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The impact of progressive chronic kidney disease on health-related quality-of-life: a 12-year community cohort study

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Abstract

Purpose Quality-of-life is poor in end-stage kidney disease; however, the relationships between earlier stages of chronic kidney disease (CKD) and are poorly understood. This study explored longitudinal quality-of-life changes in a community-based CKD cohort and assessed associations between CKD and quality-of-life over time, and between baseline quality-of-life and CKD outcomes.

Methods We used the Australian diabetes, obesity and lifestyle study—a nationally representative, prospective cohort with data collected at baseline, year 5 and year 12—to examine the relationships between CKD stage, quality-of-life and outcomes. Linear mixed regression, cox proportional hazards, Kaplan–Meier and competing risks analyses were used.

Results Of 1112 participants with CKD and baseline quality-of-life data, the physical component summary (PCS) score was significantly lower than for the general population ($p = 0.01$ age and sex adjusted), while the mental component summary (MCS) score was no different ($p = 0.9$ age and sex adjusted). In our unadjusted mixed effects model, more advanced kidney disease was associated with lower PCS and higher MCS at baseline ($p < 0.001$ and $p < 0.01$, respectively); however, this effect was no longer significant after adjustment for demographic and clinical variables. The rate of decline in PCS over the period of follow-up was greatest for those with more advanced kidney disease ($p < 0.001$ in unadjusted model, $p = 0.007$ in adjusted model). There was no association between change in MCS over the period of follow-up and severity of kidney disease in either the unadjusted or adjusted model ($p = 0.7$ and $p = 0.1$, respectively). Lower PCS, but not MCS, was associated with increased cardiovascular and increased all-cause mortality even after adjustment for key demographic and clinical variables ($p < 0.001$).

Conclusions Physical, but not mental, quality-of-life is significantly impaired in CKD, and continues to decline with disease progression.

Keywords CKD · Quality-of-life · SF36 · AUSDIAB

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Introduction

Chronic kidney disease (CKD) is common, affecting approximately one in 10 people and is associated with numerous adverse outcomes including progression to end-stage kidney disease (ESKD), cardiovascular disease and premature mortality [1]. Less well established is the impact of CKD on an individual's quality-of-life.

Quality-of-life is an important clinical and economic outcome for patients, health-care providers and payers. While quality-of-life in ESKD has been reasonably well examined, less is known about quality-of-life in early-to-moderate CKD [2]. To our knowledge, there does not exist a longitudinal study of quality-of-life in CKD among adults drawn from the general population.

Quality-of-life in kidney disease is most commonly measured with the short form 36 (SF-36) questionnaire. This has been validated in multiple populations, including an Australian population [2, 3]. Our study used longitudinal SF-36 data collected from participants in the Australian Diabetes, Obesity and Lifestyle (AusDiab) study. AusDiab is a nationally representative prospective cohort study with data, collected at baseline (1999–2000), year 5 (2004–2005) and year 12 (2011–2012). This longitudinal cohort enabled an examination of the changes in health-related quality-of-life for those with CKD and its association with disease progression.

Using data from the AusDiab study, we aimed to investigate (i) the longitudinal association between CKD and quality-of-life, and (ii) the relationship between baseline quality-of-life and future CKD outcomes including progression of kidney disease, cardiovascular mortality and all-cause mortality and (iii) utility-based quality-of-life for use in economic evaluations.

Methods

Study population

We used data from AusDiab, a national longitudinal population-based study of Australian adults. Participants were selected via a stratified cluster sampling design to provide a population representative sample of Australian adults aged ≥ 25 years. Participants completed a detailed questionnaire at home and presented to a temporary testing site for physical examination and blood and urine collection. AusDiab study methods have been described elsewhere [4]. The current analysis included only participants with CKD at baseline, defined as estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2 and/or albuminuria in accordance with current Kidney Disease—Improving Global Outcomes (KDIGO) guidelines, who were not requiring renal replacement therapy and had completed a quality-of-life questionnaire [5]. eGFR was calculated using the CKD Epidemiology Collaboration (CKD-EPI) equation. Serum creatinine was measured by the modified kinetic Jaffe method. All serum creatinine measurements were isotope dilution mass spectrometry (IDMS) standardised, either prospectively (12-year follow-up) or retrospectively (Baseline and 5-year follow-up) as previously described [6].

Ethical approval

AusDiab was approved by the ethics committee of the International Diabetes Institute. Informed consent was obtained from all participants at baseline and each follow-up.

