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Assessing environmental injustice in Kansas City by linking paediatric asthma to local sources of pollution: a cross-sectional study

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ABSTRACT

Objective A grassroots environmental-justice organisation in Kansas City has been examining the disproportionate exposure to air pollution experienced by residents living fenceline to the largest classification railyard in the USA. Prior analyses showed limited increased risk for asthma exacerbation for patients with asthma living closer to toxic release inventory (TRI) facilities and railyards. In this study, we assessed geographical asthma and environmental disparities, to further explore community-level disparities.

Design This is a cross-sectional study of population-level asthma rates, which included rates for all asthma encounters and acute asthma encounters (urgent care, emergency department, inpatient admission). Distances from census-tract centroids to nearest TRI facilities, railyards and highways were calculated. The association between asthma rates and distances was examined using Kendall's τ correlation and multivariable Poisson regression models.

Setting We used electronic medical record data from the regional paediatric hospital, census and Environmental Protection Agency (EPA) air monitoring data.

Participants Patients with 2+ asthma encounters during the EPA study timeframe were identified.

Results Residential distance from railyards exhibited a significant negative correlation with overall (−0.36 (CI −0.41 to −0.32)) and acute (−0.27 (CI −0.32 to −0.22)) asthma rates. Asthma rates were elevated among tracts north of the closest railyard (incident rate ratio: 1.38; CI 1.35 to 1.41) when compared with southern directionality. An increased distance from the nearest railyard of 3 km was associated with a decrease in overall asthma rates of 26%.

Conclusion Significant negative associations between proximity to all pollution source types and asthma rates were observed. This community-level research has served as a tool for community engagement and will be used to support proposed local policy. Environmental justice work addresses local concerns involving small, limited datasets, if the data exist at all. The academic epidemiological platform may reconsider acceptable approaches to small population research in order to better serve communities with the most need.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ Including wind direction in the assessment highlights the importance of acknowledging unique variabilities in both built and innate environments.
⇒ Use of population-level demographic data, rather than individuals' data, may have affected the accuracy of the analysis.
⇒ Research responds directly to the request of environmental justice community members questions.
⇒ Lower resolution data (census tract rather than residential data) are more applicable to conversations between community members and policy-makers.
⇒ Community asthma rates are based on asthma encounters specific to our institution, so paediatric asthma encounters from other local healthcare providers as well as those not seeking medical care are not reflected.

INTRODUCTION

It is known that mostly low-income, minority populations live in the areas surrounding the transportation corridors and railyards, including in Kansas City.[1 2] Grassroots organizer, Beto Martinez, the former co-director of environmental justice organisation, CleanAirNow in Kansas City (CANKC), has been leading a discussion around the disproportionate exposure to air pollution by residents of Kansas City, KS—specifically those neighbourhoods living fence-line to the one of the largest railyards in the nation. Kansas City is a major port and expanding hub for rail and highway infrastructure with a high amount of intermodal freight activity consisting of an annual estimate of 1 billion tons of cargo, US$216 million worth of goods moving through the city.[3]

As is the case for many environmental (in)justice communities adjacent to growing industries including freight sectors, the health hazards associated with pollution often go unaddressed until community members themselves take the initiative to identify the risks and harm. In 2012, CANKC, in partnership with community members, conducted their own monitoring of neighbourhood air pollution. This work prompted the Environmental Protection Agency (EPA) to conduct a subsequent study between 2017 and 2018 called the Kansas City Transportation and Local Scale Air Quality Study (KC-TRAQS).[4 5] According to anecdotal conversations with CANKC leadership and members, the EPA insufficiently involved the community and CANKC in the study design, communication, education, monitoring placement and reporting for KC-TRAQS. Consequently, this created distrust towards government and other outside agencies interested in doing subsequent research.

One central pillar of CANKC was to understand the potential harmful health effects in relation to the type and amount of pollution being produced by the freight sector in their neighbourhoods and how to eliminate those risks. Having evidence-based information that is accessible and easy to understand can support communities in organising, mobilising and doing advocacy work. Such information can support communities in identifying priority concerns and establishing action steps needed to influence decision-makers in policies and practices that ultimately improve the environmental conditions and health of the community. Consequently, at the behest of former CANKC leadership, this study focuses on health outcomes by investigating the link between local air pollution sources and paediatric asthma rates in the Kansas City metro area.

