IZA DP No. 18581

April 2026

Neighborhood Disorder and Dementia Risk in U.S. Older Adults: The Role of Cardiometabolic Risk

Jiao Yu
Yale University

Yi Wang
Yale University

Thomas M. Gill
Yale University

Xi Chen
Yale University, IZA@LISER and NBER

IZA DP No. 18581 April 2026

Neighborhood Disorder and Dementia Risk in U.S. Older Adults: The Role of Cardiometabolic Risk

Abstract

We estimate the effect of neighborhood disorder on dementia risk among middle-aged and older adults in the United States and identify cardiometabolic dysregulation as a mediating biological pathway. Using data from the Health and Retirement Study (HRS, 2006–2020), we show that exposure to visible neighborhood disorder is associated with higher risk of dementia (Hazard Ratio: 1.37; 95% CI: 1.08–1.74) and higher risk of cognitive impairment no dementia (CIND; HR: 1.50; 95% CI: 1.22–1.85) over a 14-year follow-up. Mediation analysis reveals that a composite cardiometabolic risk score—aggregating seven biomarkers spanning inflammatory, cardiovascular, and metabolic systems—accounts for approximately 16 percent of the total neighborhood disorder–dementia association and 19 percent of the neighborhood disorder–CIND association. These findings are robust to competing-risk regression for mortality, restriction to non-movers, age-at-onset restrictions, and exclusion of pandemic-year data. The results establish neighborhood disorder as a modifiable upstream risk factor for cognitive decline and identify cardiometabolic health as a biologically proximate mediating pathway. The findings have implications for place-based public health policy: community-level interventions that simultaneously reduce visible signs of neighborhood decay and address cardiometabolic risk may yield dementia-prevention dividends beyond what individual-level clinical strategies alone can achieve.

JEL classification

I12, I14, J14, R23

Keywords

dementia, cognitive impairment, neighborhood disorder, cardiometabolic risk, social determinants of health, mediation analysis

Corresponding author

Xi Chen

xi.chen@yale.edu

Introduction

Dementia places significant emotional, physical, and financial burdens on older adults, caregivers, and society. About 11% of U.S. adults aged 65 and older have dementia, with 12 million projected by 2040.[1] Since effective treatments are lacking, identifying upstream modifiable risk factors and pathways for dementia risk is essential.[2] As a social determinant of health, the neighborhood environment has become an important area of research.[3,4]

Grounded in the ecological theory of aging, which conceptualizes aging outcomes as dynamic interactions between individuals and their physical and social environments, late-life well-being can be achieved by optimizing the person-environment fit. Within this framework, neighborhood conditions constitute a salient environmental context. Chronic exposure to neighborhood disorder represents an environmental stressor that may overwhelm adaptive capacity, triggering sustained physiological stress responses and eventually accelerating cognitive decline and dementia risk.[5–9] Numerous studies link neighborhood environments to cognitive functioning,[10] cognitive decline,[11] and risks of cognitive impairment and dementia in later life.[7]

Despite these findings, important knowledge gaps remain. First, much of the existing literature has focused on neighborhood socioeconomic status, using census tracts as proxies for neighborhoods to capture broader area-level socioeconomic disadvantages,[12,13] while a comprehensive measure of block-level neighborhood disorder is understudied. Neighborhood disorder, characterized by persistent signs of neglect, danger, and disinvestment, is a chronic ambient stressor that contributes to psychological strain by fostering feelings of insecurity, vigilance, and helplessness. [14] Block-level disorder directly impacts the safety concerns, physical activity, and preventive healthcare access of older adults, who are particularly vulnerable due to

age-related mobility limitations. Moreover, neighborhood disorder represents a highly actionable environmental factor that can be addressed through community-level interventions. By reducing tangible signs of decay in local environments, such interventions may diminish stress exposure and promote cognitive resilience in older populations.

Second, neighborhood environments are posited to influence cognitive health through both behavioral and social pathways, but the underlying biological mechanisms are not well understood. Among multiple pathways linking neighborhood disorder to cognitive health, cardiometabolic dysregulation represents a biologically proximal and clinically modifiable mechanism through which chronic environmental stress may contribute to dementia risk.[9] Cardiometabolic risk factors are among the most important modifiable risk factors for dementia, amenable to both medical and behavioral interventions. According to the 2024 Lancet Commission report, addressing cardiometabolic risks alone could reduce dementia prevalence by 12%.[2] Existing literature frequently employs composite scores of cardiovascular, metabolic, and inflammatory biomarkers as summary indicators of cardiometabolic risk and overall physiological health. These composite scores are associated with an elevated risk of cognitive decline and dementia.[15,16] Despite these established relationships, there is limited evidence testing their mediating roles in the pathway between neighborhood disorder and dementia risk. Preliminary support for this mediating hypothesis comes from clinical studies. For instance, research in a clinical cohort found that the association between neighborhood disadvantage and brain atrophy was mediated by a summary score of cardiovascular risk.[17] They found that a sustained stress response, stimulated by environmental stressors, may lead to physiological dysregulation across multiple systems (e.g., inflammatory, cardiovascular, and metabolic systems), which in turn disrupts homeostasis, accelerates neurodegeneration, and contributes to

cognitive decline and dementia.[18–20] Nevertheless, such evidence remains scarce and is primarily from small clinical samples. A significant gap persists in large-scale epidemiological studies that test whether cardiometabolic risk mediates the association between neighborhood disorder and dementia in community-dwelling older adults.

Understanding the association between neighborhood disorder, cardiometabolic risk, and dementia may help guide prevention strategies and support policies that promote supportive neighborhoods for aging populations. Accordingly, this study leverages the nationally representative, population-based Health and Retirement Study with 14 years of follow-up (2006–2020). This research builds on previous findings by using inverse probability weighting methods to balance participant characteristics across levels of neighborhood disorder. A directed acyclic graph (DAG) is presented in Appendix Figure S1 to illustrate the hypothesized pathways. This study aims to answer two questions: First, to what extent is neighborhood disorder associated with dementia risk among middle-aged and older adults? Second, to what extent does cardiometabolic risk, a composite measure of inflammatory, cardiovascular, and metabolic biomarkers, mediate this relationship? The study hypothesized that exposure to neighborhood disorder is associated with a higher risk of dementia and that this association operates, in significant part, through cardiometabolic risk factors.

Methods

Data Source

The Health and Retirement Study (HRS) is a nationally representative study of middle-aged and older adults aged 51 and older in the contiguous US. The HRS was initiated in 1992 and collects data every two years on participants' physical, mental, cognitive, economic, and social well-

being. Details about the HRS study design and procedures are available elsewhere.[21] Starting in 2006, a random half of the sample was selected to participate in the Enhanced Face-to-Face (EFTF) interview and to provide dried blood spot samples. From these participants, additional data on residential information, anthropometric measures, and blood-based biomarkers were obtained.[22,23] The other half were asked to complete the EFTF in 2008. Participants who completed the EFTF in either 2006 or 2008 were included in this study. This study was exempt from IRB approval and informed consent as it used publicly available and de-identified data.

Analytical Sample

The target population for this study was community-dwelling, dementia-free adults aged 51 years and older. The sample selection process is shown in Appendix Figure S2. Of 12,371 community-dwelling participants aged 51 years and older who completed the HRS EFTF in either 2006 or 2008, 51 participants without neighborhood disorder measures, 2,123 participants without valid biomarker data, 340 participants with any missing covariates, and 431 participants living with dementia at their baseline assessment were excluded, resulting in a final analytic sample of 9,426 participants for dementia analysis. In addition, a secondary sample of 7,455 participants with normal cognition who were free from both CIND and dementia at baseline was derived. This secondary sample was analyzed to assess early-stage cognitive impairment associated with neighborhood disorder.

Cognitive Outcomes

Cognitive impairment and dementia were assessed biennially from 2006 to 2020 using the Langa–Weir classification algorithm.[24] This algorithm has been validated against the clinical dementia diagnoses from the Aging, Demographics, and Memory Study and has shown high sensitivity and adequate specificity. [25] The Langa–Weir Classification relies on the 27-point

modified Telephone Interview for Cognitive Status (TICSm) scale to assess cognitive health. It classifies individuals into categories of normal cognition (12-27), cognitive impairment no dementia (CIND, 7-11), or dementia (0-6). For participants unable to complete the interview, cognitive status was determined using proxy respondent assessments (see Appendix Table S1 for a detailed description of the classification). Additionally, participants who self-reported a physician diagnosis of Alzheimer's Disease or Alzheimer's disease and related dementias (AD/ADRD) were classified as having dementia (n = 217, 2.3%), regardless of their TICSm-derived scores.

Neighborhood Disorder

Exposure to neighborhood disorder was assessed at baseline using interviewers' ratings of eight neighborhood environment items.[26] Interviewers recorded whether each of the following conditions was present within sight of participants' housing units: "vandalism", "boarded houses", "abandoned cars", "abandoned or demolished houses", "trash, litter, or junk in the street", "trash, litter, or junk around buildings", "run-down or poorly kept yards or communal areas", or "poorly kept nearby structures." A total score was calculated by summing the number of visible signs interviewers endorsed (range: 0–8). Because signs of disorder were relatively rare, 87% of the scores were 0. Following prior literature, individuals living in neighborhoods with no disorder (0) were coded as unexposed, while those with any disorder (≥ 1) were coded as exposed.[27]

Cardiometabolic Risk

Cardiometabolic risk was constructed with seven biomarkers indicating physiological dysregulation across multiple biological systems, including systolic blood pressure (SBP), diastolic blood pressure (DBP), glycosylated hemoglobin (HbA1c), high-density lipoprotein

(HDL), total cholesterol (TC), C-reactive protein (CRP), and waist circumference. Additional information about the protocol, instructions, and criteria for biomarker collection is available elsewhere.[22] Consistent with earlier research, each biomarker was dichotomized to indicate high risk (1) versus low risk (0) using clinical thresholds (see Appendix Table S1 for thresholds). Regardless of measured values, a biomarker was classified as high risk if participants reported a diagnosed condition (e.g., high blood pressure, diabetes, etc.) or the use of medication for that condition. The cardiometabolic risk score was calculated by summing the indicator biomarkers (range: 0–7), with higher scores representing higher risk.

