



**ENVIRONMENTAL HEALTH
& ENGINEERING, INC.**

**REVIEW OF
LOGAN AIRPORT HEALTH STUDY**

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REVIEW OF LOGAN AIRPORT HEALTH STUDY

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EXECUTIVE SUMMARY

BACKGROUND

Air pollution and noise from airports, such as Logan Airport, have long been a source of concern for surrounding communities. Aircraft, other airport operations, and airport-associated road traffic are known sources of not only noise but also a variety of air pollutants that have been shown to have harmful effects on human health. The impacts of airport-related emissions and noise on the health of surrounding communities are less well understood. To address this issue, the Logan Airport Health Study (LAHS) was conducted by the Massachusetts (MA) Department of Public Health Bureau of Environmental Health (BEH). The LAHS is a cross-sectional study intended to ascertain the prevalence of specific health conditions within neighborhoods surrounding Logan Airport and to examine their association with air pollution and noise originating from Logan Airport. A final report of the study and its findings was published by BEH in May 2014.

At the request of AIR Inc., an East Boston community group, Environmental Health & Engineering, Inc. (EH&E) reviewed the LAHS as well as related background materials and scientific literature. EH&E was asked to determine whether the LAHS provides a definitive characterization of community health impacts associated with Logan Airport.

CONCLUSIONS

The LAHS showed that of the many health outcomes examined, airport-related exposures were significantly associated with only two—1) probable asthma in children, and 2) chronic obstructive pulmonary disease (COPD) in adults, and not with any of the cardiac or auditory health measures. However, for the reasons summarized as follows, the LAHS does not provide a definitive assessment of potential airport-related health impacts in communities located in close proximity to Logan Airport.

Design

While providing a useful “snapshot” of the health and exposures of Logan Airport neighbors, due to its design the LAHS cannot discern whether exposures to airport pollution and noise occurred before or after diagnosis of disease. As a result, the study cannot address whether or not there is a cause-and-effect relationship between airport emissions and probable asthma in children or COPD in adults. Nonetheless, the LAHS provides important background information on the pollution and noise profiles and disease prevalence near a major airport, which can be used to generate new hypotheses for further study.

Because most of the LAHS health outcomes develop slowly with some outcomes relatively rare, studies of airport-related health impacts may require the observation of many participants over a

long period, rather than a snapshot in time. Additional surveys should be administered to the same cohort, with these surveys asking not only about doctor-diagnoses of diseases but also about intermediate health effects that can be markers for or precursors of disease, such as blood pressure and sleep disturbance.

Participant-Specific Exposures

The BEH analyses were all conducted on categories of exposure classified as low, medium and high for air pollution as a whole and also for noise levels. Additional analyses of LAHS data may be useful for learning more from the study. By using the actual participant-specific estimates of air pollution and noise, health analyses will be able to better capture spatial variation in exposures, reduce exposure misclassification, and increase the ability of the LAHS to detect exposure-health associations. For noise, alternate exposure measures, such as peak noise, should be explored, as these measures may be more variable and may better capture participant's airport-related noise exposures, which are intermittent and generally associated with airplane takeoffs and landings. These analyses are critical given findings from recent studies of airport noise and cardiovascular disease, which consistently show threshold effects.

Specific Markers of Airport Exposures

New analyses should also be conducted using air pollutant and noise measures that are more specific markers of airport-related activities. The LAHS indicated that Logan Airport emissions led to a small contribution to fine particle, nitrogen oxide, and volatile organic compound (VOC) levels in surrounding neighborhoods, all of which are emitted by many non-airport related sources. As a result, the ability of the study to separate the impacts of airport- from non-airport exposures was likely poor, even with controls for background pollution and distance to road. The scientific literature supports the use of ultra fine particles (UFP), elemental carbon (EC), and certain polycyclic aromatic hydrocarbons (PAHs) as more specific markers of airport related activities.

Selection and Recall Bias

The LAHS administered a one-time telephone survey of health outcomes, demographic and risk factors for a random sample of individuals living in surrounding communities, with a response rate of survey participants estimated between 35 and 60%. This response rate raises some concerns about possible selection bias within the LAHS, where non-respondents who had phones that were always busy or never answered differed systematically from the study participants. In addition, the impact of possible recall bias associated with the self-reported outcomes is important to consider, as is the case with all studies involving survey data. This could have occurred in the LAHS if participants living in the high exposed area reported their health outcomes or risk factors differently than participants living in the low exposed area. Recall bias

in the LAHS cannot be ruled out, as no verification of the self-reported information was performed.

Non-Specific Health Outcomes

As with its exposure measures, the LAHS health outcomes are non-specific to the impacts of the measured air pollutants, as many risk factors for the disease exist beyond air pollution. Importantly, the LAHS controlled for many risk factors and predictors of the outcomes assessed in its health models. It may not have been possible, however, to account for all risk factors in the health models given the general nature of the health outcomes examined.

Relationship to Previous Findings

The significant findings in the LAHS for probable asthma and to a lesser extent COPD are consistent with observed associations reported in cohort studies examining impacts of exposures to motor vehicles, a source common to airport operations and with similar pollution emission profiles as aircraft. These findings point to the need for additional studies that focus on airport-related respiratory impacts, particularly in children and older adults.

Previous epidemiological studies of traffic have consistently shown strong associations between traffic pollution and cardiac outcomes. The null findings in the LAHS suggest that the impacts of airport-related pollution on cardiac health warrant further study in neighborhoods surrounding Logan Airport, given the inability of a cross-sectional study design to examine exposure-effect associations.

Many studies have linked noise exposures to a variety of adverse health impacts, including sleep disruption, heart disease, hypertension, stress, annoyance, hearing impairment, hospital admissions, and impaired learning in children. Although these and other sleep and cardiac outcomes were not examined in the LAHS with relation to noise, evidence from other studies points to the need for additional analysis of the LAHS data to assess noise impacts on cardiovascular, sleep, and learning outcomes.