Paediatric asthma is one of the costliest and most disparate paediatric diseases in the Kansas City metro area (Children's Mercy, internal data). Paediatric asthma is frequently used to study the impacts of pollution exposure on child health.[6] It is one of the most common childhood diseases and occurs frequently enough to identify health impacts even for individual neighbourhoods. There are multiple key factors to justify the necessity to better understanding paediatric asthma: (1) it is one of the costliest childhood diseases (as an impact on our healthcare system overall and therefore attracts attention from politicians and local health officers); (2) it is demonstrative of health inequities in several demographic factors throughout the USA[7 8]; and (3) it is directly impacted by pollution exposure.[6]

Previous analyses completed by the authors, using Children's Mercy Kansas City Hospital Electronic Health Record (EHR) data, showed limited correlations between (1) patient-level asthma exacerbation (as indicated by acute care visits to a Children's Mercy provider) and residential proximity to toxic release inventory (TRI) facilities; and (2) residential proximity to a railyard and asthma rates overall.[9] These analyses were challenged by the community specificity of the study, and therefore potential limitations of external validity. And while the limited correlations supported what epidemiological studies have shown for years—children residing closer to pollution sources have higher rates of asthma and asthma exacerbation,[9] we were not able to present the data in a way that prioritised certain neighbourhoods for intervention over others; what was missing from previous analyses, according to community organisers, was applicability of the data for community organisers and decision-makers (outcomes were not divided by districts or discreet neighbourhoods). In response to community discussion and to address the latter, the aim of this study was to examine asthma rates, not at the asthmatic patient level, but among neighbourhoods/community census tracts in relation to proximity to pollution sources in the Kansas City metro area. In this study, we hypothesised that those census tracts geographically closest to pollution sources would have higher rates of asthma as well as higher rates of asthma exacerbation. Because of the highly localised quality of the data, we were also able to focus on unique geospatial features of the region, specifically wind direction proximal to the largest local railyard.

METHODS

Study design

This is a cross-sectional study of population-level asthma rates, which included rates for all asthma encounters and acute asthma encounters (urgent care, emergency department, inpatient admission).

Population data

Children between the ages of 0 and 19 who were seen at Children's Mercy Kansas City between October 2016 and October 2019 with a discharge diagnosis of asthma (ICD-9: 493.XX or ICD-10: J45.XX) in their EHR were identified. This timeframe was selected to reflect the timing of the KCTRAQS study, which we initially intended to use as our primary exposure data source. Patient-level observations were provided by Children's Mercy Kansas City and the study was determined exempt by the Children's Mercy, Kansas City Institutional Review Board (IRB number: STUDY00001525). As the only paediatric hospital in the Kansas City Metro Area, Children's Mercy Kansas City provides care to 50%–75% of patients with paediatric asthma in its catchment area (internal catchment data). Patients who had only one asthma encounter during the study time period were excluded. Patients of all ages 0–19 were included to reflect the population estimates provided by census datasets. Children who resided outside the four KC metropolitan counties of interest (MO: Jackson and Clay; KS: Wyandotte and Johnson) were also excluded.

The home address of patient with asthma at the time of encounter was geocoded and assigned to census tracts. Asthma encounter data were aggregated to the census tract to create two summary measures: (1) the frequency of all asthma encounters; and (2) the frequency of acute asthma encounters, which was defined as an asthma visit to the urgent care, emergency department or inpatient admission. Census tract-level asthma encounter frequencies were standardised to asthma rates using the U.S.

