine sample was obtained from 88% of men and 86% of women aged 16 and over who had a nurse visit, and a non-fasting blood sample from 77% of men and 73% of women. Approval was obtained from the Oxford B Research Ethics Committee for both surveys (HSE 2009 ref 08/H0605/103, HSE 2010 ref 09/H0605/73).

Socioeconomic factors selected included: (i) occupation using National Statistics Socioeconomic Classification[23] [NS-SEC, in three categories: high (managerial and professional occupations), middle (intermediate occupations) and low (routine and manual occupations)]; (ii) qualifications grouped as: degree (NVQ4/NVQ5/Degree or equivalent), below degree (higher education below degree or NVQ3/GCE A Level equivalent or NVQ2/GCE O Level equivalent or NVQ1/CSE other grade) and none (no qualification); (iii) household income tertiles; (iv) household tenure (owned or rented accommodation); (v) access to motor vehicle within the household (any versus none) and (vi) area-level deprivation [using Index of Multiple Deprivation 2007 (IMD) national quintiles: 1 least deprived (IMD 0.37–8.32), 2 (8.32–13.75), 3 (13.75–21.22), 4 (21.22–34.42) and 5 most deprived (34.42–85.46)].[24]

Ethnicity was self-defined using 2001 census categories. Hypertension was defined as self-reported pre-existing doctor diagnosis, survey-defined [high blood pressure identified (BP systolic $\geq$140 mmHg and/or diastolic $\geq$90 mmHg and/or taking medication for hypertension) at survey examination] and 'total' (doctor + survey diagnosed). Diabetes was treated similarly: survey-defined diabetes was HBA_{1c} $\geq$6.5% at clinic visit. Body mass index (BMI) was classified as normal ($<$25 kg/m^2), overweight (25–29.9 kg/m^2) and obese ($\geq$30 kg/m^2).[25] Waist circumference was classified as: $<$94 cm, 94–102 cm (high) and $>$102 cm (very high) for men, and $<$80 cm, 80–88 cm (high) and $>$88 cm (very high) for women. For South Asians, the waist circumference threshold was 90 cm (men) and 80 cm (women).[25]

Serum creatinine was assayed using an IDMS traceable enzymatic assay in a single laboratory [Clinical Biochemistry Department at the Royal Victoria Infirmary (RVI), Newcastle-upon-Tyne]. Albuminuria was assessed using urinary albumin creatinine ratio (ACR), measured on a single random urine sample. Abnormal levels were divided into microalbuminuria (ACR 2.5–30 mg/mmol in men and 3.5–30 mg/mmol in women) and macroalbuminuria [ACR $>$ 30 mg/mmol (in either sex)].[26] CKDEPI eGFR values were derived using the standard equation,[22] a more accurate measure of true eGFR than the Modification of Diet in Renal Disease (MDRD) equation in routine use in the UK.[27] Details of laboratory analysis, internal quality control and external quality assurance are provided in HSE documentation.[17,18] The Kidney Disease: improving Global Outcomes classification of CKD based on level of eGFR was used: Stage 1: eGFR 90 ml/min/1.73 m^2 or more with albuminuria, Stage 2: 60–89 ml/min/1.73 m^2 with albuminuria, Stage 3a: 45–59 ml/min/1.73 m^2, Stage 3b: 30–44 ml/min/1.73 m^2, Stage 4: 15–29 ml/min/1.73 m^2 and Stage 5: $<$15 ml/min/1.73 m^2.[28] Current guidelines recommend that CKD be defined on the basis of reduced eGFR present for at least 3 months.[5,26,28] However, because of the cross-sectional nature of the HSEs, a single eGFR $<$60 ml/min/1.73 m^2 was used to define CKD stage 3–5 in these analyses.

