Community Health and Airport Operations Related Noise and Air Pollution: Report to the Legislature in Response to Washington State HOUSE BILL 1109

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Public Health
Seattle & King County

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EXECUTIVE SUMMARY

This report responds to a proviso in Washington state's House Bill 1109 which requests that Public Health – Seattle & King County produce a) airport community health profiles for a one-mile, a five-mile, and a 10-mile radius of the airport; b) a comprehensive literature review assessing the strength-of-evidence for health effects of airport operations; c) a summary of findings of the University of Washington School of Public Health study on ultrafine particulate matter; and d) recommendations to address health issues related to the impact of the airport on the community. The purpose is to understand the community health effects of pollution related to Seattle-Tacoma International Airport (SeaTac) operations.

WHAT IS THE HEALTH OF AIRPORT COMMUNITIES COMPARED TO THE REST OF KING COUNTY?

The majority of people in King County identifying as Black/African American, Hispanic/Latino, and Native Hawaiian/Pacific Islander live in communities within 10 miles of the airport (hereafter referred to as airport communities). A greater proportion of people in these communities are immigrants, and a slightly higher proportion are children, compared to elsewhere in the county.

People living within 10 miles of SeaTac airport face disparities in health, resources, and risk factors compared to the rest of the county. They were significantly more likely to be living below 200% of the federal poverty level, to not receive needed medical care due to the cost, and not to have health insurance compared to the remainder of King County. They were also more likely to have risk factors that increase their vulnerability to more serious health outcomes, such as a higher prevalence of smoking, obesity, physical inactivity and high blood pressure. A greater percentage of adults in airport communities than elsewhere in the county reported inadequate amounts of sleep.

Airport communities are associated with higher rates of pervasive health concerns. Compared to the rest of the county, communities within 10 miles of SeaTac report:

- A greater percentage of infants born prematurely and/or with low birthweight;
- Higher hospitalization rates for asthma, stroke, chronic obstructive pulmonary disease (COPD), heart disease, and diabetes;
- Lower life expectancy; and
- Higher rates of death overall, as well as death from heart disease, unintentional injury, chronic lower respiratory disease, diabetes, chronic liver disease, and homicide.

In several measures, the rates of poor health outcomes were worse the closer you are to the airport. For example:

- Higher hospitalization rates for heart disease;
- Higher rate of death from all causes;
- Higher rate of death from heart disease; and
- Lower life expectancy.

This examination of community health is a snapshot of health conditions experienced by people living within 10 miles of SeaTac airport. Findings demonstrate that disparities are present throughout the life course, beginning at birth.

WHAT POLLUTANTS RESULT FROM AIRPORT OPERATIONS AND WHAT ARE THE LIKELY HEALTH IMPACTS?

Airport operations result in noise and air pollution, which are linked to many of the health outcomes experienced by airport communities. Noise pollution contributes to hypertension and heart disease and likely causes poor school performance among children. Air pollution impacts numerous organ systems, and multiple pollutants are associated with cardiovascular and respiratory problems. The air pollutants

related to airport operations include particulate matter of various sizes, ozone, carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur oxides (SO_x), and other hazardous air pollutants. Fine particulate matter ($PM_{2.5}$) causes cardiovascular and respiratory problems, and likely causes cancer and central nervous system conditions, including dementia and neurodegeneration. Existing research is less conclusive about larger and smaller particulate matter in comparison, though recent studies link exposure to increased risk of preterm births and respiratory concerns, among other issues. Ozone, NO_2, and SO_x cause short-term respiratory issues. NO_2 likely causes long-term cardiovascular problems, and CO causes short-term cardiovascular concerns and likely affects lung functioning.

These pollutants are especially concerning for people with underlying respiratory or cardiovascular issues because they worsen existing conditions, though long-term exposures increase risk in the general population for developing problems. The hazardous air pollutants (HAPs) found at airports are known or suspected carcinogens and/or cause birth defects. Lead exposures are more common at Boeing Field, Renton, and Auburn airports than at SeaTac airport, because small planes with piston engines still use leaded gasoline. Lead causes central nervous system problems and is damaging even at low levels; thus it is worth mentioning despite lower prevalence at SeaTac airport. Noise and air pollution have been documented near SeaTac as well as other airports at levels higher than recommended for population health.

WHAT WERE FINDINGS FROM THE UNIVERSITY OF WASHINGTON PUBLIC HEALTH STUDY OF ULTRA-FINE PARTICULATES?

Researchers with University of Washington's Environmental and Occupational Health Sciences Department conducted the first study of ultrafine particle (UFP) concentrations near the SeaTac airport during 2018–19 and found higher concentrations of UFP below aircraft flight paths, with the highest concentrations associated with aircraft landings. Pollution near roadways showed high concentrations of UFP and black carbon. Findings support the conclusion that communities underneath and downwind of the flight path are exposed to aircraft-related UFP concentrations.

RECOMMENDATIONS TO ADDRESS HEALTH ISSUES

Prevention and mitigation of airport-related pollution exposures is critical for these communities, given their increased risk. People living in airport communities are more likely to be exposed to airport-related air and noise pollution. They are more likely to have underlying conditions like diabetes, heart disease, and respiratory conditions, which increases vulnerability to more serious health outcomes resulting from pollution exposures. Epigenetic changes from exposures to previous generations may increase susceptibility to health effects from air pollution today.

