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LETTER

# Air pollution is linked to higher cancer rates among black or impoverished communities in Louisiana

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## Abstract

Despite longstanding concerns about environmental injustice in Louisiana's industrialized communities, including the area known as Cancer Alley, there is a lack of environmental health research in this state. This research gap has direct consequences for residents of industrialized neighborhoods because state regulators have cited a lack of evidence for adverse health outcomes when making industrial permitting decisions. We investigated how cancer incidence relates to cancer risk from toxic air pollution, race, poverty, and occupation across Louisiana census tracts, while controlling for parish-level smoking and obesity rates, using linear regression and Akaike information criterion model selection. We used the most recent cancer data from the Louisiana Tumor Registry (2008–2017), estimates of race, poverty, and occupation from the US Census Bureau's American Community Survey (2011–2015), and estimated cancer risk due to point sources from the US Environmental Protection Agency's 2005 National Air Toxics Assessment (accounting for cancer latency). Because race and poverty were strongly correlated ($r = 0.69$, $P < 0.0001$), we included them in separate, analogous models. Results indicated that higher estimated cancer risk from air toxics was associated with higher cancer incidence through an interaction with poverty or race. Further analysis revealed that the tracts with the highest (i.e. top quartile) proportions of impoverished residents (or Black residents) were driving the association between toxic air pollution and cancer incidence. These findings may be explained by well-established disparities that result in greater exposure/susceptibility to air toxics in Black or impoverished neighborhoods. Regardless, our analysis provides evidence of a statewide link between cancer rates and carcinogenic air pollution in marginalized communities and suggests that toxic air pollution is a contributing factor to Louisiana's cancer burden. These findings are consistent with the firsthand knowledge of Louisiana residents from predominantly Black, impoverished, and industrialized neighborhoods who have long maintained that their communities are overburdened with cancer.

## 1. Introduction

Residents of Cancer Alley and other industrialized areas of Louisiana have long maintained that they are disproportionately impacted by cancer and other health problems from chronic exposure to industrial pollution (e.g. [1–4]). Cancer Alley has been defined as the ~130 mile, winding corridor along the Mississippi River between Baton Rouge and New Orleans [4], where, according to the state emissions inventory, more than 200 industrial facilities release significant amounts (i.e. >5 tons per year) of harmful air pollution [5]. Notably, this definition of Cancer Alley (alternatively labeled the Industrial Corridor) does not capture some of Louisiana's most heavily industrialized communities (e.g. Mossville [3]). In absolute terms, more pounds of industrial toxic air pollution are released annually in Louisiana than in any

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other state, based on 2019 data from the US Environmental Protection Agency (EPA) [6]. These emissions translate to a disproportionate burden of health risks for Louisiana's industrialized neighborhoods. Nearly every census tract between Baton Rouge and New Orleans ranks in the top 5% nationally for cancer risk from toxic air pollution and in the top 10% for respiratory hazards [7]. Concerns about Louisiana's pollution burden have been dismissed by state regulators and politicians, who maintain that there is no evidence of adverse health outcomes (e.g. [8]), or that lifestyle choices play a larger role in the state's cancer burden (e.g. [9]). Such perspectives fail to consider the lack of pollution-related research in Louisiana, or the complex and interactive pathways through which discrimination and inequities influence behaviors, toxic exposures, and health outcomes [10].

While cancer risk from air toxics is uniformly high across Louisiana's Industrial Corridor by national rankings, this burden is unevenly distributed among neighborhoods. Recent estimates of pollution-related cancer risk for census tracts from Baton Rouge to New Orleans range from 24.8 per million (Tract 279.02, Jefferson Parish) to 1505.1 per million (Tract 708, St John the Baptist Parish), with Black and impoverished tracts being disproportionately impacted [11–13]. (Louisiana parishes are equivalent to US counties.) This disparity is part of a larger pattern of inequities in pollution exposure across the United States [14–21] and globally (reviewed in [22]). In Louisiana, industrial facilities are often located on former plantation sites, where the adjacent neighborhoods are predominantly Black (i.e. African-American) and often include descendants of the emancipated settlers who founded the community [23]. The pollution risks faced by these and other Black communities are not simply products of their lifestyles; Black Americans are exposed to an estimated 56% more fine particulate ($PM_{2.5}$) pollution compared to the amount that would be generated by their consumption of goods and services, while White Americans benefit from 17% less exposure relative to their consumption [24]. Compounding this racial disparity, Black Americans do not receive a proportionate share of the economic benefits from industrial polluters, in terms of employment opportunities [17].

In Louisiana and across the United States, the most racially segregated neighborhoods tend to experience the highest cancer risks from air toxics [13, 16]. Racial residential segregation is considered to play a major role in health disparities, including those related to air toxics [25, 26]. This form of segregation is caused by structural mechanisms of discrimination that result in political, economic, legal, and social disparities [26, 27]. In turn, these disparities result in a complex network of factors in the built environment, social environment, and individual situation that can increase exposure or susceptibility to pollution [26]. For example, the lack of local grocery stores in the built environment and food insecurity in the social environment can contribute to poor nutrition in communities of color, increasing susceptibility to diabetes or heart disease [26]. These diseases can be triggered or worsened by chronic exposure to particulate matter pollution [28], which is consistently higher in communities of color across the United States [19, 29]. Existing disparities in segregated communities can be worsened by industrialization, for example, when grocery stores or recreational centers are closed to make way for petrochemical plants. In Louisiana, the dismantling of built and social environments for industrial development has led to the relocation of entire communities (e.g. Diamond, LA), while members of other communities (e.g. Mossville, LA) still seek equitable relocation [30]. Relocation can (theoretically) reduce or circumvent toxic exposures and certain other inequities in segregated communities; however, buyout programs present their own challenges with respect to distributive, procedural, and interactional injustice [31].

From a public health perspective, regulatory decisions related to air quality should be informed by information about emissions, exposures, risks, and corresponding health outcomes. The need for such information in Louisiana is particularly acute, where hundreds of new pollution sources are permitted each year [32] and where there is minimal data about health outcomes associated with pollution exposure. Even when faced with strong evidence of pollution disparities, Louisiana's political leaders and decision-makers may require evidence of disparate health *outcomes* before taking corrective action [33]. The few studies of health outcomes from residential exposure to environmental pollution in Louisiana have generally reported non-significant findings (but see [34]). However, these studies were statistically underpowered [35, 36] or lacked any quantitative measure of pollution exposure [37]. Despite being cited as evidence against pollution-related cancer disparities [8, 38], the annual reports from Louisiana's cancer registry do not quantify pollution exposure or control for confounding variables in their statistical comparisons of 'Industrial Corridor' cancer rates (e.g. [39–41]). To address the lack of empirical research about pollution-related health outcomes in Louisiana, we evaluated cancer incidence among Louisiana census tracts relative to estimated cancer risk from air toxics, while accounting for race, poverty, and certain health and occupational factors using publicly available data from state and federal agencies. Our goals were to elucidate the drivers of cancer rates in Louisiana and to determine whether the firsthand experiences of industrialized communities, which indicate a disproportionate burden of cancer, are evident in Tumor Registry data.