Statistical analyses

The prevalence by CKD stage included participants with both serum creatinine and urinary ACR data. Analyses of

CKD and albuminuria associations used all participants with relevant data to maximize power and to allow analysis of albuminuria individually. Logistic regression models were used to examine the relationships between CKD and demographic, socioeconomic, lifestyle and clinical factors, adjusted for age and sex. Age was categorized as <65 and ≥65. An age × sex interaction term was included in multivariable regression models following identification of an age × sex interaction for CKD 3–5 early in our analyses. Overall, CKD prevalence estimation accounted for weighting within gender to allow for gender differences in response. Non-response weights were used in all analyses. Despite low numbers from ethnic minorities, ethnicity is associated with variation in RRT rates,[13,14] and ethnicity was therefore included as a potential confounder in multivariable analyses. Three dichotomized-dependent variables were investigated: CKD defined by the CKDEPI equation as eGFR <60 ml/min/1.73 m^2 (Stage 3–5); the presence of micro- or macro-albuminuria and CKD stage 1–2 defined as eGFR >60 ml/min/1.73 m^2 with evidence of albuminuria. Sensitivity analyses were conducted in the white-only population, and, for albuminuria, in people without diabetes. For CKD 3–5, analyses were also conducted using the MDRD equation to define CKD.

Interactions of socioeconomic variables with age and sex were examined and also with diabetes in the albuminuria models. The final models were (i) age, sex and age × sex, (ii) age, sex, ethnicity and age × sex and (iii) age, sex, ethnicity, age × sex, smoking, BMI, doctor diagnosed hypertension and doctor diagnosed diabetes.

Odds ratios are presented with 95% confidence intervals (CIs) and *P* values of <0.05 were considered statistically significant. All analyses, adjusted for the complex survey design, were performed using IBM SPSS Statistics version 19.

Results

The total combined sample size (unweighted) for the 2009 and 2010 HSE was 13 065 individuals aged 16 and over. Sample characteristics (weighted for non-response) are shown in Table 1. In total, 5799 (44.4%) respondents had a valid serum creatinine value, 7592 (58.1%) had a valid ACR and 5318 (40.7%) had both. Of the unweighted sample of 5799 individuals, 3186 (54.9%) were female (51.2% of the weighted sample). Of those excluded because they lacked a valid serum creatinine, 1994 (27.6%) had no formal qualifications (compared with 20.6% in those included). Of those without valid ACR, 1239 (22.4%) had no access to a motor vehicle and 1660 (30.2%) had no qualifications (compared with 16.7 and 20.3% in those included). Otherwise those included and those excluded were comparable with regard to SES.

The overall weighted prevalence of CKD stage 3–5 was 303/5786 (5.2%). The prevalence of any albuminuria was 8.2% in men and 7.5% in women [for macroalbuminuria, 0.3 and 0.5% respectively (only 22 people)]. Both CKD 3–5 and albuminuria prevalence was higher in people with low income, no access to a vehicle and no formal qualifications. Prevalence patterns for CKD 1–2 and CKD 3–5 with albuminuria were similar to those for overall albuminuria [although the number of people with CKD stage 3–5 with albuminuria was low ($n = 66$)] (Table 2).

Age–sex-adjusted CKD 3–5 was associated with lack of qualifications [odds ratio (OR) 2.27 (95% CI 1.40–3.69)], low income [OR 1.50 (1.02–2.21)] and renting household tenure[OR 1.36 (1.01–1.84) versus ownership]. Tenure remained significant in fully adjusted models. Albuminuria remained associated with several SES measures on full adjustment: low income [OR 1.55 (1.14–2.11)], no vehicle [OR 1.38 (1.05–1.81)], renting [OR 1.31 (1.03–1.67)], most deprived area-level quintile [OR 1.55 (1.07–2.25)] (Fig. 1 and Supplementary data, Appendix Table S1).

Table 3 shows the prevalence and associations for lifestyle and clinical factors, which might act as confounding/explanatory factors for the SES–CKD relationship. BMI, diabetes and hypertension were positively associated with CKD and albuminuria, whereas total cholesterol was not associated with either. HDL cholesterol was negatively associated with both. All SES measures were associated with smoking, type 2 diabetes, hypertension and obesity after age–sex adjustment (Supplementary data, Appendix Table S2). CKD 1–2 was associated with smoking, BMI, waist circumference, HDL cholesterol, diabetes and hypertension (data not shown).