Outcomes of interest

The primary outcome of interest was quality-of-life over time by CKD stage. The secondary outcome was the relationship between quality-of-life and disease progression, cardiovascular and all-cause mortality [7].

Health-related quality-of-life

Quality-of-life was measured at each time point with the self-reported SF-36 (version 1) questionnaire. The SF-36 comprises eight domains: physical functioning, role limitations due to physical issues, bodily pain, general health, vitality, social functioning, role limitations due to emotional issues and mental health. These eight domains are combined into two summary measures: physical component summary (PCS) score and mental component summary (MCS) using the Australian scoring algorithm [8]. The mean PCS and MCS scores are standardised to 50 in the general population with a standard deviation of 10. Higher scores indicate better quality-of-life. A difference in PCS or MCS of ≥ 5 is considered clinically significant [9–11].

Quality-of-life measures for economic evaluation are often discussed in terms of utility. Utilities are commonly used to calculate quality adjusted life years (QALYs) and are of particular relevance to cost–utility analyses. Utility is measured on a 0–1 scale, where 0 represents death and 1 represents full health [12]. Utility is the numerical value attached to the strength of an individual's preference for a specific health state. We transformed SF-36 scores into SF-6D utilities using a published algorithm for Australian tariffs (weights) [13]. A commonly used minimum clinically important difference of 0.03 was used in our analysis [14].

Biomedical and behavioural variables

Our analysis incorporated the following independent variables: age, sex, eGFR, albuminuria, diabetes, hypertension, anaemia, smoking status, daily alcohol intake, physical activity and body mass index (BMI). These variables were included in our analysis due to their potential association with health-related quality-of-life, described in more detail below.

CKD stage was defined according to the 2012 KDIGO guidelines: Stage 1 albuminuria and eGFR of ≥ 90 mL/min/1.73 m^2; Stage 2 albuminuria and eGFR of 60–89 mL/min/1.73 m^2; Stage 3 eGFR of 30–59 mL/min/1.73 m^2; Stage 4/5 eGFR of < 30 mL/min/1.73 m^2 [5].

Albuminuria categories were defined as no albuminuria (urinary albumin creatinine ratio (ACR) < 2.5 mg/mmol for males and < 3.5 mg/mmol for females), microalbuminuria (2.5–25 mg/mmol for males and 3.5–35 mg/mmol for females), and macroalbuminuria (> 25 mg/mmol for males and > 35 mg/mmol for females).

Diabetes was defined as those who (i) answered yes to the question: "Have you ever been told by a doctor or nurse that you have diabetes?"; (ii) were taking glucose-lowering medication; (iii) had a fasting blood glucose ≥ 7.0 mmol/L (≥ 126 mg/dL); (iv) had 2-h post load glucose ≥ 11.1 mmol/L (≥ 200 mg/dL) or (v) had a haemoglobin A1c ≥ 6.5% [15].

Hypertension was defined by (i) answering yes to the question: "Are you currently taking tablets for high blood pressure?"; (ii) systolic blood pressure ≥ 140 mmHg or (iii) diastolic blood pressure ≥ 90 mmHg when measured by a study nurse in a seated position following at least 5 min of rest, recorded as the average of the last two of three measurements made at 1-min intervals.

Anaemia was defined as serum haemoglobin < 130 g/L for males and < 120 g/L for females at baseline.

BMI was grouped into underweight (BMI < 18.5 kg/m^2), healthy weight (BMI 18.5–24.9 kg/m^2), overweight (BMI 25–29.9 kg/m^2) and obese (BMI > 30 kg/m^2) categories [16]. Physical activity was grouped into sufficient (≥ 150 min per week of leisure time physical activity), insufficient (> 0 and < 150 min per week of leisure time physical activity) and sedentary (0 min per week of leisure time physical activity) [17].

Smoking, dietary protein and alcohol consumption were based on self-report questionnaire. Smoking status was categorised as never smoked, former smoker and current smoker. Alcohol consumption was grouped into non-/light drinkers (< 10 g/day), moderate drinkers (≥ 10 and < 30 g/day) and heavy drinkers (≥ 30 g/day) [18]. Protein intake was categorised as low (< 0.6 g/kg), normal (0.6–1.5 g/kg) or high (> 1.5 g/kg).

