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Historical Neighborhood Redlining and Cardiovascular Risk in Patients with Chronic Kidney Disease

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The leading cause of death among individuals with Chronic Kidney Disease (CKD) is Cardiovascular Disease (CVD). Social risk factors, including the effects of neighborhoods and systemic racism, have been as associated with CVD risk. During the 1930s, the United States government-sponsored Home Owners' Loan Corporation (HOLC) implemented a grading system to evaluate neighborhood risk for mortgage applications.[1] Neighborhoods rated "A", depicted in green, represented the wealthiest areas, while areas rated "D", depicted in red, represented the poorest neighborhoods and were referred to as "redlined." This practice resulted in residential segregation and has contributed to persistent systemic racism.[1] We have previously demonstrated a link between redlining and increased neighborhood-level cardiometabolic risk, including CVD and CKD, throughout the US.[2] The aim of this study is to investigate the association between redlining and the prevalence and incidence of CVD in a contemporary prospective cohort of patients with CKD.

The data that support the findings of this study are available from the corresponding author. Data were obtained from participants that enrolled in the Chronic Renal Insufficiency Cohort in 2003–2008. The study protocol was approved by institutional review boards at the participating institutions.[3] All human subjects provided informed consent to participate in the study. Briefly, adults with mild to moderate CKD were recruited from 7 sites and followed prospectively for incident CVD. Events are adjudicated via phone calls and review of medical records. The residential addresses of participants were geocoded into census block groups at study entry and linked to HOLC-graded maps generated from digitized maps. We examined the association of participant neighborhood HOLC grade with prevalent

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(logistic regression) and incident (cox regression models in patients without prevalent CVD) CVD events (myocardial infarction, peripheral arterial disease, stroke, heart failure, atrial fibrillation, and all-cause death), adjusting for age, sex, race, ethnicity, estimated glomerular filtration rate (eGFR), smoking, low density lipoprotein cholesterol (LDL), diabetes, systolic and diastolic blood pressure (BP) and urine albumin-creatinine ratio. We also employed Bonferroni corrections to account for multiple comparisons.

A total of 1720 participants were included (553 in HOLC D). Worsening neighborhood HOLC grade was associated with an increased proportion of Black and Hispanic residents, younger age, higher proteinuria, higher systolic BP, and higher high-sensitivity troponin levels (all $P<0.01$), as determined using analysis of variance for continuous and Chi-Square for categorical variables. In multivariable-adjusted models, group B (Odds Ratio [OR] 2.34 [95% CI: 1.32–4.15]), group C (OR 2.31 [1.34–3.98]) and group D (OR 2.10 [1.21–3.65]) were associated with increased baseline CVD (reference, HOLC group A) (Table 1). These relationships were not attenuated after further adjustment for household income and education (B vs A: OR 2.31 [1.30–4.10]; C vs A: OR 2.25 [1.30–3.88]; D vs A: OR 2.04 [1.17–3.56]). Among 1118 participants without baseline CVD, group D was associated with increased risk for HF or all-cause death (adjusted hazard ratio [HR] 2.63 [1.31–5.28]), (table 1) which remained unchanged after further adjustment for B-type natriuretic peptide (BNP) and high-sensitivity troponins (D vs A: HR 2.55 [1.27–5.13]), or education and income (D vs A: HR 2.42 [1.20–4.86]).

Historical residential segregation policies continue to impact present-day health risk in patients with CKD. This study suggests that patients with mild-moderate CKD, current residents of historically redlined neighborhoods have a 2-fold higher risk of HF, independently of established CVD risk factors. The mechanisms of these associations remain speculative. We have previously demonstrated that current residents of historically poor HOLC groups have worse adverse environmental exposures[4] which may mediate increased CVD risk. These conditions may constitute geographical footprints that significantly impact life expectancy and disease burden, even in high-income countries.[5] As such, potential mechanisms linking redlining with intergenerational CVD risk, and interventions to reduce risk, need to be elucidated in future studies.

This study has important implications for both policymaking and clinical practice. The results suggest that policies surrounding residential segregation can have a persistent, multi-generational impact on chronic health outcomes. As a result, the health effects of neighborhoods and mortgage legislation should be carefully considered, particularly among minority populations. The identification of high-risk neighborhoods through redlining highlights the need for targeted community engagement strategies and healthcare investments. Furthermore, neighborhood characteristics such as redlining risk should be considered in the assessment of individual patient risk for future events, allowing for the implementation of intensive risk reduction strategies. This may necessitate the incorporation of neighborhood disadvantage in risk prediction models and the establishment of calibration across different patient populations.