Covariates

Demographic information, health behaviors, housing information, and geographic factors were included as covariates.[27,28] A detailed description of all covariates is available in Appendix Table S1. These covariates included age, gender, race and ethnicity, marital status, education, labor force status, household income, smoking, alcohol consumption, activity of daily living difficulty, Body Mass Index (BMI), hypertension, diabetes, Medicare insurance coverage, home ownership, whether living in single-family housing, number of household members, and geographic region. Since hypertension and diabetes may lie on the pathway to dementia through cardiometabolic risk, adjusting for them in outcome models could potentially attenuate the association between neighborhood disorder and dementia. Therefore, these variables were included in the inverse probability weighting models but excluded from the outcome regression models.

Statistical Analysis

Descriptive statistics were assessed at baseline across neighborhood disorder exposure status. Statistical comparisons used Chi-square tests for categorical variables and Wilcoxon rank-sum tests for continuous variables. Descriptive characteristics stratified by cognitive health status are

available in Appendix Table S2. Kaplan−Meier curves were used to determine the survival probabilities for dementia and CIND, stratified by neighborhood disorder, using age as the time scale.[29] Log-rank tests were performed to examine the statistical differences between the exposed group and the unexposed group.

Next, doubly robust estimation was conducted using inverse probability weighted Cox proportional hazards models, adjusting for individual-level covariates.[30] Inverse probability weights were generated using the covariate balancing propensity score (CBPS) method to adjust for residential selection. [31] Censoring was further addressed by constructing inverse probability weights separately for mortality and dropout. Final analytic weights were calculated by multiplying three inverse probability weights by HRS survey weights to account for the complex study design, attrition, and non-responses. Additional details regarding model estimation are provided in the Supplemental Material. Two separate models were estimated to assess the risk of dementia and CIND, respectively. Survival time was defined as age at the first EFTF interview through age at ascertainment of dementia or CIND, death, or the end of follow-up in 2020. The proportional hazards assumption was examined through the inspection of Schoenfeld residuals. Results are reported as hazard ratios (HRs) with 95% confidence intervals (CIs).

Lastly, mediation analyses were conducted to examine whether cardiometabolic risk mediated the association between neighborhood disorder and cognitive outcomes. The total effect was decomposed into direct and indirect (mediation) effects. Model coefficients, bootstrap confidence intervals, and diagrams illustrating the estimated mediation effects are reported.

A series of sensitivity analyses was conducted. First, analyses were restricted to participants who did not move during follow-up to address residential mobility. Second, Fine-Gray proportional hazards regression was performed to account for death as a competing risk.[29]

Additional sensitivity analyses included restricting incident dementia to cases occurring at 65 years or older and excluding data from 2020 to evaluate robustness to age-related risk concentration and potential COVID-19–related disruptions. Statistical tests were two-sided, with a significance level of $p < 0.05$. Data analyses were conducted between January and March 2025, with RStudio version 4.2.2,[32] and STATA 18.[33]

Results

Sample Characteristics

Table 1 presents descriptive statistics of the overall sample and subgroups stratified by exposure to neighborhood disorder. Of the 9,426 participants included in the dementia analysis, the mean (SD) age was 68.9 (9.3) years and 59.2% were female. The mean (SD) years of education was 12.8 (2.9). Significant sociodemographic and health differences were observed between groups exposed to neighborhood disorder versus those unexposed. Participants residing in disordered neighborhoods were disproportionately non-Hispanic Black (exposed: 28.3% vs. unexposed: 8.0%, $p<.001$), Hispanic (exposed: 15.1% vs. unexposed: 6.0%, $p<.001$), or from other racial and ethnic groups (exposed: 2.4% vs. unexposed: 1.8%, $p<.001$). They also had fewer years of education [exposed: 11.3 (3.2) vs. unexposed: 13.0 (2.8), $p<.001$], a higher prevalence of health conditions, such as hypertension (exposed: 63.9% vs. unexposed: 57.6%, $p<.001$), and diabetes (exposed: 26.5% vs. unexposed: 19.2%, $p<.001$), and higher levels of cardiometabolic risk [exposed: 3.2 (1.5) vs. unexposed: 2.8 (1.6), $p<.001$]. Similar patterns were observed in the CIND subsample (Appendix Table S3).

Associations Between Neighborhood Disorder and Risks of Dementia and CIND

Overall, 1,747 of the 9,426 participants (18.5%) developed dementia between 2006 and 2020 (Appendix Figure S2). Kaplan–Meier curves (Figure 1) stratified by neighborhood disorder indicated higher risks of both dementia (Figure 1A) and CIND (Figure 1B) among those exposed to any neighborhood disorder, compared to the unexposed group. Log-rank tests revealed statistically significant differences in survival probability between these two groups.

The adjusted risks for dementia and CIND by exposure status are presented in Table 2. The application of IPWs successfully balanced the covariates (Appendix Figure S3). In a series of hierarchical IPW-adjusted Cox proportional hazards regression models, neighborhood disorder was significantly associated with higher risks of dementia and CIND. In the fully adjusted model (Model 3), those who were exposed to neighborhood disorder had a higher risk of dementia (HR, 1.37; 95% CI: 1.08-1.74), compared to the unexposed participants. Similarly, exposure to neighborhood disorder was associated with an increased risk of CIND (HR, 1.50; 95% CI, 1.22-1.85).

The Mediation Role of Cardiometabolic Risk

The associations between neighborhood disorder and cardiometabolic risk, as well as the associations between cardiometabolic risk and the risk of cognitive outcomes, were statistically significant (Appendix Figure S4). As shown in Table 3, mediation analysis revealed that neighborhood disorder had a significant total effect on dementia risk (HR = 1.42, 95% CI: 1.12–1.80). Cardiometabolic risk mediated approximately 16.2% of this association, with a significant indirect effect (HR = 1.06, 95% CI: 1.00–1.12). Similarly, neighborhood disorder exhibited a significant total effect on the risk of CIND (HR = 1.45, 95% CI: 1.13–1.80), with 19.3% of the total effect mediated through cardiometabolic risk.

When the analyses were repeated after excluding participants who moved during the study period, the results were largely unchanged (Appendix Table S4). Additionally, employing competing risk regression models yielded consistent findings (Appendix Table S5). Findings restricting incident dementia to age 65 or older and excluding 2020 data were consistent with the primary results (Appendix Tables S6 and S7).

Discussion

Using data from a large, nationally representative sample of 9,426 middle-aged and older adults in the United States, this study investigated the association between neighborhood disorder and cognitive health. The findings of this study reveal that residing in neighborhoods with visible signs of disorder was associated with increased risks of dementia and CIND, even after accounting for important covariates. Cardiometabolic risk significantly mediated the associations between neighborhood disorder and both dementia and CIND.

These findings contribute to the literature linking neighborhood environments to dementia risk. [17,29] While most previous studies examined neighborhood conditions at the census tract or zip code level and produced mixed findings, neighborhood disorder captures localized stressors (e.g., vandalism) at a finer, block-level scale. This granular measure may reflect localized environmental stressors that are highly visible to older residents and more directly experienced. [28] The significant association between neighborhood disorder and dementia risk is broadly consistent with previous research linking neighborhood stressors to cognitive health.[8,27] However, using a regional Chicago-based sample, a study found no significant association between neighborhood disorder and cognitive function.[10] Studies in European cohorts have found that area deprivation was not significantly associated with dementia.[34] Such discrepancies

may reflect differences in geographic scope, outcome measures, and study design. Using a nationally representative cohort, this study provides new insights into the association between neighborhood disorder and dementia, distinguishing it from studies relying on smaller clinical samples, [17,35] or cross-sectional studies.[3,14,23]

This study further examined neighborhood disorder as a risk factor for the onset of CIND, a critical precursor to dementia. Regression findings reveal a significantly positive association between neighborhood disorder and CIND, suggesting that environmental stressors may be linked to an early manifestation of cognitive impairment.[36] Unlike older adults living with dementia, who are more likely to be homebound and thus have limited interaction with their surroundings, older adults with CIND may engage more frequently with their neighborhoods.[37] Therefore, neighborhood disorder may be particularly relevant in the early stages of cognitive impairment. Identifying this association may provide a potential window for early interventions prior to the onset of dementia.

Cardiometabolic risk significantly mediated the relationships between neighborhood disorder and both dementia and CIND, suggesting a potential biological pathway operating through multiple physiological systems that leads to increased dementia risk. Older adults living in disordered neighborhoods may experience greater physiological demands to cope with multiple environmental stressors.[38] Chronic stress activates the hypothalamic-pituitary-adrenal (HPA) axis.[39] The resulting low-grade inflammatory state is associated with cardiovascular dysregulation, epigenetic aging acceleration, and metabolic neurodegeneration.[38,40–42] Indeed, substantial studies have found that exposure to neighborhood disorder is associated with elevated cardiometabolic risk, a known risk factor for neurofunctional alterations such as hippocampal

atrophy and impaired prefrontal cortex, both of which accelerate cognitive decline and contribute to dementia risk.[17,40]

While these results are observational, the findings suggest that efforts to reduce neighborhood disorder, such as improving safety, maintenance, and clean environments, may complement existing dementia prevention strategies focused on individual-level risk factors.[43] Further integrating chronic disease management in high-risk neighborhoods may mitigate the cumulative biological burden of cardiometabolic risk. Ultimately, policies targeting both environmental risk factors for dementia and its biological mediators could facilitate healthy aging in place.

