Logan Airport Health Study

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Massachusetts Department of Public Health

Bureau of Environmental Health

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EXECUTIVE SUMMARY

Background

Chapter 159 of the Acts of 2000 included a line item directive that stated "*the Director of the Bureau of Environmental Health Assessment* [presently named the Bureau of Environmental Health] *of the department shall conduct an environmental risk assessment of the health impacts of the General Lawrence Logan Airport in the East Boston section of the city of Boston on any community that is located within a 5 mile radius of the airport and is potentially impacted by the airport.*" The 17 communities located either fully or partially within the five-mile radius of the airport include Boston, Brookline, Cambridge, Chelsea, Everett, Hull, Lynn, Malden, Medford, Melrose, Milton, Nahant, Quincy, Revere, Saugus, Somerville, and Winthrop. Based upon this directive the Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH) designed and conducted the Logan Airport Health Study (LAHS).

In the early stages of design of the LAHS, the MDPH/BEH formed a Community Advisory Committee (CAC) composed of area residents, local health officials and technical experts in the areas of epidemiology, biostatistics, survey design and administration, and air modeling. With input from the CAC, the MDPH/BEH designed and implemented a cross-sectional disease and symptom prevalence study that investigated the associations between opportunities for exposure to airport emissions and adverse health outcomes. Environmental exposure data included noise and air emissions. Air pollution emissions are primarily from aircraft operations, ground service equipment, transportation vehicles on airport property, and the airport power plant. The primary source of noise from the airport is that of aircraft takeoff and landing operations. Three categories of health outcomes were evaluated: respiratory, cardiovascular, and auditory effects.

Surveyed population and health questionnaire

Following a pilot study aimed to test survey methods initiated in 2002, interviews for the LAHS commenced in 2005. A total of 6,072 eligible residents representing households from the 17 communities that make up the study area were interviewed. These adult respondents also provided information for 2,215 children living in those respective households. Therefore, the results of the LAHS represent information for 8,287 individuals living within five miles of Logan Airport. The telephone interviews, conducted in English and Spanish, collected information on the prevalence of targeted health outcomes as well as relevant demographic and risk factor information. Information was collected for one adult in each of the interviewed households and for any children aged 3-17 years.

Study participants were selected randomly so that the survey results could be considered representative of the study area. A strategy was also employed to oversample residents living closest to the airport to ensure an adequate sample size representing those with the highest potential exposure. Statistical weighting methods were then employed to account for the oversampling.

Modeled after nationally and internationally recognized health surveys, including the Behavioral Risk Factor Surveillance System (BRFSS) and the International Study of Asthma and Allergies in Childhood (ISAAC), the LAHS survey contained questions designed to assess the following categories of asthma and respiratory disease: lifetime asthma, current asthma, current asthma with medication use, probable asthma, asthma hospitalizations, and chronic obstructive pulmonary disease (COPD). Cardiovascular outcomes included non-fatal heart attack, angina, and coronary heart disease. Auditory effects included adult-onset hearing impairment and tinnitus. In addition to assessing the presence of health outcomes, the survey also included questions on risk factors associated with the targeted health outcomes, on potential exposures inside the home and at work, and questions reflecting demographic and socio-economic status. A ten-year residential history was also taken in order to provide some measure of each respondent's length of residency in the area.

Exposure assessment: Air pollutants

Air pollutant emissions typically associated with airport operations are largely due to incomplete combustion of fuel from aircraft, ground service equipment, and passenger automobiles on airport property. To estimate potential air pollution exposure specifically from airport-related operations (and thereby exclude possible exposure from non-airport related sources), advanced high-resolution air dispersion modeling (US FAA EDMS model version 5.1.3) was applied to predict ambient concentrations across the study area of five primary air pollutants (CO, NOx, $PM_{2.5}$, SOx, VOCs). The air dispersion modeling was based on 2005 emissions data, meteorological inputs, and aircraft takeoff and landing information for over 350,000 aircraft operations (94% of total 2005 operations). The modeling analysis also estimated emissions along flight paths up to an altitude of 3000 feet for takeoffs and landings. Data on emissions and airport operations were provided by Massport.

Using ArcGIS to map the 6,072 households included in the study, air pollutant concentrations were assigned to each respondent based on inverse-distance weighting of concentrations predicted from the air dispersion modeling. Given the very high correlation of estimated concentrations of the five pollutants across the study area, a combined exposure variable was developed that encompassed all pollutants. Annual average pollutant concentrations were selected for developing cut-points for the creation of three exposure areas estimating low, medium, and high potentials for exposure to airport-related air pollution.

Exposure assessment: Noise

MDPH/BEH also evaluated noise exposure across the study area using noise contours from aircraft operations provided by Massport. Using US FAA's Integrated Noise Model (INM), Massport models noise by considering the number of operations, types of aircraft operating during the day and night, use of runway configurations, and location and frequency of flight paths to and from the runways. Massport produces annual Day-Night Sound Level (DNL) contours that range from 60-75 dBA at five dB increments. The WHO

health-based guideline to protect against hearing impairment is 70 dBA. This guideline value indicates that the risk for hearing impairment would be negligible for a cumulative noise exposure below 70 dBA on a daily basis over a lifetime. Review of the 2005 INM noise contours indicated that the 70 dBA contour did not include a sufficient number of respondents to assign as the high noise exposure category. As a result, the 65 dBA contour was selected as the high noise exposure area. The medium noise exposure area was defined by households located in the 60-64 dBA noise contour and the low noise exposure area was defined by households located outside the 60dBA noise contour.

Statistical analysis

All analyses were conducted using SUDAAN, a statistical package designed for use with complex sampling methodologies, which incorporates weighting and variance calculations associated with the complex random digit dialing (RDD) sample design. Descriptive analyses were conducted separately for adults and children to assess the frequencies (percent of the population) with various socio-demographic characteristics. The prevalence of other potential factors (covariates) that may be associated with each specific outcome among adults and children were also estimated. The prevalence of each health outcome of interest was examined in the total population and among those living in each category of estimated airport-related air pollution or noise exposure.

Multivariate analysis (multiple logistic regression) was used to assess the association between the prevalence of targeted health outcomes and residence in low, medium, or high exposure areas while accounting for the impact of other potentially influential factors (confounders). Controlling for other factors known to be strong predictors of the health outcome being investigated is a statistical method to evaluate the association of interest, while adjusting for differences across exposure areas for other risk factors such as age, race, smoking status, family history of heart disease, or residential proximity to major roadways.

Results / Conclusions

The major conclusions of the Logan Airport Health Study are as follows:

- Air dispersion modeling of airport related emissions using a state-of-the-art model indicates that the highest predicted pollutant concentrations associated with airport-related operations are near the perimeter of Logan Airport and fall off rapidly with increased distance. This is a characteristic of the impact of sources that are primarily located near the ground surface.

- Consistent with findings of other airport studies, modeled concentrations of air pollutants are low relative to measured background air pollution concentrations.

- Evaluation of associations between airport-related pollutant concentrations and targeted health outcomes among the study area population detected some elevations in respiratory health outcomes in the high exposure area.

  Specifically:

- Among children, study results identified some respiratory effects indicative of undiagnosed asthma (i.e., probable asthma); children in the high exposure area were estimated to have three to four times the likelihood of this respiratory outcome compared with children in the low exposure area.

- Among adult residents, individuals diagnosed with chronic obstructive pulmonary disease (COPD) were statistically significantly more likely to have lived in the high exposure area for three or more years.

- There were no statistically significant differences in cardiovascular outcomes in the study population across the high, medium, and low exposure areas.

- There were no statistically significant differences with respect to hearing loss in either adults or children for those living in the high exposure area compared to the lowest exposure area.

Recommendations

- The results of this study should be reviewed by Massport and others to determine mitigating steps that can be taken across the study area.

- Massport has undertaken initiatives to reduce air pollution impacts within their control (e.g., providing infrastructure for compressed natural gas (CNG) fuels and electricity charging stations, Alternative Fuel Vehicle Program). Similar initiatives could be considered in consultation with local communities that would serve to further reduce the burden of indoor and outdoor sources of air pollution on residents in closest proximity to the airport.

- Massport has also been working with the East Boston Neighborhood Health Center (EBNHC) to address workforce issues among Massport employees. Massport could expand these efforts with the EBNHC as well as other community health centers to better address respiratory health notably among children in closest proximity to the airport.

- While air dispersion modeling indicates that the contribution from Logan Airport operations across the study area is relatively small, air pollution levels are higher in urban areas. Predicted pollutant concentrations were higher near the perimeter of the airport; thus, any methods that can be implemented to continue to reduce airport-related air pollution should be explored.

- MDPH/BEH should work with communities within the high exposure area (in whole or in part) on initiatives that would serve to further reduce exacerbation of pre-existing respiratory diseases (e.g., asthma and COPD) among residents.

  Specifically:

  - MDPH/BEH will continue to support MassDEP's efforts to reduce motor vehicle emissions including implementation of the Low Emissions Vehicle program and diesel engine retrofit initiatives;

  - Upon request MDPH/BEH's Indoor Air Quality (IAQ) Program staff will work with local municipalities to conduct IAQ assessments in schools and public buildings;

  - Upon request MDPH will work with local officials to address concerns that may be associated with local development initiatives;

  - MDPH/BEH will collaborate with the MDPH Bureau of Community Health and Prevention's Tobacco Cessation and Prevention Program on their efforts to work with local boards of health and tobacco-free community partnerships. These efforts enforce youth access and secondhand smoking laws and provide educational/outreach resources to support smoke-free workplace and housing programs.

1 PROJECT OVERVIEW

1.1 BACKGROUND

The Acts of 2000 of the Massachusetts General Court included a line item directive that stated "*the Director of the Bureau of Environmental Health Assessment* [presently named the Bureau of Environmental Health] *of the department shall conduct an environmental risk assessment of the health impacts of the General Lawrence Logan Airport in the East Boston section of the city of Boston on any community that is located within a 5 mile radius of the airport and is potentially impacted by the airport.*" Given the economic instability, the study has had varying levels of resources over the past decade. Despite irregular funding, the Massachusetts Department of Public Health, Bureau of Environmental Health (MDPH/BEH) conducted the Logan Airport Health Study (LAHS) to respond to that directive.

In the early stages of design of the LAHS, the MDPH/BEH formed a Community Advisory Committee (CAC) composed of residents, local health departments in the study area, and technical experts in the areas of epidemiology, biostatistics, survey design and administration, and air modeling. With input and assistance from the CAC, the MDPH/BEH designed and implemented a cross-sectional disease and symptom prevalence study investigating associations between potential exposure to airport emissions and adverse health outcomes among residents living in cities and towns located within a 5-mile radius of Logan Airport.

In order to address some of the challenges associated with conducting such a large prevalence study and to ensure the survey design would be sophisticated enough to detect an effect, if present, MDPH/BEH conducted a pilot study beginning in 2002. The draft survey instrument was pilot tested with a population living near an airport in Portland, Maine to improve the survey instrument. The refined survey instrument was again pilot tested in 2005 with 25 randomly selected individuals located in the LAHS area to ensure questions were clear and valid for the study area itself.

MDPH/BEH considered the potential health impacts on local communities of both noise and air emissions from Logan Airport. Air pollution sources at the airport include aircraft (takeoff, landing, taxiing, and use of auxiliary power units), ground service equipment, passenger and commercial motor vehicle fleets operating and parking on airport property, and the airport power plant. The primary source of noise from the airport is that of aircraft takeoff and landing operations. After review of the scientific literature on the health effects most frequently associated with exposure to the types of emissions typical of airports, MDPH/BEH identified respiratory, cardiovascular, and auditory effects as those of greatest potential public health significance. These outcomes also appeared to be of greatest interest to residents within the five mile study area.

MDPH/BEH gathered available environmental data in the study area related to airport operations. These included available ambient air quality monitoring data within the 5-mile radius of the airport (e.g., the Massachusetts Department of Environmental Protection–mandated monitoring stations for criteria air pollutants). Additionally, MDPH/BEH acquired an abundance of data provided by Massport that is not mandated to be reported such as complete flight path and scheduling information and detailed airport maps.

To address both the complexity in assessing environmental impacts of airport operations and any existing data gaps, MDPH/BEH designed a cross-sectional study to collect information on the prevalence of targeted health outcomes and various demographic and risk factor characteristics among residents living in the designated 5-mile radius study area. In addition, MDPH/BEH utilized available environmental and airport operations data to develop estimates of airport-related air pollution and noise exposure across the study area, thus allowing for an evaluation of the possible relationships between targeted health outcomes and these exposure opportunities.

1.2 GOALS AND OBJECTIVES

The overall goal of the Logan Airport Health Study was to determine whether residents living in areas with greater potential for airport-related exposures were more likely to experience respiratory, cardiovascular, or auditory effects compared to those residents living in areas with lesser potential for airport-related exposures.

The primary objectives were:

- To geographically stratify the study area into distinct exposure areas based on the best available data for predicting ambient concentrations of air pollution and noise associated with operations at Logan Airport.
- To collect information on specific health outcomes (i.e., respiratory, cardiovascular, and auditory endpoints) and other relevant information from a statistically representative sample of residents in the study area population.
- To evaluate associations between environmental exposures arising from airport operations and targeted health outcomes among the study population.

To meet these objectives, four major activities were carried out:

- A draft health survey was pilot-tested with a population living near an airport in Portland, Maine in April 2002 – April 2005.
- Based on the findings of the survey pilot study, a refined survey instrument was administered to residents within a 5-mile radius of Logan Airport to collect health outcome and relevant demographic and lifestyle data.
- Available air quality data related to airport operations were compiled and air dispersion modeling was conducted in order to estimate exposure areas impacted by air pollution associated with Logan Airport operations.

- Available noise modeling data were compiled and used to estimate exposure areas impacted by noise associated with Logan Airport operations.

Logan Airport is located within metropolitan Boston with various sources of ambient air pollution not related to the airport. Air pollutant emissions typically associated with airport operations are largely due to incomplete combustion of fuel and are the same as those emitted from other urban mobile sources (e.g., passenger automobiles, airport equipment). Available environmental monitoring data do not allow for precise apportioning of the contribution from airport sources and from other sources of air pollution in the study area. Thus, high-resolution air dispersion modeling was applied to predict ambient concentrations in the study area based solely on emission estimates associated with airport operations. For exposure to noise associated with airport operations, noise modeling of aircraft activities provided airport-related noise contours in the study area. Development of air and noise exposure modeling was made possible in part by Massport, who provided MDPH with access to a variety of data not mandated to be reported by MA regulations.

2 SUMMARY OF HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO AIRPORT-RELATED AIR POLLUTANTS AND NOISE

2.1 INTRODUCTION

Logan Airport's expansive aviation infrastructure contains a complex mix of transportation-related sources of air pollution and noise. Air pollution from airport-related activities is generated from aircraft engine emissions from passenger and cargo planes, ground service equipment (GSE), auxiliary power units (APU), aircraft refueling, and the airport's power plant. Passenger, commercial, and airport fleet vehicles traveling within the airport boundaries are also sources of air pollution at all airports. Noise is generated primarily from aircraft landing and takeoff phases and along flight paths in the study area. Emissions from airport operations are primarily from combustion of aviation fuel from aircraft and combustion of diesel fuel or gasoline from mobile source emissions (e.g., motor vehicle fleets, ground service equipment, and auxiliary power units, APUs, power plant). Fossil-fuel combustion contains a complex mixture of oxides of nitrogen (NOx), sulfur dioxide (SO_2), volatile organic compounds (VOCs), carbon monoxide (CO), carbon dioxide (CO_2), and particulate matter (PM_{10}, $PM_{2.5}$, ultrafine particles – collectively referred to as PM). In addition, numerous speciated VOCs, including hazardous air pollutants (e.g., benzene), are emitted from these sources. Fuel vapors and aerosols are also emitted during aircraft refueling, mobile source refueling, and from fuel storage tanks located on the grounds of the airport.

A review of the scientific literature was conducted to inform the development of the survey instrument. Based on this review, it was determined that cardiovascular, respiratory, and auditory health effects are the primary outcomes associated with exposure to combustion-related pollutants and noise. Thus, the primary health outcomes of interest in the LAHS survey were cardiovascular outcomes (heart attack, angina/coronary heart disease), respiratory outcomes (asthma, respiratory symptoms, COPD, bronchitis), and auditory outcomes (hearing impairment, tinnitus).

Given the voluminous nature of the scientific literature associated with air pollutants, the LAHS literature review focused on major scientific studies and compendiums by the US Environmental Protection Agency (US EPA) and the World Health Organization (WHO) on the health effects of air pollution (primarily criteria air pollutants) and noise. References are provided at the end of this report for additional information.

2.2 SUMMARY OF HEALTH STUDIES OF AIR POLLUTION

Few studies have been conducted to evaluate the potential health effects of exposure to airport-related emissions, specifically, but a significant body of evidence exists in the scientific literature that links certain health outcomes with exposure to specific pollutants contained within the complex mixtures of air pollution sources in general. For many of these studies, ambient concentrations of PM ($PM_{2.5}$, PM_{10}, ultrafine particles) and the gaseous co-pollutants (i.e., CO, sulfur dioxide (SO_2), NO_2 and ozone) are highly correlated with certain health outcomes. Although the studies do not provide clear evidence to distinguish impacts associated with individual pollutants or a combination of pollutants within the complex ambient mixture as the putative agent(s), research efforts over the past decade have focused on particulate matter as the primary pollutant of concern (US EPA, 2009). "Particulate matter," also known as PM or particle pollution, is a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. The size of particles is directly related to possible health effects associated with exposure to air pollution because small particles can pass through the throat and nose and enter the lungs. Once inhaled, these particles can affect the heart and lungs and cause serious health effects. Particulate matter is characterized by the aerodynamic size of the particles. The inhalable course fraction has an aerodynamic diameter ranging from 10 to 2.5 micrometers. The fine particle fraction has an aerodynamic diameter of 2.5 micrometers and smaller. Ultrafine particles are those particles below 0.1 micrometers.

Ultrafine particles (UFPs) are also important to consider because they are the major particulate fraction emitted from aircraft engines. In addition, UFPs are capable of efficiently carrying and transporting large amounts of absorbed or condensed toxic air pollutants into the respiratory tract (Sioutas et al., 2005). Current epidemiological evidence supports associations between inhalation of fine (≤2.5 μm) and ultrafine (≤0.1 μm) ambient particulate matter and increases in cardiovascular and respiratory morbidity and mortality (Delfino et al., 2005; Penn et al., 2005). Recently promulgated ambient air standards by the US EPA for nitrogen dioxide (NO_2) (US EPA, 2008) and SO_2 (US EPA, 2008a) are based on studies that have demonstrated respiratory health effects from short-term exposure to NO_2 and SO_2, including airway inflammation in healthy people; increased respiratory symptoms in people with asthma, and increased visits to emergency departments and hospital admissions for respiratory illnesses. Thus, people with heart or lung diseases, children and older adults are the considered at-risk populations for these health impacts.

Numerous scientific studies over the past 20 years have linked PM exposure to cardiovascular and respiratory health effects, including: premature death in people with heart or lung disease, nonfatal heart attacks, irregular heartbeat, aggravated asthma, decreased lung function, and increased respiratory symptoms, such as irritation of the airways, coughing or difficulty breathing.

CARDIOVASCULAR HEALTH OUTCOMES

According to Wilson and Culleton (2005) cardiovascular disease (CVD) is common in the general population, affecting most adults in the US over the age of 60 years. As a diagnostic category, CVD includes five major areas: coronary heart disease (CHD) (manifested by myocardial infarction (MI), angina pectoris, heart failure and coronary death); cerebrovascular disease (stroke and transient ischemic attack); and peripheral vascular disease (PVD). Coronary heart disease (CHD) contributes approximately one-third to one-half of the total CVD. The Framingham Heart Study reported a lifetime risk of coronary heart disease at age 40 years was 48.6% (95% CI 45.8–51.3) for men and 31.7%

(29.2–34.2) for women. At age 70 years, lifetime risk was 34.9% (31.2–38.7) for men and 24.2% (21.4–27.0) for women (Lloyd-Jones et al., 1999). Most individuals with coronary heart disease show no evidence of disease as the disease progresses before the first onset of symptoms, which may present as angina or a MI. Angina is a symptom of CHD and defined as chest pain or discomfort that occurs due to an inadequate supply of oxygen to the heart muscle.

The biological mechanisms linking air pollution to heart disease involve direct effects of air pollutants on the cardiovascular system, blood, lungs and/or indirect effects mediated through pulmonary oxidative stress and inflammatory responses (Peters et al., 2001). Studies on the latter topic seem to point to initiation of pulmonary and systemic oxidative stress and inflammation with a subsequent cascade of physiological responses that are capable of instigating cardiovascular effects (Brook et al., 2004). These effects include, but are not limited to, myocardial infarction and angina (Brook et al., 2004). Elderly patients, those with pre-existing cardiac or respiratory conditions, and diabetics have been identified as the primary individuals who may be at increased risk (Katsouyanni, 2003).

Historically, the primary health outcome observed in relation to particulate matter exposures has been mortality. In one of the first studies identifying the health impacts of particulate matter exposure, Dockery et al. reported in the Harvard Six Cities Study that long-term exposure to air pollutants is independently associated with cardiovascular mortality, with $PM_{2.5}$ and sulfates showing the strongest relationship to cardiovascular disease (Dockery et al., 1993). Similar results were found in a seminal study of American Cancer Society (ACS) data (Pope, 1995) in which long-term exposure to increases in mean $PM_{2.5}$ concentrations were associated with increases in cardiopulmonary mortality. A follow-up study by Pope et al. in 2002 found that fine particulate and sulfur oxide–related pollution were associated with all-cause mortality, lung cancer, and cardiopulmonary mortality. Follow-up studies of the Six Cities Study cohort (Laden et al., 2006) and ACS Study (Krewski et al., 2009) have also shown a decrease in mortality risk with decreases in $PM_{2.5}$ that have occurred in these study areas over the past few decades

Hospitalizations and emergency department (ED) visits for cardiovascular outcomes

Short-term exposure to $PM_{2.5}$, particularly in patients with underlying coronary artery diseases, has also been linked to acute coronary events, including MI (Peters et al., 2004; Miller at al., 2007; von Klot et al., 2005), angina/other ischemic heart disease (IHD)(Schwartz et al., 1995; Miller at al., 2007; von Klot et al., 2005; Pope et al., 2006b), dysrhythmias (Schwartz et al., 1995; Rosenlund et al., 2008), and heart failure (Schwartz et al., 1995).

US EPA reviewed several large multicity hospital admission and emergency department (ED) visit studies (MCAPS, Dominici et al., 2006; SOPHIA, Metzger et al., 2004; Peel et al., 2005; Tolbert et al., 2000; APHEA and APHEA-2, Le Tertre et al., 2002; HEAPSS, Von Klot et al, 2005; Multicity Studies in Australia and New Zealand, Barnett et al., 2006). They concluded that large studies from the US, Europe and Australia/New Zealand provide support for an association between short-term increases in ambient levels of $PM_{2.5}$ and PM_{10} and increased risk of hospitalization for total cardiovascular disease. US EPA cited studies showing associations between short-term increases in $PM_{2.5}$ and IHD, which represents a subset of all cardiac disease hospitalizations and is sometimes, termed "coronary heart disease," although the extent of the association varies considerably between studies. This category typically includes acute myocardial infarction (MI), acute and sub-acute forms of IHD, angina pectoris and other forms of chronic IHD. For example, the positive associations ranged from early studies showing a 0.6% excess risk of hospitalization for IHD per 10 $\mu g/m^3$ increase in mean PM_{10} (Schwartz et al., 1995) to a study in Salt Lake City, Utah (Pope et al, 1996) that found a 4.8% excess risk of acute MI or unstable angina per 10 $\mu g/m^3$ increase in $PM_{2.5}$. A 2001 study in Boston by Peters et al. found that a 10 $\mu g/m^3$ increase in the 2-hour average $PM_{2.5}$ levels was associated with a 17% excess risk of MI and a 10 $\mu g/m^3$ increase in the 24-hour average levels was associated with a 27% excess MI risk. In contrast, a study using the same methodology in Washington State (Sullivan et al., 2005) found no association, although US EPA cites other studies suggesting that substantial heterogeneity of effects are to be expected across different locations.