Biochemical markers were defined as hyperuricaemia: serum uric acid ≥ 0.42 mmol/L; Vitamin D deficiency: serum vitamin D < 60 nmol/L; hypercholesterolaemia: total cholesterol > 6.2 mmol/L or patient self-report of taking medications to lower cholesterol.

Mortality

Death was determined by linking the AusDiab cohort to the Australian Institute of Health and Welfare's National Death Index (NDI) [19]. The follow-up period for all-cause mortality was the date of death or 30 November 2012, whichever occurred first.

Statistical analysis

We reported categorical variables as frequencies and percentages, with mean and standard deviation (SD) reported for continuous variables. Pearson's chi-squared statistic, or Fischer's exact test for small samples, was used to compare categorial variables. Student's *t* test was used to compare continuous variables.

To explore the association between the independent variables described above and PCS and MCS, we determined the rate of change in PCS and MCS per year using a linear mixed effects regression model. The mixed effects model with random intercepts and random slopes estimated the rate of change in PCS over time, while incorporating the varying number of PCS collection points and different follow-up periods for each individual. We included all variables with a p value of < 0.25 in univariate analysis in addition to age, sex, CKD stage and albuminuria. Mixed effects model assumptions of normality, homoscedasticity and linearity, were tested graphically.

The log-rank test was used for univariate analysis of factors associated with survival. We then used the Kaplan–Meier method to compare the survival time between the four quartiles of PCS and MCS and a Cox proportional hazards model to determine the predictors of cardiovascular and all-cause survival and of quality-of-life declines of ≥ 30%. The proportional hazards assumption was tested for each variable and the model as a whole.

All statistical analyses were performed with Stata 15 (StataCorp, College Station, TX, USA). Study results were reported according to the STROBE statement for cohort studies [20].

Results

Baseline patient characteristics

The participant flow chart is presented in Fig. 1 and participant characteristics at study entry are described in Table 1. Baseline eGFR and ACR were available for

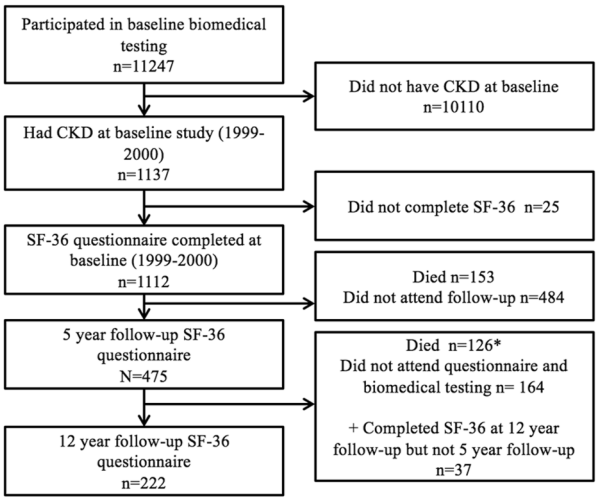


Fig. 1 Flow chart of study participation and data availability

11,205 participants. CKD criteria were met by 1137, of whom quality-of-life data were available for 1112 (98%) and these participants were the focus of the present study. Across the 1112 participants, we had information for 12,462 person-years. The longest follow-up was 14.5 years (mean follow-up 11.2 years, median 13.2 years), after censoring at death.

Females comprised 553 (50%) and males 559 (50%) of the 1,112 participants. The mean ± standard deviation age was 64 ± 15 years. There were 421 (38%) participants with stage 1 CKD, 314 (28%) with stage 2 CKD, 346 (31%) with stage 3 CKD and 31 (3%) with stage 4/5 CKD (Table 1).

Of the 1112 participants, 185 (17%) had quality-of-life scores available for baseline, year 5 and year 12 follow-up periods, 327 (29%) had quality-of-life scores available for baseline and either the year 5 or year 12 follow-up period, and 600 (54%) had baseline quality-of-life scores only. Lower baseline quality-of-life was strongly associated with loss to follow-up from either non-attendance of death after adjusting for age and sex (Table 2).

PCS at baseline was mean ± standard deviation 43(± 11), significantly lower than the PCS mean ± standard deviation of 50(± 10) for the general population ($p = 0.01$ age and sex adjusted). Mean ± standard deviation PCS scores by CKD stage were 47(± 10) for stage 1, 42(± 11) for stage 2, 40(± 11) for stage 3 and 35(± 13) for stage 4/5 ($p < 0.001$, $p = 0.01$ age and sex adjusted) (Fig. 2). The mean ± standard deviation MCS was 51(± 10), not significantly different from the mean ± standard deviation MCS of 50(± 10) for the general population ($p = 0.9$ age and sex adjusted). There was no significant difference in MCS by CKD stage after adjustment for age and sex ($p = 0.4$) (Fig. 2).