This study has notable strengths. While based on U.S. data, the framework linking neighborhood disorder to cognitive aging through cardiometabolic risk is not context-specific and may be relevant to aging populations globally.[4,44] Using nationally representative longitudinal data, this study provides evidence illuminating one plausible biological pathway in neighborhood disorder-dementia associations. Methodologically, this study demonstrates how inverse probability weighting can be applied in observational studies to address residential selection in neighborhood health research. This analytic approach is reproducible and can be readily applied to comparable datasets in other national contexts.

This study has several limitations. First, neighborhood disorder was measured at baseline and may not fully capture changes in exposure over time. Because older adults are often less mobile than younger cohorts, those who relocate often move to similar neighborhoods and therefore tend to be exposed to relatively stable neighborhood environments, suggesting that baseline measures may reasonably approximate longer-term exposure. [45] Future research should incorporate repeated measures of neighborhood conditions to capture potential dynamic changes.

Second, while inverse probability weighting reduces bias due to residential selection on observed characteristics, residual confounding from unmeasured factors may persist. Additionally, most participants in this study lived in neighborhoods with no disorder. This limited variability in the exposure likely reduced statistical power. Third, although proxy assessments and censoring weights were applied to reduce bias due to attrition, some under-ascertainment of dementia between interview waves may persist. Thus, results should be interpreted with caution. Fourth, neighborhood disorder is likely associated with dementia risk through multiple pathways, and cardiometabolic risk represents only one potential mechanism. Future studies should examine a broader set of potential mediators to fully characterize neighborhood–cognition associations. Lastly, cardiometabolic risk reflects cumulative physiological processes shaped by long-term exposures that may also influence residential selection. Time-varying confounders such as psychosocial strain may also affect both cardiometabolic risk and dementia. These interdependencies limit the ability to identify causal mediation effects with observational data. Therefore, the results should not be interpreted as causal.

Conclusions

In this nationally representative longitudinal cohort study of community-dwelling middle-aged and older adults, living in neighborhoods with visible signs of disorder is associated with higher risks of dementia and CIND. Cardiometabolic risk significantly mediates the association between neighborhood disorder and cognitive health outcomes. Addressing neighborhood disorder and cardiometabolic conditions through community initiatives has the potential to delay cognitive impairment and dementia onset and promote healthy aging in place.

Funding/Support: This study was supported in part by the National Institute on Aging of the National Institutes of Health (R01AG077529, P30AG021342) and the University of Minnesota Life Course Center on the Demography and Economics of Aging (P30AG066613), funded through a grant from the National Institute on Aging. The Health and Retirement Study is sponsored by the National Institute on Aging (U01AG009740) and is conducted by the University of Michigan. The IZA Discussion Paper Series serves as a preprint server to deposit latest research for early feedback.

References

1. Zissimopoulos JM, Tysinger BC, St.Clair PA, Crimmins EM. The Impact of Changes in Population Health and Mortality on Future Prevalence of Alzheimer's Disease and Other Dementias in the United States. *J Gerontol B Psychol Sci Soc Sci*. 2018;73(Suppl 1):S38-S47. doi:10.1093/geronb/gbx147

2. Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. *The Lancet*. 2024;404(10452):572-628. doi:10.1016/S0140-6736(24)01296-0

3. Gill TM, Leo-Summers L, Vander Wyk B, Becher RD, Liang J. Neighborhood Disadvantage in a Nationally Representative Sample of Community-Living Older US Adults. *JAMA Netw Open*. 2024;7(12):e2450332. doi:10.1001/jamanetworkopen.2024.50332

4. Besser L, McDonald NC, Song Y, Kukull WA, Rodriguez DA. Neighborhood Environment and Cognition in Older Adults: A Systematic Review. *Am J Prev Med*. 2017;53(2):241-251. doi:10.1016/j.amepre.2017.02.013

5. Ross CE, Mirowsky J. Neighborhood Disorder, Subjective Alienation, and Distress. *J Health Soc Behav*. 2009;50(1):49-64. doi:10.1177/002214650905000104

6. Duchowny KA, Diaz-Ramirez LG, Boscardin WJ, et al. The Neighborhood Environment and Handgrip Strength: Longitudinal Findings From the Health and Retirement Study. *J Gerontol Ser A*. 2024;79(11):glae242. doi:10.1093/gerona/glae242

7. Chen X, Lee C, Huang H. Neighborhood built environment associated with cognition and dementia risk among older adults: A systematic literature review. *Soc Sci Med*. 2022;292:114560. doi:10.1016/j.socscimed.2021.114560

8. Wong R, Wang Y. Role of Neighborhood Physical Disorder and Social Cohesion on Racial and Ethnic Disparities in Dementia Risk. *J Aging Health*. 2022;34(9-10):1178-1187. doi:10.1177/08982643221101352

9. Qin W, Yu J, Brown T. Perceived Neighborhood Disorder, Health Behaviors, and Inflammation Among Older Adults: A Mediation Analysis. *J Aging Health*. Published online June 30, 2025:08982643251356732. doi:10.1177/08982643251356732

10. Clarke PJ, Ailshire JA, House JS, et al. Cognitive function in the community setting: the neighbourhood as a source of 'cognitive reserve'? *J Epidemiol Community Health*. 2012;66(8):730-736. doi:10.1136/jech.2010.128116

11. Clarke P, Weuve J, Barnes L, Evans DA, Mendes de Leon CF. Cognitive decline and the neighborhood environment. *Ann Epidemiol*. 2015;25(11):849-854. doi:10.1016/j.annepidem.2015.07.001

12. Powell WR, Zuelsdorff M, Keller SA, et al. Association of Neighborhood-Level Disadvantage With Neurofibrillary Tangles on Neuropathological Tissue Assessment. *JAMA Netw Open*. 2022;5(4):e228966. doi:10.1001/jamanetworkopen.2022.8966

13. Cadar D, Lassale C, Davies H, Llewellyn DJ, Batty GD, Steptoe A. Individual and Area-Based Socioeconomic Factors Associated With Dementia Incidence in England: Evidence From a 12-Year Follow-up in the English Longitudinal Study of Ageing. *JAMA Psychiatry*. 2018;75(7):723-732. doi:10.1001/jamapsychiatry.2018.1012

14. Pearlin LI, Menaghan EG, Lieberman MA, Mullan JT. The Stress Process. *J Health Soc Behav*. 1981;22(4):337-356. doi:10.2307/2136676

15. Chanti-Ketterl M, Stebbins RC, Obhi HK, Belsky DW, Plassman BL, Yang YC. Sex Differences in the Association Between Metabolic Dysregulation and Cognitive Aging: The Health and Retirement Study. *J Gerontol Ser A*. 2022;77(9):1827-1835. doi:10.1093/gerona/glab285

16. Wu Q, Ailshire JA, Kim JK, Crimmins EM. The Association Between Cardiometabolic Risk and Cognitive Function Among Older Americans and Chinese. *J Gerontol Ser A*. 2024;79(7):glae116. doi:10.1093/gerona/glae116

17. Hunt JFV, Buckingham W, Kim AJ, et al. Association of Neighborhood-Level Disadvantage With Cerebral and Hippocampal Volume. *JAMA Neurol*. 2020;77(4):451-460. doi:10.1001/jamaneurol.2019.4501

18. Buschmann RN, Prochaska JD, Cutchin MP, Peek MK. Stress and health behaviors as potential mediators of the relationship between neighborhood quality and allostatic load. *Ann Epidemiol*. 2018;28(6):356-361. doi:10.1016/j.annepidem.2018.03.014

19. Franks KH, Bransby L, Saling MM, Pase MP, Sindi S. Association of Stress with Risk of Dementia and Mild Cognitive Impairment: A Systematic Review and Meta-Analysis. *J Alzheimer's Dis*. 2021;82(4):1573-1590. doi:10.3233/JAD-210094

20. White S, Mauer R, Lange C, et al. The effect of plasma cortisol on hippocampal atrophy and clinical progression in mild cognitive impairment. *Alzheimers Dement Diagn Assess Dis Monit*. 2023;15(3):e12463. doi:10.1002/dad2.12463

21. Heeringa S, Connor J. *Technical Description of the Health and Retirement Survey Sample Design*.; 1995.

22. Crimmins E, Kim JK, McCreath H, Faul J, Weir D, Seeman T. Validation of Blood-Based Assays Using Dried Blood Spots for Use in Large Population Studies. *Biodemography Soc Biol*. 2014;60(1):38-48. doi:10.1080/19485565.2014.901885

23. Crimmins E, Guyer H, Langa K, Ofstedal MB, Wallace R, Weir D. Documentation of physical measures, anthropometrics and blood pressure in the Health and Retirement Study. *HRS Doc Rep DR-011*. 2008;14(1-2):47-59.

24. Langa KM. Langa-Weir classification of cognitive function (1995-2020). *Surv Res Cent Inst Soc Res Univ Mich Httpshrsdata Isr Umich Edusitesdefaultfilesdocumentationdata-Descr Pdf*. Published online 2023.

