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By all measures: an examination of the relationship between segregation and health risk from air pollution

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Abstract A great deal of evidence suggests that African-Americans in more racially segregated communities are at a higher risk for a variety of health problems. Scholars have argued that these health inequalities might be explained by racial differences in exposure to air toxins. However, there are a number of ways to measure segregation, each representing different pathways of exposure. There has yet to be a systematic evaluation of how exposure to air toxins varies by these different measures, making it difficult to begin to theorize about the causal story linking segregation, pollution and health. This paper addresses this gap by examining how the health risk from industrial toxins varies by the 19 most commonly used segregation measures. Results show that, with the exception of two segregation measures, living in metro areas with relatively higher segregation levels, is associated with significantly greater health risk from industrial air toxins for all racial groups. Moreover, African-Americans in more segregated metro areas typically experience an added risk of exposure compared to non-Hispanic whites.

Keywords United States · Racial segregation · Environmental inequality · Air pollution · Health risk · Multilevel models

Introduction

The concept of "residential segregation" has played an important role in the place and health literature (Meijer et al. 2012; Kramer and Hogue 2009). Specifically, African-Americans in communities with higher levels of racial segregation experience a diverse set of negative health outcomes, such as higher rates of preterm births (Britton and Shin 2013); infant-mortality (LaVeist 1993), lung cancer

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(Hayanga et al. 2013), hypertension (Kershaw et al. 2011), and all-cause morbidity (Collins and Williams 1999). In addition, there is evidence that non-Hispanic whites also have worse health outcomes in more segregated communities (Morello-Frosch and Jesdale 2006; Collins and Williams 1999). The knowledge that more segregated communities have worse health outcomes raises the question: What makes segregation bad for your health? Over the past decade, scholars have shown a great deal of interest in exposure to air pollution as one possible underlying mechanism (Kramer and Hogue 2009; Gee and Ford 2011; Gee and Payne-Sturges 2004; Evans and Kantrowitz 2002; Williams and Collins 1995). Nonetheless, researchers today still note that "little is known regarding the association between racial segregation and air pollution exposure" (Jones et al. 2014, p. 2135).

Previous work has produced inconsistent findings, with some showing that segregation increases risk of exposure to environmental toxins and others showing no association. Earlier works found a positive association between the dissimilarity index of 44 large metropolitan areas in the United States, and the unequal distribution of the 148 chemicals listed in the 1990 Environmental Protection Agency (EPA) Air Toxics Data (Lopez 2002). Using the same data, but for an expanded set of 309 metro areas, Morello-Frosch and Jesdale (2006) found that an increase in the multi-group dissimilarity index amplified the cancer risk from air toxins for all racial groups. In addition, the racial disparity in cancer risk widened with increasing levels of segregation. More recently, scholars have come to different conclusions. Using 2000 census data, and over 600 chemicals listed in the 2000 Toxic Release Inventory (TRI) data, Downey et al. (2008) linked the average toxic concentration for 329 metropolitan areas in the United States with dissimilarity index measures for several racial/ethnic groups. The authors concluded that, "metropolitan area residential segregation levels and metropolitan area income inequality levels are relatively poor predictors of metropolitan area environmental inequality outcomes" (Downey et al. 2008; p. 288). This study was an expansion of Downey's previous work (2007), which used modeled TRI data for 61 of the largest metro areas in the United States. His results showed that those cities with the highest degree of segregation were not the same cities with the largest racial disparities in pollution exposure. Similarly, a more recent study of six cities estimated exposure to nitrogen oxides and fine particulate matter (PM 2.5) at the census tract level found that those areas with clusters of African-American populations greater than the city average did not have significantly higher amounts of exposure to pollution (Jones et al. 2014).

The discrepancy in these findings makes it clear that more evidence is needed in order to understand the extent to which segregated communities are at risk for greater exposure to air toxins. Examination into this form of structural racism is important if we are to begin to unpack what it is about "place" that affects individual health outcomes. To this end, the analysis presented here examines how the combined health risk from over 500 industrial air pollutants varies according to the 19 most commonly used measures of segregation. A series of multilevel models were run with different segregation measures for non-Hispanic whites (herein referred to as white) and non-Hispanic blacks (herein refeered to as

African-Americans), in order to compare how the relationship between pollution and segregation varies by the measure used.

Results show that all but two measures of segregation are significantly associated with a greater risk of exposure to air toxins for everyone in that metro area. The extent to which African-Americans within more segregated metro areas experience an added health risk from air toxins is also examined. Results show that block groups with African-American populations greater than the metro average have an added risk of exposure if they live in more segregated metro areas. However, a moderation of this relationship was seen in the interaction between the relative proportion of African-American and three segregation measures: isolation index, distance-decay isolation, and absolute clustering. Moreover, this association was complicated when sensitivity analyses were run for spatial correlation. When standard errors were clustered by metro areas this variable lost significance, but primarily for those measures that reflected the evenness dimension of segregation. This suggests this dimension, which is currently the most used measure of segregation, is sensitive to spatial clustering. The analyses presented here will provide a foundation for scholars to start the process of critical thinking about what segregation measures are actually capturing in terms of health risk.

