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A Prospective Study of Socioeconomic Status, Prostate Cancer Screening and Incidence Among Men at High Risk for Prostate Cancer

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Abstract

Purpose—Higher socio-economic status (SES) men are at higher risk of prostate cancer (PCa) diagnosis, an association commonly interpreted as a function of higher rates of prostate screening among higher-SES men. However, the extent to which screening explains this association has not been well quantified.

Methods—Within a Detroit-area cohort of 6,692 men followed up after a benign prostate procedure, a case-control study was conducted of 494 PCa cases and controls matched on age, race, duration of follow-up, and date of initial benign finding. 2000 Census data were used in a principal component analysis to derive a single factor, labeled the Neighborhood SES Index (NSESI), representing zip code-level SES.

Results—Among cases, higher SES was associated with a younger age at initial biopsy: −1.48 years (95% CI, −2.32, −0.64) per unit NSESI. After adjustment for confounders and duration of follow-up, higher SES was associated with more PSA tests and DRE during follow-up; 9% (95% CI, 2, 16) and 8% (95% CI, 1, 15) more respectively, per unit NSESI. Higher SES was associated with a higher risk of PCa diagnosis during follow-up, multivariable adjusted $OR = 1.26$ per unit increase in NSESI (95% CI, 1.04, 1.49). Further adjustment for screening frequency somewhat reduced the association between SES and PCa risk (OR $= 1.19$ per unit NSESI, 95% CI, 0.98, 1.44).

Conclusions—Differences in screening frequency only partially explained the association between higher zip code SES and PCa risk; other health care related factors should also be considered as explanatory factors.

Keywords

socioeconomic status; prostate cancer; incidence; screening

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Introduction

In 2012 in the United States, 241,740 new cases of prostate cancer (PCa) are expected, along with 28,170 deaths from the disease, making PCa the most commonly diagnosed non-skin cancer among men in the United States and the second leading cause of cancer death among men (1). In addition, PCa screening and detection efforts absorb a large amount of health resources; approximately 1 million biopsies are performed annually in the US due to suspicion of PCa, two-thirds of which reveal benign conditions (2). Autopsy studies have shown that more than half of all men over age 50, and approximately two thirds over age 70, die with undiagnosed PCa, suggesting that there is a large population of men with relatively benign undiagnosed PCa (3, 4). Screening behavior and medical utilization are likely to have a substantial influence on who is ultimatley diagnosed and treated (5, 6).

While age, race, and family history are the primary risk factors for PCa, there is evidence that men who have higher socioeconomic status (SES) or who live in higher-SES neighborhoods have a higher incidence of PCa $(7-12)$, but that their tumors are more likely to be localized and/or low grade at diagnosis (5, 7, 13–16) and that they have lower mortality (13, 17, 18). Analyses of data for 1973–2001 from the SEER-National Longitudinal Mortality databases show that higher personal income and educational attainment are associated with higher PCa incidence and lower risk of late-stage diagnosis (7). A study of 70,899 cases diagnosed between 1988 and 1992 from the San Francisco Bay Area cancer registry found that higher neighborhood SES measured at the Census block group level was associated with higher PCa incidence in African American and Hispanic men (8). However, the NIH-AARP Diet and Health study of 292,688 men found that higher Neighborhood Deprivation Index score measured for the residential Census tract was associated with lower PCa incidence among Caucasian but not African American men (12). Analyses of 4,332 men in the National Program of Cancer Registries Patterns of Care Study found that higher neighborhood-level income and educational attainment measured at the Census tract level is associated with lower stage PCa at diagnosis (13). Recent analyses of SEER-Medicare data from 18,067 men diagnosed with PCa between 1994 and 2002 show that higher Zip code-level median household income is protective against advanced stage disease at diagnosis (5). Recent work with 1995 to 2007 cancer registry data from 21,808 men in the Philadelphia metroplitan area shows that higher neighborhood deprivation at the Census tract level is associated with poorer tumor grade at diagnosis (16).