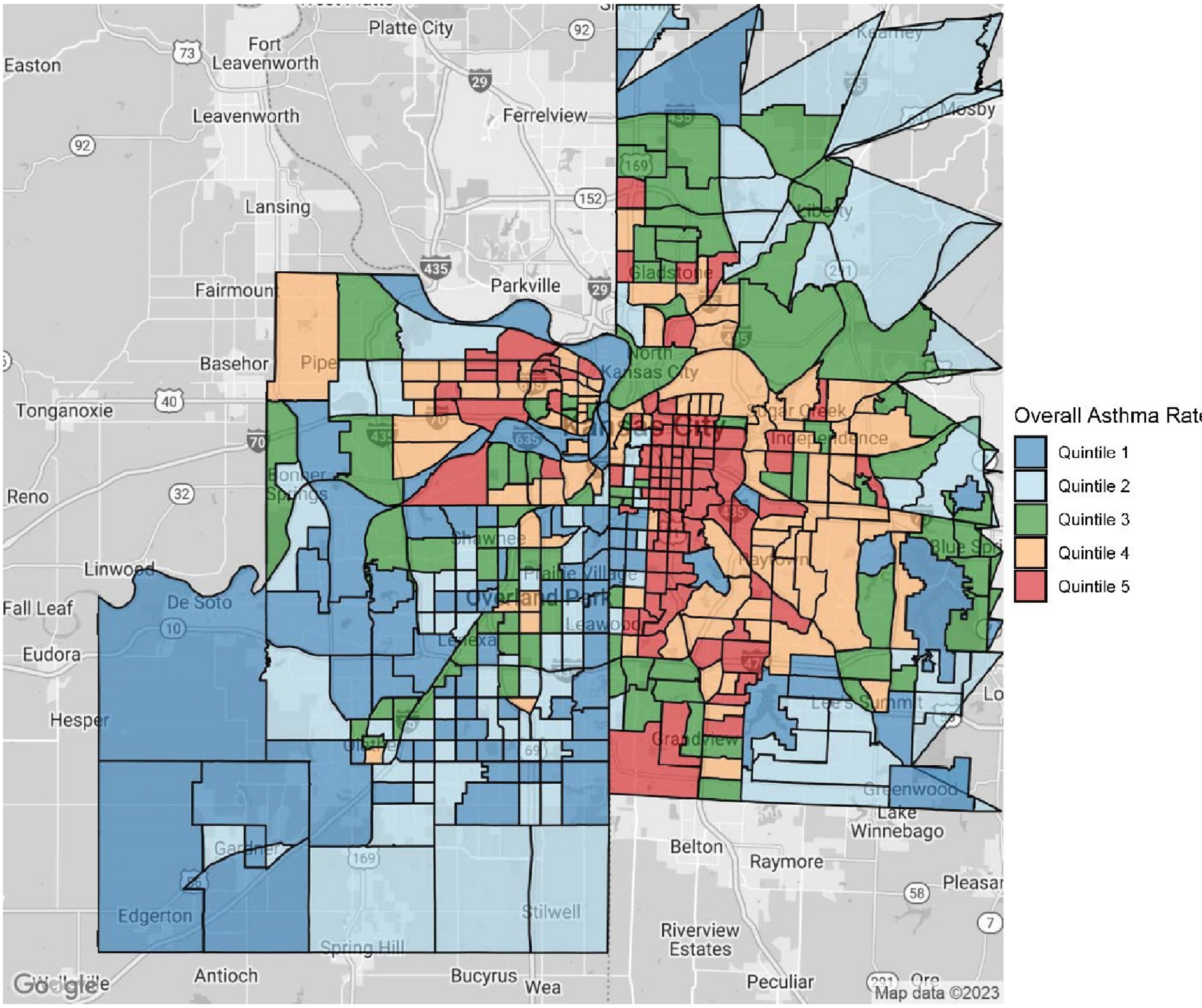

Figure 1 TractsMap (census tracts in quintiles of overall asthma rates).

Census Bureau's American Community Survey (ACS) 5-year 2015–2019 population estimates of children aged 0–19 years (figure 1, TractsMap). There are 443 tracts initially included in the four study counties; however, we excluded 15 tracts due to population estimates of zero. The remaining 428 census tracts were used for all analyses. Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) cross-sectional reporting guidelines were used for this report.[10]

Exposure data

Residential proximity to pollution-emitting sites was used as a proxy measure for pollution exposure.[11 12]

To provide the best estimate of pollution exposure(s) without a dense network of reliable long-term measurements, several pollution sources were included when assessing if proximity to those sources is related to overall asthma rates and rates of asthma exacerbation. TRI site locations were obtained from the EPA Facility Registration Service geodatabase.[13] The locations of major roadways were obtained from the Mid-America Regional Council. The locations of railyards were obtained from the US Department of Transportation, Federal Railroad Administration.

For each census tract, the spatial centroid of the polygon was determined. The closest distance of the centroid to the particular pollution feature was taken to be the nearest Euclidean distance. Distances were calculated for each pollution feature (railyard, TRI, and highway) and subsequently log-transformed and then standardised (mean=0, SD=1) in order to demonstrate how asthma rates change with a one-standard deviation change in distance. The determination of whether a census tract spatial centroid was north or south of the nearest pollution feature was based simply by comparing the latitude of the centroid to the nearest feature. This southerly wind direction is noted during the 1-year mobile measurement campaign and distributes air pollution asymmetrically around railyards.[4 14]

Analyses

Kendall rank correlation coefficients, with percentile-based bootstrapped confidence limits, were calculated to compare relationships between the distance of the centroid to the three pollution features with the overall and acute asthma rates. Multivariable Poisson regression models were completed to calculate the asthma incidence rate ratios (IRR). Rates were standardised by including the census tract-level population estimates as an offset term.

While proximity to pollution sources is frequently considered sufficient as a proxy for exposure, the community-specific focus of this study provided an opportunity for us to investigate highly localised concerns. A comparison of the north–south directionality from railyards is included to determine what, if any, the role that the predominantly southerly wind direction plays in exposure to local patients with asthma residing particularly close to a railyard. This was tested with the Poisson generalised linear model by comparing the centroids south and north of railyards. All analyses were completed using R software (R Foundation for Statistical Computing; Vienna, Austria).