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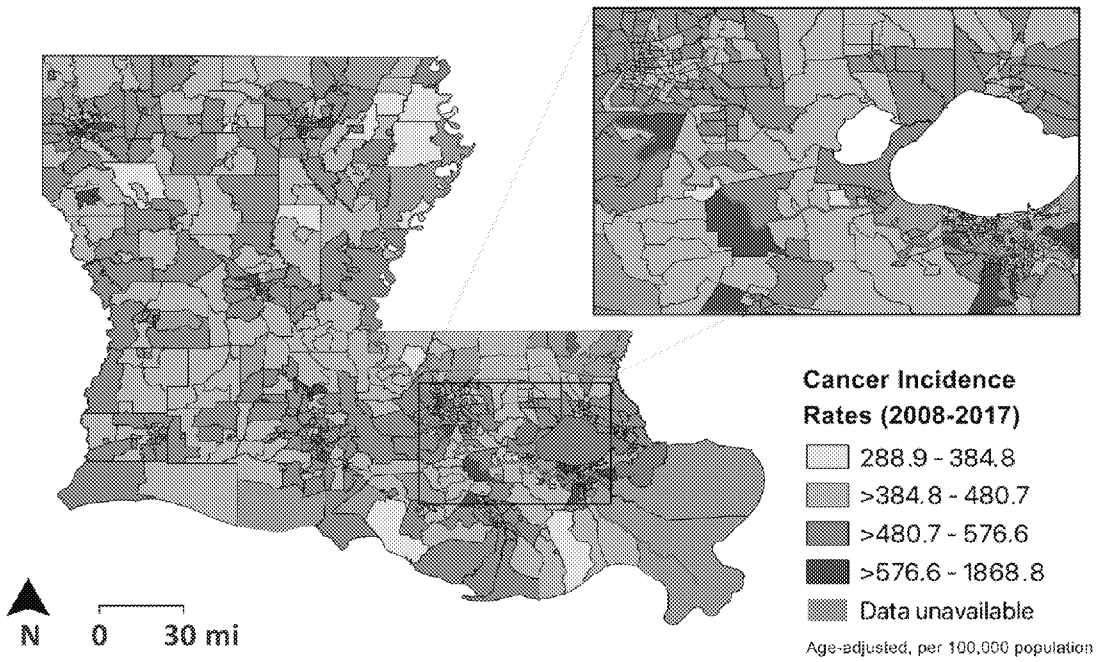


**Figure 1.** Age-adjusted annual cancer incidence rates, averaged from 2008 to 2017, as reported by the LTR. Data breaks correspond to quartiles. Inset depicts the Industrial Corridor from Baton Rouge to New Orleans. The Mississippi River and the Pontchartrain Lake System are shown in blue.

## 2. Methods

### 2.1. Cancer incidence rates

We used 10 year average annual cancer rates for all malignant tumors combined from the Louisiana Tumor Registry (LTR)'s most recent annual report, reflecting cases diagnosed from 2008 to 2017 [42]. We did not examine individual cancer types because these data are not published for most census tracts in Louisiana due to the relatively small numbers of cases [42]. Even for the most prevalent cancers (i.e. lung and breast), the low case counts (typically 2–5 cases per year) result in unreliable cancer rates, i.e. with extremely wide confidence intervals. Thus, our analysis is limited to overall cancer incidence, which is directly comparable to estimated cancer risk from air toxics. Notably, specific cancers that are excluded from reporting are still included in the overall cancer rate, which is available for 932 of 1148 census tracts in Louisiana (figure 1). These rates are age adjusted by the LTR and presented per 100 000 population. For simplicity, we subsequently refer to age-adjusted cancer incidence rates as 'cancer incidence'.

### 2.2. Pollution-related cancer risk

We used estimates of pollution-related cancer risk from the EPA's 2005 National Air Toxics Assessment (NATA), which reflect toxicity-weighted pollution levels in 2005 (figure 2). Because EPA improves its NATA methodology continually, the 2005 NATA provided more a refined approach compared to the previous NATAs (1996, 1999, and 2002), while still allowing a reasonable time gap relative to the 2008–2017 cancer rate dataset to account for cancer latency [43]. Additionally, in selecting the dataset, we considered that changes in census tract boundaries occur during each decennial census (e.g. 1990, 2000, and 2010). To account for these changes, we excluded significantly-changed census tracts from our analysis, as described below.

We used NATA's estimates of Point Source Cancer Risk because Louisiana's industrialized communities are characterized by a high density of point sources. These represent stationary sources for which locations are known, including industrial plants, electric utilities, and large waste incinerators [44]. In the 2005 NATA, Point Sources did not include airports, homes, wildfires, vehicles, or other mobile or diffuse sources of pollution [44]. For simplicity, we subsequently refer to 2005 NATA Point Source Cancer Risk as 'estimated cancer risk from air toxics' or, where a more concise descriptor is helpful, 'air toxics'.

### 2.3. Demographic and health indicators

Our analysis included 5 year estimates of race, poverty, and occupation at the census tract level from the US Census Bureau's 2015 American Community Survey (ACS; representing data collected from 2011 to 2015; figure 3). These estimates included percentages of Black or African-American residents (alone or in combination with another race), percentages of residents living below the federal poverty threshold,



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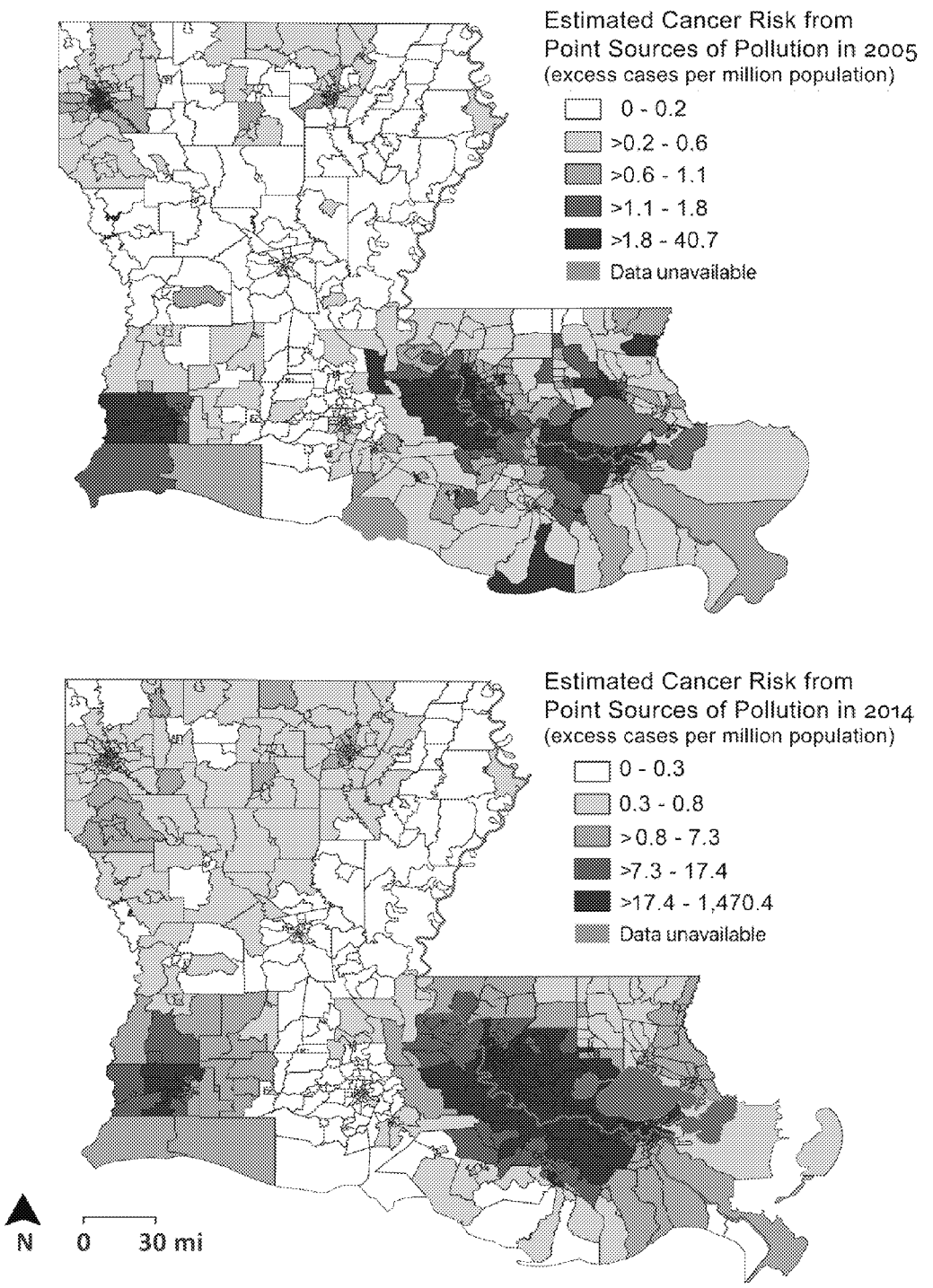