A significant age × sex interaction ($P < 0.05$) was identified in the CKD models, with younger (<65) females having greater odds of CKD compared with younger males but with no difference in older age groups. There were no significant interactions between age and SES in the CKD models or diabetes and SES in the albuminuria models.

There were no differences in these results in the sensitivity analyses for the white-only population, and, for albuminuria, in people without diabetes (data not shown).

The use of the MDRD equation in place of CKDEPI resulted in slightly different associations of CKD 3–5 with SES, with qualification level and vehicle ownership remaining associated in the fully adjusted model (Supplementary data, Appendix Table S3).

Discussion

Main findings of this study

This study found socioeconomic disparities in the prevalence of CKD stage 3–5, using the CKD-EPI equation to define

Table 1. Sociodemographic and clinical characteristics of the weighted study sample

Variable	*Category*	*People with valid serum creatinine value*		*People with urine albumin creatinine ratio value*	
		Number	*Column %*	*Number*	*Column %*
All	Aged 16+	5799	100	7592	100
Age	16–34	1756	30.3	1949	25.7
	34–54	2037	35.1	2844	37.5
	55–64	856	14.8	1218	16.0
	65–74	615	10.6	871	11.5
	75+	522	9.0	655	8.6
Ethnicity	White	5244	90.4	6884	90.7
	South Asian	243	4.2	285	3.8
	Black	154	2.7	200	2.6
	Other	139	2.4	160	2.1
Sex	Male	2823	48.7	3667	48.3
	Female	2963	51.1	3870	51.0
Income tertile	Lowest	1393	24.0	1517	20.0
	Middle	1617	27.9	1963	25.9
	Highest	1829	31.5	2224	29.3
Access to motor vehicle	Yes	4728	81.5	6280	82.7
	No	1056	18.2	1256	16.5
Qualification	Degree	1295	22.3	1761	23.2
	Below degree	3296	56.8	4238	55.8
	None	1197	20.6	1531	20.2
Occupation (NS-SEC)	High	1894	32.7	2646	34.9
	Middle	1203	20.7	1611	21.2
	Low	2619	45.2	3207	42.2
IMD Quintile	1. Least deprived	1197	20.6	1683	22.2
	2.	1204	20.8	1601	21.1
	3.	1228	21.2	1627	21.4
	4.	1105	19.1	1442	19.0
	5. Most deprived	1051	18.1	1184	15.6
Housing Tenure	Own/mortgage	3955	68.2	5389	71.0
	Rent/other	1817	31.3	2148	28.3
Smoking	Never	3126	53.9	4089	53.9
	Ex	1429	24.6	2007	26.4
	Current	1210	20.9	1423	18.7
Body mass index (BMI)	Normal	1956	33.7	2468	32.5
	Overweight	2047	35.3	2683	35.3
	Obese	1314	22.7	1815	23.9
Waist circumference	Low	2120	36.6	2701	35.6
	High	1347	23.2	1761	23.2
	Very High	2242	38.7	2938	38.7
Total cholesterol	<5 mmol/l	2675	46.1	2984	39.3
	≥5 mmol/l	3110	53.6	3719	49.0
HDL cholesterol	<1.2 mmol/l	1301	22.4	1591	21.0
	≥1.2 mmol	4485	77.3	5809	76.5
Albuminuria	None	4837	83.4	6896	90.8
	Micro	399	6.9	601	7.9
	Macro	22	0.4	39	0.5
Diabetes	No diabetes	5370	92.6	6957	91.6
	Doctor diagnosed[a]	305	5.3	450	5.9

Table 1. Continued

Variable	*Category*	*People with valid serum creatinine value*		*People with urine albumin creatinine ratio value*	
		Number	*Column %*	*Number*	*Column %*
	Survey defined[b]	316	5.4	442	5.8
	Total[c]	429	7.4	581	7.7
Hypertension	No HT	3800	65.5	4854	63.9
	Doctor diagnosed[a]	1387	23.9	1992	26.2
	Survey defined[d]	1542	26.6	2112	27.8
	Total[c]	1980	34.1	2683	35.3
Chronic kidney disease	Yes	303	5.2	—	—
	No	5483	94.6	—	—

[a]Self-reported doctor diagnosis.
[b]$HBA_{1c} \geq 6.5\%$.
[c]Doctor or survey diagnosed.
[d]Identified as high blood pressure (BP systolic $\geq$140 mmHg and/or diastolic $\geq$90 mmHg and/or taking medication for hypertension).