Patient and public involvement

None.

RESULTS

There are 443 census tracts initially identified from the metro region. Fifteen tracts with children populations estimates of zero were excluded. Nearly all census tracts had up to 25 paediatric asthma encounters for every 100 residing children; 27 census tracts (6.3%) had number of encounters at or higher than 30 (per 100 children) (online supplemental figure 1). Likewise, almost all census tracts had acute asthma visits less than 10 (per 100 children) (online supplemental figure 2); however, there were 25 (5.8%) census tracts with acute care visits at or higher than 10.

Figure 1 shows the distribution of acute asthma events during our period of study. In this map, one can see a cluster of higher rates of paediatric asthma central to the Kansas City metro area, on the Missouri side of the state line. These areas align well with the distribution of industry and railyards in the city as well (figure 2: pollution sources map).

Among the census tracts, the shortest distance between the centroid of a tract and a railyard, TRI facility and highway is 0.01 km (100 m), 0.07 km and 0.01 km, respectively (table 1). Those tracts in the closest quartile were up to 3.93 km, 1.48 km and 0.62 km, respectively. Census tract centroids ranged between 0.0098 and 42.647 km from the closest railyard, between 0.0678 and 28.669 km from the closest TRI facility and 0.0061 and 6.340 km to the closest highway. Eleven census tracts are north of a railyard (25.9%).

There are significant negative correlations of all three pollution sources with both the overall and acute asthma rates in Kansas City census tracts (table 2) which suggests that as distance from any of the three pollution factors increased, there is a significant decrease to both the overall as well as the acute paediatric asthma rates. Notably, the distance from the railyard exhibited the strongest correlation with overall (−0.36 (CI −0.41 to −0.32)) and acute (−0.27 (CI −0.32 to −0.22)) asthma rates.

Residential distance from railyards exhibited a significant negative correlation with overall (−0.36 (CI −0.41 to −0.32)) and acute (−0.27 (CI −0.32 to −0.22)) asthma rates. Asthma rates were elevated among tracts north of the closest railyard (IRR: 1.38; CI 1.35 to 1.41) when compared with southern directionality. Increasing the distance from a railyard by 3 km was associated with a decrease in overall asthma rates of 26%.

Directionality from railyard

Poisson GLM analyses show that overall asthma rates were significantly higher among tracts north of the closest railyard (IRR: 1.38; CI 1.35 to 1.41; $p<0.001$) when compared with a southern directionality (table 3). This railyard directionality association remained (adjusted IRR: 1.13; CI 1.11 to 1.16; $p<0.001$) even after adjusting for distance from the three pollution sources. Directionality from a railyard was significantly associated with rates of acute asthma exacerbation in an unadjusted model (IRR: 1.19; CI 1.15, 1.24; $p<0.001$), though this relationship was attenuated when adjusting for distances (IRR: 1.01; CI 0.97 to 1.05; p=0.675).

DISCUSSION

This paper provides an assessment of community-level asthma health disparities regarding environmental pollutants and land use in Kansas City. Asthma disproportionately affects communities living closer to industrial and transit pollution sources, specifically TRI facilities, highways and railyards. Census tracts with closer proximity had higher rates of both asthma and acute asthma healthcare utilisation than those with less proximity to these industrial pollution sources. The strongest predictor of acute healthcare utilisation for paediatric asthma among census tracts was proximity to a railyard. In the Kansas City metro, there are 29 railyards, including the second largest intermodal railyard in the nation. A unique feature of this study was that in addition to assessing the relationship between proximity to pollution sources and asthma rates, we ascertained whether wind direction was a key predictor of asthma rates and exacerbation. While the adjusted rate ratio for acute asthma was considered non-significant, the risk for a community having both higher rates of paediatric asthma as well as rates of acute asthma exacerbations is higher for census tracts downwind than upwind of a railyard.

There is a substantial and rapidly growing body of literature connecting exposure to pollution on both the development and exacerbation of asthma.[6 15–18] Khreis *et al*, in their systematic literature review and meta-analysis, found variability amongst different pollutants (nitrogen oxides (NO_x) vs sulphur oxides (SO_x) vs particulate matter equal to 2.5 µm in diameter (PM2.5) vs black carbon (BC));