The primary interpretation of the association between SES and PCa incidence and with stage and pathology at diagnosis is that higher-SES men have greater access to health and screening services, leading to earlier and over diagnosis $(7, 8, 13–15, 19)$. Low-SES men are more likely to be uninsured and to live in areas that have fewer medical facilities and/or facilities facing higher burdens of indigent care. A California study found that higher educational attainment and a higher ratio of income to the Federal Poverty Line were associated with having ever had a PSA test (20). Neighborhood SES may also mediate the effects of race-based segregation on access to health care (21). However, to our knowledge no prior study has directly tested whether differences in screening behavior explain the link between higher SES and PCa risk and the aggressiveness of tumors at diagnosis.

To address this gap, we examine the association of neighborhood-level SES with PCa screening behavior, PCa incidence, and tumor aggressiveness at diagnosis. The study uses a case-control design nested within a longitudinal cohort study of men followed up for PCa incidence after having undergone a biopsy or transurethral resection of the prostate (TURP) that found benign conditions. The study includes data on PSA and Digital Rectal Exam (DRE) screening during cohort follow-up and the matching of controls to cases on date of

the initial procedure and duration of follow-up effectively adjusts for temporal changes in screening practices.

Methods

Using incidence density sampling, a nested case-control study was assembled from a retrospective cohort of 6,692 men from the Detroit, Michigan, metropolitan area who had a prostate biopsy or TURP procedure with a benign finding between January 1990 and December 2002 at Henry Ford Health System (22). During follow-up of the cohort through July 2007, 808 potentially eligible cases diagnosed with prostate cancer were identified. The incidence of PCa within this high-risk cohort was approximately twice that of the general Detroit SEER population, although the ratio of African American to White cases in the cohort (ratio $= 1.62$) was similar to that in the overall SEER data (ratio $= 1.53$). To be eligible for the nested case-control study, the men had to be free of prior PCa diagnoses, a PSA level within a year of cohort entry had to be present in the medical record data and a benign tissue specimen from the initial procedure had to be available for pathological review. Patients diagnosed with PCa less than one year after the date of initial benign procedure were ineligible for the study. 'Date of cohort entry' was defined as the date of the initial benign procedure; 'date of case diagnosis' was the date of first cancer-positive tissue specimen or the date a clinician first reported a clinical diagnosis of PCa. Controls were matched to cases on age at entry into cohort $(\pm 2 \text{ years})$, date of entry into cohort $(\pm 2 \text{ years})$, race (African American or White), and type of specimen (biopsy or TURP; 7% of cases underwent an initial TURP).

We were able to match 802 of 808 potentially eligible cases to controls. Further review of medical record data reduced the final analytic sample to 574 case–control pairs. Exclusions were primarily due to problems with availability of tissue blocks ($n=126$; 55%), including lack of analyzable prostate tissue, wrong specimen type, or missing specimens. Other exclusions included absence of a PSA test within 1 year of cohort entry $(n=37; 16%)$ and evidence of malignancy $(n=29; 13%)$ after re-review of specimens initially characterized as benign. Further medical record review found earlier benign specimens (outside the cohort window) that made 12 (5%) pairs ineligible. The remaining pairs $(n=24; 10%)$ were excluded for reasons related to incomplete records at the time of cohort entry or diagnosis.

Data

Data for this study were abstracted from the HFHS medical records. The presence of any notation in the medical record of a family history of PCa in the subject's father or brothers was used to indicate a positive family history. Data on all PSA tests were abstracted and the PSA test value immediately prior to the initial benign procedure at the HFHS was used as the baseline PSA level. If a PSA test result immediately prior to the initial benign procedure was not available, the score from the first PSA test after the procedure was used. Screening intensity was measured as the total number of PSA tests and DRE during follow-up; for cases the period between the initial procedure and diagnosis was examined and for controls the period between the initial procedure and matching date (corresponding to the time interval between initial procedure and diagnosis for the matched case) was examined. Pathology data were retrieved from the medical records and high-grade disease was defined as having a biopsy (or surgical) Gleason score of 7 (4+3) and higher. Advanced stage disease was defined as pathologic or clinical stage of T3a or higher. Aggressive prostate cancer was defined has having either high-grade or advanced stage disease (23).