To address the health disparities of airport communities, we recommend the following:

- Implement focused efforts to address the health disparities of airport communities, including mitigating the health impacts of airport operations.
- Continue development and implementation of strategies to mitigate airport-related air and noise pollution.
- Expand the systematic monitoring of pollutants (both outdoor and indoor exposures) in residences, schools, childcare settings, and long-term care facilities, including the implementation of new technologies to improve measurement of exposures indoors and outdoors.
- Support research to address gaps in knowledge, including the levels of pollutant exposure resulting from airport operations, the extent to which outside pollutants infiltrate indoor settings, and the precise mechanisms and degree of harm caused by air and noise pollution.

I. INTRODUCTION

Washington State sought to better understand the community health effects of pollution related to Seattle-Tacoma International Airport operations. The legislature thus included the following proviso in HOUSE BILL 1109 for the Washington State operating budget.[1]

a) $62,000 of the general fund – state appropriation for fiscal year 2020 and $63,000 of the general fund – state appropriation for fiscal year 2021 are provided solely for the King County local health jurisdiction, as part of the foundational public health services, to conduct a study on the population health impact of the SeaTac airport communities.
b) By December 1, 2020, the King County local health jurisdiction shall submit a report to the appropriate committees of the legislature that must include:
    i. An analysis of existing data sources and an oversample of the Best Start for Kids child health survey to produce airport community health profiles within a one-mile, five-mile, and ten-mile radius of the airport;
    ii. A comprehensive literature review concerning the community health effects of airport operations, including a strength-of-evidence analysis;
    iii. The findings of the University of Washington School of Public Health study on ultrafine particulate matter at the airport and surrounding areas; and
    iv. Any recommendations to address health issues related to the impact of the airport on the community.

This report summarizes findings from these efforts. The following section reviews community health profiles for communities within radii of one, five, and 10 miles from the airport. The purpose of the community health profiles is to describe the communities proximal to the airport to understand their health. The next section summarizes findings from the literature review, including background information about airport operations and key pollutants; strength-of-evidence methods and findings; evidence of relevant, effective mitigation efforts; and a summary of findings from the University of Washington study on ultrafine particulate matter. The report concludes with recommendations based on the community health profiles and a review of the evidence to date.

II. WHAT IS THE HEALTH OF AIRPORT COMMUNITIES COMPARED TO THE REST OF KING COUNTY?

Report requirement 1: Community health profiles by distance from airport

> Proviso language: An analysis of existing data sources and an oversample of the Best Start for Kids child health survey to produce airport community health profiles within a one mile, five mile, and ten mile radius of the airport

A. SUMMARY

The community health profiles of areas one, five, and 10 miles from SeaTac airport (Zones A, B, and C) describe disparities in health experienced in airport communities compared to elsewhere in King County. A majority of the people in King County identifying as Black/African American, Hispanic/Latino, and Native Hawaiian/Pacific Islander live within 10 miles of the airport. In comparison with the Balance of County (the non-overlapping areas of the county beyond 10 miles of the airport), people living in airport communities are disproportionately more likely to experience poor health and less access to resources. For example:

- Between 34% and 51% of children were living at less than 200% of the federal poverty level, compared to 15% of children in Balance of County.
- The percent of adults with no health insurance in Zones A and B was more than twice the rate in Balance of County.
- Hospitalization rates for asthma, stroke, diabetes, and heart disease were higher in airport communities than in Balance of County.
- Life expectancy was between 1.7 and 5.0 years lower than that of Balance of County and decreased the closer you are to the airport.
- Residents of airport communities had higher rates of death by heart disease, unintentional injury, chronic lower respiratory disease, diabetes, chronic liver disease, and homicide than did people in Balance of County.

Many of these conditions have been linked in the literature to airport-related air and noise pollution. These include preterm births, depression, high blood pressure, and hospitalizations for asthma, heart disease, COPD, and stroke.

A greater percentage of adults in Zones B and C reported inadequate amounts of sleep compared to Balance of County. Parents/caregivers of children in elementary school or younger living in airport communities had inadequate sleep amounts similar to those of others in the rest of the county. Results differ from the prior measure, and this is likely due to the difference in who was asked, as these results are from parents/caregivers of younger children and not adults of all ages.

An analysis of excess deaths examined how many fewer deaths would occur if the communities near the airport had the same risk of death as Balance of County. Across four of the leading causes of death in the county (heart disease, cancer, unintentional injuries, and stroke), the number of deaths in airport communities exceeded the expected number of deaths if airport communities had the same death rates as the Balance of County. The closer you are to the airport, the higher the number of excess deaths associated with these causes.

A community asset not reflected in the current population health data is the robust community organization and advocacy in support of environmental justice that exists in airport communities. People living in airport communities are acutely aware of the data and evidence regarding air and noise

pollution and actively advocate for decisions that lead to healthy and safe environments (see, for example, the Duwamish River Cleanup Coalition and the Beacon Hill Seattle Noise Team).

The following profiles are descriptive and cannot be linked to specific causes. Findings demonstrate, however, that disparities are present throughout the life course, beginning at birth.

B. COMMUNITY HEALTH PROFILE METHODS

To understand the health of communities near SeaTac airport, we analyzed data from multiple sources. These include:

- Washington State Department of Health's Community Health Assessment Tool (CHAT), a repository of population health information including birth, hospitalization, and death data;
- The U.S. Census Bureau's annual American Community Survey;
- King County Best Starts for Kids Health Survey of parents/caregivers of infants and young children through fifth grade;
- Washington State's Healthy Youth Survey of eighth-, 10th- and 12th-grade students in public schools;
- Washington State Department of Health's Behavioral Risk Factor Surveillance System survey of adults; and
- Education measures for K–12 students from Washington State's Office of the Superintendent of Public Instruction.