RESPIRATORY EFFECTS ASSOCIATED WITH EXPOSURE TO AIR POLLUTANTS

Asthma is a physician-diagnosed chronic inflammatory disorder of the airways and is one of the most common chronic diseases of childhood, affecting 4 million children nationwide (NHLBI, 2007). Asthma is a complex disorder characterized by variable and recurring symptoms, airflow obstruction, bronchial hyper-responsiveness, and an underlying inflammation. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible, either spontaneously or with treatment (NHLBI, 2010).

According to the National Asthma Education and Prevention Program Expert Panel (2007), the development of asthma appears to involve the interplay between host factors (particularly genetics) and environmental exposures that occur at a crucial time in the development of the immune system. Two major factors are the most important in the development, persistence, and possibly the severity of asthma: exposure to airborne allergens (particularly sensitization and exposure to house-dust mite and plant pathogens [Alternaria]) and history of viral respiratory infections (including respiratory syncytial virus [RSV] and rhinovirus). Other environmental factors under study include: tobacco smoke (i.e., exposure in utero is associated with an increased risk of wheezing, but a link to subsequent development of asthma has not been established), air pollution (e.g., ozone and particular matter) and diet (e.g., obesity or lower intake of antioxidants and omega-3 fatty acids). The specific association of these factors with the onset of asthma has not been clearly defined.

Hospital admissions and emergency department (ED) visits for respiratory outcomes

Epidemiological studies have reported significant positive associations between measured ambient air pollutants, notably particulate matter, and increased respiratory-related hospital admissions, emergency department and physician visits. As noted previously, US EPA recently promulgated 1-hour ambient air standards for NO_2 and SO_2

based primarily on studies that found respiratory health effects. These effects include airway inflammation in healthy people, increased respiratory symptoms in people with asthma, and increased visits to emergency departments and hospital admissions for respiratory illnesses.

Numerous epidemiologic studies have found excess risk for hospitalizations and ED visits associated with exposure to $PM_{2.5}$ and PM_{10} for all respiratory diseases combined, COPD admissions, and larger excess risks for asthma as well as increased physician visits. US EPA reviewed over 90 studies published since 2002 of the potential association between PM and hospital admissions and ED visits. In studies of children, the greatest risks observed by Barnett et al. (2005) using a 0-1 day lag, were increases in respiratory hospital admissions of 6.4% among infants (<1 year of age) and 4.5 % among children 1-4 years of age per 10 $\mu g/m^3$ increase in $PM_{2.5}$. Other studies of respiratory hospitalizations and ED visits have reported increased risk to children in association with $PM_{2.5}$, $PM_{10-2.5}$, and PM_{10} (US EPA, 2009). In adults, a study in Atlanta (Study of Particles and Health in Atlanta, SOPHIA) reported an excess risk of 1.3% per 10 $\mu g/m^3$ increase in 24-hour average PM_{10} levels for ED visits for combined respiratory causes among all ages (Peel et al., 2005). Similar findings were reported for additional years of data from the SOPHIA Study and in a study in Idaho (Ulirsch et al., 2007). However, a study of respiratory admissions and ED visits in Spokane, Washington found no association with any size fraction of PM (Slaughter et al., 2005). This is consistent with the finding that air pollution is associated with hospital admissions for respiratory diseases throughout the world but the magnitude of the effect differs across locations (US EPA, 2009).

Hospital admissions and emergency department (ED) visits for asthma

Many studies of PM exposure and hospitalizations for asthma have found a positive effect, but results often vary by age and PM size fraction studied. US EPA concluded that the effect estimates from studies of $PM_{2.5}$ and hospital admissions and ED visits for asthma for 10 pediatric studies are imprecise and not consistently positive across different age groups and lag times (US EPA, 2009). However, for studies of adults or adults and children

combined, associations of asthma hospital admissions and ED visits with $PM_{2.5}$ were observed in most studies. For both adults and children, studies of associations with PM_{10} are more consistently positive. Overall, US EPA has concluded that recent studies on $PM_{2.5}$ and respiratory hospitalizations and ED visits have been consistently observed. Most effect estimates were in the range of about 1-4% increased risk of hospitalization in areas with mean 24-hour $PM_{2.5}$ concentrations between 6.1 and 22 $\mu g/m^3$.

Respiratory symptoms

Multiple studies conducted throughout the world (North America, Europe, and Germany) have established significant associations of respiratory symptoms (increase in cough, wheeze, and bronchitis), and impairment of lung function among individuals exposed to PM (cited in Kappos et al., 2004). A growing body of evidence indicates that a substantial number of children and adults experiencing asthma-like symptoms are not diagnosed with asthma (Yeatts et al., 2003, Lee et al., 2007; US EPA, 2009).

Respiratory symptoms and medication use

Epidemiologic studies of asthmatic children have observed increased respiratory symptoms and asthma medication use in those exposed to higher concentrations of $PM_{2.5}$ and PM_{10} (US EPA, 2009) and ozone (Millstein et al., 2004). Similar studies among asthmatic adults are less consistent and these associations have not been demonstrated for healthy individuals.

Respiratory health effects from other criteria pollutants

Oxides of nitrogen (NO_x) is the general term that describes a mixture of highly reactive gases that contain nitrogen and oxygen in varying amounts (e.g., nitric oxide [NO] and nitrogen dioxide [NO_2]) that affect the respiratory system. Symptoms include wheezing, cough, reduced lung function, and increased airway responsiveness in normal and asthmatic individuals. NO_x may also be a co-factor in the tissue damage associated with exposure to ambient levels of ozone. High indoor NO_x exposure in children has been

linked to a reduced resistance to respiratory infections and an increased likelihood of respiratory illness including wheezing and persistent cough (van Strien et al., 2004). NO_x emissions have also been associated with increased severity of virus-induced asthma exacerbation (Chauhan et al., 2003) and respiratory illness in children and proximity to roadways (Brunekreef et al., 1997).

Sulfur dioxide (SO_2) is a direct respiratory irritant and contributes to the formation of sulfate and sulfuric acid adsorbed onto particulate matter. Compared to children from less polluted areas, children residing in industrial communities with high concentrations of SO_2 and total suspended particulates had higher lifetime prevalence of allergies, eczema, bronchitis, wheeze, shortness of breath, and cough without cold (Heinrich, 2003).

Volatile organic compounds (VOCs) are also linked to respiratory morbidity. In particular, the respiratory irritant acrolein and several aldehyde compounds are thought to interact within the complex mixture of ambient pollutants to exacerbate asthma and asthma-related symptoms (Leikauf, 2002). VOCs contribute to the formation of ozone and airborne secondary particles. Ozone is formed in the atmosphere from the reaction of combustion by-products - NOx, VOCs, and ultraviolet light.

Chronic obstructive pulmonary disease (COPD)

Although precise definitions vary, the American Thoracic Society (ATS) has defined COPD as "a disease state characterized by the presence of airflow limitation due to chronic bronchitis or emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and may be partially reversible." COPD is a nonspecific term that refers to a large group of lung diseases characterized by airflow obstruction with related symptoms (e.g., chronic cough, exertion dyspnea, expectoration, wheeze) (Mannino, 2002). Estimates of COPD prevalence primarily refer to patients with a diagnosis of either chronic bronchitis or emphysema (Sunyer, 2001).

COPD is a leading cause of death and disability/morbidity in the United States (Mannino, 2002). The burden of disease associated with COPD is largely underestimated

because a diagnosis of COPD is associated with increased risk for hospitalization and in-hospital mortality from other common diagnoses. For example, hospital discharges with primary or secondary COPD are more frequently diagnosed with other co-morbid conditions, including cardiac and pulmonary vascular disease, pneumonia and thoracic malignancies. Even in patients with severe COPD, a large number are admitted to the hospital for other co-morbidities, with COPD labeled as a secondary diagnosis (Holguin et al., 2005; Peel at al., 2007).

While cigarette smoking is the primary risk factor for an estimated 80-90% of COPD cases, occupational exposures and exposure to indoor and outdoor air pollutants are also risk factors for COPD. Other risk factors for COPD include second-hand smoke, history of childhood respiratory infections, and heredity. COPD has been associated with smoke from biomass fuel, history of pulmonary tuberculosis, chronic asthma, and respiratory tract infections during childhood (Salvi et al., 2009).

Decline in lung function/airway obstruction is a hallmark of COPD and studies suggest that ambient air pollution may play an important role in new onset and exacerbation of COPD (MacNee et al., 2000; MacNee et al., 2003). In a review of studies assessing the association between air pollution and COPD, Sunyer (2001) concluded that air pollution is related to the following: the increase of self-reported diagnosis of chronic bronchitis or emphysema as reported in two studies (NHANES and Adventist Health Air Pollution Study); an increased prevalence of breathlessness and mucous hypersecretion as reported in the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA) study; and lower lung function levels in adults as reported in cross-sectional studies. A number of studies have looked at the effect of air pollution on reduced or impaired lung function, or accelerated decline in lung function, and the majority have found that exposure to ambient air pollutants is associated with decreased lung function (as measured either by Forced expiratory volume in 1 second (FEV_1) or Forced vital capacity (FVC)) (Sunyer, 2001). Thus, individuals with COPD are more susceptible to the effects of air pollution (Anzueto et al., 2007). Ambient air pollution (PM_{10} and $PM_{2.5}$, ozone, NO_2, and SO_2) has been implicated in the exacerbation of COPD symptoms and increased hospital

admissions primarily in the elderly (Peel et al., 2007; Ko et al., 2007; Chuang et al., 2007; Lagorio et al., 2006; Schwela, 2000). Associations with COPD have been consistently observed in areas with high concentrations of $PM_{2.5}$ and PM_{10} in multicity and single city studies in the US and Canada (US EPA, 2009).

While most of the literature related to air pollution and COPD report significant associations between outdoor air pollution and COPD exacerbation including increased COPD hospital admissions (NMMAPS study) and mortality (Harvard six-cities study), only a small number of studies have evaluated the role of air pollution in the prevalence of COPD. COPD is a complex chronic disease with an evolving definition in epidemiological studies. The Global Initiative for Obstructive Lung Disease (GOLD) established guidelines in 2002 to address the need to streamline the definition for epidemiological studies. The definition recommended the use of a respiratory questionnaire and a post-bronchiodilator FEV_1/FVC ratio of <0.7 to diagnose COPD (Salvi et al, 2012).

Although smoking is a primary risk factor for COPD, findings from NHANES III study using post-bronchodilator spirometry (ratio of (FEV_1)/[FVC] <0.70) suggested that 25% of the COPD cases in the US are in never-smokers. Similar findings were reported in the UK and Spain (Salvi et al., 2009).

In the last decade as the concern of the burden of non-smoking COPD has increased from such factors as worldwide use of biomass fuel (Salvi, et al., 2009), studies have found an association between long-term exposure to air pollutants associated with traffic and COPD diagnosis. A study of 4757 women 55 years of age living in Germany that used a questionnaire to asses symptoms and risk factors (and the GOLD criteria to define COPD) found that a 7 $\mu g/m^3$ increase in five year mean PM_{10} concentrations was associated with a 5.1% decrease in FEV_1, a 3.7% decrease in FVC, and a 33% increase in prevalence of COPD. Women living less than 100 meters from a busy roadway (>10000 vehicles per day) also had significantly decreased lung function and COPD was 1.79 times more likely (95% CI 1.06-3.02) than those living farther away. Levels of PM_{10} and NO_2 were significantly associated with COPD. The results were consistent with the Swiss SAPALDIA except that

they appear stronger, which was attributed to women being more susceptible to COPD and respiratory symptoms caused by environmental factors than men. Another study in Sweden also found that living closest to traffic was associated with prevalence of COPD (Lindgren et al, 2009).

In the largest study conducted in Denmark of over 50,000 eligible subjects, of which 1786 participants had a first hospital admission for COPD, COPD incidence was associated with the 35-year mean NO_2 levels (hazard ratio, 1.08; 95% confidence interval 1.02–1.14, per interquartile range of 5.8 $\mu g/m^3$), with stronger associations in subjects with diabetes (1.29; 1.05–1.50) and asthma (1.19; 1.03–1.38) (Andersen et al., 2011). The investigators discussed the plausible biological mechanism of repeated inhalation injury to the lungs from long-term exposure to air pollutants and the chronic and progressive nature of COPD.

Although epidemiological evidence demonstrates an association between air pollution exposure and exacerbation of both asthma and COPD, a comprehensive review of existing studies of long-term exposure primarily to traffic-related pollutants and COPD among adults found that the evidence overall was suggestive but not conclusive (Schikowski et al., 2013).

SUMMARY OF HEALTH EFFECTS ASSOCIATED WITH TARGETED AIR POLLUTANTS

In the absence of specific studies of the health impacts of the complex mixture of air pollutants emitted from airport operations, the health effects associated with targeted air pollutants from airport operations were considered in this study. In summary, numerous epidemiological studies have shown an association between both short-term and long-term exposure to air pollutants, most notably particulate matter, and cardiovascular mortality as well as non-fatal cardiac events (myocardial infarction, angina/other ischemic heart events, dysrhythmias, and heart failure). The elderly and persons with pre-existing cardiopulmonary diseases, including coronary heart disease, are most susceptible.

In addition, epidemiological and toxicological studies have demonstrated an association between exposure to air pollutants and respiratory effects, notably

exacerbation of physician-diagnosed asthma (number of episodes, emergency department visits, and asthma hospitalization) in both children and adults. The increase in respiratory symptoms, including wheezing, in areas with higher pollution levels also suggest environmental factors may be critical in the development and exacerbation of asthma and related symptoms. Exposure to air pollution is also an environmental risk factor associated with exacerbation and prevalence of COPD.

2.3 HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO NOISE

In addition to contributing to air pollution, airport operations also result in significant noise. The dominant source of noise from airport-related activities is aircraft engines. The quantitative measure typically used to assess the effect of noise on the environment is expressed as sound energy produced over the entire noise event during a standard time period. The method used to quantify the sound level of a transient noise event, (e.g., from takeoff and landing of aircraft) is the Sound Exposure Level or SEL. The SEL sums individual sound level readings over the duration of the event. For airport-related noise assessment, the Day-Night Sound Level (DNL) sums the individual flyover SELs over the day (24-hours) with a nighttime noise weighting of 10 decibels (dB) added to the SEL for operations occurring from 10 pm to 7 am. Since 1974, the standard approach for assessing exposure to environmental noise levels from aircraft operations is the time-weighted daily average exposure index or DNL. Conceptually, the DNL represents the total accumulation of all noise energy spread out uniformly over a 24-hour period.

STUDIES OF NOISE IMPACTS OF AIRPORTS

Auditory

Noise-Induced Hearing Loss

There is a significant body of evidence on the association between noise exposure levels and auditory impairment such as noise-induced hearing loss (NIHL). NIHL is hearing loss that develops slowly over a long period of time as a result of exposure to continuous or intermittent exposure to loud noise. NIHL is a complex disorder caused by a combination

of environmental and genetic factors. Most of the health literature regarding NIHL is associated with occupationally induced hearing loss (WHO, 2004, ACOEM, 2002). In addition to occupational exposure studies, studies have reported reduced hearing ability in adults and school-age children living close to a commercial airport (Chen et al., 1997; Chen et al., 1993), and a military airport (Miyakita et al., 2001). Recent studies have focused on the genetic association in humans (e.g., oxidative stress genes, inner ear potassium recycling pathway genes, and monogenic deafness genes) for NIHL (Konigs et al., 2009, Sliwinska-Kowalska et al., 2013).

Hearing impairment is caused by morphological changes in the inner and outer hair cells of the cochlea, where the stereocilia become fused and bent. The symptoms of NIHL increase gradually over time as sounds may become distorted or muffled and it may become difficult for a person to understand speech (NIDCD, 2005). The development of NIHL progresses through two phases. Phase one is characterized by temporary threshold shift (TTS). This is a brief hearing loss that occurs after noise exposures and hearing is completely restored after a rest period. After repeated exposure to noise intense enough to produce TTS, a permanent threshold shift (PTS) will occur. This is an irreversible increase in hearing thresholds caused by irreversible hair cell damage. Hearing loss is variable within the population but it is not known why some individuals are more susceptible than others (Quinn et al., 2001). There is some indication that hearing loss may be accelerated with co-exposure to environmental chemicals and cigarette smoke (El-Shazly, 2006; Pouryaghoub et al., 2007).

Guidelines for community noise developed by the WHO (1999), US EPA (1974), and the National Academy of Sciences (NAS) (2005) determined independently that a maximum exposure level to noise that is protective against NIHL are noise levels equal to or less than 70 decibel (dB) over a 24-hour time-average period (Leq (24) <70 dBA-weighted sound). This assumes exposure to all environmental noise does not exceed 70 dB throughout daily activities irrespective of where and under which conditions this exposure is received, including exposure to occupational levels of noise. With respect to community noise impacts, WHO determined in 1999 that evidence strongly suggests that the

calculation methods used by the International Organization for Standardization (ISO) 1999 for occupational noise should also be applied to environmental and leisure time noise exposures. The guideline states that health impacts are unlikely for individuals exposed to cumulative noise exposure over a 24-hour period of less than or equal to 70 dB [Leq (24) =<70 dBA-weighted noise]. WHO also notes that the uncertainties associated with this assessment suggest that a margin of safety is needed when applied in a community setting. The uncertainties associated with this guideline value that were not considered include the (1) increased risks due to greater vulnerability of children in acquiring NIHL than adults, (2) increase risk from an exposure when noise is combined with vibrations (as is the case with lower frequency noise from aircraft), and (3) increased risk from exposures from ototoxic substances (having a toxic effect on the ear or its nerve supply) including certain chemicals (e.g., opioids, organic solvents, carbon monoxide), smoking, and having high blood pressure or high cholesterol (Konings et al., 2009).

Tinnitus

Tinnitus is a symptom associated primarily with noise-induced hearing loss. Tinnitus is defined as the perception of sound for which there is no external acoustic source. It is often referred to as "ringing in the ears" and may manifest itself as a buzzing or whistling sound. Tinnitus can be persistent or transient, and may be perceived in one or both ears. There is a clear correlation between hearing loss and tinnitus; however, not all persons with hearing loss have tinnitus (NAS, 2005; Henry et al., 2005). According to a review by Henry et al. (2005), there are no uniform or reliable clinical measures for defining the negative impact on individuals that experience tinnitus. Individuals that experience clinically significant effects from tinnitus report a range of health problems including sleep disturbances (in about half of individuals), and effects on cognition, emotional status, and hearing. Hearing aides are a common type of treatment for tinnitus.

There are wide-ranging theories of the pathological mechanisms for tinnitus. The most prevalent theories involve loss of hair cells or hair cell function, discontinuity of activity across the auditory nerve, and disruption of the central auditory pathways (NAS,

2005). However, the mechanism that causes sustained tinnitus is unknown (Henry, et al., 2005). Tinnitus may be caused by loud noise, which results in hair cell damage, certain ototoxic medicines, or medical conditions such as nutritional status, vascular disease, middle-ear disease, diabetes, hypertension, autoimmune disorders, and degenerative neural disorders (Perry et al., 2000). Synergistic effects have been reported between noise, diuretics, and common aminoglycoside antibiotics such as gentamicin (NAS, 2005).

SUMMARY OF HEALTH EFFECTS OF NOISE

In summary, there are a wide-range of health effects associated with exposure to various levels and sources of noise including noise-induced hearing impairment, interference with speech communication, disturbance of rest and sleep; psychophysiological effects, mental-health effects, impairment of performance of school children in cognitive tasks, and annoyance. Although recent studies have reported a possible increased risk of hypertension from exposure to aircraft-related noise, this finding still requires additional study. To date, a significant body of evidence has demonstrated an association between high noise levels and auditory impairment (NIHL). Tinnitus is a symptom associated primarily with noise induced hearing loss. Guidelines for community noise levels developed by the WHO and US EPA found that auditory effects, including noise-induced hearing loss, are not expected to occur at noise levels equal to or less than 70 dB over a 24-hour time-average period (Leq (24) <70 dB).

HEALTH STUDIES OF AIRPORT-RELATED AIR POLLUTION STUDIES

Lin et al. (2008) evaluated hospital admission rates for respiratory outcomes from 1995-2000 and residential proximity to three large New York airports (Rochester Airport in Rochester, LaGuardia Airport in New York City, and MacArthur Airport in Long Island). The respiratory outcomes examined were hospital admissions for asthma, chronic bronchitis, emphysema, chronic obstructive pulmonary disease, children's bronchitis (<4 years of age), and children's bronchiolitis. Findings suggested that residential distance within 5 miles of two of the three airports was associated with an increase in hospitalization admission rates for respiratory conditions (1.96; 95% CI 1.16-3.29 for

Rochester and 1.68; 95% CI 1.36, 2.07 for LaGuardia) after adjusting for potential neighborhood-level confounders from census block data (poverty level, African-American race, Hispanic ethnicity, education less than higher school). Wind flow patterns were analyzed to identify census block groups receiving predominant wind flow from the airports, but this was not found to be a factor in hospitalization rates of the respiratory outcomes studied. Due to the nature of hospitalization records, the study was limited, however, by the lack of individual-level information on important individual-level confounding factors, including smoking status, which may be related to respiratory hospitalizations.

Of the two studies identified in the literature that evaluated specific health outcome data in residents living near an airport, one of the most notable are the series of studies conducted since the 1990's in the communities near Schiphol Airport in the Netherlands (Franssen et al., 2003). A broad range of self-reported health outcomes have been evaluated using a postal survey of 11,812 residents living within approximately 2.5 miles from Schiphol Airport (Amsterdam). The survey included questions on annoyance, sleep disturbance, self-rated general health status, respiratory complaints, and medication use. In general, investigators concluded that air traffic emissions contributed only a few percentage points to local air pollution levels and that there was no evidence that air traffic emissions contributed to respiratory disorders. In a follow-up study, the investigators found associations between general health status, use of medication for cardiovascular diseases or increased blood pressure, and use of sleep medications or sedatives and aircraft noise exposure. Those residents who were severely annoyed by aircraft noise were more likely to have poor self-perceived health and more likely to report higher blood pressure.

3 HEALTH SURVEY DESIGN AND METHODS

3.1 STUDY AREA AND SAMPLE POPULATION

STUDY AREA

Consistent with the intent of legislative language, the study population was defined as communities located, either fully or partially, within a 5-mile radius of the General Lawrence Logan Airport. Based on the 2000 US census, seventeen communities, with populations totaling over 1 million residents 18 years of age and older, are located within a 5-mile radius of Logan Airport and were, therefore, considered part of the study area (Figure 3-1). As shown in Figure 3-1, the study area includes the airport itself and extends outward to the geographic borders of each of the seventeen communities located within the 5-mile radius. The communities are: Boston, Brookline, Cambridge, Chelsea, Everett, Hull, Lynn, Malden, Medford, Melrose, Milton, Nahant, Quincy, Revere, Saugus, Somerville, and Winthrop. Two communities directly abut the airport property, Winthrop which has residential properties located within 800 feet to the east of the airport, and East Boston, which immediately borders the airport to the west with some residential properties directly abutting airport property.

SELECTION OF HOUSEHOLDS TO SURVEY (SURVEY SAMPLE)

The presence of air pollutants and noise associated with airport operations varies greatly across the five-mile radius study area. For that reason, it was expected that portions of the study area closer to the airport would have the potential for higher levels of airport-related exposures than portions of the study area farther away from the airport.