Background

To begin to understand how exposure to environmental hazards might operate as the mechanism through which segregation creates and perpetuates health inequality, we need to recognize the centrality of the manufacturing industry in the spatial patterning of African-Americans in US cities. During the Great Migration, a large portion of the African-American population was displaced into America's cities to work as labor for the growing manufacturing industry (Grossman 1991). After World War II, redlining prevented African-Americans from moving into America's rapidly developing suburbs. This process allowed whites to leave urban centers, segregating African-Americans in these more industrial areas (Farley et al. 2002; Massey and Denton 1988; Quillian 2003; Brodkin 1998). It is argued in the 1960s and 1970s there was increasing evidence and awareness that emissions from such facilities were detrimental to health (Saha and Mohai 2005). Research has shown that during this time in the Detroit metro area, factories were increasingly likely to be sited in areas with greater percentages of African-Americans (Saha and Mohai 2005). This pattern was also consistent in New York City; Maantay (2001) found from 1961 to 1998 areas with a higher than average proportion of minorities were more likely to be zoned, and rezoned, for industrial uses.

America's racial minorities, particularly African-Americans, have lived, and continue to live, in more industrial areas (Ard 2015; Taylor 2014). However, it is reasonable to ask if these industrial factories are still a major driver of health risk today, and there are several reasons to think this is the case. Firstly, because industries prompted the building of residential areas, roads and other infrastructure, their location is correlated with other types of pollution source (e.g., mobile sources) (Lee et al. 2010; Weber 1909). Secondly, and perhaps most importantly, many of

these industrial facilities still exist today and have been able to opt out of newer, more stringent environmental regulations. According to data from the U.S. Energy Information Administration (2015), of the 3772 boilers used in electric generators operating in the United States in 2013, roughly 49 % of them were not held to updated emission standards, and roughly a third of those grandfathered out of newer regulations were built between 1925 and 1970. The ability to grandfather out of stricter environmental regulations incentivizes the continued operation of older facilities which are operating under less stringent standards (Gerrard 1994). In 2002, the EPA's National Scale Air Toxics Assessment program estimated that all stationary sources (i.e., a building, structure, facility, or installation that emits air toxins) combined to make up 24 % of the cancer risk, over 52 % of neurological risk, and 18 % of respiratory risk from air toxins (see Table 1).

Health inequality scholars have noted the difficulty in measuring the precise geographic context in which environmental factors influence health (Kwan 2012; Acevedo-Garcia and Osypuk 2008). Kwan (2012) terms this the Uncertain Geographic Context Problem. What geographic delineation best represents the pathway through which place affects health? To answer this question adequate models are needed that take into account the spatial and temporal complexities of people's lives (Kwan 2012). Therefore, scholars need to ask: Where do people spend their days and how does this contribute to their exposure to different environments? Temporally, the investigation presented here is based on the idea that people spend the bulk of their daily lives in activities conducted inside their home, such as sleeping and household activities (Bureau of Labor Statistics 2015). Thus, the dependent variable in the analyses presented below is the average annual residential exposure. This measure is derived from plume modeled data of industrial air toxins. Of course, individuals also spend time away from their home. The exposure experienced in these places is not captured in the following analyses; thus, only a partial picture of risk is presented here.

A goal of this paper is to encourage further discussion in the environmental inequality literature about which spatial configuration is the best measure of segregation As Massey and Denton (1988, p. 283) noted, "groups may be 'segregated' in a variety of ways." Their analysis of segregation measures helped to delineate five dimensions of segregation that conceptualized different ways in which demographic groups live apart from one another. Health and place scholars have yet

Table 1 Percentage of health risk contributed by EPA's National Air Toxics Assessment Data in 2002

Health risk	Stationary sources Major and area sources	Non-stationary sources On-road and non-road sources	Background Sources farther than 50 km away, unidentified and natural sources
Cancer	24.07	30.04	45.89
Neurological	52.30	19.53	28.17
Respiratory	18.33	77.49	3.92

to adequately explain how these conceptions of spatial patterning might account for the role pollution exposure has in the link between segregation and health inequality. It is essential that this theoretical discussion take place before the creation, and application, of more ways to measure spatial patterning of demographics. Methodologists within sociology have begun to apply techniques developed in geographic science to the study of social inequality (Lee et al. 2008). For example, Reardon and O'Sullivan (2004) estimate population density with census tract data in order to blur the arbitrary boundaries census delineations provide. Other scholars have utilized the Getis-Ord statistic to measure clustering of African-Americans (Jones et al. 2014). However, even with more accurate measures of where people are located, unless we begin with a theoretical basis for why one type of separation of racial groups is more important to health than another, the field will be no closer to understanding why segregation is related to health inequality. This paper uses the dimensions of segregation delineated by Massey and Denton (1988) as a starting place for this discussion.

There have primarily been 19 measures of residential segregation that are widely used in the racial segregation literature (U.S. Census 2013). Factor analyses done by Massey and Denton (1988) showed that these measures reflect five different dimensions of segregation (a) centralization, the concentration of a minority race around the urban center; (b) clustering, which reflects the tendency for those of the same racial group to live within the same geographic area; (c) concentration, measures the degree to which a minority race is occupying less physical space than the majority; (d) exposure, the tendency of individuals to interact with those outside of their race; and (e) evenness, how distributed a minority group is across a broader urban area (Massey and Denton 1988).