Data on the patient's own SES were not directly available. Patient SES was proxied using data on SES in the residential zip code noted in the medical record data at the time of cohort entry. Some of the zip codes listed in the medical record data could not be matched to zip

codes in the Detroit metropolitan area and may represent post office boxes. SES is a multidimensional construct that typically includes measures of educational attainment and income (24, 25). For each zip code tabulation area, US Census data for 2000 were downloaded and the measures "proportion of residents 25 years or older who had completed high school or better", "proportion of residents 25 years or older who had completed college or better", "proportion of residents living in poverty", "median household income", "per capita income", "proportion of the residents receiving public assistance" and "proportion of

residents who were African American" were calculated. These indicators were selected based on the author's review of previous studies of neighborhood SES and prostate cancer outcomes. Income and education represent the most commonly used measures of neighborhood SES in this literature, and racial composition was selected due to the salience of racial segregation in the Detroit area (9, 10, 12, 26–32). Indicators with relatively low variation in the Detroit metropolitan area, such as the percentage of Hispanics and the percentage of households lacking plumbing or kitchen facilities, were excluded from consideration.

Statistical Analysis

Based on prior research among men treated for PCa at the Henry Ford Health System, we expected the zip code-level measures of SES and racial composition to be highly correlated with each other, an expectation that was tested using Spearman Rank Correlation analyses (33). Similar to prior work using multiple Census data based measures of SES we applied principal component analyses to the seven zip code-level measures to estimate a comprehensive index of zip code-level SES (9, 10, 12, 26–31). Principal component analyses of the zip code-level data identified a single factor with an eigenvalue greater than 1. This factor was labeled "Neighborhood SES Index" (NSESI) and accounted for 80% of variance in the Zip code-level measures. A higher NSESI score reflected a higher neighborhood SES.

Among cases we used linear regression analyses to assess whether neighborhood SES was associated with age at baseline benign biopsy. These analyses adjusted for PSA at cohort entry, race, and family history of PCa and as controls were matched to cases on age at initial biopsy these analyses were restricted to cases only. Among cases and controls combined generalized estimating equation (GEE) analyses were used to assess whether neighborhood SES predicted the number of PSA tests during cohort follow-up (34). Count of PSA tests was modeled using a Poisson distribution and the GEE analyses accounted for nonindependence of observations that occurred because some individuals served as both a control and a case or as a control matched to multiple cases with different periods of followup. These analyses adjusted for age at cohort entry, PSA at cohort entry, race, family history of PCa, and duration of follow-up. The same analytical strategy was used to assess associations between neighborhood SES and the number of DRE tests during follow-up. Conditional logistic regression analyses were used to determine whether neighborhood SES was associated with PCa risk during follow-up, after adjustment for family history of PCa and PSA level at cohort entry.

Results

As observed previously, there were strong correlations among the six measures of zip code level-SES and the proportion of residents who were African American, with Spearman rank correlations ranging from −0.89 to −0.25 for inverse correlations and 0.64 to 0.95 for positive correlations (33). The NSESI score was positively associated with the proportion of residents 25 years or older who had completed at least high school (r=0.97) and at least college ($r=0.90$), median household income ($r=0.96$), per capita income ($r=0.98$), and negatively associated with the poverty rate (r=−0.88), the proportion of residents receiving

public assistance (r=−0.88) and the proportion of residents that are African American (r= −0.50).

Complete data were available from 494 case-control pairs, with zip code data missing for 82 case or control subjects. Age, family history of PCa, PSA at baseline, and case-control status were not associated with subjects having missing data zip code data. African American men were less like to have missing data, although the association was of only borderline significance (p=0.07). Table 1 presents descriptive statistics for cases and controls.

Among cases, after adjustment for race, family history, and PSA at initial procedure, increasing NSESI score was associated with a lower age at cohort entry, −1.48 years (95% CI, −2.32, −0.64) per one unit increase in NSESI score. Among cases and controls combined, after adjustment for race, age at cohort enrollment, PSA at initial procedure, family history of PCa, and duration of follow-up, increasing NSESI was associated with a higher count of PSA tests during follow-up, with men receiving 9% more PSA tests (95% CI, 2%, 16%) per one unit increase in the index. Similarly, men received 8% more DRE (95% CI, 1%, 15%) per one unit increase in the index.

