Chronic kidney disease (CKD) is rising in prevalence, increasingly in clinical practice, treatment guidelines, screening and education campaigns, health care utilization planning, and goal development for risk reduction and preventative behavior. 

Although the absolute risk of disease development in a given year may be small, the lifetime risk (for an individual at birth) and residual lifetime risk (for an individual currently free of disease) of common diseases can be high. The lifetime risk of diabetes is 33%-39%, and the residual lifetime risks of hypertension and diabetes for a middle-aged man are 11.5%, and 3.6%, respectively. Women experienced greater CKD risk yet lower ESRD risk than men; blacks of both sexes had markedly higher CKD stage 4 and ESRD risks (lifetime risks for white men, white women, black men, and black women, respectively: CKD stage 3a+ 53.6%, 64.9%, 51.8%, and 63.6%; CKD stage 3b+ 29.0%, 36.7%, 33.7%, and 40.2%; CKD stage 4+ 9.3%, 11.4%, 15.8%, and 18.5%; and ESRD, 3.3%, 2.2%, 8.5%, and 7.8%). Risk of CKD increased with age, with approximately one-half the CKD stage 3a+ cases developing after 70 years of age.

Limitations: CKD incidence was modeled from prevalence estimates in the US population. ESRD incidence and prevalence were modeled from US population mortality and morbidity rates.

Conclusions: In the United States, the lifetime risk of developing CKD stage 3a+ is high, emphasizing the importance of primary prevention and effective therapy to reduce CKD-related morbidity and mortality.

INDEX WORDS: Chronic kidney disease; end-stage renal disease; incidence; lifetime risk.
kidney disease than whites, particularly in the more severe stages. Women live longer than men and accordingly may face a greater lifetime CKD burden. The objective of this study thus was to estimate age-, race-, and sex-specific residual lifetime risks of CKD stages 3a+, 3b+, and 4+, and to update US estimates for the residual lifetime risk of ESRD.

METHODS

Lifetime Risk Estimates

The residual lifetime risks of 4 kidney disease outcomes were independently estimated: CKD stage 3a+ (eGFR <60 mL/min/1.73 m²), as estimated using the CKD-EPI [CKD Epidemiology Collaboration] creatinine [2009] equation), CKD stage 3b+ (eGFR <45 mL/min/1.73 m²), CKD stage 4+ (eGFR <30 mL/min/1.73 m²), and ESRD (chronic kidney failure treated by dialysis or transplantation). For each outcome, a separate Markov chain model was designed to simulate progression of an initially outcome-free individual of given sex, race, and baseline age through the mutually exclusive states of no kidney disease, kidney disease, and death, with kidney disease and death treated as absorbing states (Fig 1). State transition probabilities were specified as: (1) the probability of dying prior to the development of kidney disease (defined as CKD stage 3a+, 3b+, 4+, or ESRD; QnoCKD), and (2) the probability of developing kidney disease (lCKD). The formulas for calculating each state transition probability are provided as online supplementary material; estimates are provided in Tables S1 (CKD stage 3a+), S2 (CKD stage 3b+), S3 (CKD stage 4+), and S4 (ESRD). Monte Carlo simulations were conducted in a simulated cohort of 10,000 individuals of specified race, sex, and baseline age, with a lifetime horizon (capped at 90 years), a cycle length of 1 year, and an individual perspective. Age-specific transition probabilities were assumed to be unchanged throughout the lifetime of the simulated cohort; progression through CKD stages was not modeled.

Data Sources

Annual probabilities of death for the US population by age, sex, and race were obtained from the National Vital Statistics Report. Sex- and race-adjusted estimates of the relative risk of mortality associated with CKD by category of eGFR and urine albumin-creatinine ratio (ACR) and in 4 age groups (18-54, 55-64, 65-74, and ≥75 years) were obtained from a previously published mortality analysis encompassing more than 2 million participants and 46 cohorts (Table S5). CKD prevalence was estimated using data from the National Health and Nutrition Examination Survey (NHANES), a multi-stage population-level survey of community-dwelling US civilians conducted by the National Center for Health Statistics. The present study included surveys conducted in 1988-1994 (2 phases, 1988-1991 and 1991-1994), 1999-2004 (3 phases, 1999-2000, 2001-2002, and 2003-2004), and in the continuous NHANES. The study population was limited to individuals 20 years and older with nonmissing serum creatinine, urine albumin, and urine creatinine values. Data were analyzed using National Center for Health Statistics–provided sample weights, primary sampling units, and strata specific to each survey.