Quality-of-life over time by baseline eGFR

In our unadjusted mixed effects model, lower eGFR was associated with lower baseline PCS ($p < 0.001$). We then adjusted the mixed effects model for age, sex, albuminuria, amount of physical exercise, BMI category and smoking status as well as comorbidities including anaemia, diabetes and hypertension. While the trend remained in our adjusted mixed effects model, it was not statistically significant ($p = 0.09$). Those with more advanced CKD experienced greater declines in PCS over the period of follow-up in both the unadjusted and adjusted models ($p < 0.001$ and $p = 0.007$, respectively) (Fig. 2a).

In an unadjusted model, lower baseline eGFR was associated with higher MCS at baseline ($p = 0.01$) and a steeper rate of decline in MCS over time ($p = 0.03$). However, in our multivariable adjusted model, eGFR was not associated with either baseline MCS or decline in MCS ($p = 0.7$ and $p = 0.1$, respectively) (Figs. 2b, 3).

Baseline quality-of-life and future health outcomes

Kaplan–Meier survival curves (Fig. 4) indicated a relationship between baseline PCS, but not baseline MCS, and survival. In a Cox regression adjusted for age, sex, eGFR and albuminuria, lower PCS was found to be associated with increased all-cause mortality ($p < 0.001$, Table 3). No statistically significant relationship was found between MCS and all-cause mortality ($p = 0.6$, Table 3). Lower PCS, but not MCS, was also associated with cardiovascular mortality in an adjusted model ($p < 0.001$ and $p = 0.6$, respectively). No relationship between PCS or MCS and disease progression was found in an adjusted model ($p = 0.9$ and $p = 0.09$, respectively).

Utility-based quality-of-life over time

The utility for CKD stage 1 at baseline was mean 0.60 (SE 0.01, $p < 0.001$) for stage 1. The utility decrement rates by stage were − 0.08 (SE 0.02, $p > 0.001$) for stage 2, − 0.10 (SE 0.02, $p < 0.001$) for stage 3 and − 0.15 (SE 0.06, $p = 0.01$) for stage 4/5 (Table 4a). The utility decrements by CKD stage were also clinically significant, exceeding 0.03 difference considered to be clinically significant. After 12 years, all participants had clinically significant declines with stage 1 CKD utility falling from a mean of 0.60 to a mean of 0.42; however, the differences between stages was no longer statistically significant (Fig. 3a).

The age- and sex-adjusted utility for CKD stage 1 was mean 0.52 (SE 0.02, $p < 0.001$) at baseline. The utility decrement by stage was − 0.02 (SE 0.03, $p = 0.39$) for stage 2, − 0.03 (SE 0.03, $p = 0.37$) for stage 3 and − 0.08 (SE 0.06, $p = 0.17$) for stage 4/5 (Table 4b). After 12 years the utility

Table 1 Baseline characteristics of 1112 participants

	Women			Men			Total		
	N	Count	Percent (%)	*N*	Count	Percent (%)	*N*	Count	Percent (%)
Age (years)	553		50	559		50	1112		100
25–44		86	16		61	11		147	13
45–64		155	28		170	30		325	29
≥ 65		312	56		328	59		640	58
eGFR (mL/min/1.73 m^2)	553			559			1112		
≥ 90		217	39		204	36		421	38
60–89		129	23		185	33		314	28
30–59		187	34		159	28		346	31
15–29		20	4		11	2		31	3
Albuminuria	525			526			1051		
None		151	29		95	18		246	23
Microalbuminuria		340	65		368	70		708	67
Macroalbuminuria		34	6		63	12		97	9
Diabetes (yes)	553	116	21	559	155	28	1112	271	24
Hypertension (yes)	543	349	64	544	406	75	1087	755	69
Body mass index	528			538			1066		
Underweight		11	2		6	1		17	2
Healthy weight		178	34		126	23		304	29
Overweight		179	34		249	46		428	40
Obese		160	30		157	29		317	30
Smoking status	532			530			1062		
Never		355	67		192	36		547	52
Former smoker		111	21		250	47		361	34
Current smoker		66	12		88	17		154	15
Anaemia	462	31	7	470	25	5	932	56	6
Physical activity	540			540			1080		
Sufficient		204	38		287	53		491	46
Insufficient		193	36		139	26		332	31
Sedentary		143	26		114	21		257	24
	N	Mean	SD	*N*	Mean	SD	*N*	Mean	SD
SF-36 PCS	553	42	11	559	44	11	1112	43	11
SF-36 MCS	553	51	10	559	51	10	1112	51	10
SF-6D utility	394	0.54	0.25	384	0.56	0.27	778	0.55	0.26