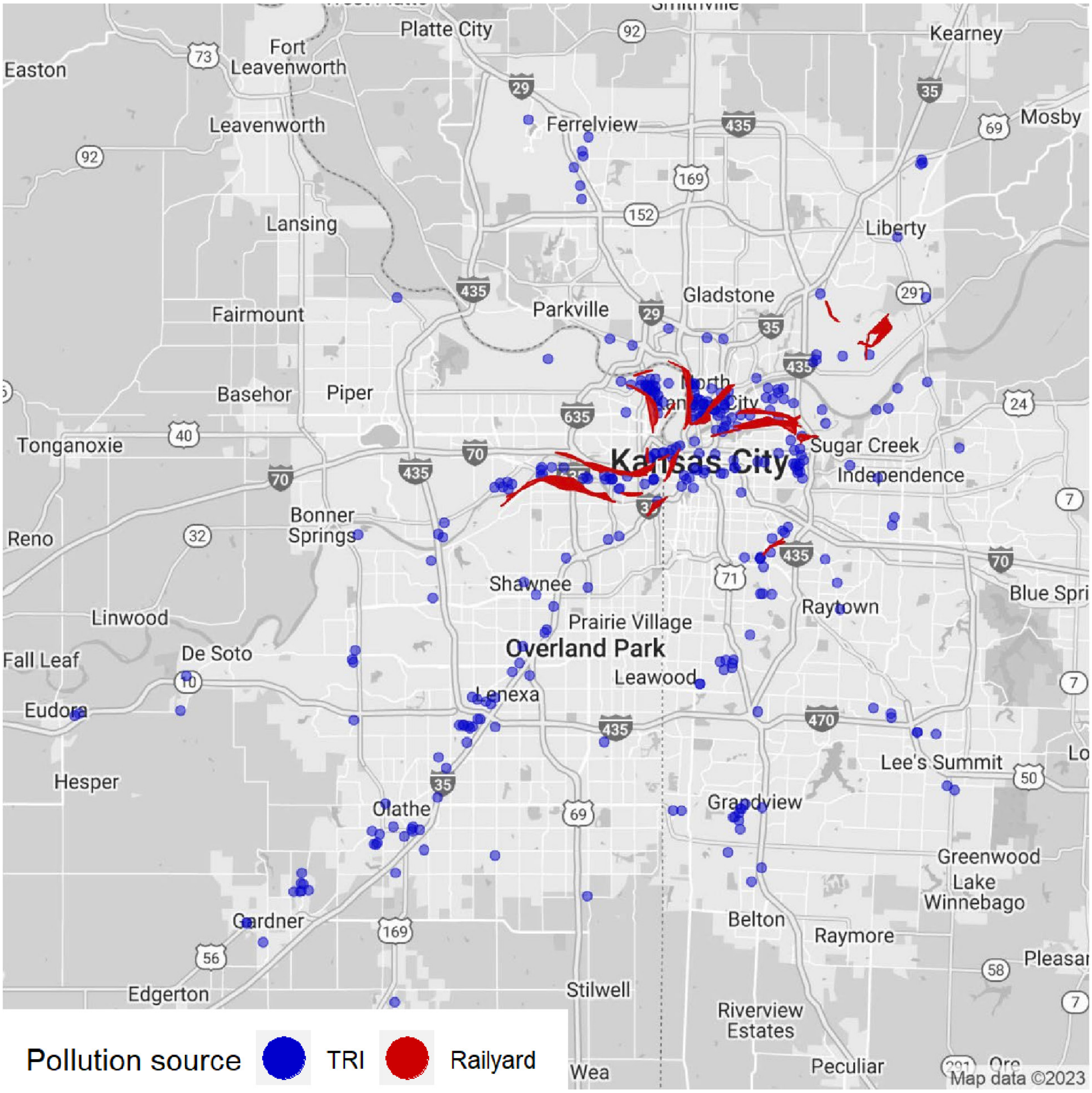

Figure 2 Pollution sources map (toxic release inventory (TRI) facilities, railyards and highways).

their overall conclusion was that early exposure to air pollutants contributes to the development of childhood asthma.[18] Orellano *et al* in their recent meta-analysis indicate that a broad spectrum of pollutants exacerbate asthma in children,[19] including the primary pollutants that come from highways, railyards and industrial sites.

Proximity to pollution sources has frequently been used as an indicator of exposure, and the reproducibility of such research indicates that this may be an effective proxy measure of pollution exposure. Liu *et al*, in their meta-analysis of publications with concentration distributions of traffic-related air pollutants as a function of the

Table 1 Census tract distance from pollution sources

	Min	Q1	Median	Mean	Q3	Max
Railyard distance	0.01	3.93	8.99	11.51	18.02	42.67
TRI distance	0.07	1.48	2.59	3.79	4.24	28.67
Highway distance	0.01	0.62	0.93	1.25	1.65	6.34

Note: distance measured in kilometres.
TRI, toxic release inventory.