The incidence, prevalence, and mortality rates for ESRD in 2009 were provided by the US Renal Data System (USRDS) in 5- to 10-year age intervals. All rates were specific to sex and race (white or black). Incidence and mortality rates (R) were converted to 1-year probabilities (Q) using the following formula: Q=R/(1-0.5×R). US population estimates for 2012 by age, sex, and race (non-Hispanic blacks and non-Hispanic whites) were obtained from US Census Bureau projections.

Disease Prevalence Estimates

Multinomial logistic regression was used to model the probability of CKD categories, defined by eGFR and ACR, to improve the precision of age-specific NHANES estimates of CKD prevalence (Fig S1). The base model for CKD stage 3a+ prevalence fit 3 eGFR categories (45-<60, 30-<45, and <30 mL/min/1.73 m²) and 4 ACR categories (<10, 10-30, >30-300, and >300 mg/g), for a total of 12 mutually exclusive CKD categories, as a function of age (a linear variable), race, and sex. No adjustment was made for survey year, a conservative approach given recent increases in CKD prevalence. For ESRD prevalence, actual sex- and race-specific figures supplied by the USRDS were used in the mortality calculations, assuming constant prevalence in each age interval provided.

State Transition Probabilities

The 1-year probability of death for persons of a given age, sex, and race and without CKD (QnoCKD) were calculated using age-, sex-, and race-specific estimates of mortality in the overall population (ie, in those with and without CKD). CKD prevalence, and hazard ratios (HRs) for mortality associated with CKD (Item S1, part A). All sex- and race-adjusted HRs used the same referent category (eGFR, 90-105 mL/min/1.73 m²; ACR <10 mg/g) and were assumed constant within each age range (18-54, 55-64, 65-74, and ≥75 years). In addition, HRs for those younger than 18 years were assumed to be equivalent to those in the 18- to 54-year age group (Table S5). Age-specific CKD incidence (lCKD) was estimated from the proportion of individuals with CKD at age x+1, less than those with CKD at age x and taking into account both the competing risk of death prior to CKD development (QnoCKD) and the 1-year probability of death in prevalent cases of CKD (QCKD: Item S1, part B).

For the model of lifetime risk of ESRD, mortality rates for persons of a given age, sex, and race and lacking ESRD were calculated using USRDS-supplied ESRD prevalence and mortality rates and converted to probabilities (QnoESRD) using established methods. The age-specific incidence of ESRD (lESRD) was estimated using rates provided by the USRDS.

Sensitivity Analyses

Forecasts of lifetime CKD risk are highly sensitive to estimates of CKD incidence (lCKD). Our equation estimates CKD incidence...
as a function of CKD prevalence (P_{CKD}), and the competing risk of death prior to kidney disease (Q_{noCKD}), which in turn is a function of CKD-associated mortality (HR_{CKD}); all assume constant probabilities for a given age over time (i.e., I_{CKD} among 30-year-olds in 2000 is the same as I_{CKD} among 30-year-olds in 2050). We therefore evaluated the robustness of our models in the following ways. First, we tested the impact of a more complex estimation of CKD prevalence (P_{CKD}), modeling prevalence as a function of age as a linear spline (knot at 70 years), race, sex, and 2 age-by-sex and age-by-race interaction terms. Second, we tested the effect of lower CKD-associated mortality (HR_{CKD}) using a 2-CKD category model (eGFR <60 mL/min/1.73 m^2 with and without ACR >30 mg/g) and the most conservative HRs in each category (eGFR of 45-60 mL/min/1.73 m^2 with ACR <10 mg/g and ACR of 30-300 mg/g, respectively). Third, we capped CKD stage 3a+ incidence at 5% (affecting white men 76 years and older, white women 75 years and older, black men 72 years and older, and black women 73 years and older). Finally, we tested the impact of increasing incidence rates by modeling CKD prevalence on survey year in addition to age, sex, and race.