In order to ensure that enough households were interviewed from all areas, those nearer and farther from the airport, the study area was divided or “stratified” into three distinct sections. These sections were designed to approximate areas that might experience high, medium, and low exposures to air pollutants and noise from airport operations.

Figure 3-1. The Logan Airport Health Study Area

For the purpose of sample selection, proximity to the airport was considered a sufficient proxy for exposure to airport-related emissions. The use of municipal boundaries, zip codes, and census tracts was also incorporated into the survey design in order to sample at the highest geographic resolution possible. The determination of distances for each of the three areas was based on knowledge of aircraft flight paths, atmospheric mixing of airport emissions, and modeled airport noise profiles.

The "high" exposure area included the area within a 1-mile radius of the center of the airport. This included emissions along the flight paths of aircraft flying at an altitude of approximately 3000 feet or less, which can contribute to ground level air pollutant

concentrations. The "medium" exposure area extends outward from the "high" exposure area to the boundary at which aircraft reach an altitude of 3000 feet, which corresponds to a ground distance of about 3-4 miles from the airport. The "low" exposure area extends from the end of "medium" exposure area to the geographical border of the outermost communities in the study area (approximately 4-12 miles from the airport). (These exposure areas were refined for the final data analysis based on air dispersion modeling results of airport-related emissions for 2005.)

Since the "high" and "medium" exposure areas cover smaller geographic areas and contain smaller populations than the "low" exposure area, the sampling strategy aimed to over-sample the populations in those areas, thus ensuring a sufficient number of households were sampled in those areas to detect statistically significant differences.

SAMPLE SIZE AND STATISTICAL POWER

The LAHS sample size was based on the statistical power needed to detect an association between air pollution exposure and heart disease prevalence. A power calculation is a method of estimating the sample size needed to detect a statistically significant association between exposure and a particular health outcome, using knowledge of the rates of the disease in the population and the predicted size and strength of the association with exposure based on previously published research. Heart disease was selected because it was found to have a lower prevalence in Massachusetts residents compared to asthma. Therefore, a sample size large enough to detect an elevation in heart disease prevalence would also be sufficient for detecting elevations in asthma or other more common health outcomes.

The sample size calculation also accounted for a study design with three exposure areas and disproportionate stratified sampling (oversampling) in the medium and high exposure areas, increasing the probability of detecting the presence of elevated rates of disease in the population living closest to the airport if indeed such disease patterns exist. Based on the power calculation, for the study to have sufficient power (80 percent, $\alpha = 0.05$) to detect an association between the exposure area of residence and the health

outcomes of interest, it was estimated that a total of 6000 participants were required, corresponding to a total of 3000 residents in the "low" exposure area, 1500 in the "medium" area, and 1500 in the "high" area. Again, these estimates were further refined once the air dispersion modeling was completed.

3.2 DESIGN OF THE SURVEY INSTRUMENT

PILOT TESTING AND OVERVIEW OF THE SURVEY

The LAHS survey instrument was developed by MDPH/BEH using validated and reliable questions from national and international health surveys, including questions on respiratory symptoms taken from the European Community Respiratory Health Survey (ECRHS) and the International Study of Asthma and Allergies in Childhood (ISAAC). Prior to its use in the study, the survey instrument was pilot tested in 2002 with a population living near an airport in Portland, Maine. Based on pilot interviews, the survey was revised to improve question clarity, to enhance specificity of responses for some items, and to provide a greater emphasis on confidentiality for respondents. After extensive review to confirm satisfactory questionnaire logic and function, an additional round of pilot interviews was conducted in 2005 with 25 randomly selected residents of the LAHS area. These respondents were asked to complete the survey and to provide feedback on questions. Specifically, they were asked to evaluate the clarity of questions, including any terminology they did not understand, and whether the flow of the survey made sense or if portions of the survey seemed confusing. Feedback from these pre-test interviews was then used to finalize the survey instrument. The 25 pilot respondents were excluded from recruitment efforts for the final LAHS survey.

The final survey instrument was comprised of four sections: (1) questions on the prevalence of respiratory, cardiovascular, and auditory health endpoints, (2) questions on risk factors associated with these outcomes, (3) questions on exposures inside the home and at work, and (4) questions reflecting demographic and socio-economic status.

HEALTH OUTCOME ASSESSMENT

The selection of health outcomes to be included in the survey was based on information gathered from a thorough review of the available scientific literature, including published literature on air pollution and potential health impacts, as well as input obtained from Community Advisory Committee (CAC) meetings and from residents and community stakeholders living in the vicinity of the airport regarding their perceptions and concerns.

The primary health outcomes of interest in the survey were asthma (lifetime asthma, current asthma, current asthma with medicine use, asthma attacks, emergency department visits for asthma, probable asthma, respiratory symptoms, and COPD); physician-diagnosed cardiovascular outcomes (non-fatal heart attack, angina or coronary heart disease) and auditory effects including hearing impairment and tinnitus. In addition to collecting information on the prevalence of these primary health outcomes, information on the age of first event, number of events, and severity was also collected.

COVARIATE DATA COLLECTED

Risk factors for the health outcomes of interest, as reported by the American Heart Association (AHA) and American Lung Association (ALA), were also evaluated for the LAHS health survey. Risk factors are characteristics (e.g., age, gender) or variables (e.g., smoking, air pollutant levels) associated with increased probability or likelihood of disease. A risk factor may be inherited, associated with a lifestyle, or due to an environmental exposure. Standardized questions associated with identified risk factors were taken from the National Health and Examination Survey (NHANES), the National Health Interview Survey (NHIS), and the Behavioral Risk Factor Surveillance System (BRFSS) survey and were included in the LAHS questionnaire. Risk factors for respiratory outcomes include genetics, exposure to indoor allergens (e.g., pollen, second hand cigarette smoke, dust, mold), parental atopic or asthmatic status, obesity, and respiratory infections. The major risk factors for cardiovascular outcomes are smoking, high blood cholesterol, high blood pressure, physical inactivity, obesity and being overweight, and diabetes mellitus. The health survey contained questions on all of these risk factors except genetics.

3.3 SURVEY ADMINISTRATION

OVERVIEW

A cross-sectional stratified telephone survey using random-digit-dial (RDD) methods was deemed most efficient to collect health outcome data representative of a study population of over 1 million residents. This type of survey provides data about a population at one point in time (e.g., 2005). Most national health surveys (e.g., BRFSS) use RDD techniques to gather information on the health status of US residents because it is a validated cost-effective sampling technique.

RDD TELEPHONE SAMPLE

As noted previously, the sampling strategy aimed to sample a greater proportion of residents living in the "high" and "medium" exposure areas compared to the "low" area. This was accomplished by using GENESYS Sampling Systems, a commercial software program that provides RDD samples. GENESYS also has the capability of identifying telephone exchanges and linking these exchanges to geographic information such as zip code areas.

The telephone numbers of households included in the health survey were drawn from an initial sample of telephone numbers generated by GENESYS. This approach ensures an equal probability of selection for every residential telephone number including unlisted numbers. The initial RDD sample of telephone numbers was then processed using specialized software to eliminate non-residential telephone numbers to the greatest extent possible.

SURVEY ADMINISTRATION

Interviews were conducted using Computer-Assisted Telephone Interviewing (CATI) technology from May to October of 2005. Interviewers were trained in interviewing protocols and procedures. In addition, interviewers received training specific to the LAHS

survey instrument using an Interviewers Manual that provided the rationale for each question of the survey. The manual was developed by MDPH/BEH after pilot-testing of the survey.

Call attempts rotated across all seven days of the week at different times of the day between 9 AM and 9 PM, according to industry standards. The only exceptions were specific, scheduled appointments outside this range. A minimum of 15 call-back attempts were made per telephone number at the screener level. Four attempts were made to convert initial refusals, except in cases where individuals requested not to be contacted again. To encourage participation, a brief message with a toll-free number was delivered to answering machines on the first, third, and seventh machine-answer call attempts. In addition, respondents who initiated the survey without completing it were re-contacted at a date and time of their choosing to complete the survey at a later time.

Standard screening questions were used to identify eligible households and to randomly select an eligible respondent to participate in the survey. Additional screening questions were used to address potential selection biases. For example, one screening question asked about the number of phone lines in the household because having more phone lines increases the probability of being selected. By collecting this information, this factor was able to be included as part of the complex sampling design.

To maximize the response rate, the survey administrators worked with MDPH/BEH to develop a number of call protocol elements designed to put responders at ease, pique interest and investment, verify the legitimacy of the caller and study, and establish trust in the confidentiality of any information given. The protocol included a standardized and appropriate call lead-in with an introductory statement about an "important environmental health study" being conducted for the purpose of learning about the respondent's health and the health of their family "to determine if there are common health problems" in their community. The statement explained the importance of participation in the study in order to help represent one's community, but did not identify Logan Airport as the subject of the study. Contact information for MDPH/BEH as well as a toll-free number

for the survey administrators and the name of the survey operation manager were given. The standardized statement also included assurances of the confidentiality of any information provided.

Surveys were conducted in English and Spanish depending upon the spoken language of the eligible respondent. For surveys conducted in Spanish, the respondent requesting the Spanish interview was re-contacted by a subcontractor within a specified period of time to complete the survey. The survey was designed to take an average of 25 minutes to complete and asked respondents to provide information about themselves and all children that might reside in the household. When an interviewer called a sampled household, one adult living in the household was randomly selected to be the respondent. If children resided in the household, the respondent was asked to complete the final section of the survey, designed to collect information for all children living in the household.

A total of 6,072 eligible residents/households were interviewed and completed surveys about themselves and 2,215 children living in those respective households. Completed surveys in each exposure area were tracked by mapping the street address, nearest landmark, or zip code provided by the respondent using GIS tools in order to ensure that the number of completed interviews per exposure area would be achieved.

4 ENVIRONMENTAL ASSESSMENT OF AIR POLLUTANTS AND NOISE ASSOCIATED WITH LOGAN AIRPORT OPERATIONS

4.1 OVERVIEW

As previously discussed, the lack of individual exposure data associated with airport emissions for this study required the use of surrogates (specifically proximity to the airport and other factors). Air dispersion modeling was performed to quantify the ambient air pollution concentrations in the study area and improve exposure classification of the participants. In addition, noise contours provided by Massport were used to classify noise exposure for the health outcome data analysis.

4.2 AIR DISPERSION MODELING ANALYSIS OF 2005 AIRPORT EMISSIONS

INTRODUCTION

Air dispersion models perform mathematical simulations of atmospheric transport and dispersion of emissions, using emission factors and meteorological information to estimate ambient air pollution concentrations associated with a source, such as Logan Airport. The US FAA's Emissions and Dispersion Modeling System (EDMS) contains two modules. The first is designed to estimate an emissions inventory, or listing, of pollutants and the amounts emitted from each of the major airport emission sources including aircraft, motor vehicles, and ground service equipment. The second module was developed to model the dispersion of these emissions in air over space and time. The model in EDMS is AERMOD and is the most advanced air dispersion model available for quantifying ambient air pollution concentrations from airport operations. AERMOD is the preferred model of environmental regulatory agencies when making air quality permitting decisions in the US. The US FAA's EDMS model has undergone several revisions over the past several years and the most recent version (EDMS v 5.1.3) was used for the LAHS (US FAA, 2010).

Air dispersion modeling of Logan Airport operations requires both the emissions inventory and the timing and location of emissions associated with airport operations.

Massport uses the emissions inventory module to calculate airport emissions on an annual basis and reports these data in the annual Environmental Data Report (EDR). The EDRs are a part of a series of annual environmental review documents submitted to the Executive Office of Energy and Environmental Affairs/Massachusetts Environmental Policy Act (MEPA) Office. They have been required since 1989 to report on the cumulative environmental effects of Logan Airport's operations and activities. EDRs provide a review of environmental conditions for the reporting year compared to the previous year. Massport also routinely logs flight operations of the exact time of every arrival and departure of aircraft by runway and operation. Massport provided MDPH/BEH both the emissions inventory input files to EDMS and the flight operations log for 2005 to determine the timing of aircraft emissions throughout the year. The location of emission sources (e.g., ground service equipment, power plant, motor vehicles) was determined from detailed maps obtained from Massport and an airport map embedded in US FAA's EDMS model. MDPH/BEH contracted with a consultant with expertise in air dispersion modeling and the EDMS model for this component of the LAHS.

SUMMARY OF AIR POLLUTANT DISPERSION MODELING ANALYSIS

MDPH/BEH used the most advanced version of EDMS that includes the most recent emission factors[1] for quantifying source emissions, particularly for aircraft, and the most recent version of AERMOD, which predicts ground-level air pollutant concentrations in a specified modeling domain. AERMOD is the regulatory model that is required for conducting dispersion modeling mandated for the permitting process for new or modified air pollutant sources required under the New Source Review regulations of the Clean Air Act. AERMOD is an advanced Gaussian plume type model with improvements primarily in the characterization of how winds speeds and turbulent mixing rates vary as a function of

[1] An emission factor is a representative value that relates the quantity of a pollutant released to the atmosphere with a particular activity. These factors are usually expressed as the weight of pollutant divided by a unit weight, volume, distance, or duration of the activity emitting the pollutant (e.g., kilograms of particulate matter emitted per gallons of fuel burned). Such factors facilitate estimation of emissions from various sources of air pollution.

height above the ground surface. The US FAA upgrade to EDMS included changing the dispersion model to AERMOD in 2006. Additional information, including the full air dispersion modeling analysis report, is provided in Appendix A. Descriptions of the major modeling parameters associated with this analysis are summarized below.

Modeling domain and topographic features

The topography of the study area is relatively flat. Therefore, a radial array was used for establishing the receptors for predicting pollutant concentrations at 10 degree intervals. The array of rings of receptors were located at radial distances of 1, 1.25, 1.5, 1.75, 2, 2.25, 2.5,2.75, 3, 3.25, 3.5, 3.75, 4, 5, 6, 8, 10, and 12 miles from the airport center. These receptors were assigned elevations of 1.8 meters above the ground surface and have base elevations set at 5.59 meters above sea level. An additional 27 receptors were placed at the aeronautical center point of Logan Airport, the Logan Statue located at the entrance of Logan Airport, and at other specific landmarks or easily identifiable locations in the LAHS area. Only receptors located off airport property were included in the modeling analysis. There were a total of 635 receptors in the modeling domain.

Pollutants of concern

The pollutants modeled by EDMS for the LAHS are nitrogen oxides (NOx), particulate matter ($PM_{2.5}$)—assumed to be all particulate matter 2.5 micrometers and smaller in aerodynamic diameter[2], sulfur oxides (SO_x), carbon monoxide (CO), and volatile organic compounds (VOCs).

[2] The primary particle sizes emitted from airport sources are $PM_{2.5}$ and ultrafine particles or UFPs (i.e., particles with an aerodynamic range of 0.1 micrometers and smaller). Currently, regulations are associated with mass fraction of particulate matter only ($PM_{2.5}$ and PM_{10}). Emissions inventories do not report UFPs and UFPs are not included in air dispersion models. Massport also assumes all PM emissions at Logan Airport are $PM_{2.5}$. To maintain consistency, this report will also refer to all PM emissions and air concentrations as $PM_{2.5}$.

Emission sources

The primary source of information on emissions from operations at Logan Airport is the annual emissions inventory for 2005 conducted by Massport using US FAA's EDMS emissions inventory module. The air pollutant sources considered in the LAHS are those associated with the routine operations at Logan Airport. These include all aircraft approaching and departing in the air, landings and takeoffs, aircraft movements on the runways, taxiways, and areas near the terminals, aircraft ground support equipment (GSE) that is needed at the terminal to load or handle arriving or departing aircraft, and auxiliary power units (APUs) at the terminals used to energize aircraft at the terminals. The emissions data also includes motor vehicle traffic on the Logan property, emissions from vehicles in the parking garages and emissions from Massport's power boilers that provide power or steam to the airport. Attachment 1 in Appendix A provides detailed descriptions and spreadsheets of all input data for the emissions inventory component of the air dispersion modeling analysis. Table 4-1 presents the 2005 emissions inventory for Logan Airport that was modeled in this study.

Table 4-1. Emissions Inventory for Logan Airport, 2005 (kg/year)

Source Category	CO	NOx	$PM_{2.5}$	SO_x	VOC
Aircraft	1149808	1193034	21368	111641	434959
GSE	2262228	254757	7425	20161	79166
APUs	48849	22971	4443	3933	3267
Parking Facilities	545896	74347	1137	N/A	111635
Roadways	378889	85137	2596	N/A	37526
Stationary Sources	11382	74169	11626	115507	663
Training Fires	1371	22	375	2	216
Grand Total	4398423	1704437	48970	251244	667432

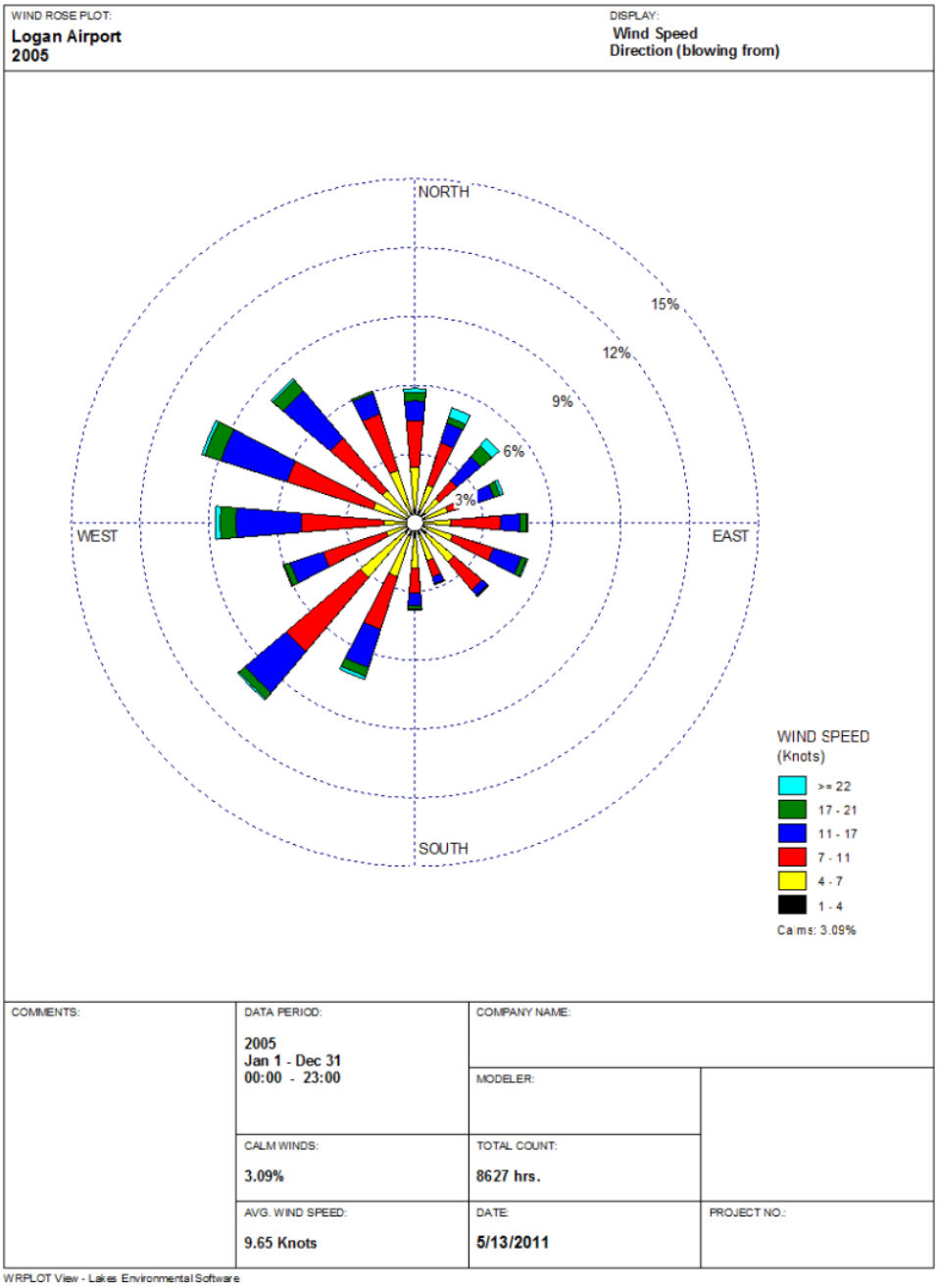
Meteorological data

AERMOD requires both surface and upper air meteorological data inputs. In addition to obtaining meteorological data for 2005 from the National Weather Service (NWS), historic meteorological data was obtained for the years 2003-2004 and 2006-2007 for the sensitivity analysis (described below). The NWS operates an Automated Surface Observation Station (ASOS) that is located amidst several of the runways and taxiways at Logan Airport. The ASOS station includes an anemometer at a height of 26 feet above the ground surface, which is well placed to represent the locations of the most important category of emissions analyzed in this study. The ASOS data includes wind speed and direction, temperature, dew point and cloud cover. See Figure 4-1 for an example of a wind rose for 2005 modeling runs. Upper air measurements are required by AERMOD for the estimation of mixing depths and are taken two times per day at the National Weather Service station located in Chatham, MA. Use of this station for this study is consistent with federal and state guidelines.

Surface characteristics

AERMOD calculates the diffusion rates and wind speed profiles using algorithms based upon an advanced understanding of air flow in the surface boundary layer and upon how the flow and the turbulent diffusion rates are dependent upon three specific parameters that characterize the ground or water surface. These parameters are the surface roughness (roughness length), the surface reflectivity of incoming solar radiation (albedo) and a measure of the importance of surface moisture in the transfer of heat to the air above the surface (Bowen ratio). The values of these parameters are obtained from land use data that was used to create maps for the study area. US EPA's AERSURFACE program uses United States Geological Survey (USGS) land use data through an interactive program to calculate average values of these three surface characteristic parameters based on latitude and longitude and estimates about seasonal vegetation and snow cover. The surface characteristics values are input into the AERMET meteorological preprocessor to determine the dispersion rates in the atmospheric boundary layer.

Figure 4-1. Wind rose from weather station at Logan Airport – 2005



MAJOR FINDINGS OF AIR DISPERSION MODELING ANALYSIS

Overall findings of modeling 2005 emissions

As mentioned, MDPH/BEH used the most advanced version of EDMS that includes the most recent emission factors for quantifying source emissions, particularly for aircraft, and the most recent version of AERMOD, which predicts ground-level air pollutant concentrations in a specified modeling domain. There have been significant improvements in the development of emission factors for aircraft and other airport-related sources incorporated into EDMS. Areas of current research include quantifying ultrafine particle emissions from aircraft, and the contribution of aircraft tires and brakes to the overall particulate matter inventory for airports (ACRP, 2008).

AERMOD modeling runs of emissions associated with Logan Airport operations in 2005 were successfully completed and validated using established protocols and methods (see Appendix A). In addition to the base model, sensitivity analyses for evaluating uncertainties in the modeling results were also conducted. For example, varying meteorological data by running the 2005 operations with 2003, 2004, 2006, and 2007 meteorological conditions resulted in concentrations that differed by only 10% from the values produced using 2005 Base year meteorological data. The average of all four years was within 5% of the 2005 Base year values. It should be noted that 2005 aircraft activity was assumed in the modeling of these additional years so the differences in the results could be attributed only to differences in meteorological conditions for these years. Extensive review of the input data was also conducted to ensure accuracy and completeness of data obtained from Massport that was entered into the AERMOD model. Quality assurance of modeling runs was conducted by reviewing the AERMET quality assurance checks of the raw observational data and extensive review of final modeling output.