Table 2. Directly age–sex standardized prevalence (%) of CKD stage 3–5 and albuminuria by sociodemographic characteristics

Variable	*Category*	*CKD 3–5 (eGFR <60 ml/min/1.73 m^2) Weighted (n = 5786)*	*Albuminuria (any) Weighted (n = 7529)*	*Albuminuria (CKD 1–2) Weighted (n = 355)*	*Albuminuria (in people with CKD 3–5) Weighted (n = 66)*	*Total numbers in row*
All	Aged 16+	5.2	8.0	7.1	1.1	
Ethnicity	White	5.6	8.1	7.2	1.3	5244
	South Asian	1.1	6.4	6.3	0.2	243
	Black	2.7	6.8	6.5	0.6	154
	Other	0.7	6.2	6.2	0.1	137
Income tertile	Lowest	6.5	8.7	7.5	1.5	1393
	Middle	6.0	8.3	7.3	1.4	1617
	Highest	3.0	6.9	6.5	0.6	1830
Access to motor	Yes	4.4	7.7	7.0	1.0	4729
Vehicle	No	8.6	9.2	7.7	2.0	1057
Qualification	Degree	2.5	7.1	6.7	0.5	1295
	Below degree	3.6	7.3	6.7	0.8	3297
	None	12.4	10.8	8.9	2.9	1192
Occupation	High	4.6	7.8	7.0	1.1	1894
(NS-SEC)	Middle	6.1	8.0	7.1	1.3	1203
	Low	5.7	8.2	7.3	1.3	2343
IMD quintile	1. (IMD 0.37–8.31) Least deprived	6.0	8.2	7.3	1.4	1196
	2. (IMD 8.32–13.74)	6.3	8.5	7.5	1.4	1204
	3. (IMD 13.75–21.21)	4.8	7.8	7.0	1.1	1229
	4. (IMD 21.22–34.41)	4.6	7.7	6.8	1.1	1105
	5. (IMD 34.42–85.46) Most deprived	3.9	7.5	6.8	0.9	1051
Housing tenure	Own/mortgage	5.7	8.1	7.2	1.3	3956
	Rent/other	3.9	7.6	6.9	0.9	1816