The following tables and text compare rates in Zones A, B, and C to Balance of County using the following definitions (Figure 1):

- Zone A: <1 mile from airport;
- Zone B: 1 to <5 miles from airport;
- Zone C: 5 to 10 miles from airport;
- Airport communities: Zones A, B, and C combined; and
- Balance of County: the area in King County more than 10 miles from the airport (the comparison area).

The zones overlap the census tract areas with the greatest average number of flights per day based on 2018 SeaTac flight data (Appendix A, Figure A1).

Appendix A reviews information for specific measures and methods of analysis. King County averages for all measures except excess deaths analysis are in Appendix B. Differences were considered statistically significant when 95% confidence intervals for rates compared did not overlap. Statistically significant differences are denoted in tables and text as the following:

- "Higher" means statistically significantly higher than Balance of County.
- "Lower" means statistically significantly lower than Balance of County.
- "Not different" means the difference from Balance of County was not statistically significant.

A difference that was not statistically significant is noted as a finding that "appeared" higher or lower.

Figure 1
Community Health Profile Zones

C. COMMUNITY HEALTH PROFILE FINDINGS

1. Demographic Characteristics

Key points:

- Nearly three-quarters of people in King County who identify as Black/African American live in airport communities. Similarly, 74.8% of the county's Native Hawaiian and Pacific Islanders, 57.6% of the county's Hispanic/Latino, and nearly half of the county's Asian population call the areas within 10 miles of the airport home.
- Compared to elsewhere in King County, airport communities (Zones A, B, and C) have a higher percentage of the population living in near poverty or poverty (up to 200% of the federal poverty level).
- Children in third grade in airport communities are less likely to have met third-grade reading standards; the on-time high school graduation rate was lower in the airport communities than in Balance of County.

Population totals and age: The average 2014–18 estimated population was 30,319 in Zone A, 260,163 in Zone B, and 605,546 in Zone C. Combined, the airport communities represent 42.6% of the county's population. The total Balance of County population is about 1.2 million, or 57.4% of King County.

The population distribution by age of residents was similar in airport communities and Balance of County with two exceptions (Table 1). The percentage of children living in the airport communities was slightly higher (between 22.8 and –3.1%) compared to Balance of County (19.5%). The percentage of adults 25–44 years of age was slightly lower; 29.5 and –32.1% of residents in airport communities were 25–44 years of age, compared to 33.9% in Balance of County.

Table 1

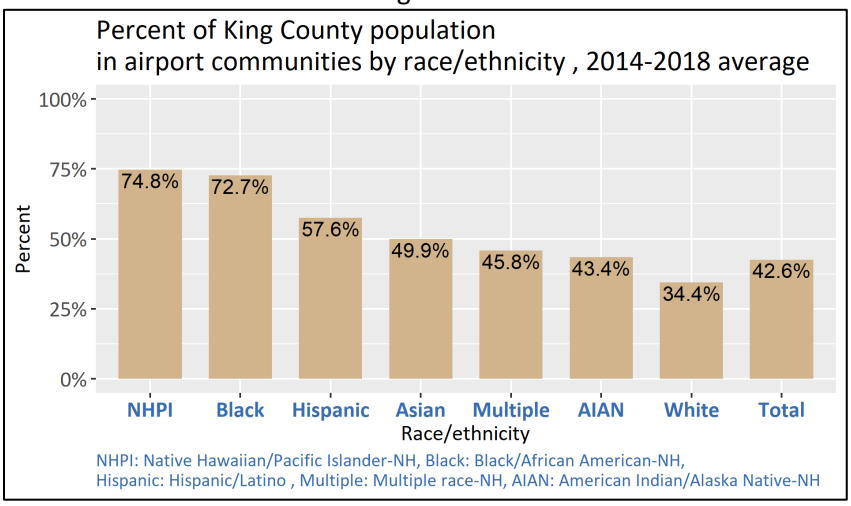
Population estimates by age group
by Zone, 2014-2018 annual average

Age	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County	
	Population	Percent	Population	Percent	Population	Percent	Population	Percent
Under 18	6,944	22.9%	60,202	23.1%	138,221	22.8%	235,712	19.5%
18 to 24	2,922	9.6%	22,250	8.6%	49,405	8.2%	111,731	9.3%
25 to 44	9,744	32.1%	79,124	30.4%	178,513	29.5%	409,925	33.9%
45 to 64	7,064	23.3%	65,759	25.3%	159,739	26.4%	306,420	25.4%
65 and over	3,645	12.0%	32,828	12.6%	79,668	13.2%	143,994	11.9%
Total	30,319	100.0%	260,163	100.0%	605,546	100.0%	1,207,782	100.0%

Race/ethnicity: The airport communities are home to the majority of King County's people of color (Figure 2). Roughly three-fourths (74.8%) of the county's Native Hawaiian/Pacific Islanders live in the airport communities. The majority of Black/African Americans (72.7%) and Hispanic/Latinos (57.6%) live in this area as well.

The percentage of the population identifying as Black/African American, Hispanic/Latino, or Native Hawaiian/Pacific Islander increased the closer you are to the airport (see Appendix B for data).

Figure 2



Foreign born: The percentage of people who were immigrants increased the closer you are to the airport (Table 3). Over one-third of Zone A residents (35.9%), 27.1% of Zone B, and 23.9% of Zone C residents were immigrants, compared to 20.5% of Balance of County residents. Place of birth for foreign-born individuals varied by zone; the highest percentage immigrated from Africa in Zone A and Asia in Zones B and C. The three most common regions of emigration (plus approximate counts and percent of total foreign-born) were:

- Zone A: Africa (4,200, 37%), Asia (3,500, 31%), Latin America (2,700, 23%);
- Zone B: Asia (34,200, 47%), Latin America (21,500, 30%), Africa (8,600, 12%);
- Zone C: Asia (81,700, 55%), Latin America (26,550, 18%), Europe (19,100, 13%).