**Figure 2.** Cancer risk from point sources of pollution, as reported in the EPA's 2005 (top) and 2014 (bottom) NATA. Data breaks correspond to quintiles. Note that the methodology differed between these two assessments, so the resulting data are not directly comparable. The Mississippi River and the Pontchartrain Lake System are shown in blue.

percentages of the workforce employed by the construction industry, and percentages employed by the manufacturing industry. We chose these industries as proxies for occupational exposure to toxic air pollutants because they are the most likely to be consistently associated with air quality hazards.

Because smoking and obesity data were not available at the census tract level, our analysis included parish-level smoking and obesity data from the 2011 Louisiana County Health Rankings (figure 4) [45]. The 2011 County Health Rankings use 2003–2009 smoking data from the US Centers for Disease Control (CDC)'s Behavioral Risk Factor Surveillance System and 2008 obesity data from the CDC's National Center for Chronic Disease Prevention and Health Protection. By necessity, our analysis used the same average parish value for all census tracts within that parish.



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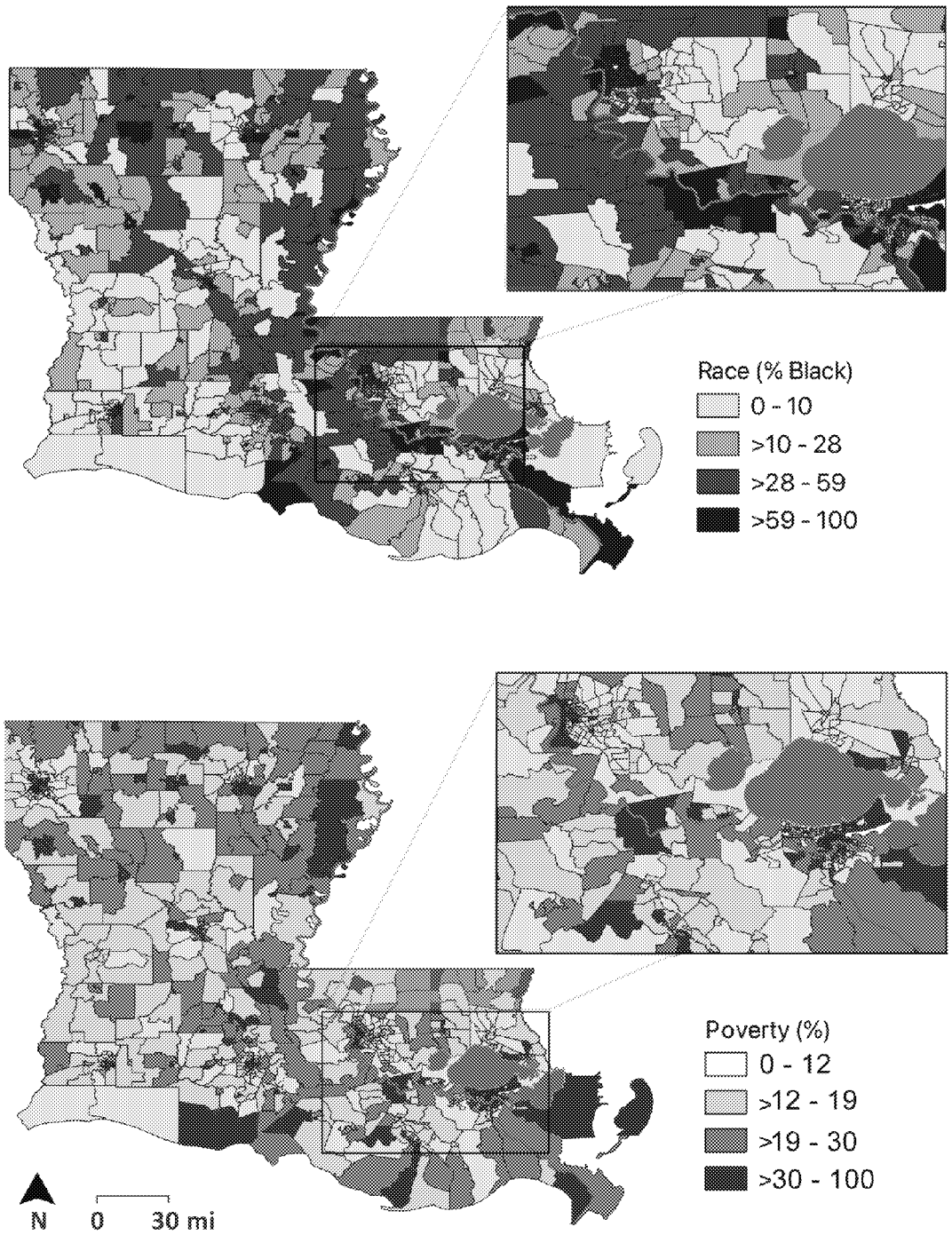


**Figure 3.** (Top) Percentage of Black residents (i.e. African American, one or more races). (Bottom) Percentage of families living below the US federal poverty threshold. Both datasets are 5 year estimates (2011–2015) from the US Census Bureau's ACS. Data breaks correspond to quartiles. The Mississippi River and the Pontchartrain Lake System are shown in blue.

### 2.4. Mapping

We mapped each dataset by percentile using QGIS Version 3.18 to visualize the geographic patterns of cancer (figure 1), toxic air pollution (figure 2), race and poverty (figure 3), and smoking and obesity (figure 4). Because our analysis relies on historical pollution values, but current pollution values are relevant from a health policy perspective, we included Point Source Cancer Risk from the most recent (2014) NATA (figure 2). Importantly, the results of different NATAs are not directly comparable due to methodological changes over time [46]. We did not use the 2014 NATA data in any statistical analysis; rather, we mapped the data for visualization only.