OVERVIEW

Air pollution and noise from airports, such as Logan Airport, have long been a source of concern for surrounding communities. Aircraft, other airport operations, and airport-associated road traffic are known sources of not only noise but also a variety of air pollutants, including nitrogen oxides and particulate pollution – both of which have generally been shown to have harmful effects on human health at sufficient levels of exposure and for susceptible populations. However, the impact of airport-related emissions and noise on the health of surrounding communities is less well understood.

To address this issue, the Logan Airport Health Study (LAHS) was conducted by Massachusetts (MA) Department of Public Health's Bureau of Environmental Health (BEH), as mandated by the Acts of 2000 of the MA General Court. The main purpose of this study was to assess the impacts of Logan Airport pollution on the health of individuals living in surrounding communities. Its design was developed by BEH in consultation with the Community Advisory Committee comprising local residents, health officials, and experts in epidemiology, biostatistics, survey design and administration, and air pollution modeling. Since the start of initial data collection in 2002, the study was conducted over an approximate ten-year time period, an extended period necessitated by varying levels of available resources. A final report of the study and its findings was published by BEH in May 2014.

At the request of AIR Inc., an East Boston community group, EH&E reviewed the LAHS as well as related background materials and relevant scientific literature. Based on this review, EH&E prepared this report that provides a high-level review of the strengths and weaknesses of the LAHS and provides guidance for additional areas of research or further actions. Included in this document are sections presenting 1) a brief review of the study and its design and findings, 2) implications of study findings and discussion of study strengths and limitations, and 3) recommendations for future research.

LOGAN AIRPORT HEALTH STUDY

The LAHS is a cross-sectional study intended to ascertain the prevalence of specific respiratory, cardiac and auditory conditions within neighborhoods surrounding Logan Airport and to examine their association with air pollution and noise originating from Logan Airport. Demographic, health, and risk factor data were obtained via a telephone-administered survey that was developed based on national and international surveys and that was pilot tested in 2002. Based on results from the pilot tests, the survey was revised to create the final survey instrument for the LAHS.

In 2005, BEH administered this final survey to 8,287 participants living in 6,072 households (6,072 respondents and their 2,215 children). All participants lived in one of 17 communities¹ located within 5 miles of Logan Airport. Participants were surveyed on one occasion, with information obtained on the respiratory, cardiac and auditory health, risk factors, and demographics of themselves and their children.

Survey respondents were randomly selected using random-digit-dialing methods intended to provide a representative sample of people living within five miles of Logan Airport. By design, a greater proportion of residents living in “high” and “medium” exposure areas, defined as living less than 1 mile and between 1-4 miles of Logan Airport, respectively, were selected for study participation.

HEALTH MEASURES

Surveys were administered via computer assisted telephone interviewing. In the interview, each respondent was asked to provide demographic, risk factor, and health information for themselves and their children. For adults, health endpoints of concern included self-reported physician diagnosis of:

- **Respiratory Outcomes:** Lifetime, current or probable asthma,² asthma hospitalizations; chronic obstructive pulmonary disease (COPD)
- **Cardiac Outcomes:** Myocardial infarction (MI), coronary heart disease (CHD)/angina
- **Auditory Outcomes:** Hearing impairment, severe hearing loss, tinnitus

¹ Boston, Brookline, Cambridge, Chelsea, Everett, Hull, Lynn, Malden, Medford, Melrose, Milton, Nahant, Quincy, Revere, Saugus, Somerville, and Winthrop.

² Probable asthma in adults was defined as individuals reporting no prior history of asthma but symptoms of wheezing and shortness of breath in the last year.

Health effects of children were assessed using parent-reported diagnosis by a physician of lifetime, current (with and without medication use) or probable asthma,³ asthma hospitalizations, chronic bronchitis/chest infections, and hearing impairment.

AIR POLLUTION AND NOISE EXPOSURE ASSESSMENT

LAHS estimated exposures for five pollutants – carbon monoxide (CO), nitrogen oxides (NO_x), fine particulate matter (PM_{2.5}), sulfur oxides (SO_x) and VOCs – the pollutants or classes of pollutants with the largest airport emissions. Noise exposures were also estimated given that it is a long-standing concern of surrounding airport populations.

Air Pollution

One-hour, 24-hour, and annual concentrations were estimated for 2005 for CO, NO_x, PM_{2.5}, SO_x and VOCs. Concentrations for each pollutant were estimated at 635 points along a polar grid superimposed across the study area using the Federal Aviation Administration's (FAA's) Emissions and Dispersion Modeling System, which estimated air pollutant concentrations over the study area using 2005 Logan Airport emissions, meteorology, and topography data as inputs into the air dispersion model, known as AERMOD. The U.S. Environmental Protection Agency (EPA) recommends AERMOD for use as a dispersion model for determining regulatory compliance for some existing and new major sources (EPA, 2005). It has not been validated for use in modeling emissions from airports and/or aircraft. AERMOD is more commonly used for estimating impacts of emissions from point sources and motor vehicle traffic.

Using these model estimates, air pollutant concentrations at the residence of each survey respondent were then calculated using inverse distance weighting that essentially averages weighted model estimates at the nearest surrounding grid points. Respondent-specific concentrations for each pollutant were then ranked with respect to those for other respondents and based on this ranking, were classified as “low,” “medium” or “high” exposure. For each pollutant, respondents with concentrations less than the median value were classified as “low,” with concentrations between the 50th and 80th percentile as “medium,” and with concentrations above the 80th percentile as “high.” Since the estimated concentrations for the five pollutants were highly correlated across the study region, pollutant classifications were generally identical for each of the five pollutants. As a result, households were classified simply as one measure of “low,” “medium” or “high” exposure. As shown on Table 1 (based on Table 4-5 of the LAHS Report), 3,034 participants lived in the low exposure area, 1,834 in the medium exposure area, and 1,204 in the high exposure area. Classifications for seven percent (or 420) of study participants were not uniform across the five pollutants, with one or two pollutants having

³ Probable asthma in children was reported by parents that their children had wheezing or a dry nighttime cough not associated with a cold in the last year.

different classifications for a given survey respondent. For these respondents, classifications were based on the exposure category for three or more of the pollutants.