To test the validity of modeling CKD incidence on estimates of CKD prevalence, we compared our modeled estimates of CKD incidence to those observed in the Atherosclerosis Risk in Communities (ARIC) Study, a population-based cohort of black and white middle-aged individuals with interval assessment of serum creatinine.

**Statistical Analysis**

All model input estimates were calculated using Stata SE, version 11.2 (StataCorp LP) and are available in Tables S1-S5. Markov models were constructed and microsimulations were conducted using C++ and TreeAge Pro 2012.

**RESULTS**

**Residual Lifetime Risk of Kidney Disease**

From birth, lifetime risks in the overall US population were an estimated 59.1% for CKD stage 3a+, 33.6% for CKD stage 3b+, 11.5% for CKD stage 4+, and 3.6% for ESRD. In general, residual lifetime risk (ie, remaining lifetime risk, conditional on disease-free status) for CKD increased until age 70 years, reflecting the generally late onset of kidney disease and the increase in life expectancy with attained age. The data in Table 1 are based on US life tables issued in 2001.

**Table 1. Life Expectancy and Residual Lifetime Risk of CKD Stages 3a+, 3b+, 4+, and ESRD by Baseline Age**

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Life Expectancy</th>
<th>Residual Lifetime Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CKD stage 3a+</td>
<td>CKD stage 3b+</td>
</tr>
<tr>
<td></td>
<td>White men</td>
<td>White women</td>
</tr>
<tr>
<td>At Birth</td>
<td>76</td>
<td>77</td>
</tr>
<tr>
<td>10 y</td>
<td>64.9</td>
<td>65.2</td>
</tr>
<tr>
<td>20 y</td>
<td>51.8</td>
<td>52.9</td>
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<tr>
<td>30 y</td>
<td>63.6</td>
<td>64.3</td>
</tr>
<tr>
<td>40 y</td>
<td>53.6</td>
<td>54.2</td>
</tr>
<tr>
<td>50 y</td>
<td>64.9</td>
<td>65.2</td>
</tr>
<tr>
<td>60 y</td>
<td>51.8</td>
<td>52.9</td>
</tr>
<tr>
<td>70 y</td>
<td>63.6</td>
<td>64.3</td>
</tr>
<tr>
<td>80 y</td>
<td>53.6</td>
<td>54.2</td>
</tr>
</tbody>
</table>

**Note:** By race, sex, and decade of age until age 90 years.

**Abbreviations:** CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease.

*b Based on Arias.28

**Values given as percentages.**

eGFR <60 mL/min/1.73 m^2.

eGFR <45 mL/min/1.73 m^2.

eGFR <30 mL/min/1.73 m^2.
baseline age (Table 1). In contrast, the residual lifetime risk of treated ESRD was highest for 20- to 30-year-olds, reflecting the relative decline in the probability of ESRD compared with death at older ages (Table S4).

**Lifetime Risk of Kidney Disease by Sex and Race**

Lifetime risk estimates differed by sex and race (Table 1). Women faced higher CKD stage 3a+ risk: from birth, predicted lifetime risks for a white man, white woman, black man, and black woman were 53.6%, 64.9%, 51.8%, and 63.6%, respectively. Blacks faced higher risks of CKD stage 4+ and ESRD: from birth, the predicted lifetime risks for a white man, white woman, black man, and black woman were 9.3%, 11.4%, 15.8%, and 18.5% for CKD stage 4+ and 3.3%, 2.2%, 8.5%, and 7.8% for ESRD. Thus, within each race, women had higher lifetime risks of CKD, whereas men had higher risks of ESRD.

The estimated onset of kidney disease was earlier in blacks than whites (Fig 2). For example, by age 60 years, 10.3% of black men and 10.4% of black women were projected to have CKD stage 3a+ compared with 6.9% and 7.8% for white men and women. Whites “caught up” in CKD stage 3a+ risk at older ages, but the earlier onset of risk for CKD stage 4+ and ESRD for blacks persisted. As such, white women had the highest risk of CKD stage 3a+ (64.9%), black women had the highest risk of stage 4+ CKD (18.5%), and black men had the highest risk of ESRD (8.5%).