The vast majority of studies examining how pollution risk varies by segregation measures have used the dissimilarity index, a measure of evenness. Despite this wide-scale use, there has been little discussion as to why this dimension of segregation would be the best indicator of pollution exposure. Theoretically, the dimension that is most likely related to African-Americans' increased risk to industrial air pollution is centralization–how close African-Americans are to the central city. This is because, as noted above, central cities were historically America's hubs of industrial manufacturing and provided low-skill jobs for African-Americans migrating from the South (Farley et al. 2002). However, in the 1970s, 1980s and 1990s, middle-class African-Americans moved away from the inner cities (Wilson 1987; Quillian 2003).

As African-American populations grew in areas adjacent to central cities, metro areas racial enclaves expanded; these areas have since been termed inner ring suburbs (Oliver 2001). This type of segregation is best captured by the clustering dimension. African-Americans living in metro areas with a high degree of clustering will have block groups with a relatively high proportion of African-American residents contiguous to one another. Investigating how the proportion of African-Americans changed in the Detroit metro area from 1970 to 1990, Downey (2005) found support for the idea that African-Americans were drawn into neighborhoods immediately adjacent to areas with already high proportions of African-Americans. However, Downey (2005, 2007) argued that this process of segregation actually

protected African-Americans from exposure to industrial toxins as newer facilities were increasingly choosing to locate in the suburbs.

While not explicitly studied in the pollution and segregation literature, the dimension concentration has the potential to reflect the history of dense African-American residential areas. Concentration is a measure of how much physical space is occupied by African-Americans. This measure is affected by the available housing stock in a block group. For example, public housing projects developed after World War II created high-rise, low-income, housing that allowed for a large number of individuals to reside in a small geographic area. These homes were largely occupied by African-Americans and spatially clustered (Dawkins 2013; Cutter et al. 2001; Venkatesh 2000). There has been little research done on how government subsidized housing varies in its risk to air toxins. However, one of the few studies to examine the issue found that in 1990, in eight large metro areas across the United States, Housing and Urban Development (HUD) Sect. 8 housing was significantly closer to hazardous facilities (Cutter et al. 2001).

As noted earlier, the vast majority of studies examining pollution and segregation have used the evenness dimension, which is a measure of how evenly distributed racial minorities are compared to the majority race in a metropolitan area. Downey et al. (2008) examined how differential exposure to modeled Risk-Screening Environmental Indicators (RSEI) data varied by the dissimilarity index across 329 metro areas in the year 2000. The authors concluded that residential segregation was a poor predictor of the ratio of exposure between African-Americans and whites. Using the same data, for 61 metro areas, Downey (2007) showed that those areas with the highest dissimilarity index were not the same metro areas with the highest environmental inequality between non-Hispanic whites and African-Americans. The author theorized that pollution and minorities might be concentrated in different neighborhoods, or environmental hazards are evenly distributed across urban areas. The latter argument runs counter to Weber's (1909) least cost theory, which holds that industrial facilities are sited near industries of similar type because these areas already have the necessary infrastructure, access to markets, and labor that companies need to effectively do business. This process clusters facilities, creating a Zone of Industry, which sociologists argue impact the economic opportunities of individuals within a metropolitan area and the spatial concentration of poverty (Wilson 1996). Because these facilities also emit air pollution, these zones of industry are also zones of exposure to air toxins.

The exposure dimension of segregation intentionally tries to reflect the experience of individuals by measuring the potential for actual contact between racial groups based on the sharing of residential areas. The segregation and health literature has debated whether this dimension is associated with positive or negative health outcomes. Some researchers have theorized, and found support for, the idea that isolation of African-Americans concentrates disadvantage in these areas (Subramanian et al. 2005; Collins and Williams 1999; Shihadeh and Flynn 1996). Others have argued that African-American social isolation can be protective, due to an increase in social capital that provides more social support and political power, which can be utilized to advocate for better services and conditions (Bell et al. 2006; LaVeist 1993). The latter parallels an argument in the environmental inequality

literature which is that the more social capital a community has, the better able they are to prevent the siting of industrial facilities through protest, affecting policy, and ensuring that polluting facilities in their area are following guidelines (Taylor 2014). While there has been very little research examining social capital and pollution, those studies that have been done have found decreased voter turn-out was associated with the expansion of hazardous waste facilities (Hamilton 1995). Downey et al. (2008) provides one of the only studies to examine how pollution exposure varies by a measure of exposure. The authors found that their results using the isolation index were not much different when compared to the dissimilarity index, and neither measure explained much of the racial disparity in exposure to air toxins.

The literature examining how health outcomes vary by segregation rates have primarily used two measures of segregation: exposure and evenness (Kramer and Hogue 2009). The overreliance on such measures has been critiqued in the place and health literature as being utilized for the sake of ease and being conceptually weak (Kramer and Hogue 2009; Acevedo-Garcia et al. 2003). A similar argument can be made in the literature examining how pollution varies according to different segregation measures. Segregation measures are indices of spatial patterning of African-Americans in metro areas. Such indices reflect the history of cities. We know that manufacturing industries have played an important role in the development of place in American cities (Taylor 2014; Farley et al. 2002; Wilson 1996) and that this has interacted in complex ways with race. To begin to understand what this means for health risk from air toxins we need to first map out how various conceptions of segregation relate to exposure. The analyses presented below can serve as a framework for future scholarship on health inequality by providing a picture of how industrial toxins vary across these indices when controlling for regional variation and metropolitan distinctness.