Air pollutant concentrations in the study area

Individual pollutants (CO, NOx, $PM_{2.5}$, SO_x, and VOCs) were modeled and concentrations were obtained from each of the 635 receptors arrayed in polar grids across the study area (see Figure 4-2). A summary of the annual average pollutant concentrations from air dispersion modeling of 205 airport operations is presented in Table 4-2.

Table 4-2. Summary of Annual Average Air Pollutant Concentrations (µg/m³) from Air Dispersion Modeling of 2005 Airport Operations

	CO	NOx	$PM_{2.5}$	SO_x	VOC
Annual Average	15.45	3.56	0.11	0.34	1.84

Distribution of air pollutant concentrations in the study area

The distribution of air pollutant concentration data is characterized by much higher concentrations near the airport that fall off rapidly with increased radial distance from the airport. This skewed distribution is consistent with the initial survey sampling design that anticipated higher exposure areas closer to the airport. This is illustrated in Figure 4-3 that shows the normalized annual average concentrations of NOx and $PM_{2.5}$ predicted for polar (or circular) receptors near the airport.

Air pollutant concentrations of individual source categories

The air dispersion modeling results provide information about the contribution of each of the major source categories (e.g., aircraft, motor vehicles, power plant) to air pollutant concentrations in the study area. The annual average concentrations for NOx and $PM_{2.5}$ for the major source categories in the modeling domain are presented in Table 4-3.

Figure 4-2. Modeling Domain for the LAHS

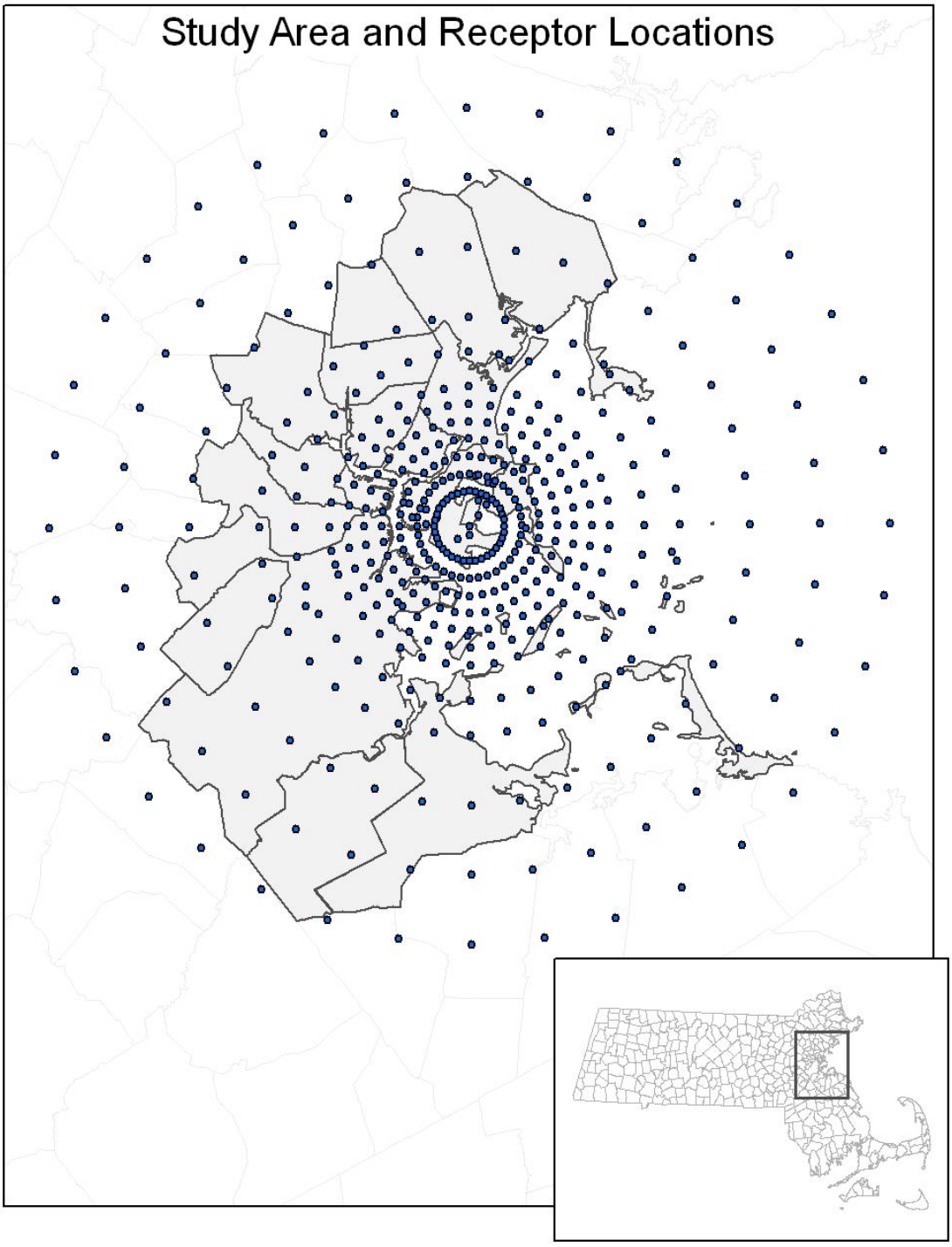


Figure 4-3. Normalized Annual Average Concentrations for NOx and $PM_{2.5}$

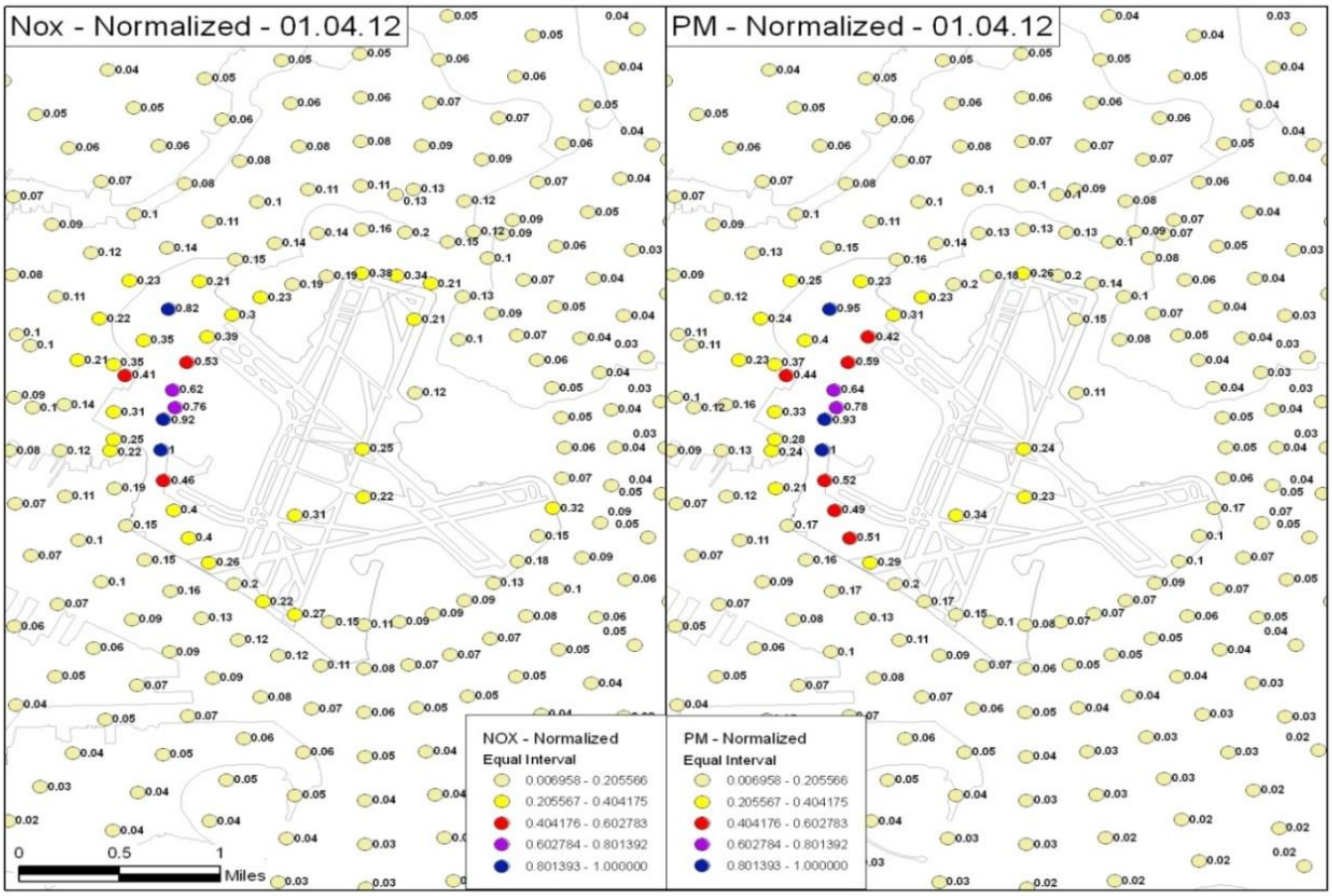


Table 4-3. Annual Average Concentrations for NOx and $PM_{2.5}$ Across the Study Area for the Major Source Categories at Logan Airport

NOx Annual Average Concentrations in Modeling Domain (µg/m³)						
	Aircraft	Gates	Parking	Roadways	Stationary	Total
Annual Average	1.65	1.08	0.25	0.54	0.05	3.6
$PM_{2.5}$ Annual Average Concentrations in Modeling Domain (µg/m³)						
	Aircraft	Gates	Parking	Roadways	Stationary	Total
Annual Average	0.03	0.05	0.004	0.02	0.01	0.11

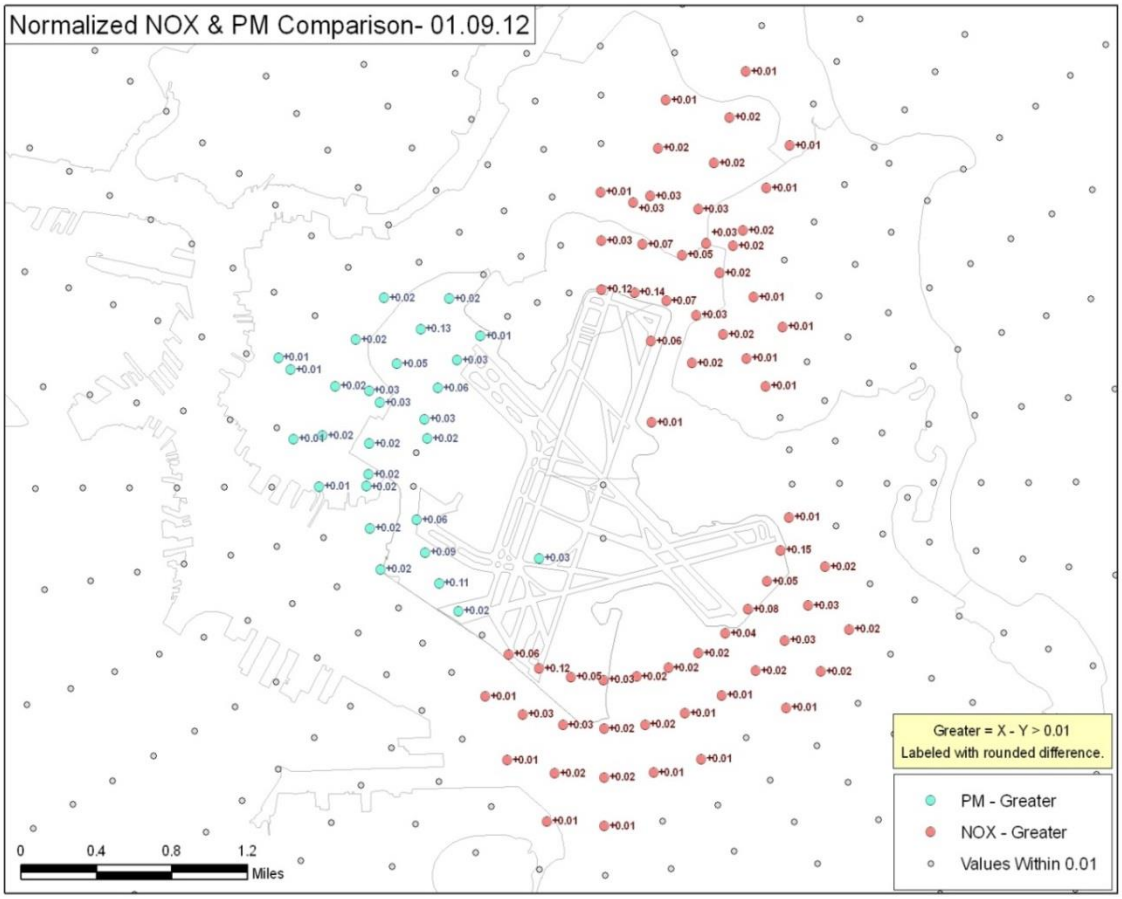
Impact of emission source locations at Logan Airport

The dispersion modeling results provide information about the relative impact of air pollutants by source location. For example, although the emissions of NOx and $PM_{2.5}$ at Logan Airport come from sources at different locations and from sources that differ significantly in emission levels, the EDMS modeling results show that the concentrations of individual air pollutants are not only strongly correlated with time (hourly, daily, and annual average values), but also with respect to geographic location of ambient air concentrations. To evaluate the degree to which the spatial patterns of pollutant concentrations are similar, the differences between the NOx and $PM_{2.5}$ normalized concentrations over the study area are plotted.

Figure 4-4 shows that although the maximum concentrations of both NOx and $PM_{2.5}$ occur just to the west of the airport, the higher NOx relative to $PM_{2.5}$ occurs at the runway ends and to the east of the airport. These results are attributed by the modeling to aircraft activities in that location. The higher $PM_{2.5}$ concentrations relative to NOx that occur to the west of the airport are presumably attributed to relatively larger fraction of motor vehicular emissions rates in those areas. Thus, operations near the terminals contribute to the air pollution concentrations in East Boston. In contrast, the aircraft takeoffs and landings are the largest contributor to concentrations north and east near Winthrop, and

near the ends of other major runways. Both sets of differences fall off rapidly to values less than 1% with increased radial distances beyond the airport perimeter.

Figure 4-4. Relative Contribution of Predicted NOx and $PM_{2.5}$ Air Pollutant Concentrations Associated with Logan Airport



4.3 AIR POLLUTION EXPOSURE CATEGORIZATION

As discussed previously, the emissions inventory was modeled using a standard atmospheric dispersion modeling system to estimate air pollutant concentrations from airport operations across the study area. The modeled concentrations of each of the five air pollutants (CO, NOx, $PM_{2.5}$, SO_x, and VOCs) were highest near the airport and were observed to decrease rapidly with increasing distance from the airport. The modeled concentrations at each receptor were analyzed using ArcGIS Spatial Analyst to create maps for each air pollutant's concentrations across the study area. Geocoded household addresses of survey respondents were overlaid with air pollutant maps to assign an air pollutant concentration to each household using inverse distance weighting (IDW) interpolation. IDW considers the values of sample points (receptors) and the distance separating them (i.e., nearest neighbor) to estimate the value at each respondent address. The inverse of the squared distance is the simplest and most commonly used format of distance-weighting, giving higher weight to closer observations (US EPA, 2004). Given the high density of the receptor grid, particularly close to the airport where concentrations have the most variability, IDW interpolation provided a consistent approach for considering local-scale pollutant variability in assigning concentrations to each of the households.

Pollutant concentrations were evaluated for several averaging times (annual average, 24-hour maximum, and 1-hour maximum) and were found to be highly correlated (Spearman correlation coefficients of 0.96 or higher) and geographically similar across averaging times. In other words, for example, areas/respondents with the highest modeled annual average NOx concentrations also had the highest modeled 24-hour and 1-hour maximum NOx concentrations. For this reason, only annual average pollutant concentrations were used in further analyses to categorize exposure to airport-related pollutants in the surrounding communities. From a statistical standpoint, the annual average is the preferred choice as it is the average of the largest number of individual predictions at each receptor and, thus, represents the lowest variability or uncertainty compared to other averaging times.

Correlation analyses of air pollutant concentrations assigned to each household also confirmed that the annual averages of all five modeled air pollutants (CO, NOx, $PM_{2.5}$, SO_x, and VOCs) were highly correlated with one another with Spearman correlation coefficients greater than 0.99 for all associations (Table 4-4). Therefore, a combined exposure variable was developed to categorize study participants based on their exposure to all five targeted compounds.

Table 4-4. Spearman Correlations[a] of LAHS Annual Average Pollutant Concentrations from Air Dispersion Modeling[b]

	CO	NOx	$PM_{2.5}$	SOx	VOC
$PM_{2.5}$	0.998	0.997	1.000	0.997	0.999
NOx	0.992	1.000	0.997	0.998	0.995
SOx	0.991	0.998	0.997	1.000	0.994
CO	1.000	0.992	0.998	0.991	0.999
VOC	0.999	0.995	0.999	0.994	1.000

[a]Pearson correlation results were similarly strong. See Appendix A, Table 12.
[b]P-values for all correlations are <0.0001.

Visualization of the concentrations of each pollutant assigned to households across the study area using histograms revealed that the distribution of concentrations followed a logarithmic function. Annual average concentrations of each pollutant were categorized into three categories: high, medium, and low areas. These categories were chosen based on the following considerations: identifying the upper distribution of pollutant concentrations, understanding the scientific literature related to the health effects of the targeted compounds, and weighing epidemiologic considerations (such as selecting a "low" group that is truly low, isolating the highest exposed group as much as possible, and maintaining sample sizes in each group that would be large enough to enable detection of any associations present). Thus, each household was categorized as follows for exposure to each air pollutant: the low category included all respondents whose modeled concentrations were less than or equal to the median value (i.e., 50th percentile); the

medium category included all respondents whose concentrations were above the median, but less than or equal to the 80th percentile; and the high category included all respondents whose concentrations were above the 80th percentile. The range of modeled air pollutant concentrations based on these cut-points and the number of respondents assigned to the high, medium, and low exposure areas for each pollutant are presented in Table 4-5.

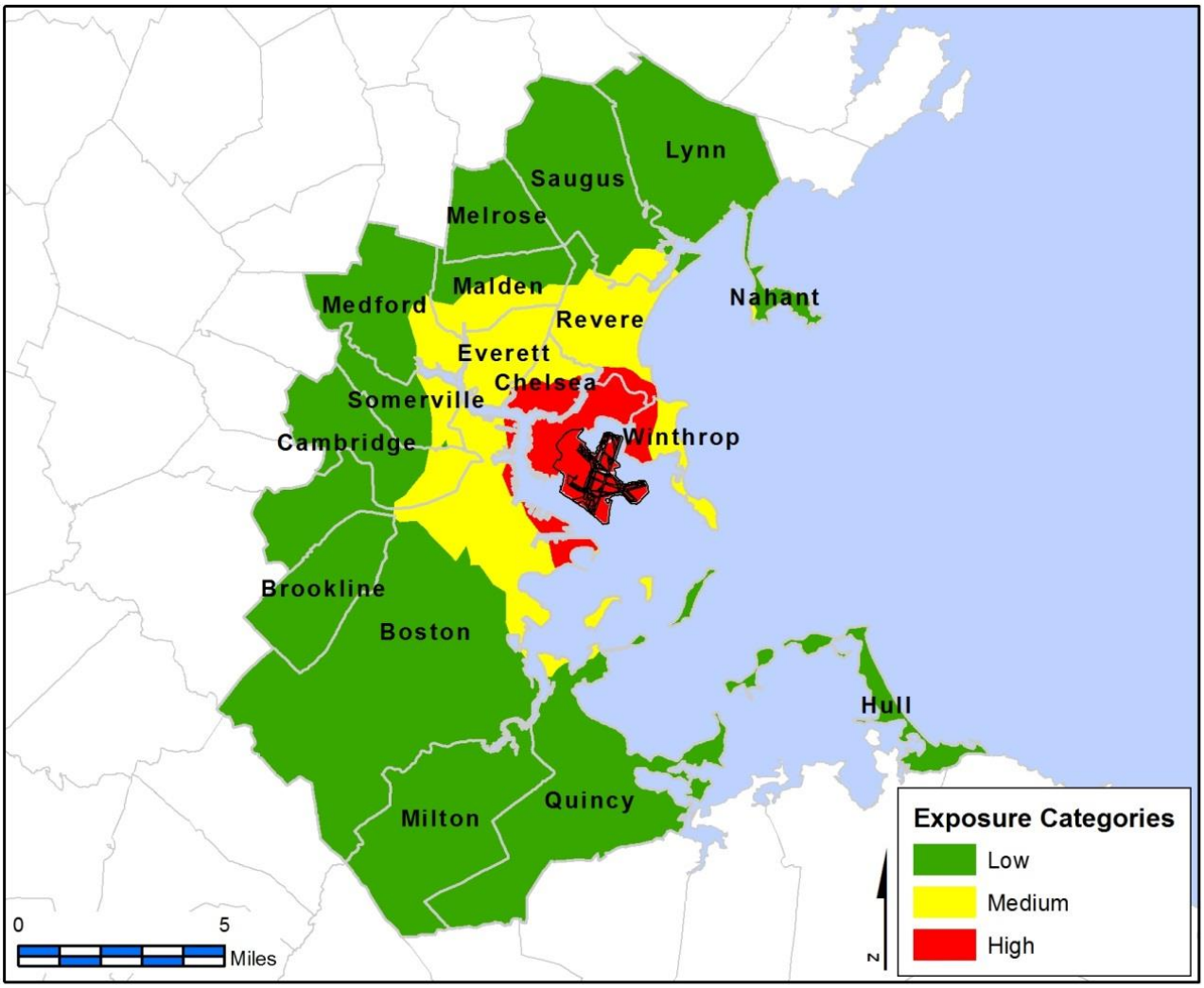
Table 4-5. Range of Modeled Air Pollutant Concentrations Associated with the High, Medium, and Low Exposure Categories for Each Pollutant

		Range of Exposure ($\mu g/m^3$)				
Exposure Level	**Number (%) Respondents**	CO	NO_x	$PM_{2.5}$	SO_x	VOCs
Low	3034 (50%)	0.57 - 3.88	0.14 - 0.77	0.005 - 0.03	0.03 - 0.11	0.08 - 0.50
Medium	1834 (30%)	3.88 - 11.4	0.77 - 2.47	0.03 - 0.09	0.11 - 0.34	0.50 - 1.53
High	1204 (20%)	11.4 - 109	2.47 - 15.0	0.09 - 0.65	0.34 - 1.39	1.53 - 11.6

Of the 6,072 individuals in the study, 93% were assigned the same categorization (high, medium, or low) for all five targeted air pollutants based on the cut-points described above. Only 420 (7%) were assigned to different categories based on different pollutants; some were categorized to both low and medium exposure groups and some were categorized to both medium and high exposure groups. None were assigned to both low and high categories. For the combined exposure variable, these 420 individuals were assigned to the exposure category most represented by the five pollutants (i.e., the exposure category assigned to three or more of the five air pollutants). The combined exposure variable was used for all subsequent analyses of associations between health outcomes and airport-related air pollution. The estimated exposure areas associated with assigning high, medium, and low exposures to respondents is illustrated in a map of the study area in Figure 4-5.

To further evaluate the choice of averaging time, combined exposure variables were created for maximum 24-hour and 1-hour pollutant concentrations, as well. Using the same percentile cut-points for high, medium, and low exposure areas, there was a 95% agreement in respondent categorization when using annual average versus 24-hour maximum concentrations and a 90% agreement when comparing annual average to 1-hour maximum concentrations.