25. Crimmins EM, Kim JK, Langa KM, Weir DR. Assessment of Cognition Using Surveys and Neuropsychological Assessment: The Health and Retirement Study and the Aging, Demographics, and Memory Study. *J Gerontol Ser B*. 2011;66B(suppl_1):i162-i171. doi:10.1093/geronb/gbr048

26. Wilkinson LR, Ferraro KF, Kemp BR. Contextualization of Survey Data: What Do We Gain and Does It Matter? *Res Hum Dev*. 2017;14(3):234-252. doi:10.1080/15427609.2017.1340049

27. Choi EY, Cho G, Chang VW. Neighborhood Social Environment and Dementia: The Mediating Role of Social Isolation. *J Gerontol Ser B*. 2024;79(4):gbad199. doi:10.1093/geronb/gbad199

28. Roberts Lavigne LC, Tian J, Hladek M, LaFave SE, Szanton SL, Samuel LJ. Residential Street Block Disorder and Biological Markers of Aging in Older Adults: The National Health and Aging Trends Study. *J Gerontol Ser A*. 2021;76(11):1969-1976. doi:10.1093/gerona/glab166

29. Dintica CS, Bahorik A, Xia F, Kind A, Yaffe K. Dementia Risk and Disadvantaged Neighborhoods. *JAMA Neurol*. 2023;80(9):903-909. doi:10.1001/jamaneurol.2023.2120

30. Yu J, Qin W, Huang W, Thomas K. Oral Health and Mortality Among Older Adults: A Doubly Robust Survival Analysis. *Am J Prev Med*. Published online September 21, 2022. doi:10.1016/j.amepre.2022.08.006

31. Imai K, Ratkovic M. Covariate Balancing Propensity Score. *J R Stat Soc Ser B Stat Methodol*. 2014;76(1):243-263. doi:10.1111/rssb.12027

32. R Core Team. *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing; 2021. https://www.R-project.org/

33. StataCorp. *Stata Statistical Software: Release 17*. College Station, TX: StataCorp LLC.; 2021.

34. Wu YT, Prina AM, Jones AP, et al. Community environment, cognitive impairment and dementia in later life: results from the Cognitive Function and Ageing Study. *Age Ageing*. 2015;44(6):1005-1011. doi:10.1093/ageing/afv137

35. Hunt JFV, Vogt NM, Jonaitis EM, et al. Association of Neighborhood Context, Cognitive Decline, and Cortical Change in an Unimpaired Cohort. *Neurology*. 2021;96(20):e2500-e2512. doi:10.1212/WNL.0000000000011918

36. Vassilaki M, Aakre JA, Castillo A, et al. Association of neighborhood socioeconomic disadvantage and cognitive impairment. *Alzheimers Dement*. 2023;19(3):761-770. doi:10.1002/alz.12702

37. Miller MJ, Cenzer I, Barnes DE, Covinsky KE. Physical inactivity in older adults with cognitive impairment without dementia: room for improvement. *Aging Clin Exp Res*. 2022;34(4):837-845. doi:10.1007/s40520-021-01999-5

38. Robinette JW, Charles ST, Almeida DM, Gruenewald TL. Neighborhood features and physiological risk: An examination of allostatic load. *Health Place*. 2016;41:110-118. doi:10.1016/j.healthplace.2016.08.003

39. Kalesnikava VA, Clarke PJ, Mukherjee B, Sen S, Mezuk B. Psychosocial Stress and Hypothalamic-Pituitary-Adrenal Axis Stress Reactivity: Variations by Race and Socioeconomic Status Among Adults at Risk of Diabetes. *Psychosom Med*. 2022;84(7):813. doi:10.1097/PSY.0000000000001112

40. Lenart-Bugla M, Szcześniak D, Bugla B, et al. The association between allostatic load and brain: A systematic review. *Psychoneuroendocrinology*. 2022;145:105917. doi:10.1016/j.psyneuen.2022.105917

41. Smith JA, Zhao W, Wang X, et al. Neighborhood characteristics influence DNA methylation of genes involved in stress response and inflammation: The Multi-Ethnic Study of Atherosclerosis. *Epigenetics*. 2017;12(8):662-673. doi:10.1080/15592294.2017.1341026

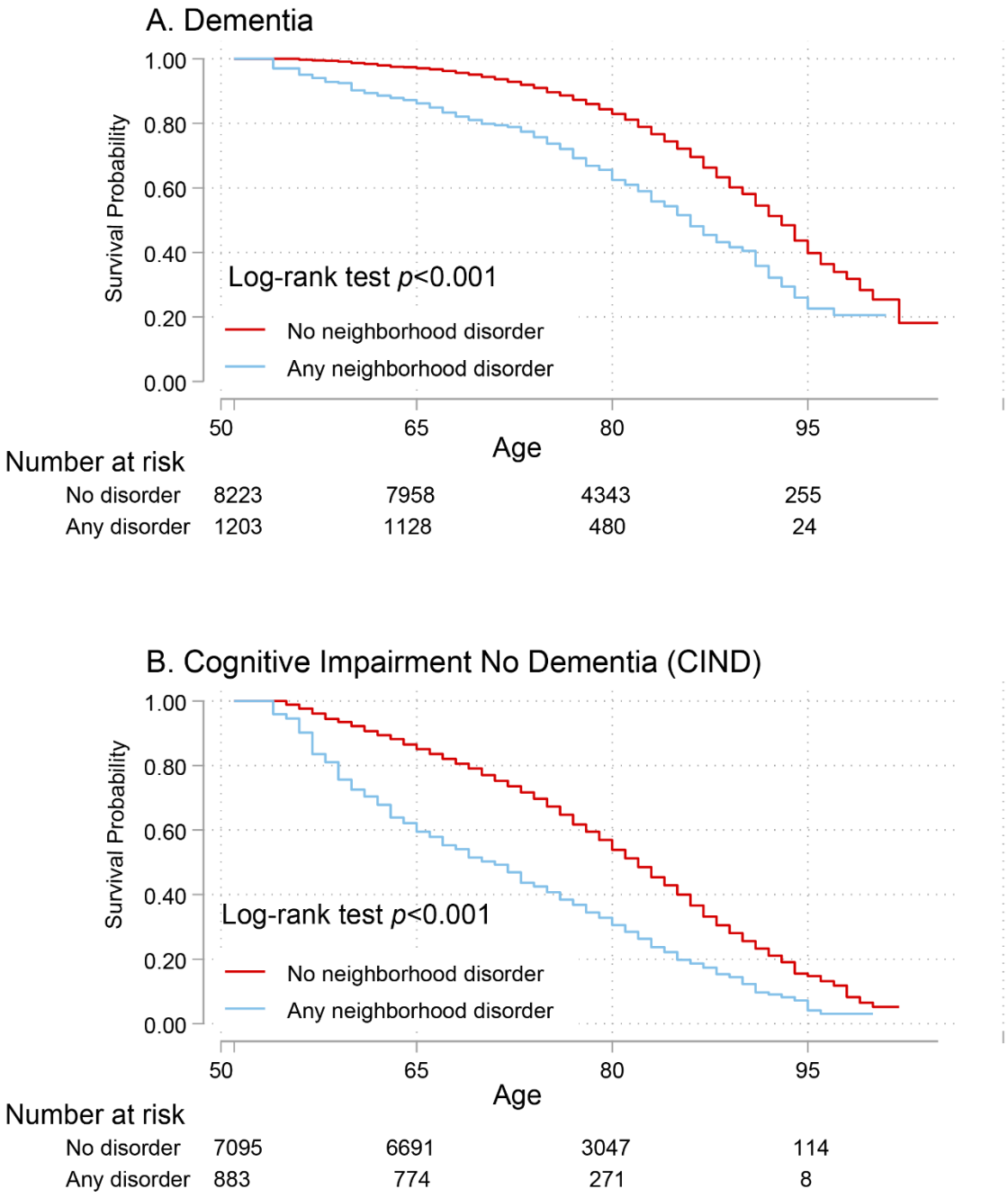
42. Keita AD, Judd SE, Howard VJ, Carson AP, Ard JD, Fernandez JR. Associations of neighborhood area level deprivation with the metabolic syndrome and inflammation among middle- and older- age adults. *BMC Public Health*. 2014;14(1):1319. doi:10.1186/1471-2458-14-1319

43. Yu J, Cudjoe TKM, Mathis WS, Chen X. Uncovering the Biological Toll of Neighborhood Disorder Trajectories: A Machine Learning Based Weighting Analysis of Biomarkers in Older Adults. *J Gerontol Ser B*. Published online November 26, 2025:gbaf242. doi:10.1093/geronb/gbaf242

44. Heide FCT van der, Valeri L, Dugravot A, et al. Role of cardiovascular health factors in mediating social inequalities in the incidence of dementia in the UK: two prospective, population-based cohort studies. *eClinicalMedicine*. 2024;70. doi:10.1016/j.eclinm.2024.102539

45. Gill TM, Becher RD, Leo-Summers L, Gahbauer EA. Changes in neighborhood disadvantage over the course of 22 years among community-living older persons. *J Am Geriatr Soc*. n/a(n/a). doi:10.1111/jgs.19172

Figure 1. Cumulative Survival Probability of Cognitive Outcomes by Neighborhood Disorder



Note: Kaplan-Meier survival curves by exposure to neighborhood disorder are shown. Numbers at risk represent unweighted values.