### 2.5. Data exclusions

Our statistical analyses excluded census tracts for which cancer rates were not available from the LTR ($n$ = 216 out of 1148 total). Additionally, we



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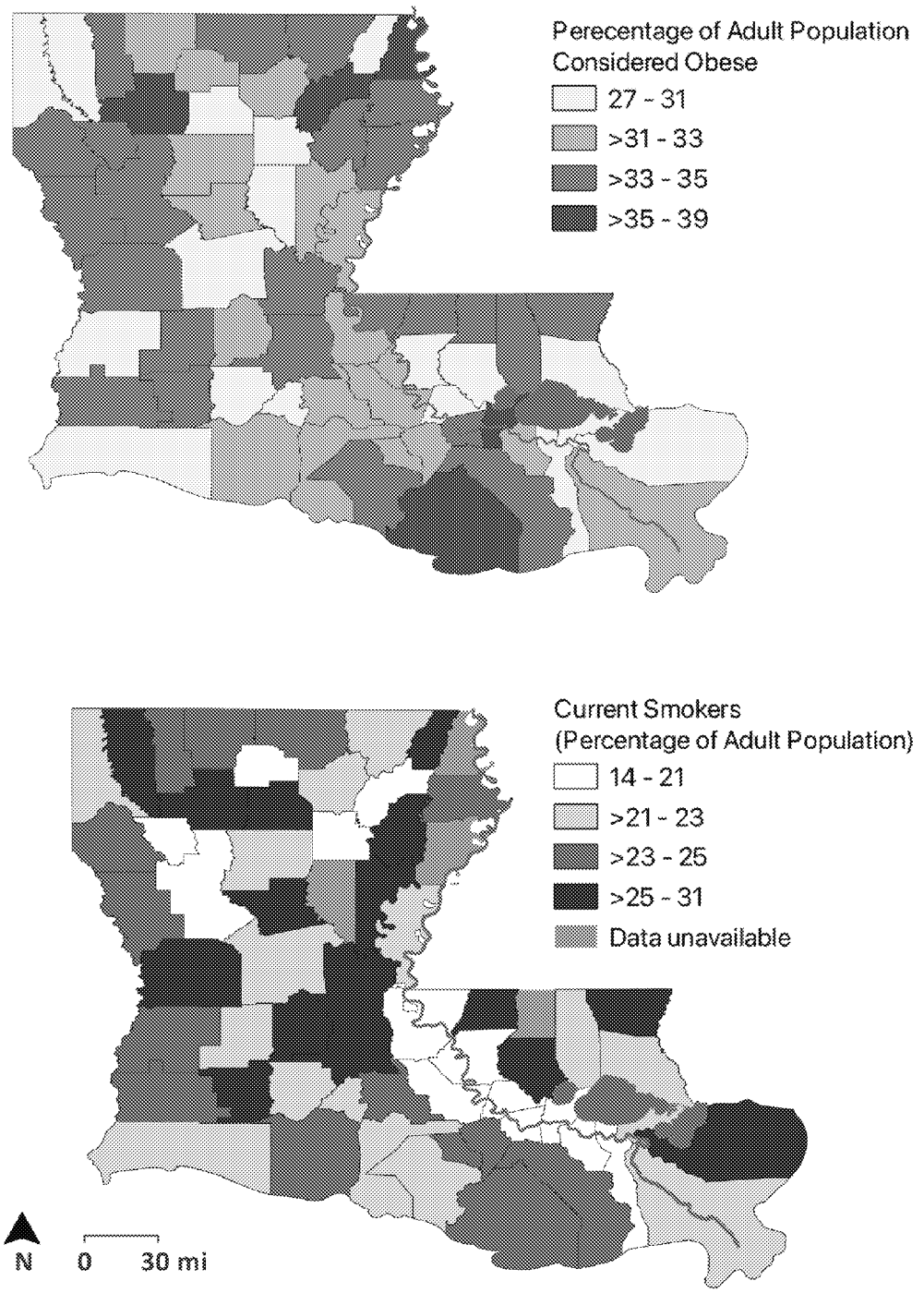


**Figure 4.** (Top) Percentage of adult population that is considered obese. (Bottom) Percentage of adult population that currently smoke tobacco products. Both datasets are from the 2011 county health rankings, which use 2003–2009 smoking data and 2008 obesity data. Data breaks correspond to quartiles. The Mississippi River and the Pontchartrain Lake System are shown in blue.

excluded tracts that the Tumor Registry designated as containing military bases ($n = 27$), because military personnel are likely to have different exposure histories compared to permanent residents. We also excluded census tracts ($n = 155$) with geographic boundaries that had changed substantially between the 2000 census and 2010 census, as identified by the US Census Bureau [47]. This exclusion was necessary because we used a pollution (i.e. estimated cancer risk) dataset that was based on the 2000 census and a cancer incidence dataset that was based on the 2010 census. After these exclusions, there were 750 census tracts remaining in the final dataset. Estimates of cancer risk from EPA's 2005 NATA were available for all of these tracts.

### 2.6. Statistical analysis

We performed all analyses in R Statistical Software [48]. With the exception of cancer rates, all variables in our datasets were non-normally distributed and were natural-log transformed (estimated cancer risk from air toxics) or arcsine transformed (race, poverty,



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and occupation) for analysis. Transformed data were mean centered [49].

Exploratory analysis revealed significant spatial autocorrelation ($p < 0.001$) in a model of cancer incidence rates with poverty, estimated cancer risk from air toxics, and the interaction of these terms, as tested by a simulation with 10 000 replicates and measured by Moran's i [50]. In order to reduce spatial autocorrelation, we include a fixed effect in the linear model for parish. This results in a model with no statistically significant spatial autocorrelation ($p = 0.40$), and therefore may have appropriate standard errors for coefficient estimates. In addition to controlling spatial autocorrelation, the addition of parish dummy variables in the model automatically controls for the combined effects of parish-level confounders, which are not of interest to this study.

We evaluated the performance of alternate linear models for predicting census tract-level cancer incidence rates using the step Akaike information criterion (AIC) function in the MASS package of R Statistical Software [51]. This function performs stepwise AIC [52] model selection through an iterative process that adds and removes variables sequentially to identify the best fit model. We chose AIC for model selection because it is a widely-used approach that performs well under a broad range of modeling scenarios, including for spatial data [53].

As described in the results, race and poverty were strongly correlated across our dataset. To avoid collinearity, we conducted parallel analyses with models that included poverty or, alternately, race. Our initial model of cancer incidence included the direct effects of air toxics, poverty, parish, construction employment, manufacturing employment, smoking, and obesity, as well as the interactive effect of air toxics and poverty. We used an analogous model to evaluate race (proportion of Black residents) in place of poverty. After identifying the top model from each analysis through stepwise selection, we used the base package of R to calculate model statistics. To determine the overall significance of air toxics in the top model, we used an $F$-test to compare versions of the same model with and without this risk.

To explore the interaction that was detected between poverty and air toxics, we calculated median cancer incidence rates for census tracts that were disproportionately impoverished versus disproportionately affluent (i.e. above or below median poverty rates, respectively). We further divided the impoverished group into air toxics quartiles, based on the full range of air toxics values represented in our dataset ($n = 750$ census tracts). We then calculated cancer incidence rates for each of the following air toxics groups: lowest quartile, below median, above median, highest quartile. We used $t$-tests to compare median cancer incidence of each of the above groups to the overall median cancer incidence for the entire dataset ($n = 750$ tracts). We generated quantitative estimates of the cancer burden from severe pollution by calculating the difference in mean cancer incidence rate for the most polluted versus least polluted census tracts (i.e. top versus bottom air toxics quartiles among tracts with above-median poverty), or, alternately, the top quartile versus the overall mean ($n = 750$ tracts). We then multiplied the result by the population represented by the corresponding data subset, to derive the total number of estimated excess cancer cases per year. Additionally, we divided the overall dataset by poverty quartile and used Pearson's correlation to test the relationship between air toxics and cancer risk for each data subset. We created scatterplots of the raw (i.e. untransformed) data to visualize these comparisons for the top and bottom quartiles by poverty. These plots included linear regression lines with 95% confidence intervals, calculated using the geom_line function in ggplot2 in R Statistical Software. We conducted parallel analyses using race instead of poverty.