Data and methods

Segregation measures

Segregation measures for the 331 Metropolitan/Primary Metropolitan (M/PMSA) in the continental United States were obtained from the U.S. Census Bureau Housing Patterns unit (U.S. Census Bureau 2011). Two of these M/PMSAs are outside of the continental U.S. and were excluded from the analysis. They were calculated for African-Americans and whites as the reference group, and block group as the unit of analysis.

Segregation as measured by the dimension evenness is maximized when no African-Americans and whites live in the same block group. The most commonly used measure of evenness, the dissimilarity index (D), reflects the degree to which the proportion of African-Americans in a block group is the same as their population in the overall metro area. Similarly, Theil's entropy index (H) reports the average block group deviation from a metro area's entropy, a measure of racial diversity, which is greatest when the two comparison groups each make up half of the

population. The final measures of evenness, the Gini index (G) and the Atkinson indices (A1, A5, and A9) all explicitly utilize the Lorenz curve. The Gini index represents the area under the diagonal of evenness, and the Atkinson indices represent this area but where areal units are differentially weighted. For a shape parameter of 0.1 (A1) block groups where the proportion of African-Americans is smaller than the metro average contribute more to the measure of segregation; for a shape parameter of 0.5 (A5) they contribute equally; and for a parameter of 0.9 (A9), block groups where the proportion of African-Americans is greater than the metro average contribute more.

The dimension exposure is measured in two primary ways. The first is the Isolation index (xPx), which can be interpreted as the probability that a randomly drawn African-American person in a metro area lives in the same block group as another African-American. The converse measure, the interaction index, provides the diametric opposite relationship of the isolation index and was thus left out of the following analyses. The next measure of exposure, the correlation ratio (V) standardizes the isolation index by the proportion of African-Americans that make up the metro area.

There are a variety of measures of the clustering dimension, two of which are measures of both relative clustering (RCL) and absolute clustering (ACL). Absolute clustering represents the average number of African-Americans in nearby block groups as a proportion of the total population in adjacent block groups. Relative clustering compares the clustering of African-Americans to the clustering of whites; RCL is zero when their clustering is equal. The spatial proximity (SP) is a measure of the average proximity between African-Americans, compared to the average proximity between whites, weighted by each racial group's proportion of the total metro area population. The final measure of clustering is a distance-adjusted probability of isolation, or distance-decay probability (DPxx). This measure can be interpreted as the probability that an African-American person will meet another African-American person if they go anywhere in their metro area. Again, the distance-decay interaction index provides the exact opposite correlation of the distance-decay isolation index and was therefore excluded, reducing the number of segregation measures presented here to seventeen.

Because the size of the central city, and thus physical capacity of residents to live there, varies, measures of the dimension centralization have been created to take into account the spatial limitations of metros. One of two such measures is the relative centralization (RCE) measure, created by ordering block groups by their increasing distance from the historic center of a city, and then comparing the cumulative proportions of African-Americans to whites. The index can be interpreted as the relative proportion of African-Americans that would have to move to equal the degree of centralization of whites around the center. The other measure of centralization is absolute centralization (ACE), which has been used to a much lesser extent. It does not compare African-Americans patterning to whites, rather it measures the extent to which African-Americans would have to move to achieve uniform distribution around the central city.

Like clustering and centralization, the concentration dimension is measured in absolute and relative terms. The absolute concentration (ACO) measure reflects the

absolute amount (or area) of physical space occupied by African-Americans compared to what is available in a metro area. The relative concentration (RCO) measure is the amount of physical space African-Americans occupy compared to whites. In addition to these two measures, the measure of delta (DEL) has also been used. Delta is the proportion of African-Americans residing in block groups with above average density of African-Americans.

Table 2 shows descriptive statistics for each of the segregation indices used, as well as their Pearson's correlation with the logged health risk and the median income of the M/PMSA. Interestingly the median income of a metro area and its