Figure 4-5. Estimated Exposure Areas Based on Assigning High, Medium, and Low Exposure Areas to Respondents



4.4 CONSIDERATION OF NON-AIRPORT RELATED BACKGROUND AIR POLLUTION

Exposure to air pollution from sources not associated with Logan Airport was taken into consideration as a potential confounding factor in the analysis. Air pollutants similar to those emitted from airport sources are present in the airshed across the study area from sources such as vehicle exhaust, power plant emissions, and other industrial processes. Given the variability of air pollutants across the study area and other factors that influence exposure (e.g., activity patterns), residents living in the study area are not likely to be equally exposed to these background air pollutants. To account for the variability of background air pollution exposure in the analysis, two measures of air pollution exposure were developed (see Appendix B).

First, each household respondent was assigned an annual average predicted background $PM_{2.5}$ concentration based on measurements from the ambient air monitoring station nearest their home. The Massachusetts Department of Environmental Protection operates air quality monitoring stations that measure and report air pollution levels across the study area. These background air pollution levels represent a general measure of the best available air quality data across the study area. Since air pollution monitoring data may be influenced by both airport and non-airport related emissions, the predicted background $PM_{2.5}$ concentrations assigned to each respondent were adjusted by subtracting the airport-related contributions of $PM_{2.5}$ that were estimated for each household using air dispersion modeling of airport operations (see Appendix B for more details on the derivation of background pollutant concentrations for this study). The derived background $PM_{2.5}$ concentrations assigned to each household were then included in all multivariate regression analyses (as discussed in upcoming Chapter 5) of associations between health outcomes and estimated airport-related air pollution exposure.

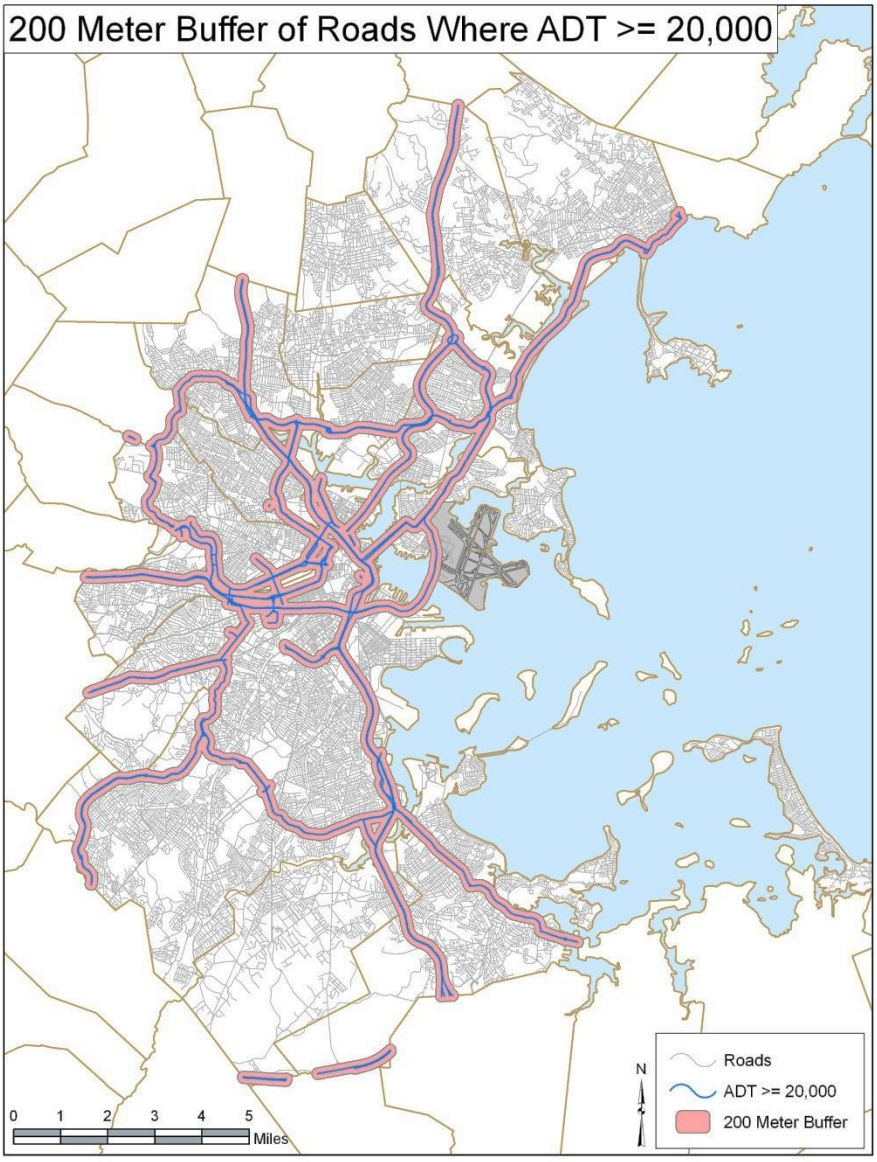
To provide context of background concentrations, the annual average predicted concentrations from modeled airport operations at Logan Airport assigned to each respondent were compared to the annual average monitored $PM_{2.5}$ background value assigned to each respondent (see Table 1 in Appendix B). The ratio of predicted $PM_{2.5}$ contributions from Logan Airport to the background air pollutant monitored values for all respondents is about 0.006[3]. Since this ratio includes a large number of predictions at large distances from the airport, ratios were also calculated for those households with the highest predicted Logan Airport concentrations. These include ratios of Logan Airport to background for the 3% of households with predicted Logan Airport $PM_{2.5}$ concentrations that exceed 50% of the maximum value and the 0.6% of households with concentrations that exceed 75% of the maximum value. For those exceeding 50% and 75% of the maximum predicted Logan Airport $PM_{2.5}$ value, the ratio of Logan Airport contributions to background estimates are also low (0.033 and 0.042, respectively).

Second, respondents were categorized based on their proximity to major roadways. Recent health studies of near-roadway exposures (100-300 meters) have found increased risk of respiratory and cardiovascular impacts (Hoffman et al, 2009; Lindgren et al, 2009). For the purposes of this study, major roadways were defined as those having average daily traffic of more than 20,000 vehicles (MassDOT, 2012).

Using ArcGIS spatial analyst tools, respondents located within 200 meters of a major roadway were identified, and an indicator variable for this was included in multivariate analyses. The map in Figure 4-6 displays the major roadways within the study area and a 200 meter buffer around them.

[3] See Table 12 in Appendix A. For example: Average Logan Contribution / Average Background Contribution: 0.07/12.21 =0.006.

Figure 4-6. Two hundred (200) Meter Buffer of Roads with Average Daily Traffic of 20,000 Vehicles or More



4.5 EXPOSURE TO NOISE ASSOCIATED WITH AIRPORT OPERATIONS

The LAHS evaluated exposure to noise from airport operations using noise contours developed by Massport. Since 1981, Federal Aviation Regulations (FAR) require airports to conduct and implement an airport noise and land use compatibility plan (FAR Part 150).

These regulations also establish US FAA's Integrated Noise Model as the noise model to be used for analyzing aircraft noise.

Noise is generated primarily from aircraft operations at Logan Airport. Using US FAA's INM model, Massport models noise by considering the number of operations, types of aircraft operating during the day and night, use of runway configurations, and location and frequency of flight paths to and from the runways. Since 2002, Massport has incorporated several enhancements to the noise modeling analysis for Logan Airport to more accurately estimate sound propagation into surrounding neighborhoods. These modeling enhancements include consideration of the surrounding water and terrain characteristics, use of advanced radar data system (i.e., PASSUR) to collect radar-based operations data, and specialized software to produce noise contours based on every individual radar trace, which improves the spatial dispersion of the radar tracks.

US FAA regulations require that airports use annual Day-Night Sound Level (DNL) contours to evaluate airport noise and adhere to land use guidelines. The guidelines restrict the building of residences, schools, hospitals or churches in areas within a DNL of 65 dB or above. A DNL of 65 dB or above is considered compatible for residential land use only if they are insulated from sound. Massport produces annual DNL contours that range from 60-75 dB at 5 dB increments and reports the estimated number of the people residing within various increments of modeled noise exposure (see Figure 4-7). Massport also provides alternative noise metrics that include the "Cumulative Noise Index" (CNI) and "Time Above" (TA) various threshold sound levels. However, since these metrics are only reported at the monitoring stations, they do not provide sufficient spatial coverage for assessing noise exposure across the entire study area.

Massport also operates a noise monitoring system at 30 locations around the airport. This system separates aircraft noise events from other local sources of noise and is limited to the measurement of airport-related noise only; it does not include background noise levels. Thus, background noise is not incorporated into the modeled noise contours and is explicitly subtracted from the noise monitoring data in order to be able to compare

the noise modeling with the noise monitoring data. Massport compares the noise monitoring data to the modeling data and presents the differences between monitoring and modeled values in the annual EDR.

For airport-related noise, modeled noise contours were readily available from the extensive noise assessment program that Massport implements using US FAA's computer modeling program (Integrated Noise Model or INM) and supplemental analysis of measured noise levels around the airport used to implement a noise abatement program.

As discussed above, the exposure level that was deemed appropriate for assessing the potential impacts of NIHL is the health-based guideline to protect against hearing impairment. This guideline states that health impacts are unlikely for individuals exposed to cumulative noise exposure over a 24-hour period of less than or equal to 70 dB [$L_{eq(24)}$ =<70 dBA-weighted noise][4] (US EPA, 1974, WHO, 1999). This guideline value indicates that the risk for hearing impairment would be negligible for a cumulative noise exposure below 70 dB on a daily basis over a lifetime. Assuming the equal energy principle[5] the annual DNL represents the daily (24-hour) cumulative noise exposure (i.e., not average) that has been averaged over a one-year period. The annual DNL differs from the cumulative noise exposure metric ($L_{eq(24)}$) because the DNL adds 10 dB to each nighttime flight (i.e., each nighttime noise event as if it occurred 10 times).

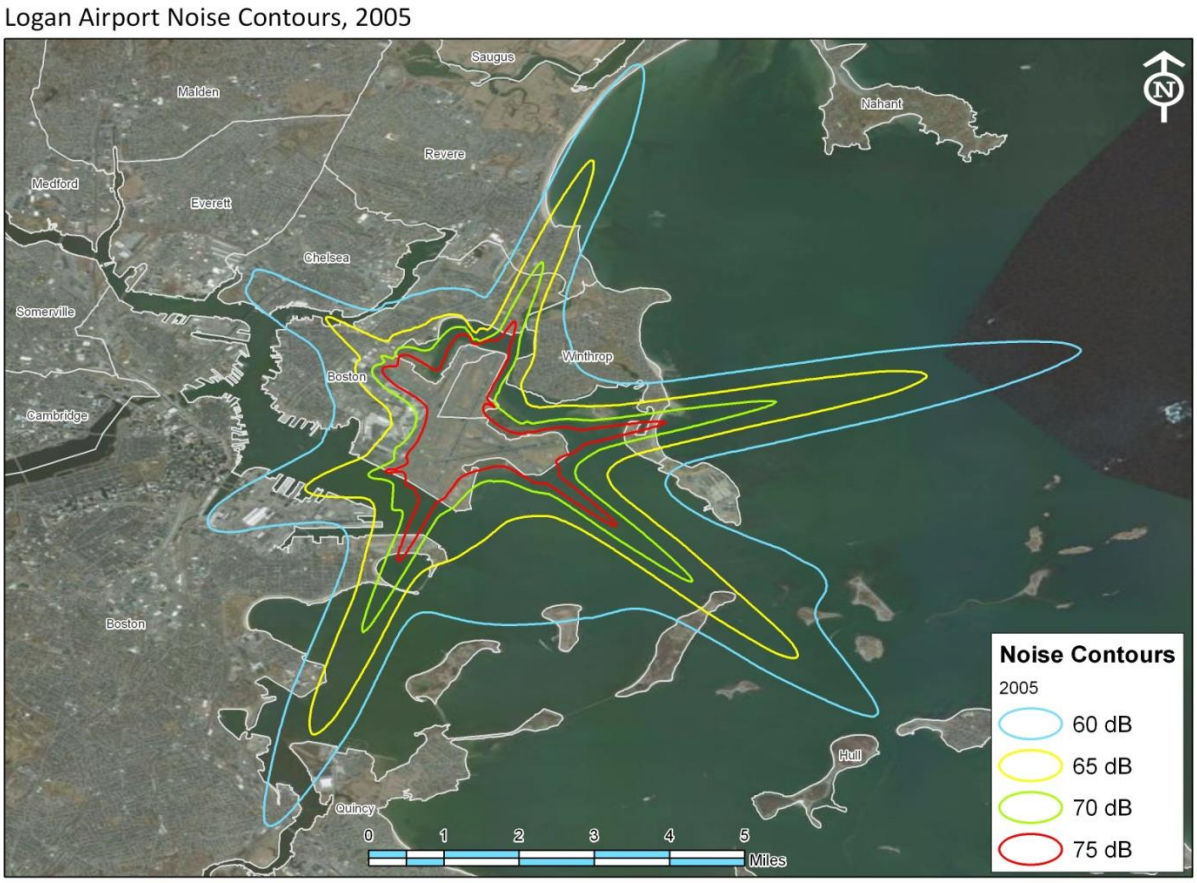
Review of the 2005 noise contours indicated that the 70 dB contour did not include a sufficient number of respondents to assign to the high noise exposure category. As a result, the 65 dB noise contour was selected as the high exposure area, the medium exposure area is defined by households located in the 60-64 dB noise contour, and

[4] US EPA identified 4000 Hz as the most noise-sensitive frequency to be protected. The noise level chosen to protect against hearing loss up to and including the 96th percentile of the population, was ranked according to decreasing ability to hear at 4000 Hz.

[5] The equal energy principle is the theory that the hazard to hearing is determined by the total sound energy (the product of sound intensity and duration) entering the ear each day.

households located in the 59 dB and less noise contour are assigned to the low exposure area.

Figure 4-7. Massport DNL Contours for 2005 Operations at Logan Airport



5 DATA ANALYSIS

5.1 OVERVIEW

Three main classes of health outcomes (respiratory, cardiovascular, and auditory) were evaluated among adults. For children, health outcomes evaluated included respiratory and auditory effects. Univariate analyses were used to describe unadjusted associations between prevalence estimates of specific health outcomes and the distribution of social, demographic, and other characteristics of the study population. Univariate analyses also identified potentially important covariates that were considered in subsequent multivariate analyses.

Multivariate analysis (multiple logistic regression) was used to determine possible associations between the prevalence of targeted health outcomes and residence in low, medium, or high exposure areas while adjusting for potentially confounding factors. In addition, dose-response analyses were conducted to determine the significance of observed trends of increased prevalence of targeted health outcomes across exposure areas.

All analyses were conducted using SUDAAN, a statistical package designed for use with complex sampling methodologies, which incorporates weighting and variance calculations associated with the complex RDD sample design.

5.2 SURVEY DATA PREPARATION

HANDLING OF MISSING VALUES

In instances where survey respondents refused to answer or did not know the answer to a particular question, missing values were present in the data set. To avoid the exclusion of these individuals in the analysis, missing values on key demographic variables (age, sex, race, ethnicity, and household income) were replaced through imputation. Data imputation is a standard procedure in RDD surveys that determines the likely value of a

given variable based upon other known characteristics, either of the respondent or of another respondent with similar characteristics. All missing values for sex and some missing values for race and ethnicity were logically imputed. The remaining missing values for race and ethnicity were replaced using the "hot deck" method in which a missing value is replaced with the value reported by a similar respondent. Lastly, regression-based imputation was used to fill in missing values for age and household income.

With the exception of household income, which was missing for 13.6 % (n=828) of respondents, the rate of missing values was less than 5% for the five key demographic variables (sex = 7 (0.1%), race = 149 (2.5%), ethnicity = 51 (0.8%), age = 303 (5.0%)). A sensitivity analysis using both the imputed and non-imputed variables for comparison was conducted to ensure reliability of the results.

Missing values for variables other than age, sex, race, ethnicity, and household income were not replaced. However, a few variables did have higher rates of missing values. For example, body weight (and, therefore, BMI) was missing for 13% of adults and household size (and, therefore, poverty income ratio or PRI) was missing for 14% of adults. For these and other important categorical covariates with sizable amounts of missing data, an extra category was added to indicate a missing response and retain individuals with missing data in multivariate analyses.

SURVEY WEIGHTING METHODOLOGY

To account for complex survey designs, such as over-sampling of particular parts of a study area, survey data are often weighted. Weighting helps to prevent any biases that might occur because of differences between respondents and non-respondents (including those that weren't available for interview such as those without a working home telephone). The probability of being selected for the study varies for different people. For example, households with more than one telephone number have a higher probability of being selected into the sample. Therefore, weights are created to adjust the survey data so that it reflects a random sample of residents living in the study area and so the results can be considered a valid representation of the entire study area population.

Sampling weights for each person were created to include three types of adjustments. The "base weight" component accounts for the size of the overall survey sample and the sizes of the "high, and "medium" exposure area oversamples relative to the overall population of the study area. The "non-response weight" component takes into account the probability of being selected into the study based on the probabilities of various events occurring at each step during the survey process. These events include the probability of having a working home telephone number, of having one's telephone number be included among the working residential numbers dialed as part of the RDD sample of telephone numbers, of being the randomly selected adult in a contacted household, and of being able and available to complete the survey. During the survey interview, some questions are designed to collect information needed to calculate the non-response component of the sampling weights. Answers to certain questions, for instance, enable an estimation of the rate of telephone service interruption in the study area.

The final component of the sampling weights, referred to as a "post-stratification weight," is designed to correct for differences in certain characteristics (age, sex, race, ethnicity) among survey respondents compared to the study area population as a whole. This adjustment normalizes the demographic data of the survey sample to match the actual demographic characteristics of the study area population. To ensure that differences in demographics across the study area were accurately represented by the post-stratification weighting, the study area was divided, by zip code, into 18 smaller sections so that no sub-section contained fewer than 20 cases. Using 2000 US Census data, weights were created to adjust demographics in each of the 18 sub-sections to the actual demographics of the populations within those zip codes. Adjustments were made for age (18-34, 35-49, 50-64, 65+), sex (male, female), race (white, black, other), and ethnicity (Hispanic, non-Hispanic).

Finally, in order to reduce inflated design effects caused by extreme values in the distribution of survey weights, a weight-trimming procedure was performed. Weights larger than the chosen cut-off value of 4 times the mean of the sampling weights were trimmed at the cut-off value. The trimmed weights were then re-distributed across the sample to maintain the total weighted population size. This procedure resulted in weights

being trimmed for only a small number of subjects; 168 adults (2.8%) and 30 children (1.4%).

5.3 HEALTH OUTCOME DEFINITIONS

RESPIRATORY

Using information gathered from the LAHS survey instrument, the prevalence of asthma and respiratory symptoms were evaluated in a number of ways designed to capture varying degrees of illness and the presence of diagnosed and un-diagnosed asthma.

1. Lifetime asthma: A "yes" response to the question, "Has a medical doctor ever told you that you have asthma?"
2. Current asthma: Those with lifetime asthma who also replied "yes" to the question, "Do you still have asthma?"
3. Current asthma with medication use: Those with current asthma, who replied "yes" to the question, "Are you currently using medications prescribed by a medical doctor for your asthma?"
4. Probable asthma, adults: Those who reported never being diagnosed with asthma, but reported wheezing with dyspnea within the previous 12 months. Wheezing was asked as, "wheezing or whistling in your chest at any time" and dyspnea was asked as, "at all breathless when the wheezing noise was present."
5. Probable asthma, children: Those who reported never being diagnosed with asthma, but were reported to have experienced "wheeze or whistle in the chest" in the previous 12 months AND either "a dry cough at night apart from a cough associated with a cold or chest infection" or having "sounded wheezy during or after exercise."
6. Asthma hospitalization: Those with current asthma were asked to report the number of times during the previous 12 months that they either a) visited an emergency room or urgent care center or b) were hospitalized overnight for

asthma. Those who reported at least one incident of either are considered to be positive for this outcome.

Individual respiratory symptoms such as wheezing, chest tightness, and shortness of breath were also evaluated. Children were considered to have chronic bronchitis and/or chest infections if they were reported to have been diagnosed with bronchitis or a chest infection by a doctor on two or more occasions in the previous 12 months. Lastly, the prevalence of chronic obstructive pulmonary disease (COPD) was evaluated in adults. A person was considered to have COPD if they reported ever being told by a medical doctor that they have COPD, emphysema, or chronic bronchitis.

CARDIOVASCULAR

The prevalence of two cardiovascular outcomes was evaluated in adults, myocardial infarction (MI) and coronary heart disease (CHD). For MI, survey respondents were asked to report having ever been told by a medical doctor that they had a "heart attack or myocardial infarction." For CHD, they were asked whether they had ever been told by a medical doctor that they have "angina or coronary heart disease."

AUDITORY

For adults, auditory outcome questions included a follow-up component to ascertain age at diagnosis, and only diagnoses at or after age 18 were evaluated. First, adults were asked if a medical doctor had ever told them that they have a "hearing impairment." Those who reported having a hearing impairment were then asked if they currently use a hearing aid that they began using at or after age 18. Lastly, those with tinnitus were defined as persons who reported being told by a medical doctor that they have "tinnitus or ringing in the ears." For children, those with a hearing impairment were defined by a "yes" answer to the question of a medical doctor ever having diagnosed the child with a hearing impairment.

5.4 COVARIATE DEFINITIONS AND CLASSIFICATIONS

COVARIATES IN HEALTH SURVEY

The survey instrument was designed to collect information on as many factors, or covariates, as possible that have been identified in previous studies as being possibly related to the health outcomes of concern (i.e., respiratory, cardiovascular, and auditory).

Basic demographic factors that were considered include sex (male or female), age (18-39, 40-69, 70+), race (black, white, other), ethnicity (Hispanic, non-Hispanic), and Poverty Income Ratio (PIR) which is a measure of one's household income relative to the federal poverty guideline for one's household size. If a PIR is less than 1.0 then the household's income is below the federal poverty guideline. If a PIR is 2.0 then the household's income is twice the federal poverty guideline. PIR was categorized as follows: <1, 1-1.99, 2-3.5, 3.5+. Other characteristics considered include educational attainment, marital status, body mass index (BMI), smoking status (current, former, or never), gastro-esophageal reflux disease (GERD), alcohol intake, and binge drinking, which was defined as having five or more drinks on any one occasion during the previous 30 days.

Two occupational exposure measures were considered: reported current workplace exposure to dust, gas, or chemical fumes and reported lifetime exposure to loud noise at work for a duration of 3 months or more. A number of variables were considered that measure potential exposure in the home to various respiratory irritants. These include potential exposure in the home to: secondhand smoke; NO_2 from gas stoves, fireplaces, kerosene space heaters, etc.; mold; allergens from the presence of pets or cockroaches; and chemicals such as pesticides, paint, paint stripping, or polyurethane. Respondents were also asked about the presence of Massport soundproofing on their homes. Several cardiovascular risk factors were considered including high cholesterol, high blood pressure, diabetes, and family history of heart disease.

Lastly, a 10-year residential history was taken in order to provide some measure of each respondent's length of residency in the area. Respondents were asked to give the

location and year of move-in for their current residence and each previous residence in which they had lived since at least 1995. Notably, this section of the survey was one of the least completed sections as many people expressed reservations about providing residential location information. However, we were able to consider length of residence in the current home for 98% of respondents. In sensitivity analyses, final regression models were re-run after excluding residents who reported having lived in their current exposure area (based on current and previous residential address) for less than 1, 3, 5, and 10 years.

5.5 STATISTICAL ANALYSES

DESCRIPTIVE STATISTICS

Descriptive analyses were conducted separately for adults and children to assess the frequencies (percent of the population) with various socio-demographic characteristics. The prevalence of other potential factors (covariates) that may be associated with each specific outcome among adults and children were also estimated.