**Lifetime Risk Scaled to the Current US Population**

Of the 178.3 million non-Hispanic white US citizens, an estimated 13.5 million have CKD stage 3a+ and another 100.5 million were predicted to develop it during their remaining lifetime (Fig 3). Of the 34.8 million black US citizens, an estimated 1.8 million have CKD stage 3a+ and an additional 20.0 million were predicted to develop it during their lifetime. Overall, 135.8 million people (63.8%) in the current US white and black population either have (7.2%) or are expected to develop (56.6%) CKD stage 3a+ during their lifetime. Similarly, a projected 26.1 million (7.4%) either have or are expected to develop CKD stage 4+ (Figs S2 and S3).

**Sensitivity Analyses**

In sensitivity analyses using more complex models of CKD prevalence and lower rates of CKD-associated mortality, lifetime risk estimates for CKD stage 3+ ranged from 45.9%-53.6% for white men, 54.5%-64.9% for white women, 44.2%-51.8% for black men, and 55.8%-63.6% for black women (Table 2). In all scenarios, the population-weighted lifetime risk of...
kidney disease was >50%. In a sensitivity analysis using survey year in the model of CKD prevalence (and thus allowing CKD incidence to vary by year), lifetime risk estimates were much higher at 72.9%, 82.6%, 65.8%, and 77.5% for white men, white women, black men, and black women, respectively. Compared with age-, sex-, and race-specific CKD incidence observed in the prospective middle-aged population-based ARIC study, our model-based estimates were similar and generally lower, resulting in a more conservative estimate of lifetime CKD incidence. For example, our estimates of 20-year CKD stage 3a+ incidence for an initially disease-free 45-year-old were slightly lower than those observed in ARIC for white men, white women, and black women (absolute differences of −2.6%, −0.9%, and −0.1%, respectively) and slightly higher than those observed for black men (absolute difference, 0.4%). Differences of similar magnitude were observed for the incidence of CKD stage 4+ (absolute differences: 0.6%, 0.9%, −1.5%, and 1.8% for white men, white women, black men, and black women, respectively).

DISCUSSION

In this simulation study, the estimated lifetime risk of CKD stage 3a+ was >50%, lower than that of hypertension (83%-90% for a 55-year-old), but higher than those for diabetes (33%-39%), coronary heart disease (32%-49% for a 40-year-old), and invasive cancer (38%-45%). The lifetime risks of CKD stages 3b+ and 4+ and ESRD also were considerable at 33.6%, 11.5%, and 3.6%, respectively. Consistent with previous studies, the risk of CKD stage 3a+ increased dramatically with age, with approximately half the incident cases occurring after age 70 years, an observation even more pronounced in incident CKD stage 4+. In contrast, cases of ESRD developed earlier, reaching a plateau at older ages.

The strong relationship between age and incident CKD leads to an interesting result: those with the longest life expectancy had the highest risk of CKD stage 3a+. This observation, consistent with previous demonstrations of CKD incidence in older populations (eg, 20.5% incidence during a 24-month period in the Women’s Health and Aging Study I), fuels the long-standing debate about whether eGFR decline is “normal aging” or a pathologic process. Although the present study was not designed to address this controversy, it relies on HRs from a recent meta-analysis that suggested that CKD-associated risks persist in older adults, albeit with diminished relative risks. In contrast to the relationship between age and CKD, the relationship between age and ESRD incidence was not monotonic: rates declined after age 75 years, perhaps reflecting a more frequent refusal of renal replacement therapy and a higher competing risk of pre-ESRD death for older adults.

Lifetime estimates of kidney disease risk differed substantially by sex and race. White women faced the highest risk of CKD stage 3a+, yet the lowest risk of treated ESRD, possibly reflecting the older onset of kidney disease (with respect to blacks), slower CKD progression, or differences in the acceptance of renal replacement therapy. Black individuals faced higher risks of kidney disease at earlier ages. For CKD stage 4+ and ESRD, the risk difference was dramatic and persistent over a lifetime, findings consistent with the seemingly paradoxical lower prevalence yet higher incidence of moderate CKD in the US black population. This may suggest a susceptibility to CKD progression, whether due to diminished access to medical care, genetic predisposition (eg, the prevalence of APOL1 high-risk variants), differences in disease cause, or lower competing risks of death in older age.