## 3. Results

### 3.1. Quality assurance and data exclusions

After all exclusions (see section 2), data distributions were generally unchanged (table 1). One notable exception was maximum poverty rate, which was lower in our final dataset due to the exclusion of two census tracts in New Orleans with exceptionally high poverty rates (Tracts 44.02 and 48, with 87% and 100% poverty, respectively). Both tracts were geographically tiny ($\leqslant 0.16$ mi$^2$), contained fewer than 500 people, and were excluded because their boundaries changed substantially between 2000 and 2010. The other notable exception was maximum cancer rate, which was lower in our final dataset due to the exclusion of three outlying census tracts (Tracts 9507.02 and 9507.04 in Vernon Parish, Tract 109 in Bossier Parish) that contained military bases and had exceptionally high cancer rates (between 1125 and 1869 cases annually per 100 000 population). Regardless, there was no significant difference in cancer rates between census tracts that were excluded ($n = 182$) or included ($n = 750$) in our analysis ($t = -1.71$, df $= 193.11$, $P = 0.088$). If the three outlying tracts are ignored, there is even less statistical support for a difference in cancer rates between census tracts that were included versus excluded from our analysis ($t = -0.549$, df $= 236.05$, $P = 0.583$). Thus, our final dataset was representative of cancer, pollution, race, poverty, and employment in construction and manufacturing industries in Louisiana.

There were two census tracts in the dataset that were outliers, in terms of exceptionally high pollution (i.e. estimated cancer risk) values: census tract 22017023800 (Cedar Grove neighborhood, Shreveport, Caddo Parish) and census tract



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**Table 1.** Sample sizes and summary statistics for each variable analyzed[a].

| Variable | Dataset | # Census tracts | Minimum | 1st quartile | Median | 3rd quartile | Maximum |
|---|---|---|---|---|---|---|---|
| Cancer incidence | All available | 932 | 288.9 | 443.6 | 481.4 | 514.1 | 1868.8 |
| | Analyzed | 750 | 288.9 | 442.8 | 480.7 | 513.7 | 845.5 |
| Pollution-related | All available | 1105 | 0.001 | 0.25 | 0.97 | 1.47 | 40.70 |
| cancer risk | Analyzed | 750 | 0.001 | 0.22 | 0.91 | 1.57 | 30.90 |
| % Black | All available | 1128 | 0 | 10.8 | 28.7 | 60.2 | 100 |
| | Analyzed | 750 | 0 | 10.6 | 27.6 | 55.3 | 100 |
| % Poverty | All available | 1127 | 0 | 12.1 | 19.5 | 30.2 | 100 |
| | Analyzed | 750 | 0.9 | 11.9 | 18.3 | 27.9 | 62.0 |
| % Employed in | All available | 1126 | 0 | 4.3 | 7.1 | 10.5 | 29.1 |
| construction | Analyzed | 750 | 0.3 | 4.7 | 7.5 | 10.8 | 24.3 |
| % Employed in | All available | 1126 | 0 | 3.9 | 6.7 | 10.2 | 28.5 |
| manufacturing | Analyzed | 750 | 0 | 4.4 | 7.4 | 10.9 | 28.5 |
| % Current smokers | All available data | NA[b] | 14.0 | 21.0 | 23.0 | 24.0 | 31.0 |
| % Obese | were analyzed | NA[b] | 27.0 | 30.0 | 31.0 | 34.0 | 39.0 |

[a] See section 2 for data sources.

[b] Parish-level data used ($n = 64$ parishes); census-tract-level data unavailable.

22089062500 (Norco, St Charles Parish). As a conservative approach, we retained these outliers in our main statistical analysis (figure 5), but excluded them in our quartile analysis for poverty (figure 6) and race (figure A1) quartile.

### 3.2. Relationships among untransformed predictor variables

Among census tracts ($n = 750$), race (% Black) and poverty were strongly correlated ($r = 0.69$, $P < 0.0001$; figure 3). Tracts with higher proportions of Black residents generally had lower percentages of the workforce represented in the construction ($r = -0.23$, $P < 0.0001$) or manufacturing industries ($r = -0.11$, $P = 0.003$). Similarly, tracts with higher poverty rates generally had lower percentages of the workforce represented in the construction industry ($r = -0.11$, $P = 0.003$) or manufacturing industries ($r = -0.08$, $P = 0.024$). Among all census tracts, estimated cancer risk from air toxics was not significantly related to poverty rate ($r = 0.05$, $P = 0.16$), percentages of Black residents ($r = 0.04$, $P = 0.24$), or percentages of the workforce in construction ($r = 0.06$, $P = 0.12$). However, this risk was positively correlated with the percentage of the workforce in manufacturing ($r = 0.07$, $P = 0.041$). Among Louisiana parishes, smoking and obesity rates were positively correlated ($r = 0.30$, $P < 0.0001$).

### 3.3. Relationship between cancer risk from air toxics and cancer incidence

As expected, cancer incidence varied throughout the state (figure 1). Estimated cancer risk from air toxics (i.e. 2005 NATA Cancer Risk from point sources) was elevated in southwest Louisiana and in the area known as Cancer Alley in southeast Louisiana (figure 2). The top model from AIC selection included the direct effects of parish, poverty, and cancer risk from air toxics, as well as the interaction between poverty and cancer risk from air toxics (table 2).

Model statistics revealed that cancer risk from air toxics was associated with cancer incidence through an interaction with poverty, as opposed to a direct effect (table 3). Results of race models were analogous to results of poverty models (tables A1 and A2). As further evidence of an overall effect of cancer risk from air toxics, a significant difference was observed after removing pollution from the poverty model ($F = 4.46$, $P = 0.012$) and from the race model ($F = 4.06$, $P = 0.018$). A scatterplot of the overall dataset confirmed that among predominantly Black and/or impoverished census tracts, those with more toxic air generally have higher cancer rates (figure 5).

### 3.4. Further analysis of interaction effects

As described in the methods, we conducted additional analyses to better understand the observed interaction of air toxics and poverty or air toxics and race. Among the most impoverished census tracts (i.e. fourth quartile), higher estimated cancer risk from air toxics was correlated with higher cancer incidence ($r = 0.25$, $P = 0.0005$). This relationship was not observed among less impoverished census tracts (third quartile: $r = -0.01$, $P = 0.85$; second quartile: $r = 0.03$, $P = 0.72$; first quartile: $-0.11$, $P = 0.13$).

A similar pattern was observed when the dataset was broken down by Race (fourth quartile: $r = 0.13$, $P = 0.07$; third quartile: $r = 0.03$, $P = 0.71$; second quartile: $r = 0.01$, $P = 0.87$; first quartile: $r = -0.10$, $P = 0.16$). Census tracts that were disproportionately Black or impoverished had higher cancer incidence rates compared to the entire dataset ($P \leqslant 0.016$; table 4). Within each of these disproportionate groups, cancer incidence was elevated among