Table 2 Baseline quality-of-life by number of completed surveys over the follow-up period

	N	Mean (Std dev) SF-36 PCS	Mean (Std dev) SF-36 MCS	*N*	Mean (Std dev) SF-6D utility
All three surveys completed	185	48.1 (8.2)	51.8 (9.2)	41	0.53 (0.27)
Two surveys completed	327	43.7 (10.8)**	51.0 (9.8)^{ns}	249	0.58 (0.25)^{ns}
One (baseline) survey completed	600	40.9 (11.8)***	50.2 (10.4)**	488	0.52 (0.22)^{ns}

Age- and sex-adjusted linear regression

*p < 0.05, **p < 0.01, ***p < 0.001, ns = not significantly different from baseline

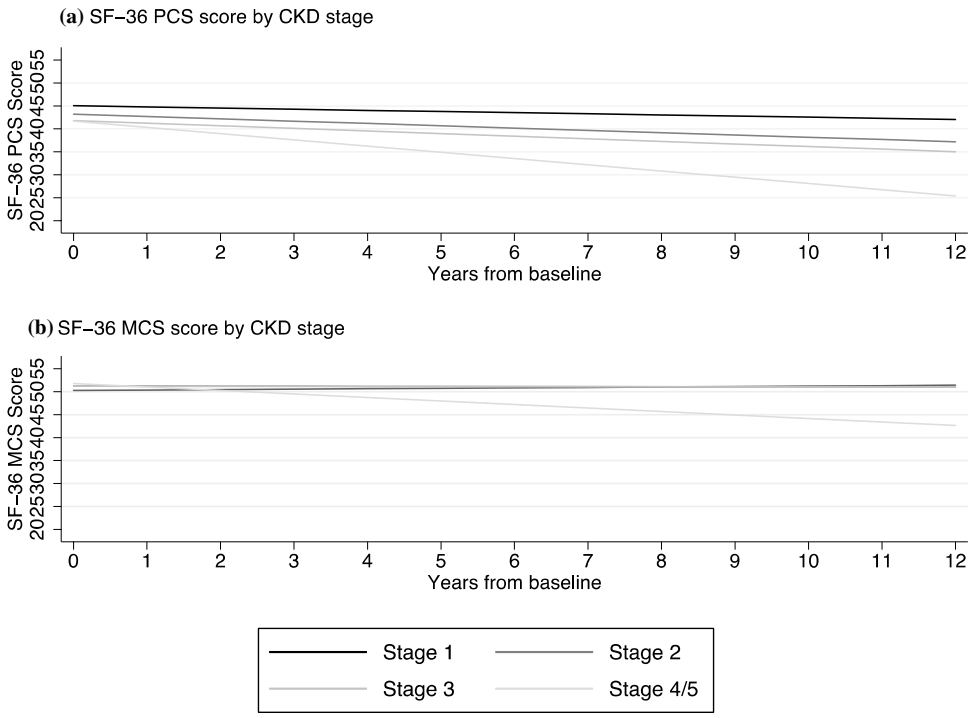


Fig. 2 Mixed effect model results, quality-of-life over time by CKD stage

for stage 1 had fallen to a mean of 0.30 (SE 0.06, $p<0.001$). The utility decrement rates by stage were +0.04 (SE 0.07, $p=0.57$) for stage 2, +0.13 (SE 0.09, $p=0.17$) for stage 3 and −0.18 (SE 0.24, $p=0.45$) for stage 4/5 (Table 4a).

Discussion

We followed the 1112 participants in our population representative CKD cohort for 12 years. The AusDiab cohort is the first to demonstrate that physical quality-of-life is both significantly impaired in CKD at baseline and deteriorates with progression, while mental quality-of-life is no different.

In our study, patients with early-to-moderate CKD had lower with physical, but not mental, quality-of-life. Our community cohort of primarily early-stage CKD patients was found to significantly lower physical quality-of-life than the general population. Those with the most advanced kidney disease experienced the greatest declines in physical quality-of-life over time, even after adjusting for relevant covariates. While we found no impact of CKD on mental quality-of-life, it is important to note that mental quality-of-life is affected by a large number of socioeconomic variables which we were not able to account for and this may have affected our results [21, 22].