Table 1 Range of Modeled Concentrations at Participant Home Locations, and Categorical Variable Assignment of Low, Medium and High Exposure Categories						
Exposure Category	Number of Respondents (%)	Modeled Concentration Range (µg/m ³) [*]				
		CO	NO _x	PM _{2.5}	SO _x	VOCs
Low (<50 th ile)	3,034 (50%)	0.57 – 3.88	0.14 – 0.77	0.005 – 0.03	0.03 – 0.11	0.08 – 0.50
Medium (50-80 th ile)	1,834 (30%)	3.88 – 11.4	0.77 – 2.47	0.03 – 0.09	0.11 – 0.34	0.50 – 1.53
High (>80 th ile)	1,204 (20%)	11.4 – 109	2.47 – 15.0	0.09 – 0.65	0.34 – 1.39	1.53 – 11.6

µg/m³ micrograms per cubic meter
CO carbon monoxide
NO_x nitrogen oxides
PM_{2.5} particulate matter that is 2.5 micrometers or smaller in diameter
SO_x sulfur oxides
VOCs volatile organic compounds

* Modeled annual average concentrations estimated at home locations.

Noise

Exposures to noise were modeled by Massport using sound contours developed from airport operations data and FAA’s Integrative Noise Model (INM), which takes into account the number of takeoffs and landings, types of aircraft taking off and landing during the day and night, runway use and flight paths. Sound contours of the annual day-night sound level (DNL) were estimated at 5 decibel (dBA⁴) increments around the airport. For the LAHS, the noise exposures were categorized into low, medium and high based on the estimated dBA of less than 60 dBA, 60-64 dBA, and greater than 65 dBA, respectively.

DATA ANALYSIS METHODS

Summary statistics of population demographics were presented as were weighted, crude prevalence estimates of health conditions in the study populations both as a whole and stratified by the exposure categories (low, medium and high).

⁴ The A-weighted decibel (dBA) is defined as “the most common unit used for measuring environmental sound levels. It adjusts, or weights, the frequency components of sound to conform with the normal response of the human ear at conversational levels. dBA is an international metric that is used for assessing environmental noise exposure of all noise sources.” (<https://www.massport.com/environment/environmental-reporting/noise-abatement/noise-glossary/>).

The association between high, medium, and low airport-related air pollution and noise exposures and health conditions were examined using multiple logistic regression models, with models run separately for pollution and noise. All models controlled for a wide range of potential confounders (Table 2), selected from previous literature and using backwards elimination procedures. For adults, adjusted odds ratios were calculated separately for several health conditions, including lifetime, current or probable asthma, COPD, MI, and CHD for associations with air pollution; and hearing impairment and tinnitus for associations with noise.

Table 2 Confounders Included in the Logan Airport Health Study Health Models					
Confounder	Adults			Children	
	Respiratory	CV	Auditory	Respiratory	Auditory
Demographics					
Age	✓	✓	✓	✓	✓
Gender	✓	✓	✓	✓	✓
Race/ethnicity	✓	✓	✓	✓	✓
Poverty income ratio (PIR)	✓	✓	✓	✓	✓
Education*	✓	✓	✓	✓	✓
Smoking status	✓	✓	✓		
Medical					
Body Mass Index (BMI)	✓				
Gastrointestinal reflux disease (GERD)	✓				
Diabetes		✓			
High cholesterol		✓			
Hypertension		✓			
Family history cardiovascular (CV)		✓			
Behavioral					
Alcohol intake	✓				
Binge drinking		✓			
Environmental					
Background PM _{2.5}	✓	✓		✓	
Proximity to road	✓	✓		✓	
Second-hand smoke	✓	✓		✓	
Household chemicals	✓				✓
Household nitrogen dioxide (NO ₂) sources				✓	
Household mold				✓	
Household allergens				✓	
Sound proofing					✓
Occupational noise			✓		
* For children, health models controlled for maternal education.					

All health models for adults were adjusted for age, sex, race, ethnicity, poverty income ratio (PIR), education, and smoking status. For respiratory and cardiovascular outcomes, models were also adjusted for background (non-airport) residential PM_{2.5} concentrations and residential proximity (<200 meters) to a major road. Additionally, respiratory models controlled for household indoor smoking, body mass index (BMI), alcohol intake, gastrointestinal reflux disease (GERD), and potential exposure to household chemicals. Cardiovascular models also controlled for family history of heart disease, high blood cholesterol, hypertension, diabetes,

binge drinking. In the case of noise exposures for adults, models also controlled for occupational noise exposures.

Health models for children were run for lifetime, current and probable asthma; current asthma with asthma medication; asthma hospitalization; and chronic bronchitis/chest infections for associations with pollution. Health models for children examined the association of noise with hearing impairments. The respiratory and noise models for children were adjusted for the following factors: age, sex, PIR, maternal education, and household indoor smoking. Respiratory models also controlled for background PM_{2.5} concentrations, residential proximity to major roads, household nitrogen dioxide (NO₂) sources, household allergens, and household mold. Noise models were also adjusted for household mold and Massport soundproofing.

Further, multivariate logistic regression models were also run to assess effect modification by length of residence, with health analyses repeated using a subset of participants who lived in their current exposure area (based on current and previous residential address) for more than 1, 3, 5, and 10 years. To examine the dose-response curves, models using a continuous measure of pollutant exposure were also run. Only those for PM_{2.5} are presented (Appendix D).