After adjustment for family history of PCa and PSA level at cohort enrollment, increasing NSESI was significantly associated with a higher risk of PCa, with an $OR = 1.26$ per unit increase in the Index (95% CI, 1.04, 1.49) (see Table 2). Further adjustment for the number of PSA tests and DRE during follow-up (see Table 2) attenuated the odds ratio slightly (OR= 1.19) and rendered it no longer statistically significant. The effects of the NSESI score on PCa risk were similar in White (OR=1.25, 95% CI, 1.00, 1.56) and African American (OR = 1.29, 95% CI, 0.94, 1.77) men. Among cases, increasing NSESI score was not associated with having a high ($>=$ 7) Gleason score (OR = 0.86, 95% CI 0.65, 1.11) or with advanced ($\overline{3}$) tumor stage (OR = 0.97, 95% CI 0.62, 1.53) or with aggressive disease as defined by Gleeson score and stage (OR = 0.86 , 95% 0.66, 1.12).

Separate sensitivity analyses of the associations between PCa risk and each of the zip codelevel indicators included in the NSESI were also conducted. Most of the income-related Census variables, including the poverty rate, median household income, and proportion of residents receiving public assistance, were associated $(p<0.10)$ with risk of PCa in the expected directions; higher SES was associated with higher risk. The measures of educational status – the proportions of residents 25 years or older who had completed at least high school and at least college – were both significantly associated with higher PCa risk. The measures "per capita income" and "proportion of residents that are African American" were not associated with PCa risk.

Discussion

In this cohort, zip code-level measures of SES were associated with a younger age at cohort enrollment, more intensive screening behavior during follow-up, and a higher risk for PCa diagnosis during follow-up. The risk of PCa associated with increasing zip code-level SES was consistent in White and African American men. These results suggest that higher SES is associated with a higher level of medical scrutiny that leads to a higher risk of diagnosis of PCa.

Previous research has found higher SES, measured at either the individual or neighborhood level, to be associated with PCa risk and with diagnosis of less aggressive tumors (5, 7–16, 19). The usual interpretation of this pattern of associations is that they reflect higher medical surveillance or more intensive screening behavior among more affluent men. This is one of the first studies to directly test this hypothesis by assessing associations of SES with age at initial biopsy and with screening behavior. Younger age at cohort enrollment was associated

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with higher NSESI score, suggesting higher medical surveillance among those from higher SES zip codes. As observed in previous studies, higher SES was associated with PSA testing (20, 35, 36). The association between NSESI score and PCa incidence was somewhat attenuated after adjustment for the number of PSA test and DRE exams during follow-up. However, after adjustment for screening behavior, a one-unit difference in the NSESI was still associated with a 19% higher odds of PCa, an effect that was borderline statistically significant. As adjustment for screening intensity does not explain all of the association between SES and PCa risk, there may still be other health care-related factors that explain the association between SES and PCa risk. For instance, higher SES men may have greater spatial access to health care facilities, or fewer transportation barriers and may be able to schedule diagnostic procedures more promptly after a rise in PSA or a suspicious DRE (12).

Use of Census data aggregated to zip codes to measure the SES is a limitation of this research (24). Individual-level data on SES were not available through the medical record system which provided most PCa risk factor data for the study subjects. Thus, it is not clear whether the results reflect individual-level differences in SES or an effect of neighborhoodlevel SES that is operating independently of individual-level characteristics. Neighborhoods in the Detroit Metropolitan Area are highly stratified by income and race, so it is likely that area-level contextual measures of SES are relatively good proxies of individual-level SES. However, Krieger et al., have argued that area-based measures of SES should be viewed as community or neighborhood-level influences, which may impact exposure and health independently from individual-level socioeconomic characteristics or may interact with them to predict health (24, 37). This hypothesis cannot be directly tested in the current study.