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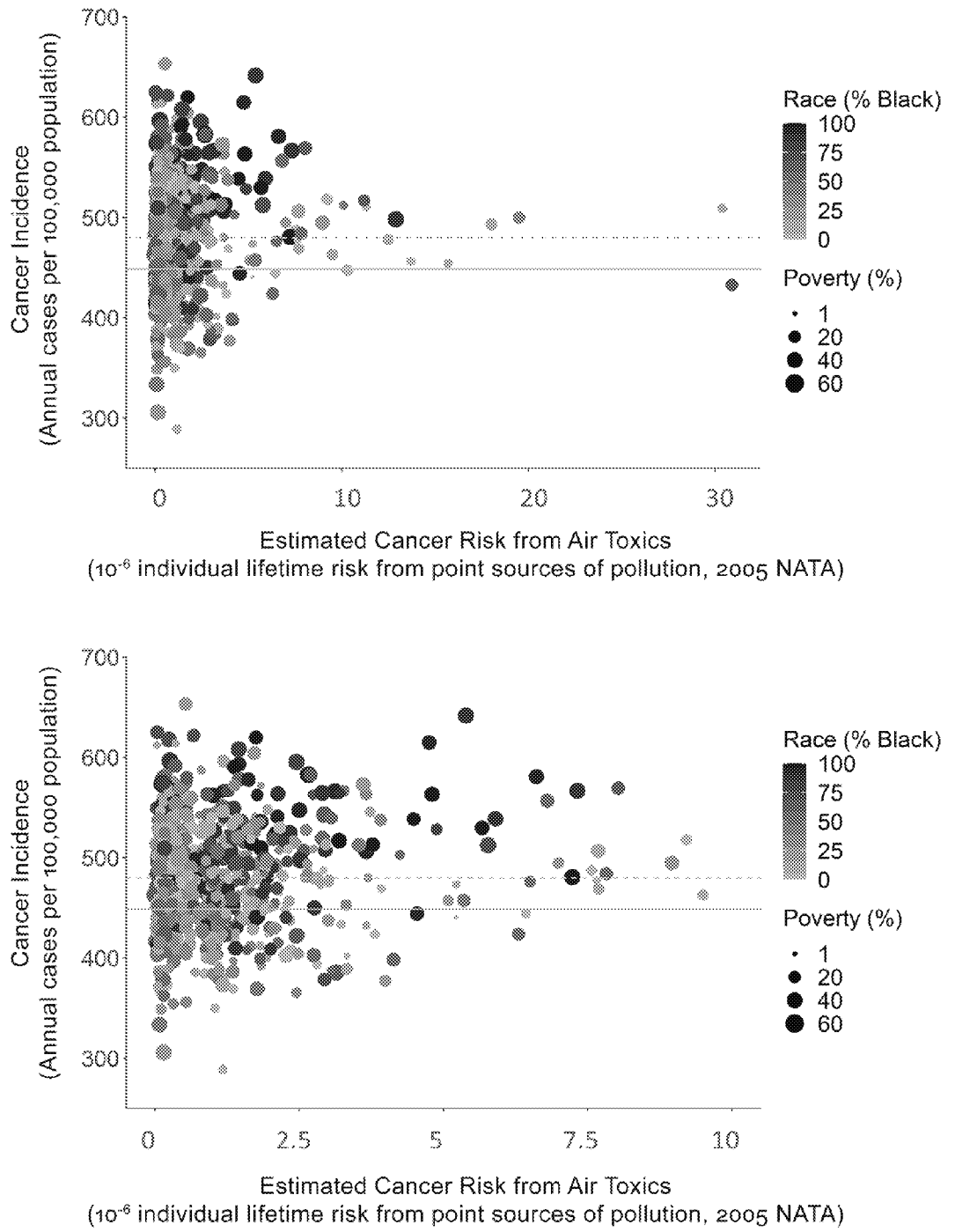


**Figure 5.** Relationships among toxic air pollution, cancer rates, poverty, and race for Louisiana census tracts ($n = 750$). The same dataset is presented in both panels, with a smaller scale on the bottom panel to better distinguish data points. All values (including poverty) are plotted on continuous scales. Solid gray line indicates US average cancer rate (448.7); dashed gray line indicates the Louisiana statewide average (481.0).

tracts with higher estimated cancer risk from air toxics ($P \leqslant 0.016$; table 4), but not among tracts with lower estimated cancer risk from air toxics (table 4; figures 6 and A1).

The (conservatively) estimated cancer burden from severe air pollution was 85 cases in Louisiana annually (among tracts with above-median poverty). More specifically, this value was 85.8 cases for the top-quartile-air-toxics versus global-average comparison ([501.7–480.3] × [400 788/100 000]), or 91.8 cases for top versus bottom-air-toxics-quartiles comparison ([501.7–478.8] × [400 788/100 000]).

## 4. Discussion

To our knowledge, this is the first statewide study of cancer incidence in Louisiana that accounts for interactions between air pollution and poverty or race. We found that higher estimated cancer risk from toxic air pollution was linked to higher cancer incidence



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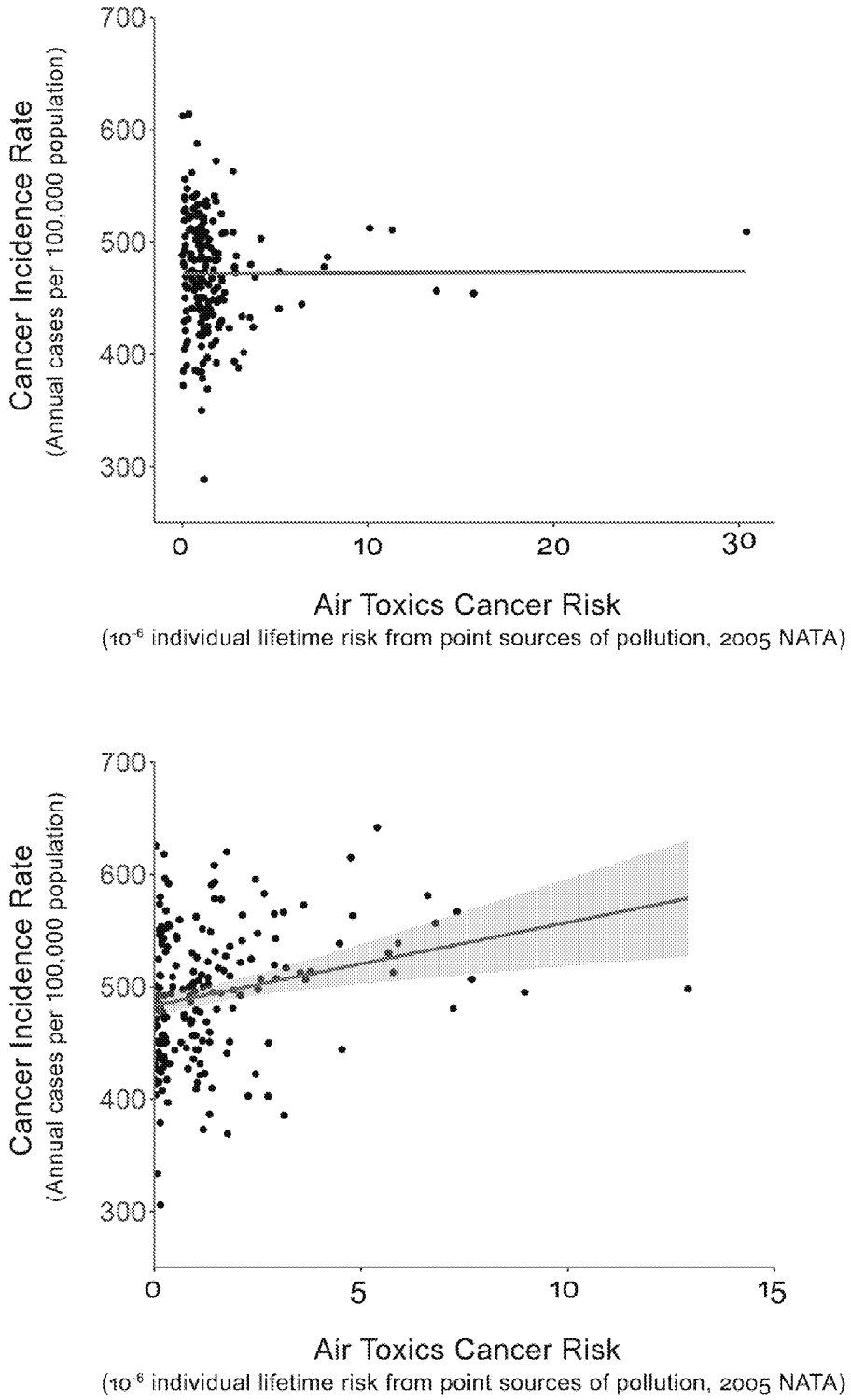


**Figure 6.** Comparison of pollution versus cancer rates for the most affluent census tracts (top panel; $r = -0.11$, $P = 0.13$) and most impoverished (bottom panel; $r = -0.25$, $P = 0.0005$) census tracts with two outliers removed (see section 3). See table 4 for quartile breaks and test statistics. Blue line represents a linear regression of untransformed data. Gray shading represents 95% confidence intervals.

among Louisiana's most impoverished neighborhoods. Because poverty and race were strongly correlated in our dataset (reflecting the disproportionate burden of poverty in Black communities), we could not reliably distinguish between the two factors in our statistical analyses. Regardless, the same patterns emerged for Black or impoverished neighborhood: higher levels of toxic air pollution were associated with higher cancer incidence.