UNIVARIATE ANALYSES

The prevalence of each health outcome of interest was examined in the total population and among those living in each category of estimated airport-related air or noise pollution exposure. Differences across exposure areas for the crude, unadjusted, prevalence of each outcome were examined by chi-square tests, which were calculated using SUDAAN survey procedures to account for complex survey design and sample weighting.

MULTIVARIATE ANALYSES / MODEL-BUILDING

Multiple logistic regression analyses were conducted to examine the associations between each health outcome of interest and exposure to airport-related air or noise pollution while controlling for potentially confounding variables. Regression models were developed to adjust for covariates known to be strong predictors of the health outcome

being investigated and/or those found to have a meaningful impact on the exposure-disease association (defined as a change in the odds ratio (OR) of 10% or more). By including these additional factors in the regression models, results were adjusted for potential confounding and the influence other factors may have on any potential relationships between exposure and a particular health outcome was removed.

All adult respiratory, cardiovascular, and auditory models were adjusted for age, sex, race, ethnicity, PIR, educational attainment, and smoking status. Respiratory and cardiovascular outcome models were also adjusted for estimated non-airport-related residential $PM_{2.5}$ concentrations and residential proximity (<200 meters) to a major road (see Section 4.4 for more details). Additionally, respiratory outcome analyses were adjusted for household indoor smoking, and cardiovascular outcome analyses were adjusted for family history of heart disease, high blood cholesterol, hypertension, and diabetes. These variables were chosen *a priori* based on information in the scientific literature regarding demographic predictors and risk factors of respiratory, cardiovascular, and auditory diseases.

Additional covariates were also examined in a backwards elimination confounding assessment performed as follows. The association between exposure and disease was assessed in regression models adjusting for all base model variables (those selected a priori) and all potential confounders. Next, each potential confounder was removed, one-by-one, from the full model and the resulting OR for the association between exposure and disease was compared to the OR from the full model. The potential confounder whose removal was found to cause the least change in the OR (up to a maximum of 10%) was removed and the process was begun again, this time using the reduced model as the "full" model in the test. This process was repeated until only those variables causing a change in effect of 10% or more remained.

The confounding assessment resulted in the inclusion of binge drinking in both cardiovascular outcome models; the inclusion of BMI, alcohol intake, GERD, and potential exposure to household chemicals in the asthma hospitalization models; the inclusion of

potential exposure to household chemicals in COPD models; and the inclusion of occupational noise exposure in auditory outcome models. Massport soundproofing was evaluated as a confounder in adult auditory analyses but was not found to be influential.

Variables included *a priori* for all children's respiratory and hearing impairment models were age, sex, PIR, maternal education, and household indoor smoking. Respiratory outcome models were also adjusted for estimated non-airport-related residential $PM_{2.5}$ concentrations and residential proximity (<200 meters) to a major road (see Section 4.4 for more details). The confounding assessment performed as described above resulted in respiratory models being adjusted for the presence of household NO_2 sources, household allergens, and household mold and the children's hearing impairment model being adjusted for household mold and Massport soundproofing.

Statistical significance was evaluated at the α=0.05 level (i.e., p-values <0.05). Results with p-values between 0.05 and 0.20 were considered to be borderline statistically significant. Borderline statistically significant results are noted particularly in cases where estimates show a consistent effect in one direction or another across several analyses or among similar health outcomes, suggesting a consistent effect.

TREND ANALYSES

As described above, multivariate logistic regression models were used to examine the likelihood of having a particular health outcome among residents in the low exposure area compared to those in the medium and high exposure areas. Additionally, trend analyses were performed to evaluate the possibility of a linear pattern in the likelihood of having a particular health outcome as the potential for airport-related air pollution exposure increases across the low, medium, and high exposure areas. Linear trends were evaluated for each health outcome by modeling the categorical airport-related air pollution variable as a continuous variable in each final, multivariate logistic regression model based on the average relative increase in air pollution concentrations that was assigned to respondents within each exposure area.

To account for the exponential distribution of air pollution concentrations across the study area, exposure values for trend analyses were assigned as follows. First, the median concentration of each of the five air pollutants was calculated for each exposure area. Second, the relative increase in concentration was calculated for each pollutant across the three exposure areas moving from low to medium to high and the average relative increase was assigned for each exposure category. Thus, the resulting values of 1, 3.09, and 12.56 were assigned in this analysis. A trend was considered significant if the p-value for this exposure variable, modeled continuously, was less than 0.05 in final, multivariate logistic regression models.

6 RESULTS

6.1 SURVEY RESPONSE RATES

The response rate of a telephone survey is the proportion of eligible households in the sample that actually complete the survey. Response rates for the LAHS were calculated using standardized methods established by the American Association of Public Opinion Research (AAPOR, 2008). A total of 37670 telephone numbers were selected by RDD methodology for inclusion in the study. Of those, 10091 (26.8%) were identified as eligible households; 17294 (45.9%) were determined to be ineligible either as a non-residential number (16745) or an ineligible household (549); and 10285 (27.3%) were classified as being either residences of unknown eligibility (5443) or numbers that were always busy, never answered, or only answered by a machine that did not indicate whether or not the number belonged to a residence (4842).

The challenge in calculating response rates is to estimate how many telephone numbers of unknown eligibility actually belong to eligible households. There are many methods of estimating this figure (Smith, 2009), but for the purposes of the LAHS we chose to apply what is known as the Council of American Survey Research Organizations (CASRO) or proportional allocation method, which formed the basis of and is equivalent to AAPOR's Response Rate 3 (RR3). The proportional allocation method assumes that the proportion of eligible to ineligible telephone numbers is the same among those that were successfully classified as it is among those that were unable to be classified. The CASRO or AAPOR RR3 method is known to be a conservative approach that generally produces underestimates of a study's response rate, but it is also the most widely used and allows for comparison across studies. Based on this method, the LAHS survey response rate was calculated to be 35%. To provide what can be considered an upper-bound estimate of the response rate, we also calculated the AAPOR Response Rate 6 (RR6), which simply assumes that none of the unresolved telephone numbers belonged to eligible households. Using this method, the LAHS survey response rate would be 60%. The true response rate is believed to fall between the two estimates.

6.2 POPULATION CHARACTERISTICS

ADULTS

Demographic characteristics are presented in Table 6-1 for the 6072 adults in the LAHS survey sample, for the study area as a whole based on survey data weighted to be representative of the overall population, and for MA statewide based on 2010 US Census data. The majority of survey respondents were white (85%) and non-Hispanic (87%). Ten percent (10%) of respondents were Black or African-American, and 5% classified their race as Asian, Pacific Islander, Native Hawaiian, American Indian, or another race. Thirteen percent (13%) of respondents identified as Hispanic or Latino. Compared to the MA population statewide, the study area population has a proportionally greater numbers of Black or African American residents (12% vs. 6%) and fewer White residents (76% vs. 82%).

More women than men responded to the survey (64% vs. 36%). The population is more evenly distributed between women and men in the study area overall (56% vs. 44%) and statewide (52% vs. 48%). A little more than half (54%) of the adults surveyed were between the ages of 40 and 69 years; 31% were aged 18-39 years; and about 15% were aged 70 years or above. The study area population overall is somewhat younger than that of the state with 46% vs. 38% of residents being aged 18-39. One third (32%) of adults in the study area lived in households with incomes less than twice the 2005 federal poverty guideline. Fourteen percent (14%) had household incomes below federal poverty guidelines compared to 11% statewide. Almost 90% of respondents had a high-school diploma or GED; 62% reported educational attainment of at least some college; and 42% had earned a bachelor's degree or higher. In the study population overall there was a somewhat higher degree of educational attainment compared to the state as a whole.

Table 6-1. Basic Demographic Characteristics for the 6072 Adult Participants of the Logan Airport Health Study (LAHS) Survey, Study Area Population, and Statewide

Characteristic[a]	Survey Respondents, Percent (%)	Study Area Population[b], Percent (%)	Statewide Population[c], Percent (%)
Race			
White	85	76	82
Black	10	12	6
Other	5	12	12
Ethnicity			
Non-Hispanic	87	90	92
Hispanic	13	10	8
Sex			
Male	36	44	48
Female	64	56	52
Age			
18-39	31	46	38
40-69	54	42	50
70+	15	12	12
Poverty Index Ratio			
< 1	15	14	11
1-1.99	19	18	13
2-3.5	23	23	n/a
3.5+	41	44	n/a
Missing	1	1	-
Education			
Less than high-school	10	8	12
High-school grad/GED	27	22	28
Some college/tech school	20	20	18
Bachelor's degree	23	26	24
Graduate school	19	23	18
Missing	1	1	-

[a]Missing values for age, sex, race, ethnicity, and household income were imputed. Poverty Income Ratio (PIR) was calculated using household income and household size; due to missing information on household size, some PIR values are missing. See Chapter 5, Data Analysis, for more information on missing values and imputation.
[b]Study area population estimates were calculated using survey data weighted to be representative of the study area in terms of race, ethnicity, age, sex, and household income.
[c]Statewide demographics are from the 2010 US Census, which does not calculate PIR prevalence for the same ranges used in this study. PIR statewide prevalence for adults in MA: 2.00-2.99=13%; 3.00-3.99=13%; 4.00+=50%.

CHILDREN

The sample population in the LAHS comprised 2215 children aged 3-17 years living in 1379 of the 6072 participating households. Information about the children was reported by adult survey responders. Presented in Table 6-2 are demographic characteristics for children in the LAHS survey sample, in the study area as a whole based on survey data weighted to be representative of the overall population, and in MA statewide based on 2010 US Census data.

Slightly more males than females (53% to 47%) were a part of the study's sample of children, although in the study area overall and in MA statewide the proportion is 51% male to 49% female. A little more than half (56%) of the survey children were aged 3-11 years, while the remaining 44% were aged 12-17 years. In the survey area overall and in MA statewide, the proportion of younger children is slightly higher (61% and 58%, respectively). Almost half (48%) of survey children lived in households with incomes less than twice the 2005 federal poverty guideline. Twenty-three percent (23%) had household incomes below federal poverty guidelines. These figures match those of the study population, in general, but are higher than the proportion of MA children living in households with incomes less than two times the poverty, which was 29% in 2005. The mothers of 81% of survey children had a high-school diploma or GED; 57% reported educational attainment of at least some college; and 36% had earned at least a bachelor's degree. These educational attainment figures were similar for the study area population overall and for the state of MA.

Table 6-2. Basic Demographic Characteristics of the 2215 Child Participants of the Logan Airport Health Study (LAHS) Survey, for the LAHS study Area, and for the State Population

Characteristic[a]	Survey Respondents, Percent (%)	Study Area Population[b], Percent (%)	Statewide Population[c], Percent (%)
Sex			
Male	53	51	51
Female	47	49	49
Age			
3-11	56	61	58
12-17	44	39	42
Poverty Index Ratio			
< 1	23	24	14
1-1.99	25	25	15
2-3.5	21	18	n/a
3.5+	31	33	n/a
Missing	0.1	0.03	-
Maternal Education			
Less than high-school	17	15	14
High-school grad or GED	24	22	19
Some college/tech school	21	21	29
Bachelor's degree	23	25	24
Graduate school	13	14	14
Missing	3	2	-

[a]Missing values for age, sex, and household income were imputed. Poverty Income Ratio (PIR) was calculated using household income and household size; due to missing information on household size, some PIR values are missing. See Chapter 5, Data Analysis, for more information on missing values and imputation.
[b]Study area population estimates were calculated using survey data weighted to be representative of the study area in terms of race, ethnicity, age, sex, and household income.
[c]Statewide demographics are from the 2010 US Census, which does not calculate PIR prevalence for the same ranges used in this study. PIR statewide prevalence for children in MA: 2.00-2.99=14%; 3.00-3.99=13%; 4.00+=44%.

6.3 DISEASE BURDEN IN THE TOTAL STUDY AREA

ADULTS

Table 6-3 presents the prevalence of targeted respiratory and cardiovascular health outcomes within the entire LAHS area using weighted sample data to be representative of the study area in terms of race, ethnicity, age, sex, and household income. For more information about the weighting process, refer to Chapter 5 (section 5.2.2).

The percentage of people having ever had a diagnosis of asthma (lifetime asthma) within the study area was found to be 15.5%. A little over one tenth of the population (11.0%) reported currently having asthma, and 7.7% had current asthma that required the use of medication. In addition, of those who had never been diagnosed with asthma, 4.6% reported symptoms indicative of asthma (probable asthma). Of adult asthmatics, 24.7% reported having had an asthma-related hospital visit in the year prior to the survey. Diagnosis of COPD was reported by 6.6% of adults. Statewide prevalence of these health outcomes is provided in the footnotes in Table 6-3.

Within the LAHS area, the prevalence of lifetime asthma, current asthma, and COPD was higher in 2005 than the statewide prevalence of these diseases; the differences are not statistically significant. For CHD and MI, the prevalence in the LAHS area is lower than that of the state as a whole. Again, these differences are not statistically significant.

Table 6-3. Prevalence Estimates of Respiratory and Cardiovascular Diseases Among Adults in the Logan Airport Health Study Area (2005)

Health Outcome	Sample Size[a]	Prevalence, Percent (%)[b, c]	95% Confidence Interval
Lifetime Asthma	6055	15.5	(14.3 - 16.8)
Current Asthma	6032	11.0	(9.9 - 12.1)
Current Asthma With Medication Use	6031	7.7	(6.8 - 8.6)
Probable Asthma	5136	4.6	(3.8 - 5.4)
Asthma Hospitalization	673	24.7	(20.2 - 29.1)
Chronic Obstructive Pulmonary Disease (COPD)	6010	6.6	(5.8 - 7.4)
Coronary Heart Disease (CHD)	6008	3.3	(2.7 - 3.8)
Myocardial Infarction (MI)	6019	3.5	(3.0 - 4.1)

[a]Sample sizes vary slightly based on the number of "don't know/refuse" responses for each outcome. Additionally, probable asthma was assessed only among those without lifetime asthma, and asthma hospitalizations were assessed only among those with current asthma.
[b]Survey data were weighted to population demographics to produce prevalence estimates representative of the study area.
[c]Statewide prevalence based on BRFSS and MMWR (for COPD) are as follows:
2005 Lifetime asthma = 14.2% (95% CI: 13.2-15.2);
2005 Current asthma = 9.6% (95% CI: 8.7-10.5);
2011 COPD = 5.4% (95% CI: 5.0-5.9);
2005 CHD = 4.0% (95% CI: 3.5-4.5);
2005 MI = 4.2% (95% CI: 3.7-4.7).

CHILDREN

Estimates of the prevalence of respiratory outcomes among children in the LAHS area are presented in Table 6-4. The prevalence of health outcomes was calculated using weighted sample data to be representative of the study area in terms of race, ethnicity, age, sex, and household income. For more information about the weighting process, refer to Chapter 5 (section 5.2.2).

The overall prevalence of lifetime asthma among children in the LAHS study area in 2005 was 21.2% compared to the statewide 2005-2007 average among children (14.4%). The prevalence of current asthma among children in the LAHS was 14.8% of children compared with the statewide 2005-2007 average of 10.3%. About 12.6% of children in the LAHS were reported to have current asthma that required the use of medication. Among children not reported to have been diagnosed with asthma, 3.6% were reported to have symptoms consistent with asthma (probable asthma). Of the children reported to have current asthma, 41.0% had visited the hospital for asthma at least once in the previous 12 months. As presented in Table 6-4, for both lifetime asthma and current asthma, the prevalence in children in the LAHS area is statistically significantly higher than among children statewide.

Table 6-4. Prevalence Estimates of Respiratory Disease Among Children in the Logan Airport Health Study Area (2005)

Health Outcome	Sample Size[a]	Prevalence, Percent (%)[b, c]	95% Confidence Interval
Lifetime Asthma	2201	21.2	(18.9 - 23.5)
Current Asthma	2192	14.8	(12.8 - 16.8)
Current Asthma with Medication Use	2191	12.6	(10.7 - 14.5)
Probable Asthma	1749	3.6	(2.5 - 4.7)
Asthma Hospitalization	328	41.0	(33.6 - 48.3)
Chronic Bronchitis/ Chest Infections	2203	6.3	(4.9 - 7.7)

[a]Sample sizes vary slightly based on the number of "don't know/refuse" responses for each outcome. Additionally, probable asthma was assessed only among those without lifetime asthma, and asthma hospitalizations were assessed only among those with current asthma.
[b]Survey data were weighted to population demographics to produce prevalence estimates representative of the study area.
[c]Statewide prevalence of children's asthma from BRFSS data as reported in MDPH (2009):
2005-2007 Lifetime asthma: 14.4% (95% CI: 13.0-15.8);
2005-2007 Current asthma: 10.3% (95% CI: 9.1-11.5).

6.4 CRUDE UNIVARIATE RESULTS: PREVALENCE OF HEALTH OUTCOMES BY AIRPORT-RELATED AIR POLLUTION EXPOSURE AREAS

ADULTS

As discussed earlier, the estimated prevalence of some health outcomes for the total study area was somewhat higher than expected based upon statewide data (refer to Tables 6-3 and 6-4). However, in order to determine if prevalence was higher due to potential exposure to air pollution related to Logan airport, disease prevalence was evaluated separately by modeled airport-related air pollution exposure categories.

The prevalence estimates for health outcomes in adults are presented in Table 6-5. The high exposure area, compared to the low exposure area, had a higher prevalence of current asthma (12.2% vs. 10.8%), current asthma that required medication (9.9% vs. 7.3%), and probable asthma (7.1% vs. 4.4%). Among adult asthmatics, report of an asthma-related hospital visit was almost twice as common in the high exposure area compared to the low (40.3% vs. 22.5%). Of these comparisons, none were statistically significant at an α=0.05 level. However, prevalence of COPD was statistically significantly different (p = 0.001) across exposure areas, with 11.0% reported in the high exposure area, 7.8% reported in the medium exposure area, and 5.7% reported in the low exposure area.

Cardiovascular diseases were reported with a similar frequency among adults in the entire study area and across air pollution exposure categories. CHD was found to be present in 3.3% of the overall study population, and non-fatal MI was experienced by 3.5% of the overall study population.

Table 6-5. Prevalence Estimates of Respiratory and Cardiovascular Diseases Among Adults by Category of Modeled Airport-related Air Pollution Exposure in the Logan Airport Health Study Area (2005)

Health Outcome	Sample Size[a]	Prevalence[b], % (95% Confidence Interval) Exposure Category Low	Medium	High
Lifetime Asthma	6055	15.5 (13.9 - 17.1)	15.5 (13.1 - 17.8)	15.9 (12.2 - 19.6)
Current Asthma	6032	10.8 (9.4 - 12.2)	11.2 (9.2 - 13.3)	12.2 (8.7 - 15.6)
Current Asthma with Medication Use	6031	7.3 (6.2 - 8.4)	8.3 (6.6 - 10.1)	9.9 (6.6 - 13.2)
Probable Asthma	5136	4.4 (3.4 - 5.4)	4.6 (3.2 - 6.0)	7.1 (4.8 - 9.4)
Asthma Hospitalization	673	22.5 (17.0 - 28.0)	26.0 (17.5 - 34.4)	40.3 (24.6 - 56.0)
COPD	6010	5.7 (4.8 - 6.7)	7.8* (6.3 - 9.4)	11.0* (8.1 - 14.6)
Coronary Heart Disease	6008	3.5 (2.8 - 4.2)	2.5 (1.7 - 3.3)	3.4 (1.1 - 5.7)
Myocardial Infarction	6019	3.6 (2.9 - 4.4)	3.4 (2.5 - 4.4)	3.2 (1.8 - 4.6)

[a]Sample sizes vary slightly based on the number of "don't know/refuse" responses for each outcome. Additionally, probable asthma was assessed only among those without lifetime asthma, and asthma hospitalizations were assessed only among those with current asthma.
[b]Survey data were weighted to population demographics to produce prevalence estimates representative of each exposure area.
*Indicates statistical significance (p-value <0.05) for comparison with the low exposure category.

CHILDREN

Similarly, the prevalence of respiratory outcomes among children was also examined for those living in each of the three modeled airport-related air pollution exposure areas (Table 6-6). The prevalence of lifetime asthma, current asthma, current

asthma requiring medication, and asthma hospitalizations among children were similar for the total study area and across all three exposure areas. No clear patterns emerged; however, probable or undiagnosed asthma was more prevalent in the high exposure area than in the medium or low exposure areas (7.3% vs. 3.4% and 3.3%, respectively). The differences were not statistically significant. Similarly, the prevalence of chronic bronchitis increased as the potential for exposure increased, but the differences were not statistically significant (8.4%, 7.1%, 5.9%, in the high, medium and low exposure areas, respectively).

Table 6-6. Prevalence Estimates of Respiratory Disease Among Children by Category of Modeled Airport-related Air Pollution Exposure in the Logan Airport Health Study Area (2005)

Health Outcome	Sample Size[a]	Prevalence[b], % (95% Confidence Interval)		
		Exposure Category		
		Low	Medium	High
Lifetime Asthma	2201	22.1 (19.2 - 25.0)	18.5 (14.5 - 22.6)	19.1 (13.6 - 24.7)
Current Asthma	2192	15.2 (12.7 - 17.7)	13.7 (10.1 - 17.3)	14.4 (9.1 - 19.6)
Current Asthma with Medication Use	2191	12.8 (10.4 - 15.1)	11.9 (8.4 - 15.3)	12.7 (7.7 - 17.7)
Probable Asthma	1749	3.3 (2.0 - 4.6)	3.4 (1.1 - 5.8)	7.3 (3.1 - 11.5)
Asthma Hospitalization	328	41.6 (32.5 - 50.6)	38.6 (25.0 - 52.3)	41.5 (23.2 - 59.9)
Chronic Bronchitis/ Chest Infections	2203	5.9 (4.2 - 7.6)	7.1 (4.4 - 9.8)	8.4 (5.1 - 11.7)

[a]Sample sizes vary slightly based on the number of "don't know/refuse" responses for each outcome. Additionally, probable asthma was assessed only among those without lifetime asthma, and asthma hospitalizations were assessed only among those with current asthma.
[b]Survey data were weighted to population demographics to produce prevalence estimates representative of each exposure area.

6.5 ADJUSTED MULTIVARIATE REGRESSION MODELS: ASSOCIATIONS BETWEEN ADVERSE HEALTH EFFECTS AND AIR POLLUTION EXPOSURE AREAS

The prevalence estimates presented in Tables 6-3 through 6-6 are not adjusted for any demographic or other risk factor information that could partially explain differences across exposure areas. In order to better isolate the relationship between targeted health outcomes and estimated exposure to airport-related air pollution, logistic regression models were developed that assess the relationships while adjusting for demographic variables and other potentially confounding risk factors. For more information regarding the development of regression models and the selection of covariates, refer to Chapter 5 (Section 5.4).

ADULTS

Presented in Table 6-7 are adjusted odds ratios for the associations between respiratory and cardiovascular outcomes and residence in low, medium, or high estimated airport-related air pollution exposure areas.

While some odds ratios were elevated in the high compared to the low exposure areas, these associations were not statistically significant. However, results were observed for the likelihood of having COPD in the high versus low exposure areas approached statistical significance (OR=1.5, 95% CI: 0.9-2.5). There was no association observed between air pollution exposure areas and other measures of asthma including lifetime asthma, current asthma not specified with respect to medication usage, and probable/undiagnosed asthma.