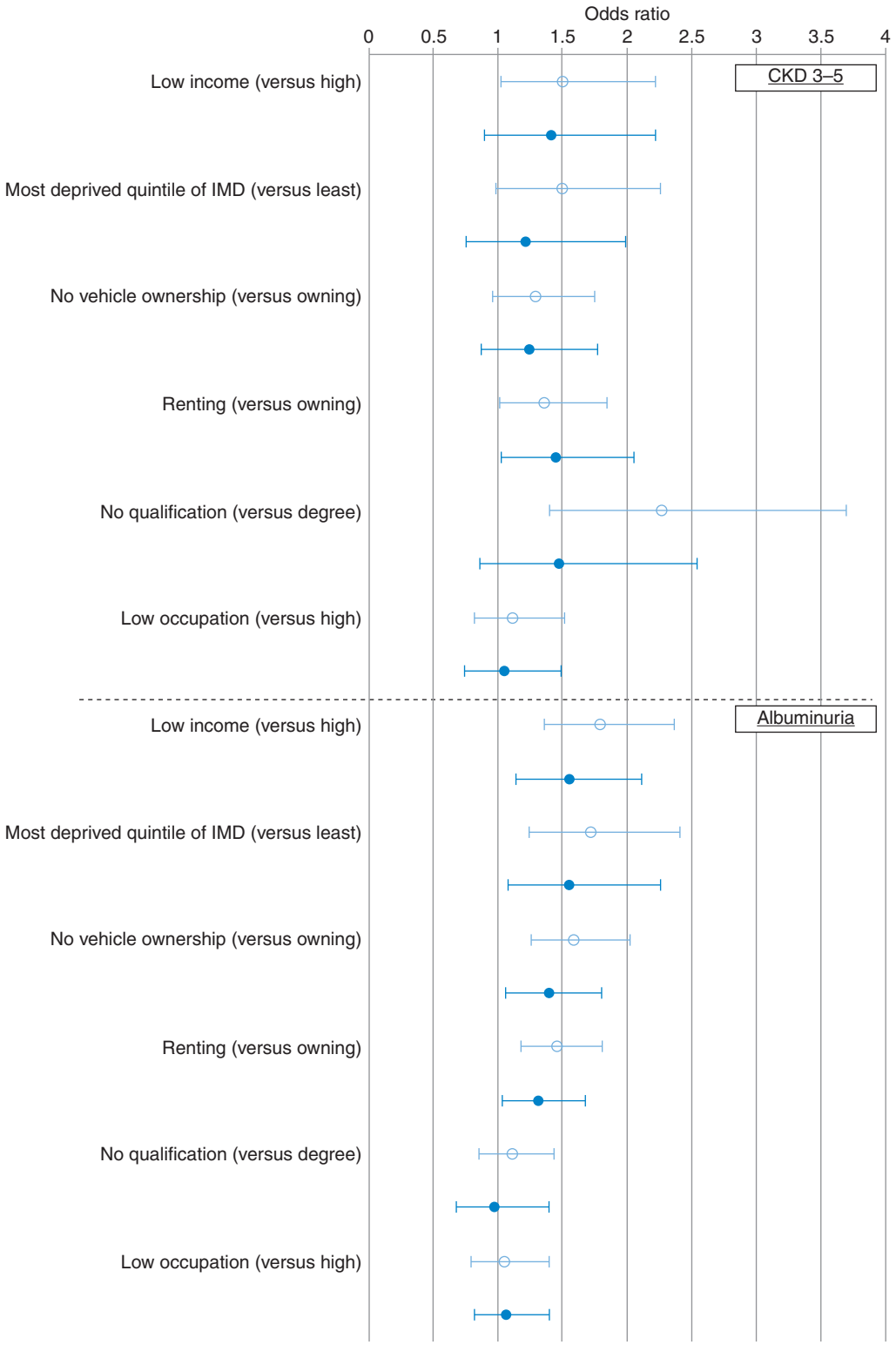

Fig. 1. Associations of CKD stage 3–5 and Albuminuria with measures of SES (age–sex and fully adjusted models). Open circle marker: age–sex adjusted. Closed circle marker: fully adjusted model.

Table 3. Prevalence and age-/sex/age × sex-adjusted associations of CKD stage 3–5 and albuminuria (all albuminuria cases) with behavioural and clinical factors