LAHS FINDINGS

In general, the contribution of Logan Airport to air pollution in surrounding communities was estimated to be small, particularly for areas beyond a one mile radius of the airport. Within one mile of the airport center, airport-related pollutant concentrations were higher and more variable, although were still low. Annual average contributions of PM_{2.5} within one mile of the airport, for example, ranged between 0.09 and 0.65 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), accounting for approximately 1-7% of the annual average PM_{2.5} concentration measured in 2005 in Lynn and City Square, Boston.

For adults, prevalences of the health outcomes of interest ranged between 3.3% (coronary heart disease) to 24.7% (asthma hospitalizations among asthmatics). For children, similar trends were observed, with prevalences ranging between 3.6% for probable asthma to 41% for asthma hospitalizations. Of note, the prevalence of each outcome was based on different sample sizes as the number of participants answering each survey question differed. The number of participants answering the question regarding asthma hospitalizations was by far the lowest, with only 673 and 328 of the over 6,000 adults and 2,000 children responding, respectively.

Epidemiologic analyses that adjusted for confounders and other covariates showed that airport-related pollution and noise had little detectable impact on the health measures assessed for residents living in surrounding neighborhoods of Logan Airport. In models adjusting for important confounders and predictors, airport-related exposures were not significantly associated with any of the cardiac or auditory health outcomes and most of the respiratory health outcomes.

Of the many outcomes examined, airport-related exposures were associated only with two outcomes, the prevalence of 1) probable asthma in children, and 2) COPD in adults. The prevalence of probable asthma in children living in the high exposure area, for example, was estimated to be three to four times higher as compared to that for children living in the low exposure area (Odds ratio [OR]=3.6, 95% confidence interval [CI]: 1.1-11), after adjusting for covariates. Correspondingly, COPD prevalence in adults was higher in the high versus low exposure areas (OR=1.5, 95% CI: 0.9-2.5), especially for participants who lived at their current residence for 3 or more and 5 or more years (3+ years $OR_{high}=1.8$, 95% CI: 1.1-3.0). Restricted analyses based on years of residence also showed a greater likelihood of current asthma with medication use in high versus low exposure areas (5+ years $OR_{high}=1.7$, 95% CI: 1.0-3.0). These stronger associations, however, disappeared when analyses were restricted to individuals living 10 or more years at their current home, possibly due to the smaller sample size.

As with the categorical exposure models, the model evaluating continuous $PM_{2.5}$ did not show higher odds of cardiovascular health effects with greater $PM_{2.5}$ exposures. For adults, only hospital admission for asthma was significant (OR=1.06, CI: 1.01, 1.10 for $0.01 \mu g/m^3$ increase in $PM_{2.5}$). For children, only probable asthma was significant (OR=1.03, CI: 1.00, 1.06 for every $0.01 \mu g/m^3$ increase in $PM_{2.5}$).

IMPLICATIONS OF LAHS FINDINGS

The LAHS is one of few epidemiology studies (Table 3) we could identify that examined the relationship of airport-related pollution and prevalence of respiratory (Miyakita et al., 2002; Lin et al., 2008; Huss et al., 2010) or cardiac conditions. It is also one of only two to examine the association of noise and auditory health of surrounding populations (Table 4), while many have associated aircraft noise with cardiovascular disease.⁵ As such, it is the first study to show airport-related associations with prevalence of probable asthma in children and on COPD in adults and null associations of airport pollution with cardiac and other respiratory outcomes. Correspondingly, it is the only study we identified that found no association of airport noise and auditory health in nearby residents, as the limited previous studies showed greater noise-induced hearing loss in children whose school was located in a flight path (Chen and Chen, 1993) and reduced hearing ability for individuals living near airports (Chen et al., 1997). Whether the LAHS's null and significant findings are generalizable or relevant to other airport settings, study populations, and time periods is not clear, given the lack of other epidemiological studies of airport-related air pollution.

By being the first study to examine associations of both airport-related pollution and noise with disease prevalence, the LAHS adds to the scientific literature regarding 1) the fate and transport of pollutants with large airport-related emissions; 2) the prevalence of self-reported cardiac, respiratory and auditory conditions near airports; and 3) the relation of these conditions to airport-related pollution and noise. In so doing, the LAHS provides important background information on the pollution and noise profiles and disease prevalence near airports, which can be used to generate new hypotheses and questions for further study. LAHS findings, however, cannot be used to infer causation or lack thereof, given its cross-sectional design, where all health, exposure, and other risk factor data were collected at one point in time. While providing a useful “snapshot” of the health and exposures of Logan Airport neighbors, they cannot discern cause and effect, because it is impossible to determine whether exposures to airport pollution and noise preceded or followed the diagnosis of disease.

Below we discuss specific components of the LAHS study design, their impacts on study findings, their relationships to previous health findings, and propose future directions.

⁵ Note that several health impact assessments for airports have been conducted. By design, however, these assessments do not examine associations of airport-related pollution and health effects. Rather these studies use modeled exposures and existing dose-response curves to estimate increases in airport-attributable mortality. Consequently, their findings are not directly comparable to LAHS results.

EXPOSURE ASSESSMENT APPROACH

The LAHS's exposure assessment approach was based on three steps: 1) modeling PM_{2.5}, CO, NO₂, SO_x, and VOCs concentrations and noise from airport activities; 2) estimating exposures for each survey respondent based on these modeled concentrations or noise contours; and 3) grouping modeled exposures into a single exposure measure indicating high, medium, or low exposures for either air pollution or noise.

Exposure Modeling

Results from the first exposure assessment step were consistent with previous airport and traffic studies that show airport-related activities to account for only small fractions of total PM_{2.5} and NO₂ concentrations in nearby neighborhoods (Adamkiewicz et al., 2010; Ashok et al., 2013; Dodson et al., 2009; Zhu et al., 2002), with these fractions decreasing sharply with increasing radial distance from the airport or roadway (Sioutas et al., 2005; Adamkiewicz et al., 2010). Notably, similar findings have been reported for airports with diverse characteristics, including the medium-sized T.F. Green Airport in Rhode Island (Adamkiewicz et al., 2010; Dodson et al., 2009) and the large Los Angeles International Airport in California (Westerdahl et al., 2008; Diez et al., 2012).