Table 2

Foreign born
by Zone, 2014-2018 average

	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Indicator	Percent	Count	Percent	Count	Percent	Count	Percent
Foreign born	**35.9%**	11,500	**27.1%**	72,600	**23.9%**	147,500	20.5%

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

Poverty and Near-Poverty: U.S. poverty thresholds define who is in poverty and are based on household income, the number of children, and family size.[i] A higher percentage of people lived in near poverty or poverty, defined as below 200% of the federal poverty level (FPL), in airport communities compared to Balance of County (Table 3). The percentage of people in near poverty or poverty increased the closer you are to the airport, ranging from 37.2% in Zone A to 16.1% in Balance of County.

Just over half of children in Zone A (51.1%) and 46.7% of children in Zone B (46.7%) were in near poverty and poverty. Just over one-third (33.5%) of children in Zone C were below 200% FPL, compared to 14.6% in Balance of County. The percentage of children living near-poverty or poverty in Zone A was over three times the rate in Balance of County.

[i] See https://www.census.gov/data/tables/time-series/demo/income-poverty/historical-poverty-thresholds.html for thresholds.

Table 3

Poverty Income <200% of the Federal Poverty Level by Zone, 2014-2018 average[1]							
	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Age	Percent	Count	Percent	Count	Percent	Count	Percent
Total	**37.2%**	11,500	**31.7%**	84,100	**24.4%**	148,800	16.1%
Under 18	**51.1%**	3,800	**46.7%**	27,800	**33.5%**	44,200	14.6%

[1] In 2020, income for a family of 4 at 200% of the Federal Poverty Level is $52,400.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

Educational success (children): Children in third grade in Zones A (43.4%), B (39.0%), and C (54.6%) were less likely to have met the third-grade reading standard than their counterparts in Balance of County (73.3%; Table 4). The high school graduation rates for students in Zones A (80.7%), B (72.3%), and C (81.0%) were lower than the rate for Balance of County (87.7%).

Table 4

Child educational sucess by Zone, 2018-2019 school year				
	Zone A: <1 mile from airport	Zone B: 1 to <5 miles from airport	Zone C: 5 to 10 miles from airport	Balance of County
Indicator	Rate	Rate	Rate	Rate
Met 3rd grade reading standard	***43.4%***	***39.0%***	***54.6%***	73.3%
High school graduation rate[1]	***80.7%***	***72.3%***	***81.0%***	87.7%

[1] Graduation from high school within 4 years

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

Note: Counts were not available for this measure.

Educational attainment (adults): Nearly seven of 10 adults in Balance of County completed an associate degree or higher, more than twice the rate in Zone A (28.1%; Table 5). The rates in Zone B (38.6%) and C (51.4%) were also much lower than Balance of County. The percentage of adults with an associate degree or higher decreased with proximity to the airport. For adults, having a high school diploma or more education was also less common in the airport communities than in Balance of County (see Appendix B for data).

Table 5

Educational attainment, age 25 and older by Zone, 2014-2018 average							
	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Indicator	Rate	Count	Rate	Count	Rate	Count	Rate
Associate's degree or more	***28.1%***	6,100	***38.6%***	71,700	***51.4%***	222,800	68.7%

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

2. Birth Risk Factors

Key points:

- The mothers of infants born in airport communities were less likely to have had adequate prenatal care than were their counterparts in Balance of County.
- Births by women in airport communities were more likely to be premature or low birthweight compared to Balance of County.

Prenatal care: Starting prenatal care early in pregnancy and continuing with regular visits improves the chances of a healthy pregnancy and birth. This indicator measures the percentage of live births for which a) prenatal care started before the end of the fourth month and b) 80% or more of the recommended number of visits occurred (Table 6). The prevalence of early and adequate prenatal care was lowest in Zone A (63.9%), lower than the Balance of County rate of 75.0%. The percentages of women with early and adequate prenatal care in Zone B (68.8%) and Zone C (71.1%) were also lower compared to Balance of County. The percentage with early and adequate prenatal care declined with proximity to the airport.

Premature birth (singletons): A premature birth is a birth at less than 37 weeks gestation and is a leading cause of disability in infants and death of newborns. Prematurity also causes developmental delays, impairment of hearing and vision and chronic respiratory problems. Exposure to PM 2.5 (fine particulate matter), an airborne toxin associated with SeaTac Airport, increases the risk of premature birth.[2–4] As with low birthweight, we limited the findings to singleton births to eliminate multiple births as a factor in the comparison. The annual percentage of premature births was higher in Zones A (9.8%), B (9.4%), and C (8.2%) compared to Balance of County (6.6%).

Low birthweight (singletons): Low birthweight, a weight at birth of less than 2,500g or roughly 5.5 lb., increases the risk of infant mortality, respiratory disorders, and neurodevelopmental disabilities. We limited the findings to singleton births to eliminate multiple births as a factor in the comparison (elective fertility treatments can increase the chance of multiple births). Low birthweight was higher in airport communities, ranging from 5.5% (Zone C) to 6.1% (Zone B) compared to Balance of County (4.7%).

Table 6

Birth risk factors
by Zone, 2014-2018 average

Indicator	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
	Percent of live births	Count	Percent of live births	Count	Percent of live births	Count	Percent of live births
Early and adequate prenatal care[1]	***63.9%***	300	***68.8%***	2,367	***71.1%***	5,042	75.0%
Premature births (singleton)[2]	**9.8%**	48	**9.4%**	334	**8.2%**	593	6.6%
Low birthweight (singleton)[3]	**5.8%**	28	**6.1%**	217	**5.5%**	400	4.7%

[1] "Early and adequate prenatal care" based on Kotelchuck Index (>= 80% of expected prenatal care visits).