Our results expand upon the published literature in several ways. Models were based on the current US general population, stratified by sex and race, and they...
estimated the lifetime risk of not only ESRD, but also more moderate forms of CKD. Risk of CKD has been simulated for a study of cost-effectiveness in the United States.19 However, the forecasts were solely among the overall population, with no stratification by sex and race and few age groups. Previous studies of ESRD risk used different populations in nationality or era.16–18 Kiberd and Clase’s16 study of ESRD risk provided useful US estimates based on mortality rates from 1998 and ESRD incidence in 1996–1998, but rates have changed significantly since that time.44 More recently published studies estimated ESRD risk among Canadian participants receiving medical care, in which the majority of the population is likely to be white.17,18 Our study updates risk estimates in US whites and US blacks, who shoulder a disproportionate amount of disease.9

The estimate that >50% of the US population will develop CKD stage 3a+ is higher than that in a 2010 simulation study,19 but consistent with the estimate that CKD prevalence nears 40% after age 70 years.5 Given that the average life expectancy is much older than 70 years for most individuals (black men being the exception28), that the risk of low eGFR increases with older age,20,21,35 and that persons who develop CKD at younger ages have a much higher mortality risk than those without CKD,27 the previously published US population lifetime CKD risk estimate of 38.5% is too low to be consistent with current prevalence estimates. This discrepancy may be secondary to difficulties estimating CKD incidence rates: the simulation study estimated incidence using projections of eGFR decline, assuming a fixed multiplier for CKD-associated mortality irrespective of age.19 This method is highly sensitive to laboratory drift in serum creatinine assays, which influences eGFR decline and the modeled distribution of eGFR slopes. CKD incidence estimated from eGFR decline is driven primarily by those who rapidly experience progression, a tail of the distribution that can be difficult to estimate precisely.

Our estimates of lifetime ESRD risk, as well as the dramatic racial disparities in risk of kidney disease, are consistent with previous studies.9,22,23,26,45 Kiberd and Clase16 estimated lifetime risks of 7.3% and 7.8% for black men and women, respectively, compared with 2.5% and 1.8% for white men and women. Using current ESRD incidence rates, we demonstrate an even higher lifetime risk for all except black women. Somewhat surprisingly, ESRD risk estimates for white Americans were only slightly higher than those recently published from Alberta, Canada.17 In their cohort of insured patients, residual lifetime risks of ESRD were estimated at 2.7% and 1.8% for 40-year-old men and women, respectively, compared with 2.5% and 1.8% for white men and women. Using current ESRD incidence rates, we demonstrate an even higher lifetime risk for all except black women. The small differences may reflect differences in disease progression, the competing risk of non-ESRD death, or practice patterns, including eGFR at renal replacement therapy initiation and the use of conservative (nondialysis) therapy, a practice that may be more prevalent in Canada.39

Unlike those generated from a prospective cohort, our estimates of CKD incidence were modeled on US

### Table 2. Sensitivity Analyses: Lifetime Risk From Birth to Age 90 Years of CKD Stage 3a+

<table>
<thead>
<tr>
<th>Category</th>
<th>Simple Prevalence Model</th>
<th>Complex Prevalence Model</th>
<th>Increasing Prevalence Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12 CKD Categories</td>
<td>Capped Incidence</td>
<td>2 CKD Categories</td>
</tr>
<tr>
<td>White men</td>
<td>53.6</td>
<td>45.9</td>
<td>47.8</td>
</tr>
<tr>
<td>White women</td>
<td>64.9</td>
<td>55.4</td>
<td>58.4</td>
</tr>
<tr>
<td>Black men</td>
<td>51.8</td>
<td>45.3</td>
<td>45.5</td>
</tr>
<tr>
<td>Black women</td>
<td>63.6</td>
<td>56.3</td>
<td>57.0</td>
</tr>
<tr>
<td>Total</td>
<td>59.1</td>
<td>50.4</td>
<td>53.0</td>
</tr>
</tbody>
</table>

Note: Values given as percentages. Simple prevalence model covariates: age (linear), sex, and race; complex prevalence model covariates: age (linear spline with a knot at 70 years), sex, race, and age-sex and age-race interaction terms; increasing prevalence model covariates: age (linear), sex, race, and survey year.