Table 1. Baseline Characteristics of Study Sample Used for Dementia Analysis According to Neighborhood Disorder [a]

	No. (%)			
	Sample used for Dementia analysis			
	Overall	No neighborhood disorder	Any neighborhood disorder	*p* value [b]
No. of observations, n (%)	9,426	8,223 (87.2)	1,203 (12.8)	
Age, Mean (SD), y	68.9 (9.3)	69.2 (9.4)	67.4 (8.9)	<0.001
Gender, n (%)				
Male	3,848 (40.8)	3,391 (41.2)	457 (38.0)	0.032
Female	5,578 (59.2)	4,832 (58.8)	746 (62.0)	
Race/ethnicity, n (%)				
Non-Hispanic white	7,585 (80.5)	6,934 (84.3)	651 (54.1)	<0.001
Non-Hispanic black	996 (10.6)	655 (8.0)	341 (28.3)	
Hispanic	672 (7.1)	490 (6.0)	182 (15.1)	
Other	173 (1.8)	144 (1.8)	29 (2.4)	
Education Mean (SD), y	12.8 (2.9)	13.0 (2.8)	11.3 (3.2)	<0.001
Household income, Mean (SD) [c]	10.6 (1.2)	10.7 (1.1)	10.0 (1.3)	<0.001
Marital Status, n (%)				
Unmarried/not partnered	3,394 (36.0)	2,778 (33.8)	616 (51.2)	<0.001
Married/partnered	6,032 (64.0)	5,445 (66.2)	587 (48.8)	
Labor Force Participation, n (%)				
Employed	3,300 (35.0)	2,902 (35.3)	398 (33.1)	<0.001
Not employed	927 (9.8)	745 (9.1)	182 (15.1)	
Retired	5,199 (55.2)	4,576 (55.6)	623 (51.8)	
Alcohol consumption, n (%)				
No drinking	6,151 (65.3)	5,240 (63.7)	911 (75.7)	<0.001
Moderate drinking	2,633 (27.9)	2,405 (29.2)	228 (19.0)	
Excessive drinking	642 (6.8)	578 (7.0)	64 (5.3)	
Smoking, n (%)				
Past smokers	4,115 (43.7)	3,642 (44.3)	473 (39.3)	<0.001
Current smoker	4,183 (44.4)	3,722 (45.3)	461 (38.3)	
Never smoked	1,128 (12.0)	859 (10.4)	269 (22.4)	
ADL difficulty, n (%)				
No difficulty	8,225 (87.3)	7,262 (88.3)	963 (80.0)	<0.001
Having difficulty	1,201 (12.7)	961 (11.7)	240 (20.0)	<0.001
Body Mass Index (BMI) Mean (SD)	28.1 (5.6)	27.9 (5.4)	29.6 (6.4)	
Medicare insurance, n (%)				
No	3,034 (32.2)	2,620 (31.9)	414 (34.4)	0.077
Yes	6,392 (67.8)	5,603 (68.1)	789 (65.6)	
Number of people in the household, Mean (SD)	2.1 (1.1)	2.1 (0.9)	2.3 (1.4)	<0.001
Household type, n (%)				
Single family housing	7,564 (80.2)	6,664 (81.0)	900 (74.8)	
Other types [d]	1,862 (19.8)	1,559 (19.0)	303 (25.2)	<0.001
House ownership, n (%)				
Not owned	1,442 (15.3)	1,134 (13.8)	308 (25.6)	<0.001
Owned	7,984 (84.7)	7,089 (86.2)	895 (74.4)	

Census region, n (%)				
Northeast	1,383 (14.7)	1,244 (15.1)	139 (11.6)	<0.001
Midwest	2,587 (27.4)	2,336 (28.4)	251 (20.9)	
South	3,657 (38.8)	3,097 (37.7)	560 (46.6)	
West	1,799 (19.1)	1,546 (18.8)	253 (21.0)	
Cardiometabolic risk, Mean (SD)	2.7 (1.5)	2.7 (1.5)	3.1 (1.4)	<0.001

Abbreviations: ADL, Activities of Daily Living; SD, Standard Deviation.

[a] The data were presented as unweighted values. Percentages may not sum up to 100 due to decimal rounding.

[b] Statistical comparisons were based on chi-square test for categorical variables and Wilcoxon rank sums test for continuous variables, with $p < 0.05$ denoting statistical significance.

[c] Household income was log-transformed to reduce skewness.

[d] Other household types included multiple unit houses, mobile home, condo, coop, rooming house, boarding house, recreational vehicle, motor home, van, car, boat, barn, convent, jail/prison, villa, in transition, garage, trailer, motel, orphanage.

Table 2. Associations Between Neighborhood Disorder and risks of Dementia and CIND in the Health and Retirement Study (2006-2020)

	Hazard ratio [95% CI]			
	Dementia		CIND	
Model	No disorder	Any disorder	No disorder	Any disorder
Model 1 [a]	1 [reference]	1.33 [1.02, 1.74]	1 [reference]	1.51 [1.22, 1.86]
Model 2 [b]	1 [reference]	1.37 [1.09, 1.75]	1 [reference]	1.48 [1.20, 1.83]
Model 3 [c]	1 [reference]	1.37 [1.08, 1.74]	1 [reference]	1.50 [1.22, 1.85]

Abbreviations: 95% CI, 95% confidence interval; CIND, Cognitive Impairment No Dementia.
Hazard ratio and 95% CI were from inverse probability weighted Cox proportional hazards regression models,

[a] Model 1 adjusted for baseline age, and gender.

[b] Model 2 adjusted for baseline age, gender, race/ethnicity, marital status, education, household income, labor force status, Medicare insurance coverage, home ownership, housing type, number of household members, and census regions.

[c] Model 3: adjusted for Model 2 covariates and smoking, alcohol consumption, activity of daily living (ADL) difficulty, BMI.

Table 3. Associations Between Neighborhood Disorder and Cognitive Outcomes Mediated by Cardiometabolic Risk

	Hazard ratio [95% CI] [a]	
	Dementia	CIND
Total effect	1.42 [1.12, 1.80]	1.45 [1.13, 1.80]
Direct effect	1.35 [1.07, 1.71]	1.35 [1.06, 1.80]
Indirect effect	1.06 [1.00, 1.12]	1.08 [1.01, 1.16]
% mediated [b]	16.2%	19.3%

Abbreviations: 95% CI, 95% confidence interval; CIND, Cognitive Impairment No Dementia.
[a]Values were calculated using the inverse probability weighting regressions generated by the covariate balancing propensity score that balances the distribution of the covariates between the exposed and unexposed groups.
[b]The proportion mediated was calculated by log (hazard ration) of indirect effect / log (hazard ration) of total effect.

Appendix

Table S1. Description of Study Variables

Variables	Type	Description
Outcomes: Cognitive health measures (2006-2020)	Categorical	Langa –Weir Classification: Applying Telephone Interview for Cognitive Status (TICSm) to assesses participants cognitive function through tasks including immediate and delayed word recall (range: 0–20 points), serial 7 subtraction test (0–5 points), and backward counting (0–2 points). Cognitive health status was classified with a combined score as: • Normal cognition: 12-27 • Cognitive impairment no dementia: 7-11 • Dementia: 0-6 Participants who were unable to complete the TICSm due to cognitive limitations were assessed with proxy reported information, including proxy assessments of memory (range: 0–4 points), interviewer assessments of cognitive impairment (0–2 points), and proxy-reported limitations in instrumental activities of daily living (0–5 points). Cognitive health status was classified with a combined score as: • Normal cognition: 0-2 • Cognitive impairment no dementia: 3-5 • Dementia: 6 and above Participants who reported a doctor's diagnosis of Alzheimer's Disease and Alzheimer's disease and related dementias (AD/ADRD) were also recorded as having dementia.
Exposure: Neighborhood disorder (2006 and 2008)	Binary	Interviewers' ratings on whether each of the following signs was present within sight of participants' housing units: "vandalism," "boarded houses," "abandoned cars," "abandoned or demolished houses," "trash, litter, or junk in the street," "trash, litter, or junk around buildings," "run down or poorly kept yards or communal areas," or "poorly kept near-by-structures," • A total score was calculated by summing the number of visible signs interviewers endorsed (range: 0–8). We further dichotomize the total score as no disorder (0) vs any disorder (>=1)
Mediator: Cardiometabolic Risk (2006 and 2008)	Continuous	Each biomarker is dichotomized based on the following clinic cutoff points to indicate high risk (1) or low risk (0). A summation score is created by adding up all binary biomarker measures. • Systolic blood pressure (SBP): >=140 • Diastolic blood pressure (DBP): >=90 • Haemoglobin A1c (HbA1c): >=6.5 • High-density lipoprotein (HDL): <=40 • Total cholesterol: >=240 • Waist circumference: male: 40cm; female: 35cm • C-reactive protein (CRP): >=3
Covariates:		
Race/ethnicity (2006)	Categorical	1= non-Hispanic White; 2= non-Hispanic Black; 3=Hispanic; 4 = Other
Age (2006)	Continuous	Age in years
Gender (2006)	Binary	1=Male, 2= Female
Education	Continuous	Years of education

(2006)		
Household income (logged) (2006 and 2008)	Continuous	Sum of household earnings, pensions and annuities, Supplemental Security Income and Social Security Disability, Social Security retirement, unemployment and workers' compensation, other government transfers, household capital income, and other income. Household income was adjusted to 2018 US dollars using Consumer Price Index.
Marital Status (2006 and 2008)	Binary	0 = Unmarried/not partnered (separated, divorced, widowed, never married) 1 = Married/partnered
Smoking status (2006 and 2008)	Categorical	1 = Past smokers (respondents who have ever smoked cigarettes but does not smoke now) 2 = Current smoker (respondents who smoke cigarettes now regardless of past smoking status) 3 = Never smoked (respondents who have never smoked cigarettes and does not smoke now)
Alcohol consumption (2006 and 2008)	Categorical	1 = No drinking (respondents who have 0 drink per day in last 3 months) 2 = Moderate drinking (men who have < 3 drinks per day and women who have < 2 drinks per day in last 3 months) 3 = Excessive drinking (men who have ≥ 3 drinks per day and women who have ≥ 2 drink in last 3 months)
Body Mass Index (BMI) (2006 and 2008)	Continuous	BMI is based on respondents' measured height (in meters) and weight (in kilograms) and it is calculated using the weight divided by the square height.
Hypertension (2006 and 2008)	Binary	Self-reported physician-diagnosed condition 0 = No hypertension 1 = Has hypertension
Diabetes (2006 and 2008)	Binary	Self-reported physician-diagnosed condition 0 = No diabetes 1 = Has diabetes
ADL difficulty (2006 and 2008)	Binary	0 = No difficulty 1 = Having difficulty in any of the following five tasks: bathing, eating, dressing, walking across the room, and getting in and out of bed
Labor Force Participation (2006 and 2008)	Categorical	1 = Employed (full-time work, part-time work) 2 = Not employed (unemployed and not in labor force) 3 = Retired
Medicare insurance beneficiary (2006 and 2008)	Binary	0 = No 1 = Yes
Census region (2006 and 2008)	Categorical	1 = Northeast 2 = Midwest 3 = South 4 = West
Home ownership (2006 and 2008)	Binary	0 = Rented home 1 = Owned home