[2] A low birth weight is a weight at birth of less than 2500g.

[3] A premature birth is a birth at less than 37 weeks gestation.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

3. Overall Health, Access to Care and Risk Factors in Children and Adults

Key points:

- Adults were more likely to be uninsured and/or not get needed medical care in airport communities than in Balance of County. A greater percentage of school-age children in Zones B and C did not have a dental checkup during the last year compared to those in Balance of County.
- Obesity was higher in airport communities for both children and adults than in Balance of County.
- Cigarette smoking and/or e-cigarette use or vaping among students in 8th, 10th and 12th grades in airport communities did not differ from that of students in Balance of County. Adults in Zones B and C were more likely to report smoking than were those in Balance of County.
- Lack of physical activity for adults was more prevalent in airport communities compared to Balance of County. School-age children in Zone B were less likely to meet physical activity guidelines as in Balance of County.
- Among adults, high blood pressure and inadequate sleep were both more common in Zones B and C than in Balance of County.

Children

Overall health status: A lower percentage of parents/primary caregivers rated their children's health as excellent or very good in Zones B (82.9%) and C (86.5%) compared to Balance of County (92.1%; Table 7). The percentage of parents/primary caregivers rating their children's health as excellent or very good in Zone A (88.4%) appeared higher compared to Balance of County.

Child did not receive needed health care: Parents/primary caregivers were more likely to report that their child had needed health care but not received it during the last year in Zone B (6.1%) than in Balance of County (2.9%). Data from the American Community Survey show that the proportion of children under 19 without medical health insurance was less than 2.5% in both airport communities and Balance of County (data not shown).

No dental checkup in the last year: Having an annual dental checkup represents preventive care. A greater percentage of 8th-, 10th-, and 12th-grade students in Zones B (19.2%) and C (16.9%) did not have a

dental checkup in the last year. compared to Balance of County (11.8%). Overall, the percentage of youth with no dental checkup in the last year increased as grade level increased (data not shown).

Cigarette smoking and e-cigarette or vape pen use: The percentage of 8th-, 10th-, and 12th-grade students who smoked in the last 30 days in Zones B and C (5.2% each) did not differ from Balance of County (4.4%).The percentage of students using e-cigarettes or vape pens during the last month in Zones B (11.3%) and C (12.8%) also did not differ from Balance of County (14.4%).

Overall, the percentage of 8th-, 10th-, and 12th-grade students smoking cigarettes one or more days out of the last 30 days increased as grade level increased (data not shown). E-cigarettes and vape pens are now the most common form of tobacco use among 8th-, 10th-, and 12th-grade students, and use increased with an increase in grade (data not shown).

Met physical activity recommendations: Being physically active helps control weight, strengthens bones and muscles, and boosts mental health and academic performance. The percentage of 8th-, 10th-, and 12th-grade students who met physical activity recommendations (being physically active for a total of at least 60 minutes for each of the last seven days) decreased as grade level increased (data not shown). Students in 8th-, 10th-, and 12th-grade in Zone B (18.2%) were less likely to report having met the recommendation than students in Balance of County (21.3%). The rate for Zone C students (19.6%) did not differ from that of Balance of County.

Obese: Obese children are more likely to have high blood pressure and cholesterol, type 2 diabetes, anxiety, and depression and to be targets of bullying. Youth are considered obese if their body mass index is in the top 5% for their age and gender. The prevalence of obesity in 8th-, 10th-, and 12th-grade students in Zones B (15.2%) and C (12.2%) was higher than the rate for Balance of County (7.5%).

Table 7

Children: Overall health status , access to care, and risk factors by Zone, various years (shown in footnotes)							
	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Indicator	Rate	Count	Rate	Count	Rate	Count	Rate
Child overall health status excellent or very good (age 6 months through 5th grade)[1,2]	88.4%	N/A	***82.9%***	N/A	***86.5%***	N/A	92.1%
Child did not receive needed health care (age 6 months through 5th grade)[2]	6.1%*	N/A	**6.1%**	N/A	3.8%	N/A	2.9%
Dental checkup: None in last year (grades 8, 10, 12)[3]	^^	^^	**19.2%**	N/A	**16.9%**	N/A	11.8%
Current cigarette smoking (grades 8, 10, 12)[3,4]	^^	^^	5.2%	N/A	5.2%	N/A	4.4%
Current e-cigarette or vape pen use (grades 8, 10, 12)[3,5]	^^	^^	11.3%	N/A	12.8%	N/A	14.4%
Met physical activity recommendations (grades 8, 10, 12)[3,6]	^^	^^	***18.2%***	N/A	19.6%	N/A	21.3%
Obese (grades 8, 10, 12)[3,7]	^^	^^	**15.2%**	N/A	**12.2%**	N/A	7.5%

[1] Children whose caregivers reported their health status as excellent or very good.

[2] 2017, 2019 average

[3] 2016, 2018 average

[4] Smoked cigarettes on one or more days in the past 30 days.

[5] Used e-cigarettes or vape pens on one or more days in the past 30 days.

[6] Active for at least 60 minutes per day for the last 7 days.

[7] Body mass index was in the top 5% for age and gender.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

*Rate is unstable; use with caution.

^^Data suppressed to meet confidentiality standard.

N/A: Estimated counts were not available for these measures.

Adults

Health status fair/poor: In Zone A, one in four (25.5%) of adults reported fair/poor health, compared to one in 10 (10.0%) adults in Balance of County (Table 8). The percentage of adults rating their health as fair or poor in Zones B (15.7%) and C (14.9%) was also higher.