Table 2 Kendall's τ

	Overall asthma rates		Acute asthma rates	
	Kendall's τ	P value	Kendall's τ	P value
Railyard distance	−0.36 (−0.41, −0.32)	0.001	−0.27 (−0.32, −0.22)	0.001
TRI distance	−0.21 (−0.26, −0.15)	0.001	−0.21 (−0.27, −0.15)	0.001
Highway distance	−0.19 (−0.24, −0.13)	0.001	−0.17 (−0.23, −0.11)	0.001

TRI, toxic release inventory.

distance from major roads, concluded that the distance to road proxy is the best and most appropriate method to characterise exposure.[19] Likewise, Choi *et al* note that distance from major roads as well as point sources is inversely related to pollution concentrations.[20]

As grassroots environmental justice movements grow, along with an increased interest in understanding cumulative impact from multiple pollution sources within individual communities interested in understanding the impact of their surroundings on health, proximity to pollution sources has become a popular way to address this curiosity.[21 22] A paucity of true measures of exposure at a neighbourhood level supports the need for proxy measures of exposure until high-resolution pollution data (with geospatial variability between city blocks) become available.

Air pollution measures are often unavailable, and proximity to pollution sources is often used as an indicator of exposure.[23 24] The correlation between geographical proximity to *certain* pollution sources, such as highways, and childhood asthma prevalence and exacerbation is relatively well established in the literature, but not for all pollution sources. This study uses proximity to multiple pollution sources as a proxy for air pollution exposure.

Highways and asthma

Residential proximity to roadways and exacerbation of asthma has been studied extensively. Khreis *et al*, in their recent meta-analysis, using 41 qualifying studies, found that traffic-related air pollution (TRAP), specifically exposure to the components BC, NO_2, PM2.5, PM10 correlate significantly with the risk of asthma development.[18] The overall body of literature suggests that there is sufficient evidence of a causal association between exposure to TRAP and asthma exacerbation and suggestive evidence of a causal association for the onset of childhood asthma.[25] In their Massachusetts study, Freid *et al* found that participants who resided closest to a major road (<100 m) during their first 3 years of life had a substantially higher risk of recurrent wheeze and asthma compared with those residing farther (>300 m) from a major road.[26] Likewise, Jung *et al* found a positive correlation between the size of the biggest road within 200 m of the home and a diagnosis of asthma.[27] Porebski *et al* also found a correlation

Table 3 Poisson GLM

	Unadjusted			Adjusted		
	IRR	P value	CI	IRR	P value	CI
Railyard directionality						
South	-ref	---	---	-ref	---	---
North	1.38	<0.001	1.35 to 1.41	1.13	<0.001	1.11 to 1.16
Railyard distance—standardised	0.69	<0.001	0.68 to 0.69	0.74	<0.001	0.73 to 0.74
TRI distance—standardised	0.80	<0.001	0.79 to 0.81	0.92	<0.001	0.91 to 0.93
Highway distance—standardised	0.85	<0.001	0.84 to 0.86	0.90	<0.001	0.90 to 0.91
Incidence rates for acute asthma (N=428)						
Railyard directionality						
South	ref	–	–	Ref	–	–
North	1.91	<0.001	1.15 to 1.24	1.01	0.675	0.97 to 1.05
Railyard distance—standardised	0.71	<0.001	0.70 to 0.72	0.78	<0.001	0.76 to 0.79
TRI distance—standardised	0.78	<0.001	0.77 to 0.80	0.89	<0.001	0.87 to 0.90
Highway distance—standardised	0.83	<0.001	0.82 to 0.84	0.87	<0.001	0.85 to 0.88

Adjusted incidence rate ratios (IRR) are based on Poisson models that included railyard directionality, railyard distance, TRI distance and highway distance. Incidence rates for all asthma (N=428).
TRI, toxic release inventory.

between residential proximity to a major roadway with more frequent asthma symptoms among children, where those living within 200 m reported more symptomatology than those living farther away.[28]

Industrial sources (TRI) and asthma

Ongoing low-level exposure to air pollution is increasingly accepted as a determinant of health.[6 29] Exposure to concentrations of pollutants from industrial emissions includes particulate matter and other hazardous air pollutants. Living near heavily polluting industries and former industrial sites has been associated with several health outcomes; literature correlates residential proximity to industrial facilities with congenital defects, childhood cancer and cardiovascular disease.[23 30] In their recent meta-analysis, Buteau *et al* were unable to find an association between exposure to air pollution from industrial point sources and asthma-related outcomes in childhood due to the heterogeneity of the available studies.[31] However, they reported on several studies that found residential proximity to point source industries (including petrochemical facilities, power plants, hazardous waste treatment plants) being inversely correlated with asthma-related utilisation of healthcare services. In their own research published prior to their meta-analysis, Buteau *et al* (2018) found correlations between residential exposure to industrial air pollutant emissions (by quantifying reported metric tons of pollutants from nearby industries) and childhood-onset asthma.[32]

Data from our study support a correlation between residential proximity to TRI facilities and the development of paediatric asthma and exacerbation of asthma symptoms.