Table 2 Descriptive statistics of segregation measures

Dimension	Index		Mean	SD	Min	Max	Correlation with:	
							Median income M/PMSA	Logged health risk
Evenness	Dissimilarly index	D	0.628	0.129	0.265	0.861	0.157	0.394
	Entropy index	H	0.405	0.167	0.036	0.726	0.142	0.374
	Gini index	G	0.777	0.122	0.369	0.954	0.146	0.397
	Atkinson index $b = 0.1$	A1	0.157	0.062	0.027	0.330	0.065	0.369
	Atkinson index $b = 0.5$	A5	0.557	0.172	0.117	0.858	0.136	0.389
	Atkinson index $b = 0.9$	A9	0.766	0.180	0.187	0.978	0.135	0.378
Exposure	Eta squared	V	0.408	0.206	0.002	0.769	0.135	0.346
	Isolation	Xpx	0.493	0.226	0.007	0.838	0.094	0.312
Clustering	Absolute clustering	ACL	0.310	0.203	0.000	0.713	0.132	0.286
	Relative clustering	RCL	2.562	2.144	−0.329	27.312	0.179	0.263
	Distance–decay isolation	DPxx	0.382	0.206	0.005	0.732	0.054	0.264
	Spatial proximity	SP	1.344	0.247	1.001	1.907	0.182	0.324
Centralization	Absolute centralization	ACE	0.731	0.167	−0.239	0.965	−0.173	0.115
	Relative centralization	RCE	0.300	0.202	−0.309	0.708	−0.020	0.323
Concentration	Absolute concentration	ACO	0.915	0.078	0.485	0.991	0.192	0.082
	Relative concentration	RCO	0.637	0.285	−1.79	0.931	0.121	0.284
	Delta	DEL	0.828	0.069	0.488	0.969	−0.110	0.070

level of segregation is positively related, with the exception of centralization measures and one measure of concentration, Delta. However, the stronger bivariate correlations are seen between the logged health risk and the segregation measures, with most being around 0.3, but range from 0.070 to 0.397. Nonetheless, all segregation measures are positively correlated with the logged health risk from air toxins.

Dependent variable

The health risk from air pollution was obtained from the Environmental Protection Agency's Risk Screening Environmental Indicators Geographic Microdata (RSEI-GM). The RSEI-GM data are based on the 2000 Toxic Release Inventory (TRI) data. TRI data are collected annually from facilities that fall within certain industries (e.g., manufacturing, mining, electric power), have more than 10 employees, and emit more than 25,000 pounds of one of the chemicals regulated under the Right to Know Act. In the year 2000, there were 17,504 facilities required to report information on the amount of 595 regulated chemicals their facility emitted over the year. These data are then used in plume models that utilize information about the height of the smokestack, the velocity at which a chemical is emitted, typical air patterns, and other place-specific information, to determine where these chemicals landed in a 100-km^2 area around the facility. The entire continental United States is broken down into over 14 million one-by-one-kilometer grid cells with each having an estimate of air pollution. Grid cell pollution estimates are then weighted by the amount of area they take up in a block group and then summed to 2000 block group boundaries. A total of 165,017 block groups made up the 329 M/PMSA areas in the continental US, of these, 5426 had no estimated pollution in them, and because zero is a meaningful number in terms of health risk from pollution exposure, 0.01 was added to all numbers before taking the log score (see Table 3). The logged pollution exposure estimate, weighted by health risk, was utilized as the dependent variable at the block group level. Results were robust to censoring the exposure variable at the 97th percentile nationwide, following Boyce et al. (2014).

Table 3 Descriptive statistics of variables used in analysis

Level		Min	Max	Mean	Std.Dev
Block group	Log of toxic concentration (dependent variable)	−4.61	11.90	1.59	2.39
	Toxic concentration (plus 0.1)	0.01	146,842.50	47.31	463.47
	Population density	0.00	332,787.20	6826.58	13,688.01
	Difference between proportion of African-Americans in M/PMSA and block group	−50.56	99.73	1.61	22.99
	Proportion of African-Americans in P/MSA	0.15	50.77	12.73	9.30
M/PMSA	Median income	25,699.00	79,127.00	46,777.83	8520.87

The weighted health risk was provided by the EPA, who uses epidemiological evidence from acute and chronic animal studies, and in vitro toxicity tests, to assign each of these chemicals a weight for how harmful they are to human health. The weights were created in order to make them commensurate across health outcomes (Blake-Hedges et al. 2007). The RSEI-GM model, "addresses chronic human toxicity (cancer and noncancer effects, such as developmental toxicity, reproductive toxicity, neurotoxicity, etc.) associated with long-term exposure" (Blake-Hedges et al. 2007; 10).

Demographic data

The proportion of African-Americans residing in a block group, and for the entire metropolitan statistical area/primary metropolitan statistical areas (M/PMSA), was calculated with 2000 Census data obtained from Social Explorer (U.S. Census Bureau 2000). There were 578 block groups with no population, so these were dropped from the analysis for a total sample of 164,439 block groups. The median income of households in the M/PMSA areas was also obtained and adjusted for inflation to 2000 dollars.

Model specification

The following model was run for each of the 19 grand-mean centered segregation measures:

$$\gamma = \alpha + \beta_1 \times \text{propaa}_{\text{M/PMSA}} + \beta_2 \times \text{logmedinc}_{\text{M/PMSA}} + \beta_3 \times \text{logpopden}_{\text{BG}} + \beta_4 \times \text{propaadiff}_{\text{BG}} + \beta_5 \times \text{SEG}_{\text{M/PMSA}} + \beta_5 \times \text{SEG}_{\text{M/PMSA}} \times \text{propaadiff}_{\text{BG}} + \varepsilon_{\text{M/PMSA}} + \varepsilon_{\text{BG}}$$

A random effect was included for M/PMSAs ($\varepsilon_{\text{M/PMSA}}$). The proportion of African-Americans in a M/PMSA controls for large-scale variation in demographics ($\text{propaa}_{\text{M/PMSA}}$), for example, metro areas inside the "black belt" in the Southern part of the United States will have a larger African-American population compared to other parts of the country. The log of a block group's population density ($\text{logpopden}_{\text{BG}}$) and the median income ($\text{logmedinc}_{\text{M/PMSA}}$) of households in a metro area is also controlled for as these have been shown to be significantly related to pollution exposure (Downey 2005; Hird and Reese 1998). The possibility that the relationship between pollution and income was nonlinear was tested with a quadratic term. However, the coefficient was virtually zero and only slightly significant in a few cases so was left out of the final models.