Variable	*Category*	*CKD 3–5*			*Albuminuria*		
		Prevalence (%)	*Odds ratio (95% CI)*	P *value*	*Prevalence (%)*	*Odds ratio (95% CI)*	P *value*
Smoking	Current	4.5	1	0.854	8.4	1	0.048
	Ex	8.9	1.24 (0.80–1.92)		9.7	0.85 (0.63–1.15)	
	Never	2.4	1.02 (0.62–1.65)		6.9	0.76 (0.59–0.99)*	
Body mass index (BMI)	Normal	2.3	1	0.001	6.6	1	0.048
	Overweight	5.0	1.72 (1.18–2.52)		6.7	1.11 (0.86–1.43)	
	Obese	7.5	2.75 (1.87–4.04)		8.4	1.33 (1.01–1.75)*	
Waist circumference	Low	2.1	1	<0.001	6.4	1	
	High	5.5	1.57 (1.05–2.34)		7.7	1.10 (0.83–1.45)	0.636
	Very High	7.9	1.97 (1.38–2.81)		9.2	1.27 (0.99–1.62)	0.051
Total cholesterol	<5 mmol/l	5.6	1	0.092	8.4	1	
	≥5 mmol/l	4.9	0.80 (0.62–1.04)		8.0	0.90 (0.73–1.09)	0.251
HDL cholesterol	<1.2 mmol/l	6.9	1	<0.001	10.0	1	
	≥1.2 mmol	4.7	0.55 (0.41–0.74)		7.6	0.73 (0.58–0.93)**	0.003
Albuminuria	None	4.6	1	<0.001	—	—	—
	Micro	16.0	2.34 (1.65–3.31)		—	—	
	Macro	30.0	7.53 (2.22–25.5)		—	—	
Diabetes	No diabetes	4.4	1	<0.001	7.0	1	<0.001
	Doctor diagnosed[a]	15.5	3.83 (2.74–5.35)		22.9	2.69 (1.95–3.70)	
	Survey defined[b]	16.8	4.33 (3.14–5.99)		16.8	2.56 (1.93–3.41)	
	Total[c]	15.4	3.99 (2.97–5.35)		20.4	2.50 (1.89–3.66)	
Hypertension	No hypertension	2.1	1	<0.001	5.2	1	<0.001
	Doctor diagnosed[a]	13.1	5.56 (4.37–7.09)		14.4	2.25 (1.81–2.81)	
	Survey defined[d]	12.4	5.11 (3.99–6.56)		9.6	2.13 (1.69–2.69)	
	Total[c]	11.3	6.04 (4.64–7.88)		13.3	2.04 (1.60–2.89)	

Age–sex-adjusted odds ratios.

[a]Self-reported doctor diagnosis.

[b]HBA_{1c} ≥6.5%.

[c]Doctor or survey diagnosed.

[d]Identified as high blood pressure (BP systolic ≥140 mmHg and/or diastolic ≥90 mmHg and/or taking medication for hypertension).

CKD, for individual measures of SES. It also identified socioeconomic disparities in the prevalence of albuminuria, an independent predictor of poor outcomes, for a wide range of both individual and an area-level measures of SES.

Higher CKD 3–5 prevalence was associated with lack of qualifications, low income and housing tenure (renting) after adjusting for age and sex. These associations were not maintained after further adjustment for ethnicity, lifestyle and clinical variables (obesity, diabetes, hypertension and smoking), which are likely to be explanatory factors on the causal pathway. Higher albuminuria prevalence was associated with low income, lack of vehicle ownership, housing tenure (renting) and IMD, and these were maintained, though attenuated, after full adjustment, demonstrating independence from these key factors on the causal pathway.

What is already known on this topic

Our results support the findings of several other studies. A population-based case–control study in Sweden found an approximately doubled adjusted odds ratio of having CKD in families with only unskilled workers compared with families with at least one professional (after adjusting for age, sex, BMI, smoking, alcohol and aspirin or paracetamol use).[29] A cross-sectional study in the UK of incident CKD presenting to renal services found increased risk of low eGFR (<30 ml/min/1.73 m^2) in areas with greater socioeconomic deprivation.[21] Cross-sectional data from the Whitehall II cohort identified higher odds of low eGFR in lower occupational grades; this association was attenuated after adjustment for BMI and components of the metabolic syndrome; similar to our findings.[30] The Atherosclerosis Risk in Communities

(ARIC) study identified an association of CKD incidence with individual SES (occupation).[31] White *et al.*, comparing findings from nationally representative surveys in the USA and Australia, showed variation between countries, and, for the USA, between different ethnic groups, in associations between SES and CKD 3–5 prevalence.[16] American non-Hispanic Whites with lower levels of education or in the lowest income quartile were more likely to have CKD compared with those with higher education levels, employed groups and those in the highest income quartile.[16] In contrast, an Australian national survey did not demonstrate an association of CKD prevalence with SES (measured by education and income) after age–sex adjustment.[16] The recent Quality Improvement in CKD trial in the UK identified the associations between deprivation (IMD) and CKD prevalence, though this was not considered 'clinically significant'.[32] Reasons for these variations are likely to be complex, but may relate to differences in health-care systems or access to health care and primary prevention.[16]