Abbreviations: ACR, albumin-creatinine ratio; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease; HR, hazard ratio.

*aThe number of CKD categories is reflected in the mortality risk: the 2-CKD category model is more conservative, classifying participants into eGFR <60 mL/min/1.73 m² with or without ACR >30 mg/g and using the HRs associated with eGFR of 45–60 mL/min/1.73 m², ACR <10 mg/g, and eGFR of 45–60 mL/min/1.73 m², ACR of 30–300 mg/g. The 12-category model uses specific HRs associated with each ACR/eGFR category; because the HRs generally increase with more CKD, this results in higher estimates of CKD mortality.*

*bThe capped incidence model is the 12-CKD category simple prevalence model, with annual incidence rates capped at 5%.

*cTotal values reflect the combination of lifetime risks, weighted by race- and sex-specific prevalence in the US population.
prevalence estimates. We chose this method for several reasons. First, national data for CKD incidence are sparse, and incidence estimates from a prospective cohort are extremely sensitive to laboratory drift, or instability in the serum creatinine assay over time. Our cross-sectional model is robust to this drift. Second, we need not extrapolate from follow-up times far shorter than the average lifetime, and we do not presume constant rates of eGFR decline. Third, using NHANES prevalence facilitates application to the general population. Our method relies on different assumptions but has the strength of providing lifetime CKD estimates that match the current US population prevalence. Reassuringly, in sensitivity analysis, our prevalence-based estimates of CKD incidence were very similar to those observed in the ARIC cohort, a population-based sample of middle-aged adults.

The central assumptions of our CKD models are: (1) a stable population (ie, no change in population structure, age-specific life expectancy, and CKD incidence rates) and (2) irreversibility of disease (ie, once CKD stage 3+ or 4+ develops, it is present until death). For a population with increasing life expectancy such as the United States, the equation we use provides a conservative estimate of CKD incidence. Similarly, an increase in CKD incidence, a real possibility given the projected increases in diabetes, hypertension, and obesity, would result in higher lifetime risk than we have presented. If CKD were to remit, we would underestimate CKD incidence, although estimates of lifetime risk might be less meaningful.

Additional limitations include the selection bias inherent in NHANES prevalence estimates; those with more severe disease may be less inclined to participate in the survey, particularly in older age groups, and a noted survey exclusion criterion is nursing home residence. However, this limitation also would apply to a prospective study and likely result in a conservative estimate of lifetime CKD risk. Finally, in estimating ESRD risk, we include those receiving dialysis or transplantation only, which may substantially underestimate those with untreated chronic kidney failure (eGFR <15 mL/min/1.73 m² and death). In summary, we estimate that >50% of Americans born today will develop CKD stage 3+ during their lifetime. Racial disparities, particularly in severe disease, are marked. Among whites, approximately 9%-11% will develop CKD stage 4+ and 2%-3% will develop ESRD; among blacks, the risks of CKD stage 4+ and ESRD are 16%-18% and 8%, respectively. Future research is needed in methods of preventing the development of CKD and ameliorating its associated morbidity.

ACKNOWLEDGEMENTS

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SUPPLEMENTARY MATERIAL

Table S1: State transition probabilities for CKD 3a+, by race, sex, and age.
Table S2: State transition probabilities for CKD 3b+, by race, sex, and age.
Table S3: State transition probabilities for CKD 4+, by race, sex, and age.
Table S4: State transition probabilities for ESRD, by race, sex, and age.
Table S5: Sex- and race-adjusted HRs of mortality associated with CKD, by age group.

Figure S1: Actual vs predicted US prevalence of CKD 3a+, by age, sex, and race.
Figure S2: CKD 4+ projections by age, race, and sex, scaled by estimated 2012 US population.
Figure S3: ESRD projections by age, race, and sex, scaled by estimated 2012 US population.
Item S1: State transition probability formulas.

Note: The supplementary material accompanying this article (http://dx.doi.org/10.1053/j.ajkd.2013.03.009) is available at www.ajkd.org.

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