Table 6-7. Estimated Exposure to Airport-related Air Pollution and Adjusted Odds of Respiratory and Cardiovascular Disease Among Adults Living in the Logan Airport Health Study Area (2005)[a]

Health Outcome	Sample Size	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value
Lifetime Asthma	5829				
Low		1.0	1.0	1.0	
Medium		1.0	0.8	1.2	*0.77*
High		1.0	0.7	1.5	*0.82*
Current Asthma	5806				
Low		1.0	1.0	1.0	
Medium		1.0	0.8	1.3	*0.93*
High		1.2	0.8	1.8	*0.43*
Current Asthma with Medication Use	5805				
Low		1.0	1.0	1.0	
Medium		1.1	0.8	1.5	*0.46*
High		1.4	0.8	2.3	*0.20*
Probable Asthma	4934				
Low		1.0	1.0	1.0	
Medium		0.8	0.5	1.2	*0.31*
High		1.1	0.6	1.9	*0.77*
Asthma Hospitalization[b]	638				
Low		1.0	1.0	1.0	
Medium		1.2	0.6	2.3	*0.63*
High		1.3	0.5	3.7	*0.56*

[a]All models were adjusted for age, sex, race, ethnicity, household income (PIR), education, smoking status, and background air pollution exposure. Respiratory outcome models were also adjusted for household indoor smoking. Cardiovascular outcome models (MI and CHD) were also adjusted for binge drinking, diabetes, hypertension, high cholesterol, and family history of heart disease. Exposure to background air pollution was adjusted using estimated residential background $PM_{2.5}$ concentrations and residential proximity to a major road (<200 meters).
[b]Analysis conducted among those with current asthma. Also adjusted for BMI, alcohol intake, GERD, and use of chemicals such as pesticides in the home.
[c]Also adjusted for use of chemicals such as pesticides in the home.

Table 6-7 *continued on page 75*

Table 6-7 *continued*

Health Outcome	Sample Size	Odds Ratio[a]	Lower 95% CI	Upper 95% CI	p-value
COPD[c]	5689				
Low		1.0	1.0	1.0	
Medium		1.2	0.9	1.7	*0.22*
High		1.5	0.9	2.5	*0.08*
Coronary Heart Disease	5603				
Low		1.0	1.0	1.0	
Medium		0.7	0.4	1.2	*0.22*
High		1.1	0.4	3.1	*0.86*
Myocardial Infarction	5608				
Low		1.0	1.0	1.0	
Medium		1.0	0.7	1.6	*0.89*
High		0.8	0.4	1.7	*0.62*

[a]All models were adjusted for age, sex, race, ethnicity, household income (PIR), education, smoking status, and background air pollution exposure. Respiratory outcome models were also adjusted for household indoor smoking. Cardiovascular outcome models (MI and CHD) were also adjusted for binge drinking, diabetes, hypertension, high cholesterol, and family history of heart disease. Exposure to background air pollution was adjusted using estimated residential background $PM_{2.5}$ concentrations and residential proximity to a major road (<200 meters).
[b]Analysis conducted among those with current asthma. Also adjusted for BMI, alcohol intake, GERD, and use of chemicals such as pesticides in the home.
[c]Also adjusted for use of chemicals such as pesticides in the home.

Although higher prevalences of current and probable asthma were observed in the high exposure area in crude, univariate, analyses (Table 6.5), when potential confounding factors were controlled for in this analysis, no differences were observed across the high, medium, and low exposure areas for the likelihood of current or probable asthma. Tests of trend were not significant for any of the targeted respiratory outcomes.

The likelihood of non-fatal myocardial infarction and coronary heart disease were not found to be different across air pollution exposure areas. Tests of trend were not significant for any of the targeted cardiovascular outcomes.

Taking length of residence into account, respiratory and cardiovascular disease associations were re-evaluated in individuals having lived in their current exposure areas for at least 1, 3, 5, or 10 years. The results for each length of residence analysis are presented in Table 6-8. Overall, restricting the analyses to individuals who had lived in their current exposure areas for 3 or more years and for 5 or more years produced stronger associations between potential airport-related air pollution exposure and most of the targeted respiratory outcomes. These stronger associations reach the level of statistical significance for COPD (3+ years OR=1.8, 95% CI: 1.1-3.0) and are of borderline significance for current asthma with medication use (5+ years OR=1.7, 95% CI: 1.0-3.0) in the high exposure area compared to the low exposure area. These stronger associations did not persist when restricting to individuals with 10+ years residence in their current exposure areas, possibly due to the loss of sample size or possibly because length of residence isn't a strong factor for these outcomes beyond 5 years or so.

For MI and CHD, there is very little change in effect when length of residency in an exposure area is considered except for the 10+ years analysis. For those longer term residents, the estimated likelihood of MI is somewhat higher for those in the medium and high exposure areas compared to those in the low, but the association is not statistically significant. Conversely, the estimated likelihood of CHD is lower in the medium and high exposure areas compared to those in the low, but again the results are not statistically significant

Table 6-8. Estimated Exposure to Airport-related Air Pollution and Odds of Respiratory and Cardiovascular Disease Among Adults Living in the Logan Airport Health Study Area Who Have Resided in their Respective Exposure Area for at least 1, 3, 5, or 10 years[a]

Health Outcome	Residence 1+ yrs					Residence 3+ yrs					Residence 5+ yrs					Residence 10+ yrs				
	n	OR	LCI	UCI	p-value	n	OR	LCI	UCI	p-value	n	OR	LCI	UCI	p-value	n	OR	LCI	UCI	p-value
Lifetime Asthma	5511					4711					4061					3076				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.0	0.8	1.2	*0.80*		1.0	0.8	1.4	*0.78*		1.0	0.7	1.3	*0.90*		0.9	0.6	1.3	*0.69*
Higher		1.1	0.8	1.6	*0.60*		1.2	0.8	1.7	*0.48*		1.1	0.7	1.7	*0.61*		1.0	0.6	1.6	*0.97*
Current Asthma	5491					4692					4043					3066				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.0	0.7	1.3	*0.97*		1.1	0.8	1.5	*0.41*		1.1	0.8	1.5	*0.54*		1.0	0.7	1.5	*0.96*
Higher		1.2	0.8	1.9	*0.32*		1.3	0.8	2.0	*0.29*		1.3	0.8	2.1	*0.29*		1.1	0.6	1.9	*0.71*
Current Asthma with Meds	5490					4692					4043					3066				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.1	0.8	1.5	*0.62*		1.2	0.8	1.6	*0.40*		1.2	0.8	1.7	*0.33*		1.0	0.6	1.6	*0.96*
Higher		1.5	0.9	2.5	*0.12*		1.6	1.0	2.7	*0.07*		1.7	1.0	3.0	*0.05*		1.5	0.8	2.7	*0.23*
Probable Asthma	4673					4007					3459					2646				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		0.8	0.5	1.3	*0.46*		0.9	0.5	1.5	*0.62*		1.1	0.6	2.0	*0.81*		1.1	0.6	2.3	*0.75*
Higher		1.1	0.6	1.9	*0.81*		1.1	0.6	1.9	*0.88*		1.1	0.6	2.1	*0.80*		1.3	0.6	2.7	*0.53*
Asthma Hospitalization[b]	598					508					436					312				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.2	0.6	2.4	*0.60*		1.2	0.6	2.7	*0.59*		1.5	0.7	3.4	*0.34*		1.6	0.6	4.4	*0.40*
Higher		1.6	0.6	4.5	*0.34*		1.5	0.5	4.0	*0.46*		1.6	0.5	4.5	*0.42*		1.5	0.4	5.2	*0.57*
COPD[c]	5394					4619					3990					3031				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.3	0.9	1.8	*0.11*		1.6	1.1	2.3	*0.01*		1.6	1.1	2.4	*0.01*		1.4	0.9	2.2	*0.13*
Higher		1.5	0.9	2.5	*0.09*		1.8	1.1	3.0	*0.02*		1.6	0.9	2.8	*0.09*		1.4	0.8	2.4	*0.22*
Coronary Heart Disease	5301					4520					3897					2950				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		0.8	0.5	1.3	*0.39*		0.8	0.5	1.3	*0.37*		0.8	0.4	1.3	*0.34*		0.6	0.3	1.1	*0.09*
Higher		1.3	0.4	3.7	*0.67*		1.3	0.4	3.7	*0.69*		1.3	0.4	4.0	*0.68*		0.7	0.3	1.6	*0.39*
Myocardial Infarction	5305					4525					3902					2953				
Lower		1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0			1.0	1.0	1.0	
Medium		1.1	0.7	1.7	*0.83*		1.0	0.6	1.6	*0.94*		1.1	0.6	1.7	*0.83*		1.2	0.7	2.0	*0.53*
Higher		0.9	0.5	1.8	*0.79*		0.8	0.4	1.7	*0.59*		0.9	0.4	1.8	*0.70*		1.2	0.6	2.4	*0.65*

[a]All models were adjusted for age, sex, race, ethnicity, household income (PIR), education, smoking status, and background air pollution exposure. Respiratory outcome models were also adjusted for household indoor smoking. Cardiovascular outcome models (MI and CHD) were also adjusted for binge drinking, diabetes, hypertension, high cholesterol, and family history of heart disease. Exposure to background air pollution was adjusted using estimated residential background $PM_{2.5}$ concentrations and residential proximity to a major road (<200 meters).

[b]Analysis conducted among those reporting current asthma. Also adjusted for BMI, alcohol intake, GERD, and use of chemicals such as pesticides in the home.

[c]Also adjusted for the use of chemicals such as pesticides in the home.

CHILDREN

Table 6-9 presents results of multivariate logistic regression models for associations between estimated airport-related air pollution exposure and respiratory outcomes in children. There were no differences across respiratory health outcomes in children in the unadjusted univariate analysis. No associations were observed across air pollution exposure areas for either lifetime asthma or current asthma that requires use of medication; nor were asthmatic children in different air pollution exposure areas more or less likely to visit the hospital for asthma-related illness.

The strongest statistically significant association between air pollution exposure area and respiratory outcomes in children was observed for the likelihood of probable (or undiagnosed) asthma, which was 3.6 times higher for children in the high exposure area compared to those in the low exposure area (OR=3.6, 95% CI: 1.1-11). There was also a statistically significant trend of higher likelihood of probable asthma in children with increasing potential exposure to airport-related air pollution (p=0.03).

Table 6-9. Estimated Exposure to Airport-related Air Pollution and Adjusted Odds of Respiratory Disease Among Children in the Logan Airport Health Study Area (2005)[a]

Health Outcome	Sample Size	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value
Lifetime Asthma	2081				
Low		1.0	1.0	1.0	
Medium		0.9	0.6	1.3	*0.51*
High		1.0	0.6	1.6	*0.94*
Current Asthma[b]	2072				
Low		1.0	1.0	1.0	
Medium		1.0	0.7	1.6	*0.90*
High		1.2	0.7	2.3	*0.52*
Current Asthma with Medication Use	2071				
Low		1.0	1.0	1.0	
Medium		0.9	0.6	1.5	*0.75*
High		1.0	0.5	2.0	*0.90*
Probable Asthma[b]	1644				
Low		1.0	1.0	1.0	
Medium		1.3	0.5	3.0	*0.58*
High		3.6	1.1	11	*0.03*
Asthma Hospitalization	319				
Low		1.0	1.0	1.0	
Medium		0.6	0.3	1.5	*0.33*
High		1.0	0.3	3.3	*0.96*
Chronic Bronchitis / Chest Infections	2082				
Low		1.0	1.0	1.0	
Medium		1.2	0.6	2.2	*0.61*
High		1.7	0.7	4.0	*0.25*

[a]All models were adjusted for age, sex, household income (PIR), maternal education, household indoor smoking, household NO2 sources, household allergens, household mold, and background air pollution exposure. Exposure to background air pollution was adjusted using estimated residential background $PM_{2.5}$ concentrations and residential proximity to a major road (<200 meters).

[b]Test of trend for this association was statistically significant ($p<0.05$).

6.6 ADJUSTED MULTIVARIATE REGRESSION MODELS: ASSOCIATIONS BETWEEN AUDITORY HEALTH EFFECTS AND NOISE EXPOSURE AREAS

Table 6-10 presents results of multivariate logistic regression models for associations between categories of airport-related noise exposure and auditory health outcomes in adults and children. Children's analyses were adjusted for the presence or absence of home soundproofing obtained from Massport as part of their noise abatement program. Massport soundproofing was evaluated in adult models, as well, but was not found to have any effect on the association between noise exposure category and hearing impairment and so was not included in final models. No statistically significant associations were observed for either adults or children and confidence intervals were wide. The direction of effect indicated by the ORs (lower than 1 or higher than 1) varied for the three outcomes evaluated in adults.

Table 6-10. Estimated Exposure to Airport-related Noise and Adjusted Odds of Auditory Impairment Among Adults and Children Living in the Logan Airport Health Study Area (2005)

Health Outcome	Sample Size	Odds Ratio	Lower 95% CI	Upper 95% CI	p-value
Adults[a]					
Hearing Impairment	5714				
Low, ≤ 59 dB		1.0	1.0	1.0	
Medium, 60-64 dB		0.6	0.4	1.0	*0.07*
High, ≥ 65 dB		0.9	0.3	2.5	*0.80*
Hearing Impairment and Uses Hearing Aid[b]	3693				
Low, ≤ 59 dB		1.0	1.0	1.0	
Medium, 60-64 dB		0.6	0.2	1.7	*0.32*
High, ≥ 65 dB		1.9	0.2	15	*0.54*
Tinnitus	5775				
Low, ≤ 59 dB		1.0	1.0	1.0	
Medium, 60-64 dB		0.8	0.5	1.5	*0.53*
High, ≥ 65 dB		0.5	0.1	2.1	*0.35*
Children[c]					
Hearing Impairment	2105				
Low, ≤ 59 dB		1.0	1.0	1.0	
Medium, 60-64 dB		0.7	0.3	2.0	*0.53*
High, ≥ 65 dB		1.7	0.4	7.5	*0.50*

[a]All adult models were adjusted for age, sex, race, ethnicity, household income (PIR), education, smoking status, and occupational noise exposure.
[b]Hearing impairment combined with hearing aid use was assessed only among adults aged 40 years or greater due to very low prevalence in younger adults.
[c]Children's hearing impairment model adjusted for age, sex, household income (PIR), maternal education, household indoor smoking, household mold, and Massport soundproofing.

7 DISCUSSION

As directed by the Massachusetts legislature, the MDPH/BEH conducted an assessment of health impacts of Logan Airport on residents of seventeen communities located within a 5 mile radius of the airport. With input from key stakeholders, including residents and local health departments, and from experts in the fields of epidemiology, risk assessment, survey design and administration, and air pollution modeling, the MDPH/BEH designed and conducted a cross-sectional disease and symptom prevalence study. The purpose of the study was to investigate associations between potential exposure to airport emissions and adverse respiratory, cardiovascular, and auditory health outcomes among residents of seventeen communities surrounding Logan Airport.

The study area was geographically stratified into three exposure areas based on the best available data for predicting ambient concentrations of air pollution and noise associated with operations at the airport. In addition, a sample of residents from across the study area, statistically representative of the total study area, was interviewed to collect information on the prevalence of the targeted health outcomes and other relevant demographic and risk factor information. The prevalence estimates of respiratory, cardiovascular, and auditory outcomes in the LAHS communities were initially compared to those of the state of MA to evaluate the general burden of disease in the study area. Finally, statistical analyses were conducted to address the primary goal of the LAHS, which was to determine whether residents living in areas with greater potential for airport-related exposures were more likely to experience the targeted health outcomes compared to residents living in areas with lesser potential for airport-related exposures. The final analyses also took into consideration the effects of confounding factors, such as smoking, which could mask or mimic the Logan-related exposure under study.

Overall, the population of the LAHS area appeared to have a greater burden of current and lifetime asthma in both adults and children than the state as a whole. In contrast, the prevalence of cardiovascular disease was lower than observed statewide. Patterns in the prevalence of these outcomes emerged as analyses looked more closely at where individuals

lived in the study area and after accounting for other risk factors for the outcomes investigated.

Even though current and lifetime asthma appeared to be high in the LAHS total adult population, when considering the potential for exposure to airport-related air pollution and other risk factors for asthma, these outcomes did not appear associated with Logan Airport. Final analyses in adults did indicate that COPD was statistically significantly higher for residents who lived 3 or more years in the high exposure area.

The more in-depth statistical analyses did not alter the initial conclusion that cardiovascular disease was not higher in the LAHS population. The reported occurrence of both heart attacks and coronary heart disease were not found to be associated with potential exposure to pollutants from the airport.

The strongest results for children were somewhat different from those observed for adults. Children who lived in the high exposure area had a greater likelihood of undiagnosed asthma (probable asthma) than children who lived in the low exposure area.

The following discussion presents how the related scientific literature and the methods employed in the LAHS support its findings. Limitations in the methods and their possible impact on the results are also discussed.

RESPIRATORY OUTCOMES

The burden of asthma in both adults and children in the LAHS area was found to be higher than that of the statewide population of adults and children. For children, the 2005 prevalence of asthma (current and lifetime) was statistically significantly higher in the LAHS area than in the state as a whole. This result is not particularly surprising considering that the LAHS area is made up of the largest metropolitan region of MA, including the city of Boston, and it is well-recognized that urban areas tend to have higher rates of respiratory disease including asthma (Sunyer et al., 1997; Galea et al., 2005; Cohen et al., 2006), particularly among children (Corburn et al., 2006; Clougherty et al., 2007; Gern et al., 2009).

Many factors have been suggested to explain the increased asthma risk in urban areas including low socioeconomic status (Gern et al., 2009), stress and violence (Williams et al., 2009), exposure to rodent and cockroach allergens, indoor exposures to mold and environmental tobacco smoke, and air pollution (Corburn et al., 2006).

The role of air pollution in the higher prevalence of lifetime and current asthma among adults and children living in the LAHS area is less clear based upon the scientific literature. Although still under investigation, research on the association between air pollution exposure and asthma prevalence and/or development has thus far been less consistent than that of air pollution and exacerbation of asthma and respiratory symptoms. In children, a large cross-sectional study of asthma (>500,000 children in 105 cities/51 countries) called the International Study of Asthma and Allergies in Childhood (ISAAC), found no association between change in prevalence in asthma and change in $PM_{2.5}$ levels (Anderson et al., 2010). On the other hand, when evaluating traffic-related exposures, most studies have observed an association with asthma prevalence (Braback et al., 2009; Jerrett et al., 2008; Lindgren et al., 2010; McConnell, 2006; McConnell et al., 2010; Salam et al., 2008); however, not all traffic-related exposure studies find an effect on asthma prevalence (Pujades-Rodriguez et al., 2009). Long term exposure to ozone (O_3), in particular, has been associated with both new-onset asthma in children and increased respiratory symptoms in current asthmatics (US EPA, 2013). Notably, from 1997-2008, the eastern part of MA including the LAHS area was designated as being in moderate non-attainment for the US EPA 8-hour ambient standard for ground level ozone (US EPA, 2013).

When specifically investigating the role of the airport on respiratory disease in the LAHS area, lifetime and current asthma prevalence were not found to be associated with predicted airport-related exposures for adults or children. However, among children not diagnosed with asthma, the odds of experiencing asthma-related symptoms (probable asthma) were 3.6 times higher for those in the high compared to the low airport-related air pollution exposure area (95% CI: 1.2-11). This finding was statistically significant even after adjusting for numerous other factors including demographic variables, background air pollution, and household exposures to tobacco smoke, NO_2, allergens, and mold.

The increased prevalence of probable asthma has also been observed in other studies evaluating the associations between exposure to air pollution and respiratory symptoms in children (US EPA, 2009). Multiple studies of children conducted worldwide have also observed significant associations between exposure to air pollution, notably particulate matter, and respiratory symptoms (increase in cough, wheeze and bronchitis) and impairment of lung function in children (Kim et al., 2012; Penard-Morand et al., 2010; Schwartz, 2004).

In this study, elevated odds of reporting physician-diagnosed COPD were observed among adults living in the high versus low airport-related air pollution exposure areas for 3 or more years (OR=1.8, 95% CI: 1.1-3.0). Although smoking is a primary risk factor for COPD, the association between COPD and the airport exposure category in this study was observed after adjusting for individual smoking status. Recent studies have suggested that a large percentage of COPD diagnoses occurring in populations of never smokers (Salvi et al., 2009). For example, Salvi et al. (2009) cites as evidence the findings from an analysis of NHANES III data suggesting that 25% of COPD cases in the US are in never-smokers.

Literature related to air pollution and COPD also report significant associations between outdoor air pollution and COPD exacerbation including increased COPD hospital admissions and mortality (Samet et al., 2000). A review by Pope et al. (2006a) cites epidemiologic evidence that long-term PM exposures are associated with lung function deficits and increased symptoms of obstructive airway disease (e.g., chronic cough, bronchitis), supporting the biological plausibility of the role of air pollution in exacerbating COPD. In addition, a small number of studies have evaluated the role of air pollution in the prevalence of COPD. In a review of studies assessing the association between air pollution and COPD, Schikowski et al (2013) concluded that results from several studies are suggestive, but inconclusive, that air pollution is related to the development of COPD.

Both short- and long-term exposures to ambient air pollution, notably $PM_{2.5}$ and NO_2, are associated with exacerbation of respiratory diseases (US EPA, 2009; US EPA 2008). In the LAHS, there was evidence, though only of borderline statistical significance, that adults

living for 5 or more years in the high air pollutant exposure area had higher odds of current asthma requiring the use of medication (OR=1.7, 95% CI: 1.0-3.0). These findings were adjusted for a number of potential confounding factors including, but not limited to, age, sex, race, household income (PIR), education, smoking status, and background air pollution.

CARDIOVASCULAR OUTCOMES

Prevalence of cardiovascular disease (CVD) in the LAHS area was found to be slightly lower than that of the state of MA as a whole, although the confidence intervals for the LAHS area and MA estimates overlap, suggesting that the differences may not be statistically significant. Although few studies have specifically examined urban versus rural prevalence of myocardial infarction or coronary heart disease in the US, the LAHS results appear to be consistent with those that have. For example, using data from the 2008 BRFSS, O'Connor and Wellenius (2012) found that the crude prevalence of coronary heart disease was 40% higher among respondents living in rural versus urban areas of the US. After adjusting for poverty, obesity, tobacco use, and other risk factors, the prevalence of coronary heart disease was still found to be higher in rural areas, but only by approximately 10% (O'Connor and Wellenius, 2012). Separately, in a study of social determinants of CVD risk factors among men in Georgia, it was found that rural men were almost twice as likely to have two or more CVD risk factors than were urban men (Quarells et al., 2012).

When looking at cardiovascular morbidity (CHD and non-fatal heart attack) in the LAHS area by airport-related air pollution exposure category, no differences were observed. After adjusting for demographic and other risk factors, including diabetes, hypertension, high cholesterol, and family history of heart disease, the likelihood of CHD and non-fatal MI was still not different across airport-related exposure categories. Although exposure to air pollution, particularly PM, has been associated with MI and CHD, more studies have reported associations with mortality rather than morbidity (Pope et al., 2006a). Given the fact that this study measured self-reported health outcomes, only persons who survived to report their MI or CHD were included in this study.

To address this limitation, MDPH/BEH analyzed mortality data from the MA Registry of Vital Records for differences in the crude 5-year average annual MI mortality rate (2001-2005) between the state of MA as a whole and each of the three airport-related air pollution exposure areas. Population data were obtained from the US Census and ArcGIS was used to assign census tracts and estimate total population for each of the LAHS exposure areas. No statistically significant differences were observed in the average annual MI mortality rates for each of the LAHS exposure areas and the state as a whole. Per 100,000 adults age 35 years and older, the annual average MI mortality rate was 99 for MA (95% CI: 95-102), 88 for the low exposure area (95% CI: 80-96), 100 for the medium exposure area (95% CI: 84-116), and 92 for the high exposure area (95% CI: 63-121).