Uninsured (non-elderly adults): Among adults 19 to 64 years of age, the percentage lacking insurance in Zones A (14.3%) and B (13.8%) was more than double that of Balance of County (5.8%). The rate in Zone C (8.9%) was also higher than in Balance of County.

Could not see doctor due to cost: Over one-quarter of adults in Zone A (26.3%) reported that they could not see a doctor when needed in the past year because of cost, more than double the rate in Balance of County (9.8%). The percentage of adults who reported not seeing a doctor due to cost in Zone B (14.1%) was also higher than in Balance of County.

Current smoker: A higher percentage of adults reported smoking in Zones B (17.7%) and C (12.1%) than in Balance of County (9.5%).

High blood pressure: The percentage of adults reporting high blood pressure in Zone B (29.3%) and Zone C (28.3%) was higher than the percentage in Balance of County (23.6%).

Obese: An obese adult has a body mass index (BMI) of 30 or higher. People who have obesity have a higher risk of death overall, high blood pressure, heart disease, diabetes, and many other serious physical and mental health conditions. Obesity among adults was more prevalent in Zones A (44.3%), B (29.2%), and C (24.4%) compared to Balance of County (19.1%).

No physical activity in leisure time: Physically inactive adults have a higher risk of cardiovascular disease, obesity, and type 2 diabetes, and generally incur higher medical costs. In Zone A, roughly one-third (31.7%) of adults reported being physically inactive (not participating in physical activity or exercise in the last month), which is 2.6 times higher than the Balance of County rate (12.1%). Physical inactivity rates in Zones B (20.5%) and C (17.5%) were also higher than Balance of County.

Inadequate sleep: Not getting enough sleep is associated with obesity, type 2 diabetes, and sleep apnea (a risk factor for hypertension, stroke, and other conditions) and can be caused by environmental noise.[5] A greater percentage of adults in Zones B (34.5%) and C (35.0%) reported inadequate amounts of sleep (less than seven hours per night) compared to Balance of County (27.0%). The prevalence of inadequate sleep in Zone A (33.5%) appeared higher than Balance of County but the difference was not statistically significant.

Parents/caregivers of children in elementary school or younger who reported inadequate sleep did not differ between airport communities and Balance of County. Results differ from the prior measure, and this is likely due to the difference in who was asked, as these results are from parents/caregivers of younger children and not adults of all ages.

Table 8

Adults: Overall health status, access to care and risk factors by Zone, various years (shown in footnotes)							
	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Indicator	Rate	Count	Rate	Count	Rate	Count	Rate
Health status fair/poor[1]	**25.5%**	6,000	**15.7%**	31,500	**14.9%**	69,500	10.0%
Uninsured (age 19-64)[1]	**14.3%**	2,800	**13.8%**	23,200	**8.9%**	35,000	5.8%
Could not see doctor due to cost[1]	**26.3%**	6,100	**14.1%**	28,200	11.3%	53,000	9.8%
Current smoker (adult)[1]	8.2%*	1,900	**17.7%**	35,400	**12.1%**	56,700	9.5%
High blood pressure[1]	27.3%*	6,400	**29.3%**	58,500	**28.3%**	132,300	23.6%
Obese[1]	**44.3%**	10,300	**29.2%**	58,300	**24.4%**	113,900	19.1%
No physical activity in leisure time[1]	**31.7%**	7,400	**20.5%**	41,000	**17.5%**	81,600	12.1%
Inadequate sleep[2,3]	33.5%	7,800	**34.5%**	69,000	**35.0%**	163,700	27.0%
Children with a caregiver with inadequate sleep[2,4]	28.9%	NA	35.0%	NA	30.0%	NA	27.3%

[1] 2014-2018 average

[2] Inadequate sleep is sleeping less than 7 hours in a 24-hour period, on average.

[3] 2013, 2014, 2016 and 2018 average

[4] 2019 only

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

*Rate is unstable; use with caution.

NA: Estimated counts not available.

4. Chronic Conditions: Asthma, Stroke, Chronic Obstructive Pulmonary Disease, Heart Disease, Diabetes and Depression

Key points:

- The self-reported prevalence of chronic conditions among adults rarely differed between airport communities and the Balance of County, with two exceptions. Compared to Balance of County, a higher percentage of adults in Zone C reported ever having had a stroke, and a higher percentage of adults in Zones B and C reported having diabetes.
- Age-specific hospitalization rates for asthma in both children and adults were higher in Zones B and C than in Balance of County.
- Hospitalization rates for stroke, diabetes, and heart disease were higher in airport communities than in Balance of County. Heart disease rates were between 1.1 and 1.9 times higher and diabetes hospitalization rates were between 1.4 and 2.5 times higher. Hospitalization rates for heart disease increased the closer you are to the airport.
- Depression was relatively common for both school-age children and adults. About one in three school-age children in Zones B and C reported depression in the last year, a higher rate than in Balance of County.

Asthma: The rates of asthma hospitalization for both children and adults were higher in Zones B and C compared to Balance of County (Table 9). In Zones B and C and Balance of County, the risk of asthma hospitalization for children was much higher than risk among adults. In Zone B, for example, the rate among adults was 28.4 per 100,000 and among children it was 100.1 per 100,000.

Asthma prevalence estimates for adults in the airport communities did not differ from Balance of County. No recent, local estimates were available for asthma prevalence in children.

Stroke: The stroke hospitalization rate for people in airport communities was higher compared to the rate in Balance of County. The percentage of people who reported ever having had a stroke in airport communities appeared to be higher than in Balance of County, but only the difference between Zone C (3.1%) and Balance of County (1.9%) was significant.