Housing type (2006 and 2008)	Binary	0 = Other (Other household types included multiple unit houses, mobile home, condo, coop, rooming house, boarding house, recreational vehicle, motor home, van, car, boat, barn, convent, jail/prison, villa, in transition, garage, trailer, motel, orphanage). 1 = Single family housing
Number of household members (2006 and 2008)	Continuous	Number of people living within the same household

Table S2. Baseline Characteristics of Study Sample According to Cognitive Health [a]

Characteristic	No. (%)							
	Sample used for dementia analysis				Sample used for CIND analysis			
	Total	Dementia free	Living with Dementia	p^b	Total	Normal Cognition	CIND	p^b
No. of observations, n (%)	9,426	7,679 (81.5)	1,747 (18.5)		7,455	4,751 (63.7)	2,704 (36.3)	
Neighborhood disorder, n (%)				<0.001				<0.001
No	8,223 (87.2)	6,762 (88.1)	1,461 (83.6)		6,649 (89.2)	4,307 (90.7)	2,342 (86.6)	
Any	1,203 (12.8)	917 (11.9)	286 (16.4)		806 (10.8)	444 (9.3)	362 (13.4)	
Cardiometabolic Risk (Mean/SD)	2.7 (1.5)	2.7 (1.5)	2.9 (1.5)	<0.001	2.7 (1.5)	2.6 (1.5)	2.8 (1.4)	<0.001
Age (Mean/SD)	68.9 (9.3)	67.9 (9.1)	73.7 (8.8)	<0.001	67.8 (9.0)	66.1 (8.6)	70.8 (8.8)	<0.001
Race/ethnicity, n (%)				<0.001				<0.001
Non-Hispanic white	7,585 (80.5)	6,338 (82.5)	1,247 (71.4)		6,302 (84.5)	4,153 (87.4)	2,149 (79.5)	
Non-Hispanic black	996 (10.6)	712 (9.3)	284 (16.3)		603 (8.1)	304 (6.4)	299 (11.1)	
Hispanic	672 (7.1)	490 (6.4)	182 (10.4)		431 (5.8)	221 (4.7)	210 (7.8)	
Other	173 (1.8)	139 (1.8)	34 (1.9)		119 (1.6)	73 (1.5)	46 (1.7)	
Gender, n (%)				0.065				0.072
Male	3,848 (40.8)	3,169 (41.3)	679 (38.9)		3,006 (40.3)	1,879 (39.5)	1,127 (41.7)	
Female	5,578 (59.2)	4,510 (58.7)	1,068 (61.1)		4,449 (59.7)	2,872 (60.5)	1,577 (58.3)	
Years of education (Mean/SD)	12.8 (2.9)	13.1 (2.7)	11.7 (3.4)	<0.001	13.2 (2.6)	13.6 (2.4)	12.5 (2.8)	<0.001
Household income[c] (logged) (Mean/SD)	10.6 (1.2)	10.7 (1.2)	10.2(1.1)	<0.001	10.7 (1.1)	10.8 (1.1)	10.5 (1.0)	<0.001
Marital Status, n (%)				<0.001				<0.001
Unmarried/not partnered	3,394 (36.0)	2,627 (34.2)	767 (43.9)		2,496 (33.5)	1,482 (31.2)	1,014 (37.5)	
Married/partnered	6,032 (64.0)	5,052 (65.8)	980 (56.1)		4,959 (66.5)	3,269 (68.8)	1,690 (62.5)	
Labor Force Participation, n (%)				<0.001				<0.001
Employed	3,300 (35.0)	2,998 (39.0)	302 (17.3)		2,950 (39.6)	2,150 (45.3)	800 (29.6)	
Not employed	927 (9.8)	694 (9.0)	233 (13.3)		645 (8.7)	376 (7.9)	269 (9.9)	
Retired	5,199 (55.2)	3,987 (51.9)	1,212 (69.4)		3,860 (51.8)	2,225 (46.8)	1,635 (60.5)	
Smoking, n (%)				0.228				0.890
Past smokers	4,115 (43.7)	3,330 (43.4)	785 (44.9)		3,298 (44.2)	2,093 (44.1)	1,205 (44.6)	
Current smoker	4,183 (44.4)	3,411 (44.4)	772 (44.2)		3,294 (44.2)	2,109 (44.4)	1,185 (43.8)	
Never smoked	1,128 (12.0)	938 (12.2)	190 (10.9)		863 (11.6)	549 (11.6)	314 (11.6)	
Alcohol consumption, n (%)				<0.001				<0.001

No drinking	6,151 (65.3)	4,853 (63.2)	1,298 (74.3)		4,648 (62.3)	2,834 (59.7)	1,814 (67.1)	
Moderate drinking	2,633 (27.9)	2,256 (29.4)	377 (21.6)		2,238 (30.0)	1,497 (31.5)	741 (27.4)	
Excessive drinking	642 (6.8)	570 (7.4)	72 (4.1)		569 (7.6)	420 (8.8)	149 (5.5)	
ADL difficulty, n (%)				<0.001				<0.001
No difficulty	8,225 (87.3)	6,808 (88.7)	1,417 (81.1)		6,647 (89.2)	4,318 (90.9)	2,329 (86.1)	
Having difficulty	1,201 (12.7)	871 (11.3)	330 (18.9)		808 (10.8)	433 (9.1)	375 (13.9)	
Body Mass Index (BMI) Mean (SD)	28.1 (5.6)	28.2 (5.6)	27.5 (5.1)	<0.001	28.2 (5.6)	28.2 (5.6)	28.2 (5.5)	0.630
Medicare insurance, n (%)				<0.001				<0.001
No	3,034 (32.2)	2,799 (36.5)	235 (13.5)		2,693 (36.1)	2,101 (44.2)	592 (21.9)	
Yes	6,392 (67.8)	4,880 (63.5)	1,512 (86.5)		4,762 (63.9)	2,650 (55.8)	2,112 (78.1)	
Number of people in the household (Mean/SD)	2.1 (1.1)	2.1 (1.0)	2.1 (1.1)	0.010	2.1 (1.0)	2.1 (1.0)	2.1 (1.0)	0.240
Household type, n (%)				<0.001				<0.001
Other types[d]	1,862 (19.8)	1,436 (18.7)	426 (24.4)		1,367 (18.3)	787 (16.6)	580 (21.4)	
Single family housing	7,564 (80.2)	6,243 (81.3)	1,321 (75.6)		6,088 (81.7)	3,964 (83.4)	2,124 (78.6)	
House ownership, n (%)				<0.001				<0.001
Not owned	1,442 (15.3)	1,076 (14.0)	366 (21.0)		977 (13.1)	544 (11.5)	433 (16.0)	
Owned	7,984 (84.7)	6,603 (86.0)	1,381 (79.0)		6,478 (86.9)	4,207 (88.5)	2,271 (84.0)	
Census region, n (%)				0.006				0.037
Northeast	1,383 (14.7)	1,142 (14.9)	241 (13.8)		1,114 (14.9)	712 (15.0)	402 (14.9)	
Midwest	2,587 (27.4)	2,152 (28.0)	435 (24.9)		2,123 (28.5)	1,403 (29.5)	720 (26.6)	
South	3,657 (38.8)	2,920 (38.0)	737 (42.2)		2,761 (37.0)	1,714 (36.1)	1,047 (38.7)	
West	1,799 (19.1)	1,465 (19.1)	334 (19.1)		1,457 (19.5)	922 (19.4)	535 (19.8)	

Abbreviations: ADL, Activities of Daily Living; SD, Standard Deviation; CIND, Cognitive Impairment No Dementia.

[a] The data were presented as unweighted values. Percentages may not sum up to 100 due to decimal rounding. Frequencies with percentages n (%) and mean with standard deviation (Mean/SD) are presented.

[b] Statistical comparisons were based on chi-square test for categorical variables and Wilcoxon rank sums test for continuous variables, with $p < 0.05$ denoting statistical significance.

[c] Household income was log-transformed to reduce skewness.

[d] Other household types included multiple unit houses, mobile home, condo, coop, rooming house, boarding house, recreational vehicle, motor home, van, car, boat, barn, convent, jail/prison, villa, in transition, garage, trailer, motel, orphanage.