There are multiple potential explanations for our finding that the link between air toxics and cancer incidence was observed in the most impoverished/Black neighborhoods, but not among more affluent/White neighborhoods. This finding



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**Table 2.** Best-supported models of tract-level cancer incidence from AIC stepwise model selection.

| Main effects[a] (all models) | Interaction terms | Other main effects | AIC | ΔAIC |
|---|---|---|---|---|
| Air toxics | Air toxics × poverty | None | 5867.8 | 0 |
| Poverty | | Employed in manufacturing | 5868.2 | 0.4 |
| Parish | | + Employed in construction | 5869.9 | 2.1 |
| | | + Smoking | 5869.9 | 2.1 |
| | | + Obesity | 5869.9 | 2.1 |

[a] Air toxics corresponds 2005 NATA Cancer Risk from point sources. See section 2 for other data sources.

**Table 3.** Statistics for best-supported cancer incidence model.

| Variable | Coefficient estimate[a] | $t$ | $P$ |
|---|---|---|---|
| (Model intercept) | 485.0 | 32.48 | < 0.0001 |
| Air toxics | −0.72 | −0.26 | 0.79 |
| Poverty | 73.07 | 5.44 | < 0.0001 |
| Air toxics × poverty | 29.14 | 2.98 | 0.003 |

[a] Coefficients correspond to transformed and mean-centered data (see section 2).

**Table 4.** Mean cancer incidence among different data subsets and results of $t$-test comparisons[a].

| | | Cancer incidence | | Versus all-data mean (480.3) | |
|---|---|---|---|---|---|
| Subset of census tracts | Population | Mean | 95% confidence interval | $t$ | $P$ |
| Disproportionately affluent[b] | 1895 934 | 471.8 | 466.8–476.7 | −3.39 | 0.0008 |
| Disproportionately poor[b] | 1542 402 | 488.8 | 483.2–494.5 | 2.97 | 0.003 |
| Bottom-quartile pollution | 461 887 | 478.8 | 468.8–488.8 | −0.30 | 0.76 |
| Below-median pollution | 876 456 | 485.9 | 478.7–493.2 | 1.53 | 0.13 |
| Above-median pollution | 663 173 | 492.9 | 484.0–501.8 | 2.80 | 0.006 |
| Top-quartile pollution | 400 788 | 501.7 | 489.7–513.7 | 3.55 | 0.0006 |
| Disproportionately white[c] | 1818 620 | 473.4 | 468.4–478.4 | −2.72 | 0.007 |
| Disproportionately black[c] | 1613 336 | 487.2 | 481.6–492.9 | 2.41 | 0.016 |
| Bottom-quartile pollution | 435 430 | 480.3 | 470.0–491.3 | 0.07 | 0.95 |
| Below-median pollution | 802 224 | 484.0 | 476.1–491.9 | 0.93 | 0.35 |
| Above-median pollution | 811 112 | 490.3 | 482.2–498.4 | 2.44 | 0.016 |
| Top-quartile pollution | 456 349 | 498.9 | 487.6–510.3 | 3.25 | 0.002 |

[a] Incidence is the number of newly-diagnosed cancer cases per 100 000 population and age-adjusted. Pollution categories are based on pollution values from the entire dataset ($n = 750$ census tracts). See section 2 for data sources.
[b] Census tracts with poverty values below (affluent) or above (poor) median poverty rate (18.25%).
[c] Census tracts with above-median values for corresponding race (Black, 27.6%; White, 67.7%).

may reflect the well-documented disparities in health risk factors and medical care that leave Black and impoverished communities more vulnerable to negative health outcomes [54–56]. For example, these communities are more likely to delay or forgo preventative medical visits due to the high cost of healthcare [57]. Empirical research indicates that physicians serving Black communities are often less effective at cancer education and cancer screening compared to physicians serving White communities [58, 59]. Ultimately, Black communities in Louisiana, like elsewhere in the US, are faced with a 'double disparity', in which they are overburdened by environmental pollution and medically underserved [60]. As an additional or alternative explanation, our dataset may have been inadequate to detect a link between air toxics and cancer incidence among affluent/White communities, perhaps because of their relatively greater geographic mobility [61]. Given the lack of widespread data on residential histories, this factor could contribute to uncontrolled variation in actual exposures and obscure a relationship between estimated exposure and cancer incidence.

While economic disparities were not the focus of our study, we found that Black residents appear to be underrepresented in the construction and manufacturing industries in Louisiana. Specifically, census tracts with higher proportions of Black residents had lower proportions of their workforce represented in the construction and manufacturing industries. Additional research is warranted to understand the full nature of this disparity, but our findings are consistent with previous research documenting that Black Americans are underrepresented in industrial employment, particularly for high-paying jobs [17].



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It is important to recognize the limitations inherent to studying environmental health disparities in Louisiana. Like elsewhere, there is limited information about factors that influence cancer incidence (e.g. drinking water contamination, or residential history), particularly for smaller geographic areas, which are the most relevant with respect to ambient air quality [62]. Accordingly, many factors that influence an individual's cancer risk cannot be evaluated in our present study. However, this ecological analysis has the benefit of a larger sample size ($n = 750$ census tracts, representing a combined population of 3.4 million people), which allowed us to detect the cancer risk from toxic air pollution in cancer incidence rates, despite the 'noise' in the dataset [63]. Some unidentified factors that influence cancer risk likely differ by parish (e.g. availability of social services) or correlate strongly with poverty/race (e.g. education) and were thus accounted for in our analysis. Finally, not all cancer risk factors contribute to census-tract-level variation in cancer incidence. For example, occupational exposures can increase cancer risk, but our analysis determined that occupation in high-risk industries (i.e. construction or manufacturing) did not explain the *geographic* variation in cancer incidence among census tracts. While we would expect geographic variation in occupational exposure, it is possible that this variation was better represented by parish, which was included as a variable in our model. Regardless, it seems unlikely that occupational exposure could explain the putative link between air toxics and cancer incidence among predominately Black (but not predominantly White) communities in Louisiana, given that Blacks are underrepresented in jobs at polluting industrial facilities [17].