Another limitation of this study is the use of zip codes to represent neighborhood areas (24, 25, 38, 39). Zip codes can vary substantially in area; their boundaries do not necessarily reflect resident's own conceptualizations of their neighborhood, nor are they defined in ways that reflect health or behavioral considerations (40). In addition, the use of zip codes to define neighborhood areas suffers from boundary effects, such that residents living close to the zip code boundaries are likely to be more affected by conditions in neighboring zip codes than are residents living more centrally within the zip code. However, the findings reported here, using relatively crude spatial measures of neighborhoods, suggest that the development of more refined spatial analyses of neighborhood SES is well justified.

SES is a multidimensional construct that reflects income, educational attainment and poverty (24, 39). Our measure of zip code-level SES, which we refer to as the Neighborhood SES Index, was developed from a principal component analysis of measures of income, education, and racial composition. This resulting measure of SES is thought to provide a better measure of SES than any single Census variable (31). Our analytical approach in this regard was similar to that used by many other groups, although the Census variables included in the principal component analysis varies extensively across studies (9, 10, 12, 26–31). The income- and education-related Census variables used in our analyses reflect multiple dimensions of SES and represent those measures most commonly used in previous studies of neighborhood SES and PCa outcomes. The extent of variation in neighborhood SES measures was somewhat higher than reported in past studies (5, 12). We additionally included racial composition in the principal component analysis because of the history of residential segregation in the Detroit metropolitan area and the role that such segregation can have in enhancing the effects of poverty.

The composition of this cohort, men at high risk who were followed-up after a prostate biopsy or TURP that yielded a benign result, reduces the generalizability of the results. Comparisons of this cohort with SEER data show that the men in the cohort had

approximately twice the incidence of PCa of the general population of men. While the findings of higher PCa risk among men in higher-SES zip codes is consistent with past studies, suggesting generalizability, prior work has also shown an inverse association between SES and tumor stage, grade and overall aggressiveness, which was not observed here. The failure to observe a significant effect on stage and grade may be a reflection of the smaller sample size, unusually comprehensive nature of the Henry Ford Health System, or the high-risk nature of the cohort. However, this cohort does reflect the situation faced by a large population of men in the U.S.; approximately 1 million biopsies are performed annually in the U.S. due to suspicion of PCa, two-thirds of which reveal benign conditions (2).

In conclusion, this work shows that zip code-level SES is associated with a higher risk of PCa diagnosis among men followed-up after receiving a benign diagnosis from prostate biopsy or TURP procedure. Zip code-level SES was also associated with more intensive PCa screening during follow-up, an association that was diminished after control for screening intensity. It is possible that additional factors such as proximity to health resources, better access to transportation and more flexible schedules may also explain associations between SES and PCa diagnosis.