This study should be interpreted within the context of limitations. First, this is a relatively small study from 7 cities and thus may not be generalizable to all patients with CKD. Additionally, there is the potential for confounding factors to influence the relationship between HOLC risk and CVD risk. The OR for prevalent CVD in group D overlaps with those of groups B and C, making it challenging to discern a clear dose-dependent relationship. A larger cohort may be needed to detect a more pronounced association. Nevertheless, this analysis is strengthened by prospective design and adjudicated outcomes.

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Non-standard Abbreviations and Acronyms:

BNP	B-type natriuretic peptide
BP	blood pressure
CKD	chronic kidney disease
CVD	cardiovascular disease
eGFR	estimated glomerular filtration rate
HOLC	Home Owners' Loan Corporation
HR	hazard ratio
LDL	low density lipoprotein cholesterol
OR	odds ratio

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Table 1.

Association between HOLC risk with prevalent and incident cardiovascular disease

	Model 1			Model 2		
	B vs A	**C vs A**	**D vs A**	**B vs A**	**C vs A**	**D vs A**
Prevalent Cardiovascular Disease (Logistic Regression Model, N=1,720)						
Any CVD	**2.08 (1.23–3.51), P=0.007***	**2.19 (1.33–3.61), P=0.002***	**1.96 (1.18–3.26), P=0.009**	**2.34 (1.32–4.15), P=0.003***	**2.31 (1.34–3.98), P=0.003***	**2.10 (1.21–3.65), P=0.009**
Myocardial Infarction or Revascularization	**2.42 (1.29–4.56), P=0.006***	**2.03 (1.11–3.73), P=0.022**	**1.97 (1.06–3.66), P=0.033**	**2.36 (1.22–4.57), P=0.011**	1.85 (0.98–3.50), P=0.059	1.83 (0.96–3.51), P=0.069
Peripheral artery disease	1.95 (0.55–6.87), P=0.298	2.91 (0.88–9.63), P=0.079	2.69 (0.80–9.03), P=0.109			
Heart failure	1.67 (0.75–3.73), P=0.21	1.52 (0.71–3.28), P=0.283	1.54 (0.71–3.36), P=0.275			
Stroke	1.83 (0.74–4.56), P=0.193	2.21 (0.93–5.25), P=0.073	2.00 (0.83–4.82), P=0.121			
Atrial fibrillation	1.61 (0.86–2.98), P=0.134	1.68 (0.93–3.02), P=0.083	1.36 (0.74–2.49), P=0.319			
Incident Cardiovascular Disease in Patients without Prevalent CVD (Cox Model, N=1,118)						
Myocardial infarction	1.17 (0.31–4.32), P=0.82	1.47 (0.44–4.93), P=0.53	1.37 (0.40–4.71), P=0.614			
Peripheral artery disease	0.94 (0.18–4.92), P=0.942	1.03 (0.22–4.74), P=0.972	1.53 (0.32–7.24), P=0.592			
Heart failure	1.56 (0.52–4.71), P=0.432	2.47 (0.89–6.88), P=0.084	**3.02 (1.08–8.51), P=0.036**	1.41 (0.47–4.28), P=0.542	2.03 (0.73–5.68), P=0.175	2.46 (0.87–6.94), P=0.089
Stroke	0.92 (0.18–4.75), P=0.916	0.84 (0.18–3.86), P=0.822	1.03 (0.22–4.79), P=0.968			
Atrial fibrillation	1.12 (0.45–2.75), P=0.809	1.52 (0.67–3.44), P=0.316	1.74 (0.74–4.10), P=0.204			
Composite outcome†	1.31 (0.59–2.90), P=0.505	1.73 (0.83–3.61), P=0.146	2.07 (0.98–4.36), P=0.056			
Death	1.22 (0.57–2.59), P=0.613	1.60 (0.80–3.23), P=0.187	**2.16 (1.07–4.39), P=0.033**	1.21 (0.54–2.70), P=0.643	1.50 (0.71–3.15), P=0.289	2.06 (0.97–4.36), P=0.061
HF or Death	1.55 (0.77–3.13), P=0.218	**2.16 (1.12–4.16), P=0.021**	**2.80 (1.45–5.43), P=0.002***	1.55 (0.74–3.23), P=0.246	1.99 (1.00–3.96), P=0.051	**2.63 (1.31–5.28), P=0.006***

Model 1: Age, Sex, Race, Ethnicity, eGFR; Model 2: Model 1+smoked at least 100, LDL, DM, SBP, DBP, UACR;

† HF, MI, Stroke, PAD;

* significant after Bonferroni correction

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