Table S3. Baseline Characteristics of Cognitive Impairment No Dementia (CIND) Sample According to Neighborhood Disorder[a]

	No. (%)			
	Sample used for CIND analysis			
	Overall	No neighborhood disorder	Any neighborhood disorder	*p* value[b]
No. of observations, n (%)	N=7,455	N=6,649	N=806	
Age, Mean (SD), y	67.8 (9.0)	68.0 (9.0)	66.4 (8.4)	<0.001
Gender				
Male	3,006 (40.3%)	2,706 (40.7%)	300 (37.2%)	0.021
Female	4,449 (59.7%)	3,943 (59.3%)	506 (62.8%)	
Race/ethnicity, n (%)				
Non-Hispanic white	6,302 (84.5%)	5,812 (87.4%)	490 (60.8%)	<0.001
Non-Hispanic black	603 (8.1%)	411 (6.2%)	192 (23.8%)	
Hispanic	431 (5.8%)	325 (4.9%)	106 (13.2%)	
Other	119 (1.6%)	101 (1.5%)	18 (2.2%)	
Education Mean (SD), y	13.2 (2.6)	13.4 (2.5)	12.0 (2.9)	<0.001
Household income, Mean (SD)[c]	10.7 (1.1)	10.8 (1.1)	10.2 (0.9)	<0.001
Marital Status, n (%)				
Unmarried/not partnered	2,496 (33.5%)	2,108 (31.7%)	388 (48.1%)	<0.001
Married/partnered	4,959 (66.5%)	4,541 (68.3%)	418 (51.9%)	
Labor Force Participation, n (%)				
Employed	2,950 (39.6%)	2,628 (39.5%)	322 (40.0%)	<0.001
Not employed	645 (8.7%)	549 (8.3%)	96 (11.9%)	
Retired	3,860 (51.8%)	3,472 (52.2%)	388 (48.1%)	
Alcohol consumption, n (%)				
No drinking	4,648 (62.3%)	4,070 (61.2%)	578 (71.7%)	<0.001
Moderate drinking	2,238 (30.0%)	2,061 (31.0%)	177 (22.0%)	
Excessive drinking	569 (7.6%)	518 (7.8%)	51 (6.3%)	
Smoking, n (%)				
Past smokers	3,298 (44.2%)	2,985 (44.9%)	313 (38.8%)	<0.001
Current smoker	3,294 (44.2%)	2,976 (44.8%)	318 (39.5%)	
Never smoked	863 (11.6%)	688 (10.3%)	175 (21.7%)	
ADL difficulty, n (%)				
No difficulty	6,647 (89.2%)	5,973 (89.8%)	674 (83.6%)	<0.001
Having difficulty	808 (10.8%)	676 (10.2%)	132 (16.4%)	<0.001
Body Mass Index (BMI) Mean (SD)	28.2 (5.6)	28.0 (5.4)	29.9 (6.5)	<0.001
Medicare insurance, n (%)				0.090
No	2,693 (36.1%)	2,380 (35.8%)	313 (38.8%)	
Yes	4,762 (63.9%)	4,269 (64.2%)	493 (61.2%)	
Number of people in the household, Mean (SD)	2.1 (1.0)	2.1 (1.0)	2.3 (1.3)	<0.001
Household type, n (%)				
Single family housing	1,367 (18.3%)	1,167 (17.6%)	200 (24.8%)	<0.001
Other types[d]	6,088 (81.7%)	5,482 (82.4%)	606 (75.2%)	
House ownership, n (%)				
Not owned	977 (13.1%)	795 (12.0%)	182 (22.6%)	<0.001
Owned	6,478 (86.9%)	5,854 (88.0%)	624 (77.4%)	

Census region, n (%)				<0.001
Northeast	1,114 (14.9%)	1,011 (15.2%)	103 (12.8%)	
Midwest	2,123 (28.5%)	1,951 (29.3%)	172 (21.3%)	
South	2,761 (37.0%)	2,422 (36.4%)	339 (42.1%)	
West	1,457 (19.5%)	1,265 (19.0%)	192 (23.8%)	
Cardiometabolic Risk, Mean (SD)	2.7 (1.5)	2.6 (1.5)	3.0 (1.4)	<0.001

Abbreviations: ADL, Activities of Daily Living; SD, Standard Deviation; CIND, Cognitive Impairment No Dementia.

[a] The data were presented as unweighted values. Percentages may not sum up to 100 due to decimal rounding.

[b] Statistical comparisons were based on chi-square test for categorical variables and Wilcoxon rank sums test for continuous variables, with $p < 0.05$ denoting statistical significance.

[c] Household income was log-transformed to reduce skewness.

[d] Other household types included multiple unit houses, mobile home, condo, coop, rooming house, boarding house, recreational vehicle, motor home, van, car, boat, barn, convent, jail/prison, villa, in transition, garage, trailer, motel, orphanage.

Table S4. Associations Between Neighborhood Disorder and Cognitive Outcomes for Participants without Residential Mobility, Health and Retirement Study, 2006-2020 [a]

	Hazard ratio [95% CI]	
	Dementia (n = 8,310)	CIND (n = 6,560)
No disorder	1[reference]	1[reference]
Any disorder	1.38 [1.07, 1.79]	1.45[1.15, 1.81]

Abbreviations: CI, confidence interval; CIND, Cognitive Impairment No Dementia.

[a] Inverse probability weighted Cox proportional hazards regression models were performed adjusting for baseline covariates including age, gender race/ethnicity, marital status, education, household income, labor force status, smoking, alcohol consumption, activity of daily living difficulty, BMI, Medicare insurance coverage, home ownership, housing type, number of household members, and geographic regions.

Table S5. Associations Between Neighborhood Disorder and Cognitive Outcomes Using Competing Risk Models [a]

	Hazard ratio [95% CI]	
	Dementia	CIND
No disorder	1[reference]	1[reference]
Any disorder	1.19 [1.02, 1.40]	1.17 [1.01, 1.35]

Abbreviations: CI, confidence interval; CIND, Cognitive Impairment No Dementia.

[a] The Fine-Gray proportional hazards regression models were estimated, adjusted for dropout weights and baseline covariates, including age, gender race/ethnicity, marital status, education, household income, labor force status, smoking, alcohol consumption, activity of daily living difficulty, BMI, Medicare insurance coverage, home ownership, housing type, number of household members, and geographic regions.

Table S6. Associations Between Neighborhood Disorder and Cognitive Outcomes Excluding Pandemic Years, Health and Retirement Study, 2006-2018[a]

	Hazard ratio [95% CI]	
	Dementia	CIND
No disorder	1[reference]	1[reference]
Any disorder	1.38 [1.08, 1.77]	1.64 [1.31, 2.05]

Abbreviations: CI, confidence interval; CIND, Cognitive Impairment No Dementia.
[a] Inverse probability weighted Cox proportional hazards regression models were performed adjusting for baseline covariates including age, gender race/ethnicity, marital status, education, household income, labor force status, smoking, alcohol consumption, activity of daily living difficulty, BMI, Medicare insurance coverage, home ownership, housing type, number of household members, and geographic regions.

Table S7. Associations Between Neighborhood Disorder and Cognitive Outcomes Excluding Participants Who Developed Dementia before Age 65, Health and Retirement Study, 2006-2020[a]

	Hazard ratio [95% CI]
	Dementia
No disorder	1[reference]
Any disorder	1.31 [1.01, 1.68]

Abbreviations: CI, confidence interval; CIND, Cognitive Impairment No Dementia.
[a] Inverse probability weighted Cox proportional hazards regression models were performed adjusting for baseline covariates including age, gender race/ethnicity, marital status, education, household income, labor force status, smoking, alcohol consumption, activity of daily living difficulty, BMI, Medicare insurance coverage, home ownership, housing type, number of household members, and geographic regions.

Figure S1. Directed Acyclic Graph (DAG) of Neighborhood Disorder, Cardiometabolic Risk, and Cognitive Outcomes