To date, quality-of-life in pre-dialysis CKD has been relatively underexamined with existing reports largely based on selected populations attending nephrology clinics. Of greater relevance, given the high prevalence of early-to-moderate CKD, is the impact of CKD on quality-of-life in the general population. This longitudinal analysis of quality-of-life in community-based CKD confirms earlier findings from the AusDiab study and builds on initial work using the baseline AusDiab study which found that eGFR < 60 was associated with a lower PCS, but not MCS, compared to those with an eGFR ≥ 60 ($p<0.001$ and $p=0.4$, respectively) [23, 24]. Our observation that the magnitude of reduction in physical quality-of-life increased with successive stages of CKD severity and declined over time within each stage of CKD strongly suggests a causal role for CKD; however, the observational nature of this study enables us to make the association but not prove causality.

Self-reported physical health, independent of other factors, is strongly associated with mortality risk in people with CKD, while mental health was not. Poor quality-of-life, particularly physical quality-of-life, has previously been shown to be associated with increased mortality in dialysis patients,[25, 26] older adults and patients with other chronic diseases such as HIV [27–29]. A study looking at quality-of-life in African American patients with hypertension and CKD participating in a randomised clinical trial found poorer quality-of-life was associated with increased risk of the composite outcome of disease progression or death [30]. Our study supports and extends these findings as it is community based in a primarily Caucasian population with mortality data on all patients, enabling a stand-alone mortality analysis.

The utilities obtained from our study, may support long-term modelling of CKD for health technology assessment. In general, cost-effectiveness studies have used utilities for