The proportion of African-Americans in a block group was cluster-mean centered, so centered at the M/PMSA level ($\text{propaadiff}_{\text{BG}}$). This helps to answer a question examined in the literature as to whether living in the parts of the metro area where there are relatively more African-Americans is associated with being at greater risk to air toxins (Jones et al. 2014). A positive coefficient on this variable would indicate that the health of those living in block groups with relatively more

African-Americans is at greater risk from air toxins than those block groups with proportionally fewer African-Americans. Sensitivity analyses were performed with the percentage of African-Americans in a block group rather than the cluster-mean centered variable and the results were essentially identical.

The interaction between segregation and the relative proportion of African-Americans was also included ($SEG_{M/PMSA} \times propaadiff_{BG}$). This allows for investigation of whether there is an added risk of living in a block group with relatively more African-Americans, in a more segregated metro area, a question previously examined in this literature (Downey 2008). If the coefficient on this variable is positive, it would mean that if you compared two block groups with the same relative proportion of African-Americans, the one in a metro area with a higher segregation level will have a greater risk from air toxins. Results were robust to sensitivity analyses with the interaction term excluded.

Results

Results from the multilevel models are displayed in Table 4. These show that the most persistent and strongly predictive factor of the extent of health risk from air toxins was whether the block group had more African-Americans living there relative to the M/PMSA average (PROPAADIFF). The coefficient ranged from 0.186 to 0.466 suggesting that those block groups with more African-Americans residents than the metro average have significantly more pollution exposure no matter what type of segregation measure is used. However, when these models were run with standard errors clustered by metro area, this coefficient lost its significance but this was largely seen with the measures of evenness. This suggests this variable is sensitive to spatial clustering.

These models show that by most measures of segregation, higher segregation is related to a higher level of health risk for everyone in the metro area. The "SEG" coefficient across all measures of evenness is significantly and positively related to pollution exposure, with the logged pollution risk effects ranging from a 0.429 increase to a 1.125 increase. There were two segregation indices that were not significantly and positively related to exposure. The two non-significant measures were absolute centralization (ACE) and delta (D), measures of centralization and concentration respectively. Notably, these are two of the three measures that are created by only looking at the distribution of the African-American population, rather than comparing the distribution of the African-American population *relative to* the white population. The only other measure that does not consider the relative distribution in some way is absolute concentration and this measure was not strongly significant.

The variable (SEGINT) is the cross-level interaction between the percentage of African-Americans in a block group and segregation of a M/PMSA. It can tell us whether there is an extra health risk associated with living in a highly African-American block group in a highly segregated M/PMSA. For all but six of the measures of segregation, there was indeed an extra health risk. It is important to remember at this point that the measure of health risk was determined by weighting

Table 4 Multilevel models with the log of the combined health risk from industrial toxins as the dependent variable

	Evenness						Exposure	
	D	H	G	A1	A5	A9	V	Xpx
Fixed effects								
MSApropaa	1.105	0.063	0.919	3.172*	1.060	0.351	−1.046	−3.763
	1.292	*1.425*	*1.298*	*1.237*	*1.312*	*1.360*	*1.631*	*2.090*
logpopden	0.237***	0.237***	0.237***	0.238***	0.237***	0.236***	0.236***	0.235***
	0.002	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*
logmedinc	0.778	0.882	0.888	1.585*	0.964	1.006	0.952	1.058
	0.724	*0.738*	*0.718*	*0.719*	*0.724*	*0.724*	*0.752*	*0.755*
propaadiff	0.221***	0.247***	0.236***	0.186***	0.231***	0.320***	0.311***	0.444***
	0.017	*0.018*	*0.018*	*0.016*	*0.018*	*0.021*	*0.020*	*0.022*
SEGINT	0.125***	0.080***	0.109***	0.180***	0.108***	−0.014	0.000	−0.159***
	0.016	*0.016*	*0.019*	*0.013*	*0.017*	*0.023*	*0.018*	*0.022*
SEG	1.004***	1.042***	0.933***	0.909***	0.995***	0.893***	1.019***	1.125***
	0.138	*0.165*	*0.127*	*0.144*	*0.143*	*0.130*	*0.187*	*0.222*
Constant	−8.588	−9.545	−9.764	−17.570*	−10.565	−10.954	−10.179	−10.989
	7.752	*7.924*	*7.695*	*7.680*	*7.753*	*7.754*	*8.088*	*8.132*
Random effects								
M/PMSA	5.048	5.232	5.030	5.230	5.112	5.124	5.379	5.436
Residual	1.353	1.353	1.353	1.352	1.353	1.354	1.354	1.353
AIC	518,695.4	518,741.5	518,720.8	518,574.7	518,715.3	518,758.5	518,774.8	518,724.3