Similarly for noise, 2005 contours for Logan Airport showed the highest noise impacts at the periphery of the airport, with airport-attributable noise generally decreasing with increasing distance from the airport. As with all airports, airport-associated noise exposures at Logan Airport primarily result from aircraft movements. As a result, noise pollution tends to follow patterns of the main flight paths at take-off and landing (Jarup et al., 2005). Noise exposures within the LAHS study area ranged narrowly, with noise exposures for study participants ranging from less than 60 to 65 dBA. These ranges are in keeping with noise studies of other airports (Jarup et al., 2005). Although not directly comparable, these values were all lower than health-based guidelines to protect against hearing impairment, which establish a lower bound 24-h cumulative noise exposure limit of 70 dBA, below which the risk for hearing impairment would be negligible (EPA, 1974; WHO, 1999).

Participant-Specific Exposures

In the second step, airport-related pollutant and noise exposures were successfully characterized for the study participants. Based on this characterization, survey respondents were grouped by their overall pollutant exposures and by their noise exposures. By creating one air pollutant exposure measure for each study participant, the study was able to reduce the number of needed health analyses by running one model to examine associations between air pollution and each health outcome rather than five individual-pollutant-specific analyses. As a result, issues associated with multiple comparisons were minimized. In addition, by categorizing exposures, the results from the health analyses were easily interpretable, allowing straightforward

comparisons of the health responses for high and low exposed participants. On the contrary, the general categorization of exposures detracts from the ability to determine the individual health impacts from separate air pollutants, which likely have different health effects. The exposure categorization also assumes, for example, that exposures of those in the high end of the medium exposure category are not equivalent to those in the low end of the high category, when they may be very close in actual exposure levels.

Importantly, LAHS participant-specific exposure estimates offer additional opportunities to expand on and improve the LAHS health analyses. For example, additional analyses should be performed using the participant-specific pollutant estimates as the exposure measure, with the analyses performed separately for each of the five pollutants.

By using the actual participant-specific estimates of pollution, health analyses will be able to better capture spatial variation in exposures. This will in turn help to reduce exposure misclassification and increase the ability of the LAHS to detect exposure-health associations. This exposure misclassification may otherwise be substantial, given LAHS findings showing substantial spatial variability in NO_x and PM_{2.5} concentrations even within the low, medium and high categories.

Correspondingly, for noise, participant-specific estimates based on the census block of residence should also be used as the exposure measure in LAHS auditory health analyses. Participant-specific estimates can be used to assess the shape of the dose-response curve for noise and to determine whether this curve has a threshold below which noise has no impact on health. This analysis is critical given findings from recent studies of airport noise and cardiovascular disease, which consistently show threshold effects. For example, in an analysis of 89 airports in the U.S., Correia et al. (2013) showed consistent, statistically significant associations between noise exposures and cardiovascular hospital admissions only when noise exposures were greater than 55 dBA. Correspondingly, thresholds ranging between 50 dBA (Eriksson et al., 2010) and 60 dBA (Huss et al., 2010) have been shown in other studies of noise and cardiovascular disease, and some have indicated no threshold for nighttime noise and some cardiovascular outcomes (Floud et al., 2013). These findings suggest that the possibility of a threshold for noise and auditory associations in the LAHS as well. It is important to note that given the relatively narrow distribution of observed modeled noise exposures, the ability of the LAHS to identify this threshold may still be limited even with participant-specific noise measures. To address this limitation, alternate exposure measures for noise, such as peak noise, should be explored, as these measures may be more variable and may better capture participant's airport-related noise exposures, which are intermittent and generally associated with airplane takeoffs and landings.

The use of participant-specific exposure measures for air pollution and noise has other potential benefits. For example, they can be used to examine the joint impacts of air pollution and noise on

health, which may be greater than those for air pollution or noise alone. Correspondingly, participant-specific exposure measures can also be used to assess the impacts of air pollution while controlling for noise and vice versa, as it is possible that each may confound the other's association with health. These new analyses are critical given that some participants may have high exposures to both noise and PM_{2.5}, while others may experience high exposures to one but not the other. Even with use of participant-specific exposure estimates these do not necessarily reflect true exposures to either air pollution or noise, since personal exposure would vary based on times spent at home, other work or transit exposures, among other factors. They would however provide estimates of potential exposures at home locations.

Specific Markers of Airport Exposures

New analyses should also be conducted using air pollutant and noise measures that are more specific markers of airport-related activities. For air pollution, these new markers would help to address a limitation of the LAHS, which assessed airport-related exposures using pollutants that are emitted by many non-airport related sources (PM_{2.5}, NO₂, and total VOCs in particular). As evidenced by the small contribution of Logan Airport emissions to neighborhood PM_{2.5}, NO₂ and VOC levels, airport activities had little influence on neighborhood concentrations of and thus exposures to these pollutants. As a result, the ability of the study to separate the impacts of airport- from non-airport exposures was likely poor, raising concerns about the possibility of confounding by non-airport sources, even with controls for background pollution and distance to road.

Although unique markers of airport related activities are not available, scientific literature supports the use of ultrafine particles (UFP), elemental carbon (EC), and certain polycyclic aromatic hydrocarbons (PAHs) as more specific markers of airport related activities. Studies of airports and major roadways, for example, show that concentrations of UFP and EC (but not PM_{2.5}) near airports are substantially higher than background levels, with strong and measurable impacts within 300-1000 meters of an airport or major roadway (Zhu et al., 2002; Sioutas et al., 2005; Westerdahl et al., 2008; Dodson et al., 2009; Hsu et al., 2012). Work by Hudda et al. (2014) showed elevated levels of ultrafine particles at distances up to 10 miles downwind of the Los Angeles International Airport, and the authors suggest that airport impacts on air quality are significantly underestimated. These findings suggest that airport-related UFP and EC concentrations will be substantially higher in the Winthrop and East Boston neighborhoods that lie within 1000 meters of the airport, offering the possibility of greater airport-related exposure contrasts and minimal confounding by non-airport related sources.