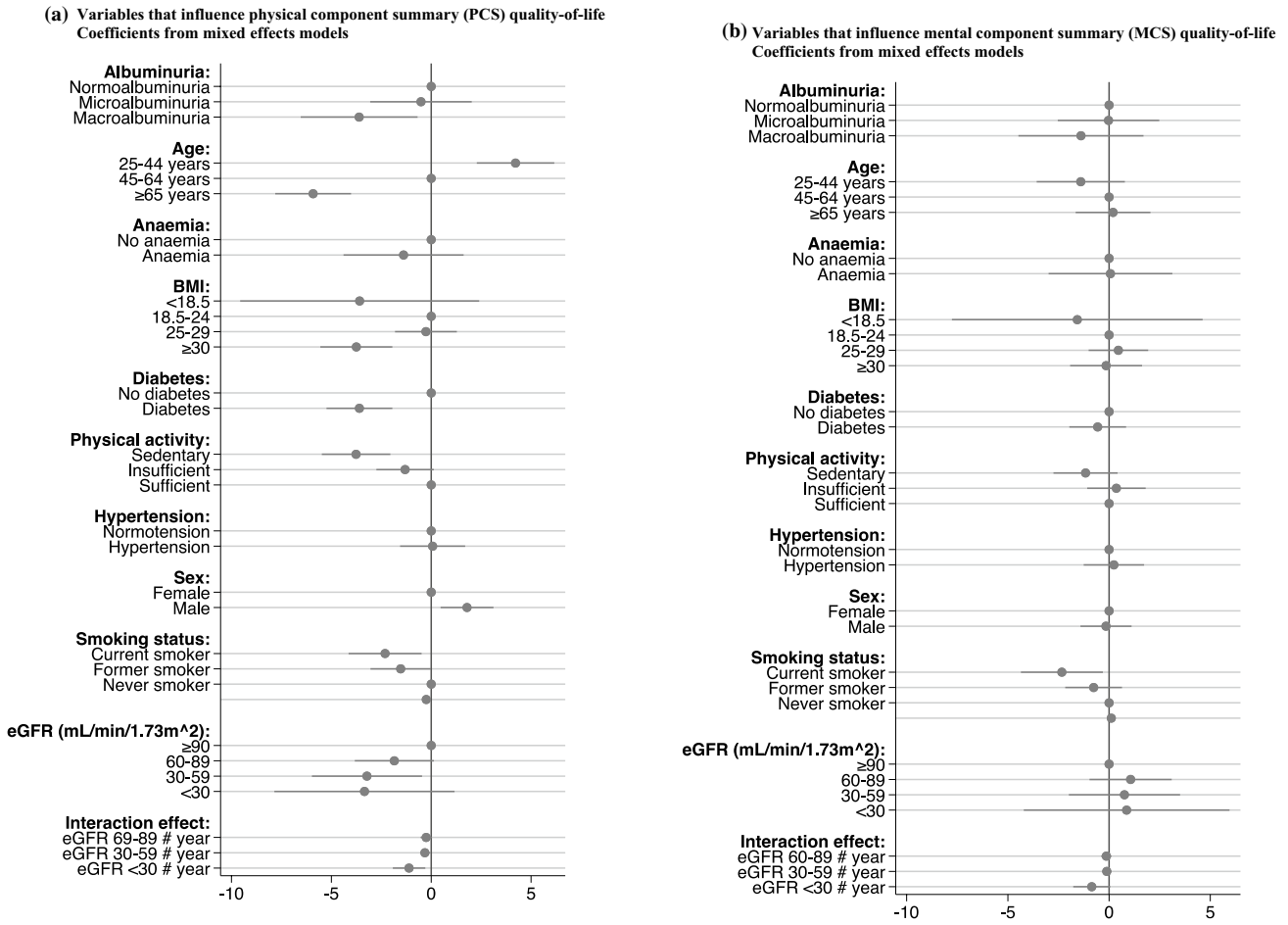
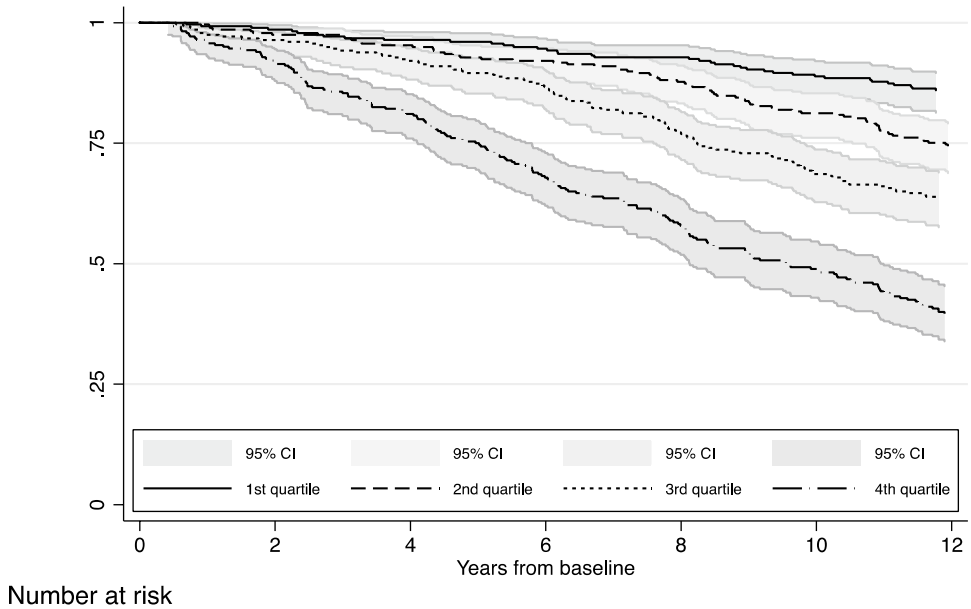


Fig. 3 Mixed effects model: Variables that influence quality-of-life. **a** Variables that influence PCS quality-of-life coefficients from mixed effects models. **b** Variables that influence MCS quality-of-life coefficients from mixed effects models

Fig. 4 Survival by PCS quartile



Number at risk

1st quartile	278	274	268	263	258	247	239
2nd quartile	277	270	264	255	243	225	206
3rd quartile	277	267	255	240	213	190	176
4th quartile	280	257	227	190	162	137	111

Table 3 Cox proportional hazards regression, all-cause mortality

Baseline variables	Hazard ratio	95% confidence interval	*p* value*
Age (years)			< 0.001
25–44	0.3	0.1–0.8	
45–64	Reference		
≥ 65	4.0	2.8–5.6	
Male sex	1.4	1.2–1.8	< 0.001
PCS			< 0.001
0–19	3.6	2.3–5.7	
20–29	3.2	2.3–4.5	
30–39	1.8	1.3–2.4	
40–49	1.5	1.1–2.0	
50–59	Reference		
≥ 60	1.1	0.3–4.6	
MCS			0.6
0–19	0.5	0.1–1.5	
20–29	0.7	0.4–1.3	
30–39	0.9	0.6–1.2	
40–49	1.1	0.8–1.4	
50–59	Reference		
≥ 60	1.0	0.7–1.3	
eGFR (mL/min/1.73 m^2)			< 0.001
≥ 90	Reference		
60–89	1.9	1.4–2.7	
30–59	2.8	1.9–4.1	
< 30	5.6	3.2–9.6	
Albuminuria			< 0.001
None	Reference		
Microalbuminuria	1.8	1.4–2.4	
Macroalbuminuria	3.3	2.3–4.8	