Table 4 continued

	Clustering				Centralization			Concentration	
	ACL	RCL	DPxx	SP	ACE	RCE	ACO	RCO	DEL
Fixed effects									
MSApropaa	0.145	5.657***	−3.185	0.888	5.086***	4.544***	8.185***	5.210***	5.471***
	1.679	*1.267*	*2.566*	*1.495*	*1.292*	*1.189*	*1.788*	*1.231*	*1.432*
logpopden	0.235***	0.236***	0.235***	0.236***	0.236***	0.235***	0.236***	0.236***	0.235***
	0.002	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*	*0.002*
logmedinc	1.145	1.590*	1.330	1.007	2.175**	1.339	1.744*	1.529*	2.100**
	0.769	*0.748*	*0.769*	*0.765*	*0.762*	*0.710*	*0.757*	*0.733*	*0.759*
propaadiff	0.397***	0.299***	0.466***	0.328***	0.298***	0.283***	0.326***	0.312***	0.305***
	0.018	*0.013*	*0.021*	*0.017*	*0.013*	*0.013*	*0.013*	*0.014*	*0.013*
SEGINT	−0.102***	0.086***	−0.179***	−0.021	0.160***	0.163***	0.081***	−0.004	0.289***
	0.015	*0.014*	*0.019*	*0.013*	*0.016*	*0.012*	*0.011*	*0.017*	*0.014*
SEG	0.933***	0.489***	1.024***	1.006***	0.157	0.932***	0.429**	0.588***	0.122
	0.225	*0.122*	*0.287*	*0.215*	*0.116*	*0.129*	*0.161*	*0.111*	*0.125*
Constant	−12.439	−18.147*	−14.046	−11.013	−24.429**	−15.284*	−20.131*	−17.442*	−23.660**
	8.278	*7.978*	*8.315*	*8.228*	*8.134*	*7.578*	*8.064*	*7.827*	*8.104*
Random effects									
M/PMSA	5.572	5.587	5.643	5.496	5.830	5.054	5.738	5.403	5.844
Residual	1.353	1.353	1.353	1.354	1.353	1.352	1.353	1.354	1.350
AIC	518,739.8	518,749.2	518,697.7	518,779.2	518,701.6	518,559.0	518,744.7	518,776.2	518,393.5

For the fixed effects coefficients, the figures in italics are standard errors

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

the total amount of a chemical estimated to be in the air, by its toxicity to human health on a variety of health outcomes. This means the magnitude of the effect can only be understood when comparing across models. The results in Table 4 show that for all of the centralization measures, being in a block group that has a larger proportion of African-American residents than the metro average is associated with an increased health risk, with the relative centralization measure having the strongest relationship. There are three measures where the interaction is significantly negative, absolute clustering (ACL), distance–decay isolation (DPxx) and isolation (Xpx). A negative slope tells us that the overall effect is not as great as the two effects independently; for these measures the effect of segregation needs to be adjusted downward. That is, those block groups with a relatively high African-American population, in metro areas with these three measures, did not show an added risk for exposure, rather their combined effect moderated the health risk from exposure downward.

The median income of a metro area rose as the average pollution exposure increased. Although this relationship was not always statistically significant, the positive association is consistent across models. In addition, when the median income was significant, the segregation index lost explanatory power in a few cases. This led to the question of whether there was a strong correlation between a M/PMSA's median income and segregation. This did not seem to be the case, as the bivariate correlation between all segregation measures and median income ranged from 0.001, for ACE, to 0.027, for RCL.

Discussion

Health inequality scholars have called for a more thoughtful use of segregation indices, with an eye to understanding the mechanisms that link segregation to health risks (Kramer and Hogue 2009). The results presented here help shed light on one such mechanism–exposure to industrial air toxins. With two exceptions, all measures of black/white segregation in metro areas were associated with significantly higher health risk from air toxins. This means that everyone living in more segregated communities during this time were exposed to a greater health risk from air toxins than those living in less segregated communities. The strength of this relationship was relatively stable and robust to sensitivity analyses. These results are consistent with Morello-Frosch and Jesdale (2006), who showed that the segregation of a metropolitan area was associated with an increase in the health risk from air toxins for all racial groups. However, these results dispute more recent conclusions that suggest those metropolitan areas with higher levels of racial segregation of African-Americans are no more unequal in their exposure to air toxins than areas with lower segregation (Downey et al. 2008).

The two segregation measures that were shown to not be significant were absolute centralization (ACE), a measure of the centralization dimension, and delta (DEL), a measure of the concentration dimension. These two measures are unique in that they do not compare the African-American population distribution directly with the white population but rather consider only the arrangement of African-

Americans. The first of these measures, absolute centralization (ACE), reflects the proportion of African-Americans living in a metro area that would have to move to achieve uniform distribution around the central city, i.e., population center. The second non-significant measure is delta. This measure of the concentration dimension reflects the proportion of African-Americans in a metro area that are living in block groups with above average density of African-Americans. These results suggest measures that only consider the patterning of the African-American population are less predictive of exposure to air toxins than those that consider this distribution relative to the distribution of the white population. Therefore, to understand health inequality that might be arising from differential exposure to air pollution scholars need to examine white space relative to black space rather than just looking at the distribution of the African-American population alone.