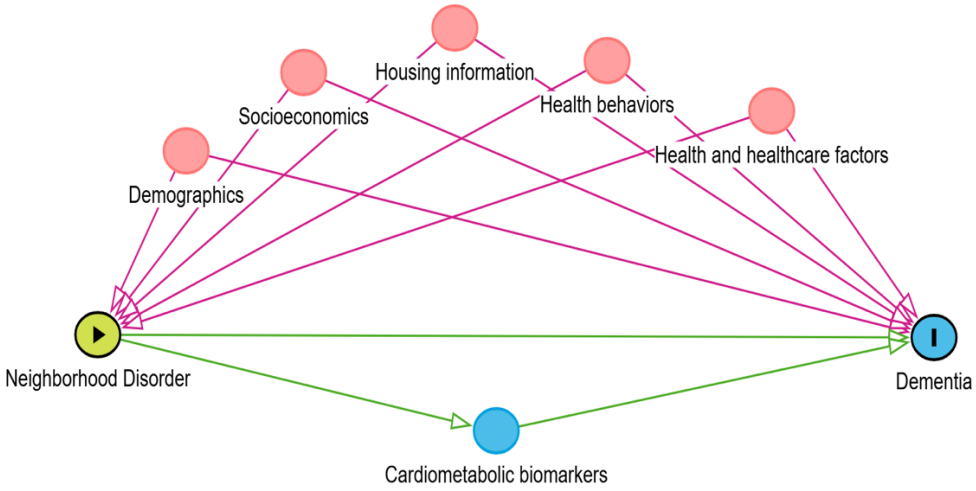
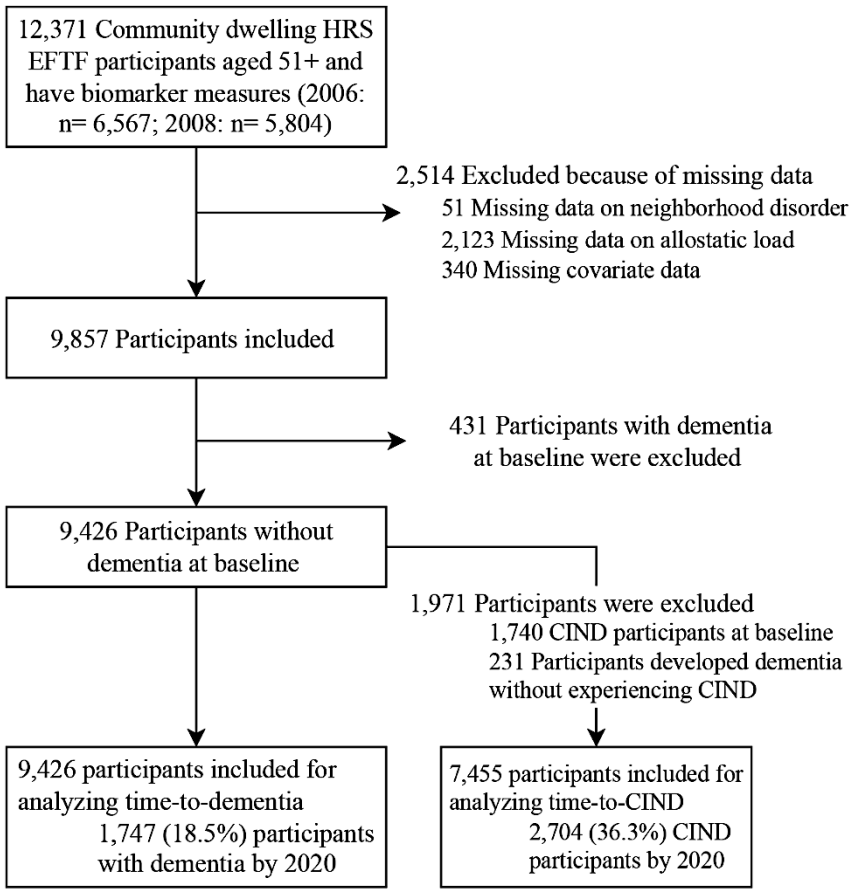
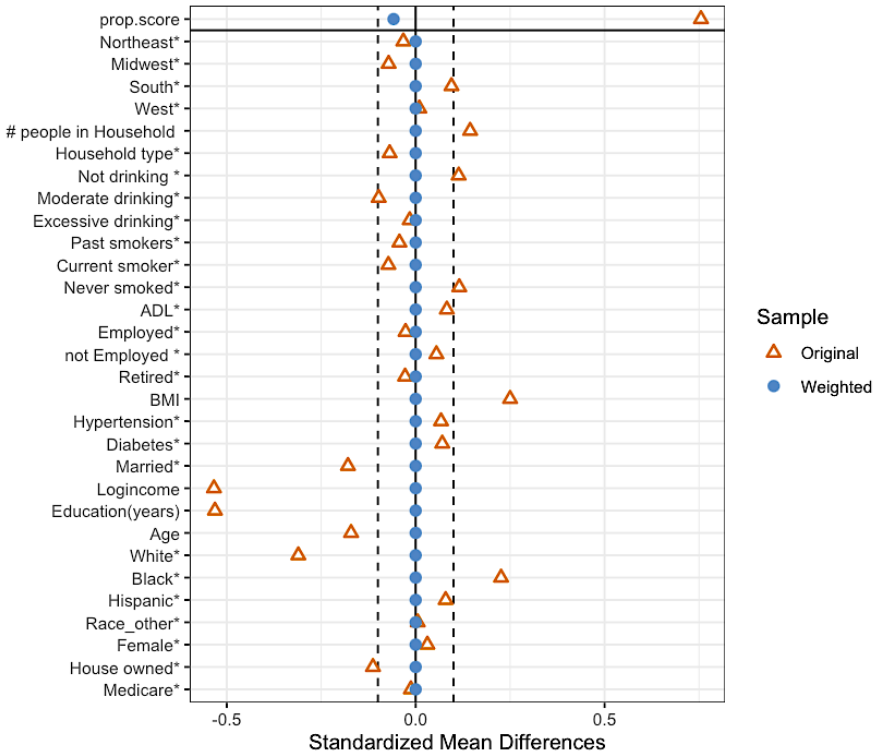


Figure S2. Analytic Sample Selection Process



HRS EFTF indicates the Health and Retirement Study Enhanced Face-to-Face interviews; CIND indicates Cognitive Impairment No Dementia.

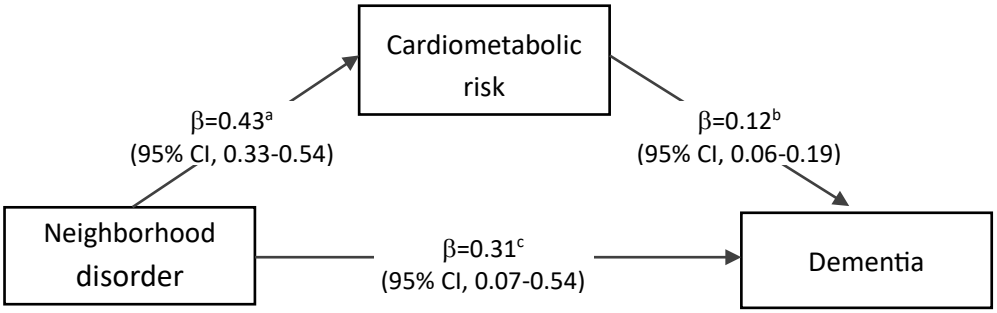
Figure S3. Covariate Balance Plot Before and After the Application of IPW



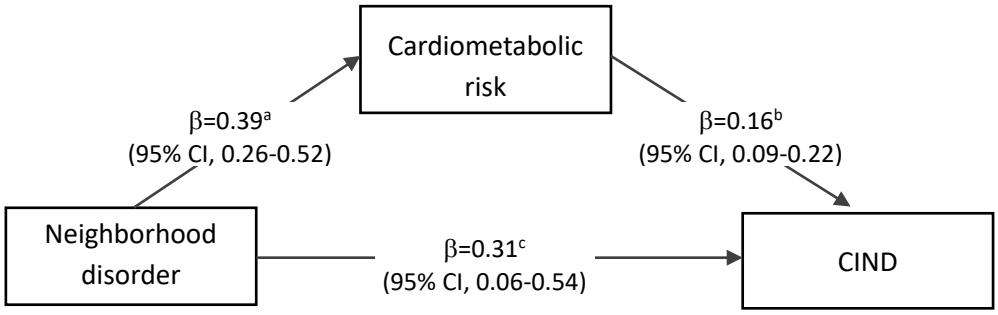
The figure shows standardized differences in the distribution of baseline covariates between the exposed and unexposed groups before and after the application of inverse probability weighting (IPW) adjustment. IPW was estimated using Covariate Balancing Propensity Score (CBPS) method. All covariates had a standardized difference of less than 0.1, indicating covariate balance was achieved with no statistically significant differences between the exposed group and the unexposed group after weighting.

Figure S4. Illustrative Diagrams of Cardiometabolic Risk Mediation of Associations Between Neighborhood Disorder and Cognitive Outcomes

A Cardiometabolic risk mediation and dementia risk



B Cardiometabolic risk mediation and the risk of cognitive impairment no dementia (CIND)



A, The estimated coefficients from the mediation analysis of cardiometabolic risk on the association between neighborhood disorder and dementia risk. B, The estimated coefficients from the mediation analysis of cardiometabolic risk on the association between neighborhood disorder on the risk of cognitive impairment no dementia (CIND).

[a] indicates the coefficient from the linear regression model assessing the association between neighborhood disorder and cardiometabolic risk (X → M). [b, c] indicate coefficients from the Cox regression models assessing the associations between neighborhood disorder and cardiometabolic risk on the risk of cognitive health (X + M→Y). Inverse probability weighting was applied in all model estimation.

Supplemental Material

Procedures of the Inverse Probability Weights (IPW) Cox proportional hazards regression analysis

The IPW Cox proportional hazards regression analysis proceeded in two steps. To construct the IPWs for residential selection, propensity scores that reflect each participant's predicted probability of being in the treated group (exposed to neighborhood disorder) was calculated. We regressed the binary indictor of neighborhood disorder on the baseline covariates, including age, gender race/ethnicity, marital status, education, household income, labor force status, smoking, alcohol consumption, activity of daily living difficulty, BMI, hypertension, diabetes, Medicare insurance coverage, home ownership, housing type, number of household members, and geographic regions. Instead of employing a traditional logistic model, we employed a Covariate Balancing Propensity Score (CBPS) method. The CBPS approach typically results in better covariate balance than traditional logistic methods in the presence of high-dimensional data and is less sensitive to model misspecification.

Next, we calculated stabilized inverse probability weights (IPW) to reduce the impact of extreme values. For treated individuals, the stabilized weight was calculated as $\frac{\Pr(T=1|X_i)}{e(X_i)}$, and for untreated individuals as $\frac{1-\Pr(T=1|X_i)}{1-e(X_i)}$, where $\Pr(T=1|X_i)$ is the predicted probability of treated and $e(X_i)$ denotes the propensity score. Once stabilized IPWs were calculated, covariates balance was assessed by comparing the distribution of baseline characteristic between the exposed group and the unexposed group, using a standardized difference threshold of 0.1. We compared the performance of logistic regression and CBPS. We found that CBPS balanced all included covariates, while logistic regression achieved balance for 30 of the 32 covariates. Similar approach was used to construct IPWs separately for mortality and dropout. Final analytic weights were created by multiplying the following weights: mortality weights, dropout weights, residential selection weights, and the HRS survey weights, which accounted for the complex study design, attrition, and non-responses.

In the second step, we performed the inverse probability weighted Cox proportional hazards models, controlling for the same set of covariates included in the propensity score model. The estimated effect of neighborhood disorder was unbiased given either the outcome survival regression or the propensity score model was correctly specified. In this regard, this estimator is also called a doubly robust estimator. The proportional hazards assumption was examined through visual inspections of Schoenfeld residuals. Proportional hazards model assumptions were checked and met for all final models. We assessed dementia and CIND using separate models to examine

the extent to which neighborhood disorder was associated with cognitive health outcomes as with the progress of cognitive impairment.