Chronic obstructive pulmonary disease (COPD): Hospitalization for COPD was less common than for the other chronic conditions examined except asthma among adults. Though the rate of COPD hospitalizations in airport communities appeared higher than that of Balance of County, only the rate in Zone C (68.3 per 100,000) was significantly higher than the rate in Balance of County (52.3 per 100,000). Self-reported prevalence of COPD did not differ in airport communities from Balance of County.

Heart disease: Hospitalization rates for heart disease were higher than in the Balance of County, and rates were higher the closer you are to the airport. Zone A's hospitalization rate (876.5 per 100,000) was 1.9 times the rate for Balance of County (463.3 per 100,000). The rates for Zone B (600.4 per 100,000) and Zone C (520.4 per 100,000) were also higher than the rate in Balance of County. In Zones B and C, the percentage of people who self-reported ever having a heart attack appeared higher compared to Balance of County, though the difference was not statistically significant.

Diabetes: The hospitalization rates for diabetes in airport communities were higher than in Balance of County (between 1.4 and 2.5 times higher). The self-reported prevalence of adult diabetes was more common than other self-reported, physical chronic conditions examined except for depression, and the percentage of people who reported having diabetes in Zones B (9.9%) and C (8.2%) was higher than in Balance of County (6.3%). The percentage reporting having diabetes in Zone A did not differ from Balance of County.

Depression: Self-reported depression in school-age students was relatively scommon. About one in three students in grades 8, 10, and 12 in Zones B and C reported being feeling so depressed for two weeks or more in the last year that they did not do some usual activities (data not shown). Self-reported depression was significantly more prevalent in Zones B (34.4%) and C (33.5%) than in Balance of County (29.4%).

Self-reported history of being diagnosed with a depression-related disorder was also common in adults. About one in five adults in Zones B and C, and in Balance of County reported a history of depression. The prevalence did not significantly differ by zone.

Table 9

Chronic conditions: prevalence and hospitalization
by Zone, 2014-2018 average

Cause	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
	Rate[1]	Count	Rate[1]	Count	Rate[1]	Count	Rate[1]
Prevalence							
Has asthma now[2]	5.6%*	1,300	8.2%	16,400	10.0%	46,600	8.3%
Ever had stroke[2]	2.8%*	600	3.4%	6,900	**3.1%**	14,600	1.9%
Ever had heart attack[2]	2.7%*	600	3.6%	7,100	3.4%	16,000	2.5%
Chronic obstructive pulmonary disease[2,3]	2.9%*	700	4.8%	9,600	4.1%	19,300	3.3%
Diabetes[2]	11.8%*	2,800	**9.9%**	19,800	**8.2%**	38,200	6.3%
Depression (school-age)	^^	^^	**34.4%**	N/A	**33.5%**	N/A	29.4%
Depression (adults)[2]	11.3%*	2,600	20.5%	40,900	22.2%	103,600	21.5%
Hospitalization							
Asthma (adult)	^^	^^	**28.4**	^^	**28.5**	^^	16.6
Asthma (child)	^^	^^	**100.1**	^^	**86.8**	^^	71.3
Cerebrovascular diseases (stroke)[4]	**304.8**	17	**270.3**	413	**241.9**	981	213.0
COPD[3,4]	81.7	5	59.4	94	**68.3**	286	52.3
Diabetes[4]	**179.7**	10	**133.2**	202	**102.5**	414	70.6
Diseases of the heart[4]	**876.5**	48	**600.4**	918	**520.4**	2,134	463.3

[1] Prevalence rate is percent (rate/100); hospitalization rate is rate/100,000.

[2] Age 18 and older.

[3] COPD is chronic obstructive pulmonary disease.

[4] Rates are age-adjusted.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

*Rate is unstable; use with caution.

^^Data suppressed to meet confidentiality standard.

N/A: counts not available.

5. Life Expectancy and Leading Causes of Death

Key points:

- The closer you are to the airport, the shorter the average life expectancy.
- People residing in airport communities had higher rates of death overall compared to the rate in Balance of County, and the rate increased the closer you are to the airport.
- Residents of airport communities had higher rates of death from heart disease, unintentional injury, chronic lower respiratory disease, diabetes, chronic liver disease, and homicide than did people in the Balance of County.
- The rate of death from heart disease was higher the closer you are to the airport.

- People living in Zones A and B had a higher rate of cancer compared to Balance of County. People living in Zone B also had a higher rate of lung cancer than those in Balance of County.
- Poisoning, largely due to drug and alcohol overdose, was the leading cause of unintentional injury death, and the death rate was 1.2 to 2 times higher in airport communities than in the rest of King County.

Life expectancy: Life expectancy in airport communities was lower than that of Balance of County and decreased the closer you are was to the airport (Table 10). The life expectancy of people in Zone A, closest to the airport, was 5.0 years lower than that of people living in Balance of County.

Table 10

Life expectancy at birth
by Zone, 2014-2018 average

Indicator	Zone A: <1 mile from airport	Zone B: 1 to <5 miles from airport	Zone C: 5 to 10 miles from airport	Balance of County
	Years	Years	Years	Years
Life expectancy	***77.9***	***79.4***	***81.2***	82.9

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

Leading causes of death: People residing in airport communities had higher rates of death across all causes than in Balance of County and the rate increased the closer you are to the airport (Table 11). Residents of airport communities had higher rates of death from heart disease, unintentional injury (accidents), chronic lower respiratory disease, diabetes, chronic liver disease, and homicide than did people in Balance of County. People living in Zones A and B had a higher death rate from cancer and those in Zones B and C had a higher rate from stroke compared to the rate in the Balance of County. The heart disease death rate was higher the closer you are to the airport.