The measures that made up the evenness and exposure dimensions were consistently, and strongly, related to greater health risk from air toxins. The evenness measure reflects how evenly distributed African-Americans are compared to white residents in a metro area and the exposure measure represents the likelihood an African-American resident of a metro area will come into contact with a white resident. This means that in metro areas where African-American residences have a patchy spatial patterning; they, and the whole metro area, have a greater level of health risk from air toxins. This would make sense if industrial uses are clustered wherever African-Americans are located thus supporting the idea that areas that are predominately African-American are more likely to be zoned for industrial uses (Maantay 2001; Saha and Mohai 2005). This paper measures segregation within metropolitan areas. These areas have a high degree of social and economic integration and have previously been considered a reasonable proxy for both zoning policies and housing markets (Acevedo-Garcia and Osypuk 2008; Stretesky and Hogan 1998; Maantay 2001). However, this geographic delineation is still just a proxy and therefore limited in its ability to explain whether these patterns are largely explained by zoning. Zoning is locally run by cities or townships or counties, thus zoning policies vary widely across the United States. The decentralization of zoning regulations has hampered national level research in this area (Pendall et al. 2006). To test the importance of zoning in the relationship between segregation, health and industrial air pollution requires capturing the varied geographic contexts in which in which zoning decisions are made across the United States. How to match this theoretical line of reasoning with practical measures is an important discussion that needs to be addressed in future studies of this kind. These findings support the case that further research in this area needs to incorporate the historical processes that motivate and maintain firms' incentives to locate and operate in certain locations within metro areas.

The cross-level interaction between the relative proportion of African-Americans in a block group and the segregation of the metro area (SEGINT) was generally positively correlated with pollution exposure. This finding supports the idea that there is greater inequality in exposure to air toxins in more segregated metro areas. That is, African-Americans living in more segregated metro areas experience an added health risk from air toxins. However, when spatial correlation is controlled for by using clustered standard errors, the relative proportion of African-Americans

in a block group loses significance for evenness measures primarily, which impacts the significance of the interaction effect. Although research has shown that health outcomes using spatial measures of segregation are no different when compared to standard measures of segregation (Kramer et al. 2010), these results suggests that if future studies of health risk from air pollution, especially those utilizing measures of evenness, spatial models should be considered.

This study has several limitations, which future work should address. For example, while this paper focuses on residential exposure to air toxins, the majority of the employed populations in the United States spend at least a quarter of their time every week away from their residence (Bureau of Labor Statistics 2015). Researchers who have studied this have shown that exposure away from homes complicates the picture of what environments people are exposed to (Kwan 2013; Inagami et al. 2007). For example, Ellis et al. (2004) compare the segregation of the census tract in which Los Angeles residents' work to the segregation of the tracts they in which they live. The authors find that, on average, whites work in tracts that are 60 % less segregated than where they live and African-Americans are 33 % less segregated at work. The emphasis on residential exposure of this study, therefore, does not reflect the complete picture of exposure. To overcome this limitation, future work should consider use of travel diaries and other creative measures of the daily geography of individuals' lives (Wong and Shaw 2011; Wang et al. 2012).

Future research should also examine other sources of air toxins, such as mobile sources, as well as other racial and ethnic groups, such as Hispanic/Latinos, to determine whether the relationships presented in this paper are maintained. Incorporation of several racial/ethnic minorities in the same analysis is often problematic because the theory that explains the racial geography of African-Americans, for example, is different than the theory that explains the very heterogeneous group that makes up the Hispanic demographic. Nonetheless, a focus on the clustering of non-Hispanic whites, rather than solely focusing on African-Americans, would give insight into whether the weaker correlations for the interaction variable might be in part because predominantly non-white block groups are worse off in more segregated metros, regardless of whether these block groups are predominantly African-American or just predominantly non-white.

One of the interesting findings of this study is the positive relationship between the median income of a M/PMSA and its health risk from pollution exposure. It is widely believed that industry brings in tax dollars and income for the metro areas where plants and factories are located, which incentivizes counties and metro areas to encourage polluting industries to site there (Bartik 2005). However, it is logical that individuals who are economically benefiting from such business dealings would be most inclined to support these efforts if they were able to separate their own residences from the health risk from these ventures. Future research should investigate how the perception of neighborhoods influences the siting of industrial facilities (King 2014) and where the economic benefits of industry are concentrated.

These results reinforce the importance of determining a priori which conception of segregation best captures the health risk under examination. Previous studies examining how health outcomes vary by segregation have shown that results differ by the dimension of segregation used. For example, in their analysis of birth

outcomes for African-Americans, Bell et al. (2006) found that while an individual's metro area's level of isolation was associated with lower birthweights, clustering was related to more positive health outcomes. However, Yi et al. (2014) found that after adjustment for individual-level covariates, the isolation index was not associated with biomarkers of nutritional health. The results presented here show that with few exceptions, metro areas with greater segregation also have a greater health risk from air toxins for all residents. Areas in a metro area that have a relatively higher proportion of African-American residents were also shown to be at greater risk, although this measure was sensitive to spatial clustering when using measures of the evenness dimension.

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