HEALTH OUTCOMES AND RISK FACTORS

Selection and Recall Bias

In 2005, LAHS administered a one-time telephone survey of health outcomes, demographic and risk factors for a random sample of individuals living in surrounding communities. This survey administration was somewhat successful, with a response rate of survey participants between 35% and 60%. This response rate raises some concerns about possible selection bias within the LAHS, where non-respondents who had phones that were always busy or never answered differed systematically from the study participants. While the proportion of response was similar between the exposure areas, it is possible to identify scenarios where bias may occur in this situation. For example, non-respondents may have been sicker than respondents and thus not able to answer their phone. If selection bias did occur, the LAHS may have underestimated (as in the case of this example) or overestimated disease prevalence, reducing ability to detect exposure-disease associations when they actually existed. While data are not available to ascertain whether and to what degree selection bias may have existed, it is important to interpret LAHS findings considering the possibility of selection bias.

In addition to selection bias, the impact of possible recall bias associated with the self-reported outcomes is important to consider, as is the case with all studies involving survey data. Recall bias can occur when study participants of one group intentionally or unintentionally report information differently than another group.⁶ In the case of the LAHS, this could occur if participants living in the high exposed area report their health outcomes or risk factors differently than participants living in the low exposed area. In this example, this differential misclassification by exposure group would likely bias the association between airport exposures and health away from the null. Given the involvement of the expert community panel, it is possible that the potential for recall bias was minimized during the design and performance of the study. However, recall bias in the LAHS cannot be ruled out, as no verification of the self-reported information was performed.

Non-Specific Health Outcomes

As with its exposure measures, the LAHS health outcomes are non-specific to the impacts of the measured air pollutants, as many risk factors for the disease exist beyond air pollution. Potential contributors to chronic obstructive lung disease (COPD), for example, are several, including cigarette smoking (responsible for 80% to 90% of all COPD), second hand smoke, air pollution, genetics, and severe childhood respiratory infections. Potential contributors to asthma in children are even more numerous, with the scientific literature linking asthma in children to a wide range of risk factors ranging from airborne allergens (e.g., pollen, animal dander, mold, dust mites) to

⁶ Note that inaccurate reporting by itself does not result in recall bias if these inaccuracies occur equally in exposed and non-exposed groups. Instead this inaccurate report will lead to measurement error, which, in turn, usually leads to a loss of statistical power.

respiratory infections, physical activity, cold air, second hand smoke and air pollutants, and certain medications, among other factors (NHLBI, 2007). Importantly, the LAHS controlled for many risk factors and predictors of disease in its health models, using collected information about participant demographics and behaviors, such as smoking, second hand smoke, household allergens and mold, alcohol use, and family history of heart disease. However, given the general nature of the examined health outcomes, it may not have been possible to account for all risk factors in the health models.

Single Measurements

Because most of the LAHS health outcomes develop slowly with some outcomes relatively rare, studies of airport-related health impacts may require the observation of many participants over a long period, rather than a snapshot in time. To improve the validity of the study and its ability to examine airport-related health impacts, additional surveys should be administered to the same cohort, with this survey asking not only about doctor-diagnoses of diseases, but also about intermediate or subclinical end-points that can be used as surrogates or precursors of disease, such as blood pressure, sleep disturbances, and medication usage.

RELATIONSHIP TO PREVIOUS FINDINGS

Despite its limitations, findings from the LAHS study are useful in their ability to generate hypotheses for further study. The LAHS study found significant associations between airport-related pollution and two respiratory health outcomes, probable asthma in children and COPD in adults, which is notable given the non-specific exposure and health measures. Significant findings for asthma and to a lesser extent COPD are consistent with observed associations reported in cohort studies examining impacts of exposures to motor vehicles, a source common to airport operations and with similar pollution emission profiles as aircraft. In these studies, associations between PM_{2.5} and other motor vehicle related exposures have been shown for respiratory outcomes, including asthma prevalence and severity in children (McConnell et al, 2010; Jerrett et al., 2008; Salam et al., 2008). While prevalence of COPD in adults has been associated in few previous studies with traffic- or airport-pollution (Schikowski et al., 2014), a number of studies have shown individuals with COPD to be more susceptible to motor vehicle-related health impacts (Andersen et al., 2011; Finkelstein et al., 2011; Nuvolone et al., 2011). These findings point to the need for additional studies that focus on airport-related respiratory impacts, particularly in children and older adults.

Although the examined associations between airport pollution and cardiac health outcomes were null, previous epidemiological studies of traffic have consistently shown strong associations between traffic pollution, which is also combustion-related, and cardiac outcomes. For example, Peters et al. (2004) showed greater exposure to traffic based on time-activity patterns on the day of an MI in a case-control study. Others have shown traffic-related air pollution to be associated

with increased hospital admissions for cardiovascular outcomes (Bell et al., 2014; Gan et al., 2011); mortality due to stroke (Forester et al., 2014); lung cancer (Nafstad et al., 2003; Nyberg et al., 2000); cardiovascular mortality (Jerrett et al., 2009). As discussed previously, studies have also shown higher air pollution levels near airports than those modeled in the LAHS (Westerdahl et al., 2008; Dodson et al., 2009; Adamkiewicz et al., 2010; Hsu et al., 2013). These findings suggest that the impacts of airport-related pollution on cardiac health warrant further study in neighborhoods surrounding Logan Airport, especially given the limitations in the ability of LAHS cross-sectional study design to examine exposure-effect associations.