References

- 1. Amercian Cancer Society. Society AC, ed. Atlanta, GA: American Cancer Society; 2012. Cancer Facts And Figures 2012.
- 2. Welch HG, Fisher ES, Gottlieb DJ, Barry MJ. Detection of prostate cancer via biopsy in the Medicare-SEER population during the PSA era. J Natl Cancer Inst. 2007; 99:1395–1400. [PubMed: 17848671]
- 3. Bostwick DG, Burke HB, Djakiew D, et al. Human prostate cancer risk factors. Cancer. 2004; 101:2371–2490. [PubMed: 15495199]
- 4. Gronberg H. Prostate cancer epidemiology. Lancet. 2003; 361:859–864. [PubMed: 12642065]
- 5. Carpenter WR, Howard DL, Taylor YJ, Ross LE, Wobker SE, Godley PA. Racial differences in PSA screening interval and stage at diagnosis. Cancer Causes Control. 2010; 21:1071–1080. [PubMed: 20333462]
- 6. Gornick ME, Eggers PW, Riley GF. Associations of race, education, and patterns of preventive service use with stage of cancer at time of diagnosis. Health Serv Res. 2004; 39:1403–1427. [PubMed: 15333115]
- 7. Clegg LX, Reichman ME, Miller BA, et al. Impact of socioeconomic status on cancer incidence and stage at diagnosis: selected findings from the surveillance, epidemiology, and end results: National Longitudinal Mortality Study. Cancer Causes Control. 2009; 20:417–435. [PubMed: 19002764]
- 8. Krieger N, Quesenberry C Jr, Peng T, et al. Social class, race/ethnicity, and incidence of breast, cervix, colon, lung, and prostate cancer among Asian, Black, Hispanic, and White residents of the San Francisco Bay Area, 1988–92 (United States). Cancer Causes Control. 1999; 10:525–537. [PubMed: 10616822]
- 9. Yin D, Morris C, Allen M, Cress R, Bates J, Liu L. Does socioeconomic disparity in cancer incidence vary across racial/ethnic groups? Cancer Causes and Control. 2010; 21:1721–1730. [PubMed: 20567897]
- 10. Cheng I, Witte J, McClure L, et al. Socioeconomic status and prostate cancer incidence and mortality rates among the diverse population of California. Cancer Causes and Control. 2009; 20:1431–1440. [PubMed: 19526319]
- 11. DeChello LM, Gregorio DI, Samociuk H. Race-specific geography of prostate cancer incidence. Int J Health Geogr. 2006; 5:59. [PubMed: 17176460]
- 12. Major JM, Norman Oliver M, Doubeni CA, Hollenbeck AR, Graubard BI, Sinha R. Socioeconomic status, healthcare density, and risk of prostate cancer among African American and Caucasian men in a large prospective study. Cancer Causes Control. 2012; 23:1185–1191. [PubMed: 22674292]

- 13. Byers TE, Wolf HJ, Bauer KR, et al. The impact of socioeconomic status on survival after cancer in the United States : findings from the National Program of Cancer Registries Patterns of Care Study. Cancer. 2008; 113:582–591. [PubMed: 18613122]
- 14. Xiao H, Gwede CK, Kiros G, Milla K. Analysis of prostate cancer incidence using geographic information system and multilevel modeling. J Natl Med Assoc. 2007; 99:218–225. [PubMed: 17393945]
- 15. Klassen AC, Curriero FC, Hong JH, et al. The role of area-level influences on prostate cancer grade and stage at diagnosis. Prev Med. 2004; 39:441–448. [PubMed: 15313082]
- 16. Zeigler-Johnson C, Tierny A, Rebbeck T, Rundle A. Prostate cancer severity association with neighborhood deprivation. Prostate Cancer. 2011
- 17. Chang CM, Su YC, Lai NS, et al. The combined effect of individual and neighborhood socioeconomic status on cancer survival rates. PLoS One. 2012; 7:e44325. [PubMed: 22957007]
- 18. Li X, Sundquist K, Sundquist J. Neighborhood deprivation and prostate cancer mortality: a multilevel analysis from Sweden. Prostate Cancer Prostatic Dis. 2012; 15:128–134. [PubMed: 21986984]
- 19. Jemal A, Ward E, Wu X, Martin HJ, McLaughlin CC, Thun MJ. Geographic patterns of prostate cancer mortality and variations in access to medical care in the United States. Cancer Epidemiol Biomarkers Prev. 2005; 14:590–595. [PubMed: 15767335]
- 20. Seo HS, Lee NK. Predictors of PSA Screening Among Men Over 40 Years of Age Who Had Ever Heard about PSA. Korean J Urol. 2010; 51:391–397. [PubMed: 20577605]
- 21. Guagliardo MF, Ronzio CR, Cheung I, Chacko E, Joseph JG. Physician accessibility: an urban case study of pediatric providers. Health & Place. 2004; 10:273–283. [PubMed: 15177201]
- 22. Kryvenko ON, Jankowski M, Chitale DA, et al. Inflammation and preneoplastic lesions in benign prostate as risk factors for prostate cancer. Mod Pathol. 2012
- 23. Beebe-Dimmer JL, Nock NL, Neslund-Dudas C, et al. Racial differences in risk of prostate cancer associated with metabolic syndrome. Urology. 2009; 74:185–190. [PubMed: 19428088]
- 24. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. Annu Rev Public Health. 1997; 18:341–378. [PubMed: 9143723]
- 25. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Choosing area based socioeconomic measures to monitor social inequalities in low birth weight and childhood lead poisoning: The Public Health Disparities Geocoding Project (US). J Epidemiol Community Health. 2003; 57:186–199. [PubMed: 12594195]
- 26. Wang F, Luo W. Assessing spatial and nonspatial factors for healthcare access: towards an integrated approach to defining health professional shortage areas. Health Place. 2005; 11:131– 146. [PubMed: 15629681]
- 27. James RC, Mustard CA. Geographic location of commercial plasma donation clinics in the United States 1980–1995. Am J Public Health. 2004; 94:1224–1229. [PubMed: 15226147]
- 28. Bell DC, Carlson JW, Richard AJ. The social ecology of drug use: a factor analysis of an urban environment. Subst Use Misuse. 1998; 33:2201–2217. [PubMed: 9758011]
- 29. Mares AS, Desai RA, Rosenheck RA. Association between community and client characteristics and subjective measures of the quality of housing. Psychiatr Serv. 2005; 56:315–319. [PubMed: 15746506]
- 30. Buka SL, Brennan RT, Rich-Edwards JW, Raudenbush SW, Earls F. Neighborhood support and the birth weight of urban infants. Am J Epidemiol. 2003; 157:1–8. [PubMed: 12505884]
- 31. Messer LC, Laraia BA, Kaufman JS, et al. The development of a standardized neighborhood deprivation index. J Urban Health. 2006; 83:1041–1062. [PubMed: 17031568]
- 32. Schulz AJ, Williams DR, Israel BA, Lempert LB. Racial and spatial relations as fundamental determinants of health in Detroit. Milbank Q. 2002; 80:677–707. iv. [PubMed: 12532644]
- 33. Rundle A, Richards C, Neslund-Dudas C, Tang D, Rybicki BA. Neighborhood socioeconomic status modifies the association between individual smoking status and PAH-DNA adduct levels in prostate tissue. Environ Mol Mutagen. 2012; 53:384–391. [PubMed: 22467358]
- 34. Hubbard AE, Ahern J, Fleischer NL, et al. To GEE or not to GEE: comparing population average and mixed models for estimating the associations between neighborhood risk factors and health. Epidemiology. 2010; 21:467–474. [PubMed: 20220526]