An additional consideration for our study is that the scientific understanding of cancer risk is continually improving. While we used the best available estimates of cancer risk from air toxics, NATA methods (including toxicity values) are continually refined and updated [44]. The strength of NATA is that it provides reliable information about *relative* risk, which is less influenced by changing methodology compared to *absolute* risk [44]. For example, after the release of the 2005 NATA, EPA substantially revised its toxicity values for chloroprene and ethylene oxide. As a result, Tract 708 (St John the Baptist Parish), which is impacted by both pollutants, is now estimated to have the highest NATA Cancer Risk in Louisiana by a wide margin [11]. While the absolute risk value for this tract changed dramatically, from 65 in a million to 1505 in a million, the relative risk remained similar (88th state percentile in 2005 versus 100th state percentile in 2014). The prospect of large changes in toxicity values may be one reason why EPA encourages NATA users to focus on relative risks, particularly with respect to census tracts [64]. The uncertainties involved in estimating toxicity values likely explain, at least in part, why the relationship we detected between cancer risk and cancer incidence was not more linear (figure 5). Regardless, our analysis and interpretation are robust to changes in the NATA methodology, including revisions to toxicity values, because we focus on variation in NATA Cancer Risk among a substantial number ($n = 750$) of census tracts.

Our study provides evidence that toxic air pollution is a significant driver of cancer rates in Louisiana's most vulnerable communities. We found no evidence that parish-wide smoking or obesity rates contributed to the observed link between estimated cancer risk from air toxics and cancer incidence. While it was not possible to account for smoking or obesity in this study of Louisiana census tracts, it seems improbable that average smoking/obesity rates for census tracts would vary in a pattern that correlates with toxic air pollution, but is unaccounted for by the inclusion of parish, poverty, race, and occupation in the model. Regardless, behavioral factors must be put into their appropriate social and environmental contexts because they are shaped by deeply-rooted structural inequities, such as disparities in the built environment (e.g. parks and recreational facilities) or access to quality healthcare [56]. These disparities make it more important, not less important, to understand and address the disproportionate burden of pollution in environmental justice communities. Our study contributes to this understanding by systematically documenting, for the first time, the increased cancer burden among the most polluted and marginalized communities in Louisiana. Future assessments of the industrial pollution burden in Louisiana must account for potential interactions among poverty, race, and exposure to air toxics. These conclusions are consistent with the firsthand experiences of Black residents from impoverished, industrialized neighborhoods who have long maintained that their communities are overburdened with cancer from toxic pollution.

## Data availability statement

No new data were created or analyzed in this study.

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# Appendix

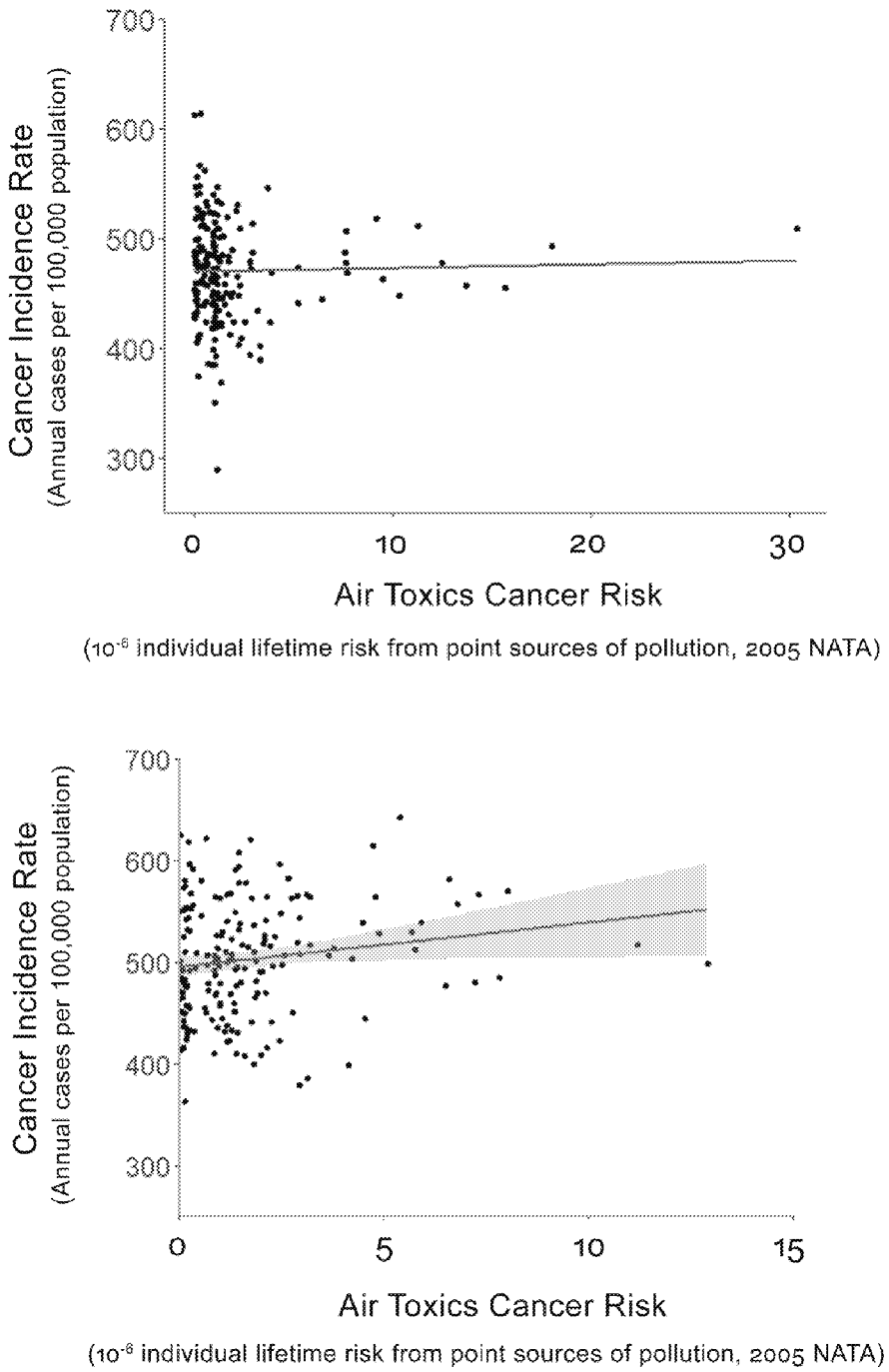


**Figure A1.** Comparison of pollution versus cancer rates among census tracts with the fewest (first quartile, top panel; $r = -0.10$, $P = 0.16$) and most (fourth quartile, bottom panel; $r = 0.13$, $P = 0.07$) Black residents as a proportion of the population. See table 4 for quartile breaks and test statistics. Blue line represents a linear regression of untransformed data. Gray shading represents 95% confidence interval.

**Table A1.** Stepwise selection results for alternate[a] cancer incidence model.

| Main effects[b] | Interaction terms | AIC | ΔAIC | Rank |
|---|---|---|---|---|
| Air toxics | Race × air toxics | 5854.3 | 0 | 1 |
| Race parish | None | 5864.4 | 10.1 | 2 |

[a] Model includes race (% Black residents) instead of poverty.

[b] Air toxics corresponds 2005 NATA Cancer Risk from point sources. See section 2 for other data sources.



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**Table A2.** Statistics for best-supported alternate[a] cancer incidence model.

| Variable[b] | Coefficient estimate[c] | $t$ | $P$ |
|---|---|---|---|
| (Model intercept) | 489.95 | 33.66 | $< 0.0001$ |
| Race (% Black) | 44.39 | 8.00 | $< 0.0001$ |
| Air toxics | −0.43 | −0.16 | 0.871 |
| Air toxics × race | 12.75 | 3.35 | 0.0008 |

[a] Model includes race (% Black residents) instead of poverty.
[b] Air toxics corresponds 2005 NATA Cancer Risk from point sources. See section 2 for other data sources.
[c] Coefficients correspond to transformed and mean-centered data (see section 2).

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