Correspondingly, many studies have linked noise exposures to a variety of adverse health impacts, including sleep disruption, heart disease, hypertension, stress, annoyance, hearing impairment, hospital admissions, and impaired learning in children (Hammer et al., 2014; Stansfeld et al., 2005). In the largest study of airport noise and health in Medicare patients conducted to date, for example, Medicare patients living near 89 US airports showed a 3.5% increase in hospital admissions for cardiovascular disease for the highest category of noise exposure compared to the lowest (Correia et al., 2013). Although these and other sleep and cardiac outcomes were not examined in LAHS with relation to noise, previous findings linking airport noise to cardiovascular outcomes points to need for additional analysis of LAHS data to assess noise impacts on cardiovascular health outcomes.

Likely the most extensive study of noise exposures on health was conducted of residents living near Heathrow Airport in London (Hansell et al., 2013). That study showed high compared to low noise exposures to be associated with a 24% increased risk of stroke and an approximately 10% increased risk for other cardiovascular diseases.

FUTURE WORK

While exploratory, the LAHS was an important first step in efforts to assess health impacts of Logan Airport on East Boston and other impacted areas. Continued efforts should be undertaken to further assess the impacts of Logan Airport on the health of surrounding populations, either through reanalysis of existing LAHS data or through additional health data collection and/or exposure reconstruction. A brief summary of these future or continued efforts is provided below:

- Air dispersion modeling should be performed to estimate airport-related concentrations of pollutants that are more specific tracers of airport emissions, such as UFP, EC, PAHs, and certain VOCs. To do so, emission inventories specific to these pollutants at Logan Airport are needed.
- The exposure modeling conducted for the LAHS estimates contributions of Logan-related traffic. Additional evaluation of traffic impacts on local neighborhoods of traffic traveling through those areas to get to Logan may have greater impacts on health due to proximity to exposed populations.
- Health models should be run using the participant-specific exposure estimates for each pollutant and noise, rather than the grouped “high,” “medium,” and “low” exposure categories. These participant specific exposures should be used to assess dose-response curves and the existence of thresholds.
- Noise exposures should be estimated for peak exposure values, with peak exposures used in health models.
- Health models should examine the joint impacts of air pollutants and noise exposures (as continuous variables) on specific health outcomes.
- Health models should examine the association between air pollution and health outcomes controlling for noise and vice versa.
- Health models should examine the association of noise with prevalence of doctor diagnosed cardiac conditions.
- The survey should be re-administered to the populations surrounding Logan Airport to allow the airport-related impacts on health to be examined for these populations over time.

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TABLES

Table 3 Summary of Airport Air Pollution and Health Studies

Year	Study	Location	Health Endpoints	Epi. Methods/Study Design	Exposure	Findings
2013	Ashok et al.	United States (multiple airports)	Risk-based mortality	Response surface model	Modeled environmental exposure to PM _{2.5} (summed from BC, CO, sulfate, nitrate, OC, ammonium)	Modeled aviation landing and takeoff emissions attributed to ~195 [90% CI: 80–340] early deaths in 2005 and ~350 [90% CI: 145–610] in 2018.
2013	Yim et al.	20 airports in United Kingdom	Risk-based mortality	Modeled PM _{2.5} , mortality concentration-response functions and varying airport emissions scenarios	Modeled exposures to PM _{2.5} , components and gases	110 deaths annually attributable to aircraft emissions in UK.
2012	Levy et al.	United State (multiple airports)	Risk-based mortality	Modeling analysis PM _{2.5} mortality concentration-response functions and population projections	Community Multiscale Air Quality model	Scenarios predicted 6.1 fold increase in aviation-related health impacts due to increased flight activity, population growth and aging, and non-aviation concentration changes
2010	Barrett et al.	Global	Risk-based mortality	Modeling analysis of cruise emissions	Modeled environmental exposure to BC, OC, sulfate, nitrate, ammonium and total PM _{2.5}	An estimated 8,000 premature deaths per year attributable to aircraft cruise emissions. Deaths represent 80% of the total deaths from aircraft emissions.
2010	Huss et al.	Switzerland	Cancer of the trachea, bronchus or lung	Cohort study focusing on cardiovascular effects of aircraft noise and PM ₁₀	Geospatial model of noise and PM ₁₀	Increased odds of cancer (OR=1.05, CI: 1.04–1.06) for every 10 µg/m ³ increase in PM ₁₀
2008	Lin et al.	Three NY airports	Hospital admissions for respiratory conditions	Cross-sectional	Residential proximity to large airports (12 miles)	Increased admissions by 37 – 52% for children aged 0-4 years living within 5 miles of airport
2002	Miyakita, et al.	Two military bases in Japan	Respiratory, general health, psychosocial indicators	Cross-sectional	Residents living in proximity to two US airbases	Increased prevalence of respiratory symptoms with increasing L _{dn} above 60 dB at home location

Epi. epidemiological
 PM_{2.5} particulate matter that is 2.5 micrometers or smaller in size
 BC black carbon
 CO carbon monoxide
 OC organic carbon
 CI confidence interval
 OR odds ratio
 µg/m³ micrograms per cubic meter
 PM₁₀ particulate matter that is 10 micrometers or smaller in size
 L_{dn} day-night average sound level (equivalent to DNL)
 dB decibel