- 35. Chiu BC, Anderson JR, Corbin D. Predictors of prostate cancer screening among health fair participants. Public Health. 2005; 119:686–693. [PubMed: 15949522]
- 36. Spencer BA, Babey SH, Etzioni DA, et al. A population-based survey of prostate-specific antigen testing among California men at higher risk for prostate carcinoma. Cancer. 2006; 106:765–774. [PubMed: 16419068]
- 37. Rundle A, Field S, Park Y, Freeman L, Weiss CC, Neckerman K. Personal and neighborhood socioeconomic status and indices of neighborhood walk-ability predict body mass index in New York City. Soc Sci Med. 2008; 67:1951–1958. [PubMed: 18954927]
- 38. Krieger N, Chen JT, Waterman PD, Rehkopf DH, Subramanian SV. Race/ethnicity, gender, and monitoring socioeconomic gradients in health: a comparison of area-based socioeconomic measures--the public health disparities geocoding project. Am J Public Health. 2003; 93:1655– 1671. [PubMed: 14534218]
- 39. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Geocoding and monitoring of US socioeconomic inequalities in mortality and cancer incidence: does the choice of area-based measure and geographic level matter?: the Public Health Disparities Geocoding Project. Am J Epidemiol. 2002; 156:471–482. [PubMed: 12196317]
- 40. Lovasi G, Grady S, Rundle A. Steps Forward: Review and Recommendations for Research on Walkability, Physical Activity and Cardiovascular Health. Public Health Reviews. 2012 (in press).

Table 1

Characteristics of the case and matched controls.

Table 2

Associations between Case-control status and Risk Factor Variables.

All OR are mutually adjusted for other variables in the table and for the matching factors.