*Wald test used to calculate *p* values

ESKD; however, CKD utilities have been largely ignored. We found the unadjusted utility of CKD was 0.60 for stage one, and there was a clinically and statistically significant decline by stage. However, when adjusted for age and sex, the differences between CKD stages were no longer statistically significant. This may be because the primary quality-of-life impact of CKD is in the physical domains, with mental quality-of-life scores proving remarkably resilient. Because utility is a single measure which combines both physical and mental domains of quality-of-life, the adverse physical impact may be diluted.

Norman et al. have previously found the SF-6D utility in the general Australian population to have a mean of 0.74 (95% CI 0.73–0.75) in those aged 61–70 years (the age bracket within which our study population's mean age falls) [31]. With mean utilities 0.14 higher than those with even the earliest stages of CKD, this suggests that CKD leads to a substantial impairment in quality-of-life. However, the Australian general population utilities may not be directly comparable to ours as Norman et al. relied on the UK algorithm to determine SF-6D utilities from SF-36 data as the Australian algorithm was yet to be developed [31].

Our study has a number of limitations. First, we used a single serum creatinine and spot urine test at baseline to determine CKD status. This may have led to overdiagnosis of CKD and a consequent reduction in the power of the associations that we found. Second, our cohort had relatively few participants with eGFR < 30 mL/min/1.73 m^2 which limits the conclusions we could draw from this population. Thirdly, a portion of the initial cohort was lost to follow-up. Whilst capture of all deaths was complete, through linkage to the National Death Index, by the end of the study 25% of the baseline population who remained alive had not presented for follow-up. It is possible that patients who did not present for follow-up were sicker than those that did (i.e. informative censoring). Any informative censoring would have the effect of reducing the power of the associations that we found.

Table 4 Utility decrement by CKD stage

CKD stage at baseline	Baseline		5-year follow-up		12-year follow-up	
	n	Coef (Std Err)	*n*	Coef (Std Err)	*n*	Coef (Std Err)
Unadjusted						
Stage 1	324	0.60 (0.01)	159	0.62 (0.02)	39	0.42 (0.04)
Stage 2 (compared to stage 1)	208	−0.08 (0.02)***	76	-0.08 (0.04)*	18	-0.05 (0.07)^{ns}
Stage 3 (compared to stage 1)	226	−0.10 (0.02)***	96	-0.11 (0.03)**	17	-0.01 (0.07)^{ns}
Stage 4/5 (compared to stage 1)	20	−0.15 (0.06)*	4	-0.20 (0.13)^{ns}	1	-0.23 (0.25)^{ns}
Age and sex adjusted						
Stage 1	324	0.58 (0.02)	159	0.60 (0.03)	39	0.30 (0.06)
Stage 2 (compared to stage 1)	208	−0.02 (0.03)^{ns}	76	0.02 (0.04)^{ns}	18	0.04 (0.07)^{ns}
Stage 3 (compared to stage 1)	226	−0.03 (0.03)^{ns}	96	0.02 (0.04)^{ns}	17	0.13 (0.09)^{ns}
Stage 4/5 (compared to stage 1)	20	−0.08 (0.06)^{ns}	4	−0.08 (0.13)^{ns}	1	−0.18 (0.24)^{ns}

*p < 0.05, **p < 0.01, ***p < 0.001, ns = not significantly different from baseline

In summary, CKD is associated with lower physical, but not mental, quality-of-life. Physical quality-of-life declines with each progressive stage of CKD, and is associated with higher risks of all-cause and cardiovascular mortality. Quality-of-life imparts key clinical information and may be used to inform decisions regarding new treatments or changes in health policy for people with CKD. Clinicians can use these findings to identify community-based individuals who are more likely to experience adverse outcomes (progression or death) and thus warrant aggressive treatment strategies and/or earlier referral to nephrology services to plan for ESKD management.

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Compliance with ethical standards

Conflict of interest All authors declare they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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