Railyards and ports and asthma

Our results show a robust relationship with the distance from railyards, even tending to show a stronger relationship than the more typical metrics of distance to highway or TRI facilities. This is a concerning result, pointing to the impact on children living close to railyards in Kansas City. Furthermore, children living in communities north of these locations are even more prone to asthma. Given the influence of wind direction on pollution concentrations, it is reasonable to investigate asymmetric health outcomes around railyards.

Though it is well established that railyard ports produce a substantial amount of air pollution and that residents living near major transportation hubs and corridors are exposed to high levels of airborne pollutants,[33] there is a paucity of studies that highlight the association between proximity to ports and the development or exacerbation of asthma or other diseases for that matter. Spencer-Hwang *et al* conducted an observational study assessing for association between residential proximity to major freight railyards in California between 2007 and 2009 and using logistic regression modelling found that children living closest to a railyard (0–5 miles) were at significant increased odds (OR 1.15, 95% CI 1.10 to 1.20) for asthma-related ER visits with even greater odds for the higher emitting railyards.[34] Likewise, in their earlier research, Spenser-Hwang *et al* identified increased likelihood of adverse respiratory health outcomes of children living and attending school near a major freight railyard.[12 35]

Study strengths and limitations

This study adds to the body of literature in a few ways. First, and most obvious, residential proximity to pollution sources is associated with the diagnosis and exacerbation of asthma—with proximity to some pollution sources showing stronger association than others (railyards and highways more so than individual industrial point sources).

Perhaps more important is the challenge that community-driven environmental justice work poses to the epidemiology pedagogy. Many communities are interested in asking for research that is not necessarily novel, but rather practical and applicable to local and regional policy and land use decisions. In order to address the community's question about neighbourhoods at risk, we used lower resolution data (census tract rather than individual's), and correlated neighbourhood proximity to pollution sources and asthma. While it may not be considered novel, the utilisation of this and previous reports by grassroots organisers to approach policymakers highlights the value of this type of academic work and partnership.

The exposure assessment above, including wind direction, highlights the importance of acknowledging unique variabilities in both built and innate environments. This aspect of our study showcases the importance of addressing local variabilities when studying a highly localised population, such as a city-specific or neighbourhood-specific study.

This study has several limitations. One key limitation of this study is the availability of data at a residential level. Population-level demographic data are most easily available at the census tract and block group levels. While recalculations might have affected the accuracy of the data, converting it to neighbourhood units, as defined by local government maps would have accordingly made the results of the report more useful for community groups to use when communicating to local decision-makers. While this would fulfil our community partner's asks, it does lend the study to ecological fallacy. Additionally, community asthma rates are based on asthma encounters for children seeking medical care at Children's Mercy hospital system. Consequently, paediatric asthma encounters from other local healthcare providers as well as those not seeking medical care are not reflected. Also, the findings are quite specific to one Midwestern metropolitan area and therefore may not generalise to other regions. Probably most important for our community partners, our aggregated asthma rates and proximity to pollution sources do not support causal claims that pollution was the cause for asthma-seeking patterns. Lastly, with this analysis, we assume that pollution exposure was ubiquitous within each census tract—this will be important to mind if the findings are used to identify areas of concern for asthma and pollution when approaching local urban planners and decision-makers.

One challenge faced when using proximity of pollution source as a proxy for exposure is recognising the type of pollution (as different chemicals affect human health differently), amount of pollution and the behaviour of that pollution can result in hugely different outcomes. In this case, pollution exposure has repeatedly been proven to correlate with asthma outcomes. There is an abundance of literature exploring the relationship between highways (and therefore transportation pollution) and asthma. It is unclear to the authors if this literature is as substantial for other health outcomes. Pollution from railyards tends to be diesel specific with elevated levels of ultrafine particulates and BC,[36] which is not so for city highway pollution (though plenty of diesel pollution does come from highway transit, it is not exclusively diesel pollution). Diesel pollution tends to be categorised as ultrafine particulate—and does not necessarily disperse the same way as other point source pollutants given their different rates of diffusion and coagulation.[37] Likewise ultrafine particulate is believed to affect health outcomes differently from PM2.5 pollution.[38] Pollution from TRI facilities likely vary substantially, depending on the industrial product.[13] In Kansas City, KS, some of the TRI facilities included in this research include (1) paint manufacturers, (2) chemical sterilisers, (3) petroleum terminals and (4) motor manufacturing, all of which produce and release different types and amounts of air pollution. Regarding this study, while the largest air pollution-producing sources were considered, other sources of pollution were not addressed. Further research is needed to better identify thresholds for exposure and background pollution levels as well as differentiating between pollution types while still acknowledging cumulative exposure. In this case, next steps may include conducting similar studies but adding sufficient, hyperlocalised, measures of pollution currently being gathered by community members.