What this study adds

There is little existing evidence on the relationship between albuminuria and SES. Data from NHANES III demonstrated the association between microalbuminuria and poverty in the USA (adjusted OR 1.18, 1.05–1.33),[19] and similar associations have been shown with various measures of SES in a Malay population.[33] To our knowledge, this is the first study to investigate the association between albuminuria and SES in a representative population sample in the UK. In unadjusted analyses, our data suggest socioeconomic inequalities in albuminuria distribution, both in those with eGFR <60 ml/min/1.73 m^2 and those with eGFR above this level, which will influence differential propensity to progress. There are few data on the relationship between SES and CKD progression. The (ARIC) study in the USA identified that, for white men, living in the lowest compared with the highest SES area-level quartile was associated with increased risk of CKD progression (hazard ratio for elevated serum creatinine 1.6 (95% CI 1.0–2.5).[34] The reasons for finding association between SES and CKD and albuminuria may be partly related to the social distribution of underlying factors associated with CKD occurrence and progression,[34] including obesity,[35] smoking, type 2 diabetes[36] and hypertension.[37] Persistence of the association for albuminuria after adjustment suggests other causal mechanisms (and or potential residual confounding) may apply. Albuminuria is a key determinant of progression and poor outcome in CKD, particularly when combined with other risk factors (type 2 diabetes and hypertension), which are more prevalent in lower socioeconomic groups. Other factors such as low birth weight[38] and health care access (with variation by health system) also show socioeconomic patterns.[39]

Limitations of this study

Strengths of this study include the nationally representative nature of the 2009 and 2010 HSE data, pooled over 2 years, increasing numbers and precision of estimates, the rigorous nature of HSE methodology with standardized protocols for measurement by trained interviewers and nurses, all samples being tested in the same laboratory with standardized assays, use of non-response weighting to reduce response bias and use of various SES measures.

The study was limited by its cross-sectional nature, reducing the ability to infer causal relationships. Reverse causation was, however, considered unlikely as the majority of people with CKD are asymptomatic. Non-response weighting is an effective method to avoid bias and maintain representativeness of the sample.[40] An important limitation was using single samples to test for serum creatinine and albuminuria. Persistence of reduced eGFR levels and elevated ACR to confirm chronicity could not be shown, which could lead to non-differential misclassification. Our methods were similar to those used in NHANES III, but repeat testing of ACR in NHANES showed reduced albuminuria prevalence.[2,19] The use of single eGFR has also been shown to elevate CKD prevalence estimates.[41] Confirmation in longitudinal studies would therefore be beneficial. There were too few cases from minority ethnic groups to give robust data on ethnic differences in CKD prevalence. South Asians and Blacks have higher rates of RRT[42] but lower prevalence of CKD than Caucasians.[43] The prevalence of CKD stage 4–5 is likely to be underestimated as, while the HSE adjusts for non-response among the general population in private households, it may not account for some in whom more severe CKD is more common (people in residential care or those unable to participate because of poor health or hospitalization) and may therefore miss individuals with ESRD. Further limitations are lack of data on prevalent CVD and family history, small numbers with macroalbuminuria, lack of information on medication use (differential use of renin angiotensin aldosterone system inhibitors by SES could result in less apparent albuminuria in those with higher SES). Accurately measuring SES in elderly populations is challenging, and non-differential misclassification may bias associations towards the null.[44] Survivor bias may have reduced socioeconomic gradients, with competing risk of mortality from premature deaths in poorer groups. Heterogeneity of our findings in terms of different measures of SES could be considered a limitation. However, given the challenges of accurately measuring SES

using any single measure, we believe that the overlap in associations shown here demonstrates support for true association rather than lack of it. A lifecourse approach to assessing SES that is beyond the scope of this study would be needed to fully understand the relationships between different measures and may be an important consideration for future research.[39,45]

Supplementary data

Supplementary data are available at the *Journal of Public Health* online.

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Conflict of interest

Dr D. O'Donoghue was National Clinical Director for Kidney Care at the Department of Health until April 2013. There are no conflicts of interest to declare. No funding was received by any of the authors to conduct this research. The results presented in this paper have not been published previously in whole or part.

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