Table 4 Summary of Airport Noise and Health Studies

Year	Study	Location	Health Endpoints	Epi. Methods/ Study Design	Exposure	Findings
2007	Aydin et al.	Frankfurt Airport – Germany	Noise perception, blood pressure, heart rate	Ecological	Noise perception and sleep quality, environmental exposure measured	Longer periods of aircraft noise experienced higher average blood pressure than those exposed to shorter periods
2006	Babisch	Multiple	Blood pressure, hypertension, ischemic heart disease, including myocardial infarction	Literature review	Transportation noise	Increased evidence from studies.
2009	Babisch et al.	Near 7 major European airports in 6 countries (HYENA study)	Annoyance	Cross-sectional	Aircraft noise INM contours	Noise annoyance associated with aircraft greater in HYENA study than 2004 EU assessment.
2012	Babisch et al.	Near 7 major European airports in London, Berlin, Stockholm (2), Amsterdam, Milan, Athens (HYENA study)	High blood pressure and noise annoyance	Cross-sectional (n=4,861)	Aircraft noise INM contours	No clear trends in noise-annoyance associations. Hypertension and aircraft noise exposure OR=1.04 (CI: 0.96–1.12)
1993	Chen and Chen	Taiwan	Hearing loss	Cohort, Ecological	Noise exposure determined by school location	Greater noise-induced hearing loss associated with location of school in flight path
1997	Chen et al.	Taiwan	Damage to the peripheral cochlear organs	Cohort	Environmental exposure determined by home location	Reduced hearing ability for individuals living near airport
2013	Clark et al.	Heathrow Airport, UK	Noise annoyance and reading comprehension	Cohort (n=461)	Noise contours for school location	Increased annoyance with increased noise exposure at school
2013	Correia et al.	United States (multiple airports)	Hospital admissions for cardiovascular disease	Retrospective cohort	Noise exposure determined by residence (zip code)	10 dB increase in aircraft noise exposure corresponds to 3.5% increase in hospital admissions
2011	Floud et al.	Near 7 major European airports in 6 countries (HYENA study)	Medication use	4,861	Aircraft noise INM contours	Nighttime aircraft noise and antihypertensive use in UK (OR=1.34, CI: 1.14-1.57) and Netherlands (OR=1.19, CI: 1.02 -1.38). Anxiety medicine use associated with daytime (OR=1.28, CI: 1.04-1.57) and nighttime (OR=1.27, CI: 1.01-1.59) aircraft noise
2013	Floud et al.	Near 7 major European airports in 6 countries (HYENA study)	Heart disease and stroke	Cross-sectional	Aircraft noise INM contours and road traffic noise; Dispersion model for NO ₂	10 dBA increase in night-time aircraft noise associated with heart disease/stroke for those living in same location for ≥20 years (OR= 1.25; CI: 1.03, 1.51)
2004	Franssen et al.	Schiphol Airport, Netherlands	General health and medication use	Cross-sectional (n=11,812)	Modeled noise contours (4 noise metrics)	Adjusted ORs ranged from 1.02 to 2.34 per 10 dBA increase in L _{den} for poor self-rated health: cardiovascular and non-prescribed sleep medication use

Table 4 Continued

Year	Study	Location	Health Endpoints	Epi. Methods/ Study Design	Exposure	Findings
2002	Goto and Kaneko	Fukuoka (Japan) airport	Blood pressure	Cohort (n=469 women cases; n=1,177 controls)	1974 Weighted Equivalent Continuous Perceived Noise Level (WECPNL) contour at home location	Systolic and diastolic BP not associated with aircraft noise levels in high and low airport noise areas.
2002	Haines et al.	Heathrow London airport	Performance tests in schools	Cross-sectional	Noise contours	Chronic aircraft noise associated with poorer school performance in reading and mathematics but may be confounded by socioeconomic factors.
2013	Hansell et al.	Heathrow – London, United Kingdom	Hospital admissions and mortality for stroke, coronary heart disease, and cardiovascular disease	Small area study	Modeled noise using UK Civil Aircraft Noise Contour Model (ANCON)	Increasing linear trends for hospital admissions with increasing noise levels
2008	Haralabidis et al.	Athens, London, Milan, Stockholm	Overnight measured blood pressure and heart rate	Small cohort study (n=149)	Measured noise in homes	Increase over 15 min intervals in which an aircraft event occurred. Non-significant increase in HR.
2011	Haralabidis et al.	Athens, London, Milan, Stockholm	24-hour blood pressure	Small cohort (n=149)	Measured noise in homes	No significant effect of aircraft noise on BP
2010	Huss et al.	Switzerland	Mortality from myocardial infarction	Cohort	Geospatial noise and air pollution models	Mortality increased with increasing level and duration of aircraft noise.
2008	Jarup et al.	Near 7 major European airports in 6 countries (HYENA study)	Hypertension	Cohort	INM noise model contours	10-dB increase in night-time aircraft noise associated with increased risk of hypertension (OR=1.14, CI: 1.01–1.29).
2014	Munzel et al.	Europe	Cardiovascular effects	Review article	Traffic and airport-related noise	--
2015	Seabi J et al.	South Africa	Reading comprehension in children	Prospective follow-up study of effects of chronic aircraft noise exposure	Noise measurements outside of 5 schools (L_{eq} , L_{amax})	Aircraft noise associated with lower reading comprehension scores, effects are sustained after cessation of exposure
2009	Selander	Near 7 major European airports in 6 countries (HYENA study)	Saliva cortisol	Cohort (n=439)	INM contours	Elevated morning saliva cortisol in women
2005	Stansfeld, et al.	89 schools located near 3 major airports in Netherlands, Spain, and UK	Children's cognition (9-10 year olds)	Multi-national cross-sectional (n=2,844)	Aircraft contours, modeled and measured noise inside and outside of schools	Associations between chronic aircraft noise and impairment of reading comprehension ($p=0.0097$), recognition memory ($p=0.0141$) and annoyance ($p<0.0001$).
2010	Swift H.	Multiple	Review of health effects associated with noise	Literature review	Cardiovascular health effects and airport noise	--

Table 4 Continued

Epi	epidemiology
HYENA	Hypertension and Exposure to Noise near Airports
INM	Integrative Noise Model
EU	Europe
n	number
OR	odds ratio
CI	confidence interval
dB	decibel
NO ₂	nitrogen dioxide
dBA	A-weighted decibel
≥	greater than or equal to
L _{den}	day-evening-night equivalent level (weighted average sound level over 24 hours that increases sound levels between 7 -11 pm by 5 dBA and by 10 dBA between 11 pm and 7 am
L _{eq}	equivalent sound level, continuous average noise level over a 24-hour period, with averaging based on sound energy
L _{Amax}	maximum A-weighted sound level over a given period, assumed to be 24 hours unless specified
p	p-value