Kansas City is a major intermodal hub that includes a large amount of rail traffic with railyards to accommodate the heavy rail activities occurring in this inland port and its surrounding industrial parks. Given the continued trajectory of development, these activities are likely to continue expanding and local communities are concerned about the impacts these developments will have on their health. These findings, though, support the community's concern and need for additional research, resources and system-level changes that reduce health burdens from the nearby railyard, highways and industry. Environmental justice work is centred on community-driven conversations and therefore community-based datasets. Our findings and our process speak to the importance and challenges of responding directly to community concerns to drive future policy and land use in this case, in the Kansas City Metro area. To this day, the grassroots drivers of this project continue to work with community members in expanding their air monitoring network and will use these findings to strategise the next action steps needed to advance local environmental justice.

CONCLUSION

A primary purpose of this research and report was a response to grassroots organisers' request for a community-oriented (geospatially pertinent) assessment on asthma and air pollution in the Kansas City metro area. The request made was for a report that addressed multiple sources of pollution for an overburdened neighbourhood adjacent to industrial parts, ports and railyards. A successful collaboration between the authors (both academics and community organisers) is reflected in this report. The work indicates that an unequal distribution of pollution in our region correlates with a health disparity (in this case asthma) that affects children around the nation.

The challenges and limitations identified point to a need both for more research—specifically ascertainment of high-resolution pollution exposure data as well as more effective means to gather community-wide health data—from several institutions, which are frequently siloed. The hope and expectation are that community partnerships like the one the authors continue to develop will be a driver for more research, collaboration and evidence-based decisions around health and well-being to drive decisions made by city planners and legislators to improve regional and local health and well-being through long-term planning and equitable distribution of resources.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Data may be made available upon request. PHI data elements would necessarily be cleaned or removed prior to sharing the data.

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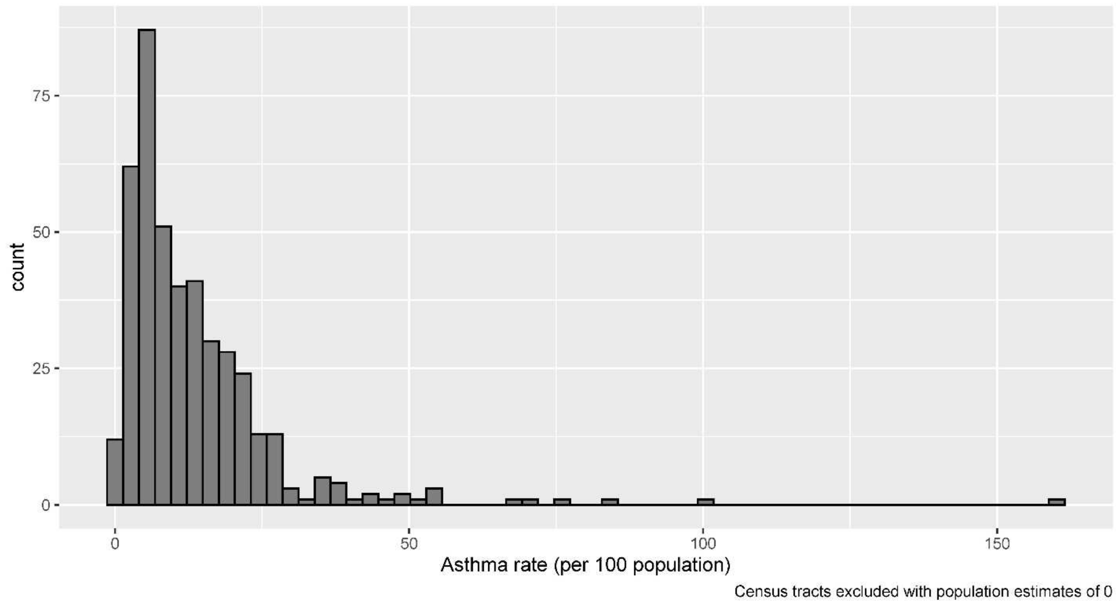
Supplemental Figures

Supplemental figure 1: Overall asthma cases per 100 population counts



Supplemental figure 2: Acute asthma cases per 100 population counts.