Despite the lack of an association between potential airport-related air pollution exposure and CVD morbidity (crude and adjusted) or mortality (crude) in this study, it is well established that both long and short term exposure to ambient air pollution, including PM and NO_2, increases risk of CVD (Atkinson et al., 2013; Peters et al., 2004; Miller at al., 2007; Schwartz et al., 1995; Wellenius et al., 2006). Therefore, the lack of an association in this study suggests that airport-related exposures were not high enough to cause detectable impacts on CHD or MI morbidity in the LAHS area. This does not rule out the possibility that small effects are present and undetectable with the current study's sample size and/or measures of outcome. For example, some studies have found associations between particulate matter exposure and sub-clinical measures of the development and progression of cardiovascular disease such as accelerated atherosclerosis, altered cardiac autonomic function, heart rate variability, and alterations in vascular tone (Brook et al., 2004; Peters et al., 2002). Also notable is the fact that effect estimates in other studies are relatively small for associations between air pollution and CVD, particularly in comparison to other CVD risk factors (Pope et al., 2006a).

AUDITORY OUTCOMES

In the LAHS, three auditory endpoints were evaluated in adults (hearing impairment, hearing impairment with hearing aid use, and tinnitus). One auditory outcome (hearing impairment), was evaluated in children. This study did not find a relationship between any of the auditory outcomes evaluated and estimated airport-related noise exposure. Effect estimates had very wide confidence intervals and the direction of effect was inconsistent across the three outcomes evaluated in adults.

A recent study has reported NIHL in workers at airports (Pepper et al., 2003) and a few studies have reported reduced hearing ability in people living near an airport (Chen et al., 1997) but the data are limited. One reason for not observing an association between airport-related noise and auditory endpoints in this study may be that residents are not exposed to sufficient noise levels to induce auditory impairment. The WHO recommendation for prevention of noise-induced hearing loss (NIHL) is that lifetime average daily exposure to environmental noise be less than 70 dB (Leq (24) <70 dB). This recommendation is estimated to prevent impairment in 95% of people exposed at or below the recommended level over an entire lifetime. Noise contours modeled for the LAHS suggested that very few residents were exposed to airport-related noise above the 70 dB WHO recommendation. Due to this fact, and to account for uncertainties in the noise modeling estimates, the high noise exposure category was defined by the 65 dB noise contour line. In addition, the WHO guideline value may underestimate auditory effects in sensitive subpopulations including children. Finally, the Massport noise modeling contours as defined only predict noise levels associated with aircraft operations in the vicinity of the airport and not cumulative noise levels that residents are typically exposed to in their neighborhoods. In a cumulative sense, exposure to levels of airport-related noise below the WHO guideline could theoretically contribute to auditory impairment. Nevertheless, no measure of hearing impairment was found to be elevated or more likely to occur among residents living in the high noise exposure area.

Another reason for not observing auditory health effects among residents living in the high noise exposure area may be due in part to the implementation of Massport's noise abatement program, which provides soundproofing of homes and apartments within the 65 dB noise contour. Analysis of survey results indicated that 44% of residents in the high noise exposure area had received Massport soundproofing and 24% of residents in the medium exposure area had received the soundproofing (Appendix C).

Although adverse auditory outcomes associated with airport-related noise exposure were not observed, other health and quality of life endpoints may be influenced by exposure to airport-related noise. Some of these effects include annoyance, interference with speech and communication, sleep disturbance, stress, and cardiovascular impacts. This study did not evaluate these endpoints. Regarding cardiovascular effects, only recently have studies begun to understand the potential impact of noise exposure on CVD health. For example, the Schiphol airport study, a multi-airport retrospective study of older people (65 years and older) in US (Correia et al., 2013, a study of residents living near Heathrow airport (Hansell et al., 2013) and a study in Switzerland (Huss et al., 2010) have reported emerging evidence of increased incidence of noise-related cardiovascular effects. A review of the literature of these studies for the US FAA/NASA/Transport Canada sponsored Center of Excellence concluded that a pattern of increased incidence of cardiovascular effects, hypertension, and ischemic heart disease associated with noise appear to have emerged (Swift, 2010).

STUDY DESIGN CONSIDERATIONS

The Logan Airport Health Study (LAHS) was a cross-sectional study. Cross-sectional studies provide an overview of the rates and patterns of diseases and/or conditions in a population and provide insight into how they may relate to other characteristics of that area. Cross-sectional studies can be thought of as providing a snapshot of the population at a specific point in time. The survey instrument (questionnaire) for this study was designed to collect information about the prevalence in 2005 of targeted health outcomes within the LAHS area as a whole and, separately, within subdivisions of the study area believed to experience different potentials for exposure to airport-related air pollution and noise in

2005. It may not be possible to distinguish whether an individual's exposure (i.e., location of current residence) preceded or followed the occurrence of a health outcome. For this reason, this study's findings cannot determine cause and effect relationships, but they can allow us to demonstrate relative differences in disease burden and to develop inferences and hypotheses based on observed associations.

The survey instrument also sought to collect information on important potential confounders such as demographic factors, health behaviors, household and occupational exposures, and other risk factors for the targeted health outcomes. Although it was not feasible to collect information on every individual in the study area, techniques of RDD sampling and post-sampling statistical methodologies (e.g., weighting of study data to population demographics) work to provide estimates of disease burden and disease-exposure associations that are meant to be generalizable to the entire study population, both those who actually participated in the survey and those who did not. Another key aspect of the survey design was that the study area was divided into estimated exposure areas for the purpose of oversampling sections of the study area nearest the airport. This was done in order to achieve sample sizes in those areas sufficient for detecting significant effects should they be present.

The survey instrument was also pilot tested both in 2002 with a population near an airport in ME and, later (in 2005), with a small sample of individuals from the LAHS area. These efforts helped to improve introductory language to encourage participation and to refine questions for clarity, increasing respondent consistency in answers. One survey limitation, however, is that health outcome data acquired through a telephone survey are self-reported and, therefore, may be subject to some error. For all outcomes in this study, persons were asked to self-report diseases and conditions; medical records review or physical examinations were not conducted. A technique used to encourage consistency of reporting for health outcomes in the LAHS was that survey questions regarding specific health outcomes began, "Have you ever been told by a doctor that you have…?" Another limitation of telephone surveys is that they generally exclude residents that only use cell phones, although prevalence of cell-phone only households was still relatively low in 2005 at

approximately 7% (Blumberg and Luke, 2007). The LAHS survey also only included only residents whose primary language was English or Spanish.

There is always the possibility of residual confounding or, in other words, factors other than the airport that are associated with the targeted health outcomes and that, when unaccounted for, could either mask effects of the airport or falsely appear as effects of the airport. However, this study benefited from the availability of data on a multitude of potential risk factors including smoking status, alcohol intake, occupational exposures to noise and respiratory irritants, and NO_2 sources in the home, to name a few. Multivariate logistic regression analyses were conducted to simultaneously adjust for these demographic, lifestyle, and other characteristics known to be risk factors for adverse respiratory, cardiovascular, or auditory outcomes.

One important example for this study was data collected on smoking status. Although the low exposure area was found to have a slightly lower prevalence of current smoking compared to the medium and high exposure areas (17% in the low compared to 21% in both the high and medium areas, Appendix C), this factor was controlled for by including smoking status as a covariate in multivariate regression models when looking at respiratory and cardiovascular outcomes. Other potential confounding factors, such as exposure to occupational dust and/or fumes, the presence of allergens in the home, and the presence of mold in the home, were assessed in multivariate regression models but were not found to have any meaningful impact on respiratory or cardiovascular results.

SURVEY PARTICIPATION

As previously discussed, statistical methodologies (e.g., weighting of the survey sample based on population demographics of the area) are employed to minimize possible response bias and help ensure that the results obtained are generalizable not only to all study participants but to the total study population from which the participants were selected. Response bias is important to minimize because its presence may reduce the study's ability to detect associations even if they are present. Survey response rates are one factor that could affect the presence of response bias.

Although survey response rates are not solely predictive of the presence or absence of non-response bias in a study (Groves and Peytcheva 2008; Wagner 2012), extremely low response rates may indicate the need for a deeper assessment of potential bias. Response rates for telephone surveys have declined steadily over the past few decades, particularly since the mid-90s when call-blocking and screening technologies skyrocketed (Curtin et al. 2005). Research examining the impact of such declines on selection bias and survey results has found, overwhelmingly, that lower response rates do not necessarily lead to biased results and most investigations have concluded that findings are not significantly different between initial responders and those recruited with more effort (Curtin et al. 2000; Davern et al. 2010; Keeter et al. 2000).

The Council of American Survey Research Organizations (CASRO) method for estimating response rates (equivalent to the AAPOR RR3) is considered the most appropriate for surveys of the type used in the LAHS. It is comparable to other RDD surveys conducted around the same time period, including for the response rate of the Massachusetts portion of the Behavioral Risk Factor Surveillance Survey (BRFSS)(CDC 2006). The CASRO or AAPOR RR3 method estimated a response rate for the LAHS of 35%.

Although useful for comparison between studies, research strongly suggests that the RR3 method produces an underestimate of the true response rate (Brick et al. 2002). The extent of underestimation varies depending on how much lower the proportion of eligible to ineligible telephone numbers actually was among unresolved numbers compared to those that were resolved. In contrast, the AAPOR RR5 response rate achieved for the LAHS (60%) can be thought of as the upper bound of the true rate.

Another useful indicator of the potential for selection bias is to examine the similarity or dissimilarity of response rates across different exposure areas. A higher response rate among communities closer to the airport, for example, could suggest that respondents in that area were more motivated to participate (although the survey instrument did not identify the airport as a subject of the study). If respondents with personal experiences of

adverse health outcomes are also more motivated to participate, then a higher response rate in areas closer to the airport could suggest a selection bias.

For the LAHS, response rates were calculated for each of the three stages of RDD telephone sampling, which were designed to oversample portions of the study area closer to the airport. For Stage 1, which included telephone numbers across the entire study area, the RR3 and RR5 response rates were 34% and 60%, respectively. For Stage 2, which included telephone numbers from the medium and high exposure areas (as defined for survey oversampling), the RR3 and RR5 response rates were 33% and 59%, respectively. For Stage 3, which included telephone numbers from only the high exposure oversampling area (within 1 mile of the airport or along a major aircraft flight path), the RR3 and RR5 response rates were 37% and 62%, respectively. Overall, response rates for the three stages of sampling were very similar with only a slightly higher rate for Stage 3, suggesting that residence near the airport was only weakly, if at all, related to the likelihood of participation in the LAHS.

EXPOSURE ASSESSMENT OF AIRPORT-RELATED AIR POLLUTION

A major strength of this study was the exposure assessment of airport-related emissions used to classify residential households into categories of low, medium, and high areas of potential exposure to air pollution from airport-related operations. This study is believed to be the only study that has assessed the relative contribution of airport-related emissions using advanced air dispersion modeling to evaluate associations with health outcomes in the surrounding community. The modeling analysis included all known sources of emissions and the temporal allocation of emissions from the takeoff and landing of aircraft by using a schedule of over 350,000 aircraft operations (94% of total 2005 operations) quantified and provided by Massport. Emissions along flight paths up to an altitude of 3000 feet for takeoffs and landings were also included in the modeling analysis.

The air dispersion modeling allowed us to investigate concentrations of five pollutants (CO, NOx, $PM_{2.5}$, SOx, VOCs) across the study area for three different averaging times, 1-hour, 24-hour, and annual. Given the extremely high correlation of estimated

concentrations of the five pollutants across the study area, a combined exposure variable was developed that encompassed all pollutants. Furthermore, although annual average pollutant concentrations were selected for developing cut-points for the three exposure areas, the spatial distribution of pollutants was extremely similar using concentrations for all three averaging times. In other words, the high exposure area (as defined by the 80th percentile of annual average pollutant concentrations) also included the highest peak concentrations (1-hour and daily).

Evaluating three categories of exposure allowed for the clearest assessment of relative effects between those in potentially high airport-related exposure areas and those in a potentially low airport-related exposure area. Nevertheless, an analysis using final multivariate adjusted regression models was also conducted to evaluate air pollution exposure on a continuous scale using $PM_{2.5}$ as the model compound in order to be sure that exposure misclassification from categorization did not limit power to detect associations. Results from continuous exposure analyses were similar to the categorical exposure analyses presented in this report (see Appendix D).

There is substantial evidence in the literature that particles are associated with respiratory and cardiovascular health effects, but there is evidence that other constituents within the complex mixture may also be relevant to human health outcomes (Hu et al., 2008; Zhou et al., 2009). The aggregate exposure metric allowed for qualitative consideration of the complex nature of the combustion of diesel, jet, and gasoline fuels that produce ultrafine, fine, and course particles, as well as gas or vapor-phase compounds. For this reason, associations observed in this study cannot necessarily be attributed to exposure to any one pollutant nor to any specific concentration. Rather, associations observed reflect differences in the likelihood of certain outcomes among those living in regions of the study area estimated to experience higher airport-related exposures relative to those living in a region of the study area estimated to experience lower airport-related exposures.

The findings of the air dispersion modeling analysis with respect to levels of predicted pollutant concentrations, as well as the spatial and temporal variability of those

concentrations across the study area, are consistent with other studies that have modeled airport operations (Ratliff et al., 2009; Adamkiewicz et al., 2010). In particular, a spatial gradient was observed such that predicted air pollutant concentrations were highest near the airport and dropped off rapidly with increasing distance from the airport. The distribution of predicted air pollutant concentrations was also consistent with monitoring studies that have detected distinct upwind and downwind emission gradients at airports for certain pollutants [e.g., NOx (Adamkiewicz et al., 2010), black carbon (Morin, 2007), and ultrafine particles (Westerdahl et al., 2008)] and a rapid reduction in pollutant concentrations with distance from the airport.

The complex mixture of air pollutants emitted from large urban airports, such as Logan Airport, are those characterized by combustion of various types of fuel (e.g., jet fuel, diesel fuel, and gasoline). As such, airport emissions are similar to pollutants typically found in urban air including criteria air pollutants and numerous air toxics (Ratliff et al., 2009; Zhou et al., 2009). As discussed previously, many of these pollutants, including $PM_{2.5}$, $PM_{10,}$ SO_2, and NO_2, have been associated with respiratory and cardiovascular endpoints similar to those measured in this study.

These health outcomes are consistent with those observed in studies of the impacts of air pollution at levels found in ambient air. However, comparison of the emissions inventory modeling of the airport done in this study to the total emissions inventory for the LAHS area indicates that the airport contributes only a small fraction to the total air pollutants emitted, which is consistent with emissions inventory studies of most other large urban airports in the US that have been estimated to contribute only 1-2% of total emissions within a given airshed (Ratliff et al., 2009). With the exception of probable asthma in children, the associations observed in this study were of either borderline statistical significance or not statistically significant. Furthermore, no associations were observed between CVD outcomes and estimated airport-related air pollution exposure. One possible reason for this is that the incremental contribution of air pollution concentrations from airport-related activities was relatively small compared to overall background concentrations.

To further assess our observations regarding the contribution of airport-related air pollution, the average airport-related $PM_{2.5}$ concentration predicted from modeling the 2005 emissions inventory across the study area (using annual average concentrations) was compared to $PM_{2.5}$ background concentrations from the nearest monitoring station, and the airport contribution was found to be less than 1 percent. For the highest LAHS airport-related modeled concentrations (those greater than 75% of the maximum), the percent contribution to background concentrations for $PM_{2.5}$ was 4.3%. The only study to model the air pollution emissions inventory for the entire US with and without aircraft emissions found that the percent increase due to aircraft emissions was about an order of magnitude less than the LAHS results (0.06% in non-attainment areas and 0.08% in all counties) (Ratliff et al., 2009). Although there are known uncertainties in the emissions inventory (e.g., mobile source PM emissions) and in the modeled concentrations, these uncertainties are not likely to significantly change the overall contribution of emissions to background concentrations. Although some associations with respiratory outcomes were observed in this study, it may be that the additional burden of airport-related air pollution to that already present in the ambient air from other sources is not strong enough for many of those associations to reach statistical significance given the sample size and study design. Similarly, as noted previously, it may be that airport-related air pollution exposures were not high enough to cause observable CVD associations in this study.

Along with the fact that estimated airport-related annual average air pollutant concentrations were relatively low in the study area, another challenge was the issue of potential confounding by background air pollution. Background air pollution exposure is not thought to be uniform across the study area, particularly from residential proximity to major roadways (Gryparis et al., 2007; Levy et al., 2003). For example, the percentage of people living within 200 meters of a major roadway was about two times higher in the medium (22%) and high (24%) exposure areas compared to the low exposure area (13%). Recent literature identifies associations between proximity to traffic sources and induction of asthma incidence in children, and evidence is suggestive of inducing asthma in adults (Braback et al., 2009; Lindgren et al., 2010; McConnell, 2006; McConnell et al., 2010; Salam et al., 2008). Recent studies have also reported an association between traffic-related

exposures and an increased prevalence of COPD (Andersen et al., 2011; Lindgren et al, 2009). For this reason, multivariate analyses in this study were adjusted for residential proximity to major roads.

To address potential confounding from other major pollutant sources in the study area, such as those arising from industrial combustion emissions, multivariate regression analyses also included a measure of monitored background $PM_{2.5}$ concentrations. Therefore, this study accounted for both overall background concentrations represented by centrally-located monitoring stations in the study area and the higher gradient near major roadways that is not captured by the central monitors. For background air pollution adjustment using air monitoring station data, $PM_{2.5}$ concentrations were used because PM has been most strongly associated with respiratory and cardiovascular health effects in epidemiologic research (compared to other pollutants such as CO). In addition, a study in Boston of the relationship between ambient measures of urban air pollution found that $PM_{2.5}$ data from centrally-located monitors was strongly associated with personal exposure to $PM_{2.5}$, supporting the use of $PM_{2.5}$ data from central monitors for assessing exposure to $PM_{2.5}$ in epidemiological studies (Koutrakis et al., 2005). While NO_x is also an important pollutant with respect to respiratory effects, a recent analysis by US EPA found that centrally-located monitors are not optimally located to characterize NO_x exposures because the monitors were located primarily to model ozone formation.

Despite adjustment for both of these variables, fully accounting for variation in personal exposure to background air pollution was not possible in this study and may be a source of residual confounding, which could either mask effects of the airport or show false positive associations. For example, the various levels of exposure related to time activity patterns, such as increased exposure from exercising outdoors could not be considered.

A further method employed to isolate the effects of exposure to airport-related air pollution included consideration of the movement of residents into or out of the study area over time. An important limitation in the exposure assessment is the potential misclassification of respondents due to different lengths of residence in the study area. For

example, some study respondents may have developed asthma or experienced a myocardial infarction prior to moving to their current residence. This type of migration effect could lead to mis-measurement of disease incidence since arrival in the study area, making it more difficult to assess true associations that may be present.

This limitation was partially addressed by performing sub-analyses in which associations were examined only among residents who had lived in their current exposure area (high, medium, low) for greater than 1, 3, 5, and 10 years. Those analyses indicated that consideration of migration may help clarify associations of exposure and disease in this population. For example, when the analyses were restricted to individuals who had lived in their current airport-related exposure area for at least 3 or more years, associations were strengthened for most of the targeted respiratory outcomes, including a statistically significantly higher likelihood of having COPD among adults in the high compared to the low airport-related air pollution exposure area. The association between airport-related air pollution exposure category and current asthma with medication use also strengthened with increasing length of residence from an odds ratio of 1.4 (high versus low; p=0.20) in the total study population to 1.7 (high versus low; p=0.05) among those with 5+ years residence (Table 6-8). These findings suggest that the respiratory impact of exposure to airport-related air pollution increases as the duration of exposure increases. For both COPD and current asthma with medication use, the association with airport-related exposure category lessened in the 10+ years of residence analysis, possibly due to loss of sample size or possibly because length of residence is not as strong a factor for these outcomes beyond 5 years or so.

For cardiovascular outcomes, attempting to refine the exposure measure by consideration of residential length did not help to elucidate any associations with potential airport-related exposure. For both MI and CHD, measures of effect (ORs) remained fairly null when restricting analyses to residents with longer lengths of residence. Since CVD outcomes involve biologic mechanisms that develop over time and involve a long lag time, one hypothesis was that assessing historical and/or longer exposures may lead to observable associations. This was not the case and, in fact, review of the scientific literature

reveals that the most robust results of the association between air pollution and CVD risk are demonstrated in studies using a case-crossover design, which examine short-term, acute exposures, particularly to PM (Peters et al., 2001;Wellenius et al., 2006).

8 CONCLUSIONS AND RECOMMENDATIONS

The major conclusions of the Logan Airport Health Study are as follows:

- Air dispersion modeling of airport related emissions using a state-of-the-art model indicates that the highest predicted pollutant concentrations associated with airport-related operations are near the perimeter of Logan Airport and fall off rapidly with increased distance. This is a characteristic of the impact of sources that are primarily located near the ground surface.

- Consistent with findings of other airport studies, modeled concentrations of air pollutants are low relative to measured background air pollution concentrations.

- Evaluation of associations between airport-related pollutant concentrations and targeted health outcomes among the study area population detected some elevations in respiratory health outcomes in the high exposure area.

  Specifically:

- Among children, study results identified some respiratory effects indicative of undiagnosed asthma (i.e., probable asthma); children in the high exposure area were estimated to have three to four times the likelihood of this respiratory outcome compared with children in the low exposure area.

- Among adult residents, individuals diagnosed with chronic obstructive pulmonary disease (COPD) were statistically significantly more likely to have lived in the high exposure area for three or more years.

- There were no statistically significant differences in cardiovascular outcomes in the study population across the high, medium, and low exposure areas.

- There were no statistically significant differences with respect to hearing loss in either adults or children for those living in the high exposure area compared to the lowest exposure area.

<u>Recommendations:</u>

- The results of this study should be reviewed by Massport and others to determine mitigating steps that can be taken across the study area.

- Massport has undertaken initiatives to reduce air pollution impacts within their control (e.g., providing infrastructure for compressed natural gas (CNG) fuels and electricity charging stations, Alternative Fuel Vehicle Program). Similar initiatives could be considered in consultation with local communities that would serve to further reduce the burden of indoor and outdoor sources of air pollution on residents in closest proximity to the airport.

- Massport has also been working with the East Boston Neighborhood Health Center (EBNHC) to address workforce issues among Massport employees. Massport could expand these efforts with the EBNHC as well as other community health centers to better address respiratory health notably among children in closest proximity to the airport.

- While air dispersion modeling indicates that the contribution from Logan Airport operations across the study area is relatively small, air pollution levels are higher in urban areas. Predicted pollutant concentrations were higher near the perimeter of the airport; thus, any methods that can be implemented to continue to reduce airport-related air pollution should be explored.

- MDPH/BEH should work with communities within the high exposure area (in whole or in part) on initiatives that would serve to further reduce exacerbation of pre-existing respiratory diseases (e.g., asthma and COPD) among residents.

  Specifically:

  - MDPH/BEH will continue to support MassDEP's efforts to reduce motor vehicle emissions including implementation of the Low Emissions Vehicle program and diesel engine retrofit initiatives;

  - Upon request MDPH/BEH's Indoor Air Quality (IAQ) Program staff will work with local municipalities to conduct IAQ assessments in schools and public buildings;

  - Upon request MDPH will work with local officials to address concerns that may be associated with local development initiatives;

  - MDPH/BEH will collaborate with the MDPH Bureau of Community Health and Prevention's Tobacco Cessation and Prevention Program on their efforts to work with local boards of health and tobacco-free community partnerships. These efforts enforce youth access and secondhand smoking laws and provide educational/outreach resources to support smoke-free workplace and housing programs.

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10 APPENDICES FOR LOGAN AIRPORT HEALTH STUDY

Appendix A: Emissions and Dispersion Modeling

Appendix B: Background Pollutant Concentrations

Appendix C: Health-Related Behaviors, Occupational Exposures, And Household Characteristics Of Adult Residents Of The Logan Airport Health Study (LAHS) Area By Airport-Related Air Or Noise Exposure Area

Appendix D: Continuous Exposure Analyses