Alzheimer's disease is the only leading cause of death that was lower in an airport community (Zone B) compared to Balance of County. The rate of suicide deaths in airport communities did not differ from the rate in Balance of County.

Table 11

Leading causes of death[1] by Zone, 2014-2018 average							
	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
Cause Of Death	Rate[2]	Count	Rate[2]	Count	Rate[2]	Count	Rate[2]
All causes	**808.6**	248	**723.4**	1,901	**639.1**	3,950	572.2
Cancer	**158.7**	48	**160.9**	428	138.6	878	135.3
Diseases of heart	**187.7**	59	**142.9**	374	**126.6**	783	115.3
Alzheimer's disease	52.2	17	***38.1***	96	43.7	259	47.8
Accidents (Unintentional injuries)	**45.8**	14	**39.1**	106	**35.7**	222	30.4
Cerebrovascular diseases (stroke)	32.7	11	**34.9**	90	**34.5**	209	28.8
Chronic lower respiratory diseases	**38.5**	10	**34.9**	90	**27.6**	168	22.5
Diabetes mellitus	**33.9**	10	**26.3**	70	**22.0**	137	14.4
Intentional self-harm (suicide)	9.8	3	13.1	35	11.8	75	11.8
Chronic liver disease and cirrhosis	**18.8**	6	**13.1**	37	**10.2**	69	7.9
Influenza and pneumonia	14.3	4	**13.5**	35	10.0	61	8.6
Essential hypertension and hypertensive renal disease	10.9	4	**10.6**	28	**9.1**	55	6.8
Nephritis[3]	^^	^^	**8.6**	22	**6.6**	41	4.6
Septicemia	7.0*	2	**9.1**	24	**7.0**	42	4.1
Assault (homicide)	**6.6***	2	**7.9**	20	**4.0**	24	1.1
Certain conditions originating in the perinatal period	^^	^^	**4.0**	11	**3.2**	17	1.8

[1] The list of leading causes was developed by including any cause of death that is one of the 10 leading causes in any race group.

[2] Rates are age-adjusted rates per 100,000 population.

[3] Nephritis, nephrotic syndrome and nephrosis

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

*Rate is unstable; use with caution.

^^Data suppressed to protect confidentiality.

Cancer deaths: Though rates of cancer deaths overall were higher among people living in Zones A and B compared to Balance of County, rates for leading causes of cancer deaths did not show the same pattern (Table 12). Residents of Zone B had a higher rate of death from lung cancer and colorectal cancer compared to the Balance of County.

Table 12

Leading causes of cancer death[1]
by Zone, 2014-2018 average

Cause Of Death	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
	Rate[2]	Count	Rate[2]	Count	Rate[2]	Count	Rate[2]
Lung cancer[3]	36.1	11	**36.5**	96	29.9	188	27.7
Colorectal cancer	10.8	3	**14.8**	40	11.0	70	11.2
Breast cancer (female)	20.5	3	20.0	28	19.4	68	18.1

[1] Leading causes of cancer death were ranked by number of deaths in King County. The leading 3 were chosen.

[2] Rates are age-adjusted rates per 100,000 population.

[3] Cancer of the trachea, bronchus and lung

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

Unintentional injury deaths: Poisoning, falls, and traffic crashes were the most common causes of unintentional injury deaths. Nationally and locally, the majority of poisoning deaths are from drug abuse. The annual report from the King County Medical Examiner's office on drug and alcohol overdose deaths shows a substantial increase from 2014 to 2018, the time period that forms the dataset of deaths for our report.[ii] Rates of death by poisoning were higher in airport communities compared to Balance of County and were higher the closer you are to the airport (Table 13). The rate of traffic deaths was higher in Zone B than in Balance of County.

Table 13

Leading causes of unintentional injury death[1]
by Zone, 2014-2018 average

Injury Mechanism	Zone A: <1 mile from airport		Zone B: 1 to <5 miles from airport		Zone C: 5 to 10 miles from airport		Balance of County
	Rate[2]	Count	Rate[2]	Count	Rate[2]	Count	Rate[2]
Poisoning	**20.1**	6	**14.8**	41	**12.4**	80	10.2
Falls	8.4	3	10.4	27	11.1	68	11.1
Motor Vehicle-Traffic	8.4	3	**7.1**	19	5.5	34	4.2

[1] Leading unintentional injury mechanisms were ranked by number of deaths in King County. The leading 3 were chosen.

[2] Rates are age-adjusted rates per 100,000 population.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

[ii] For more information, see https://www.kingcounty.gov/depts/health/~/media/depts/health/medical-examiner/documents/2018-overdose-death-report.ashx.

6. Excess Deaths: How Many Fewer Deaths Would Occur if the Airport Communities Had the Same Risk of Death as the Balance of King County?

Key points:

- Across four of the leading causes of death in the county (heart disease, cancer, accidents, and stroke), the number of deaths in airport communities exceeded the expected number of deaths if airport communities had the same death rates as the Balance of County. Observed deaths from these causes ranged from 1.1 to 2.3 times higher than expected in the airport communities.
- Across the airport communities, deaths from cancer were between 1.1 and 1.4 times higher than expected, heart disease deaths were between 1.3 and 2.3 times higher, unintentional injury deaths were between 1.2 and 1.8 times higher, and stroke deaths were between 1.4 and 1.9 times higher than expected.

We estimated the number of excess deaths in airport communities by calculating the number of deaths that would occur if the population of airport communities was dying at the same rate as in Balance of County (see Appendix A for details). We included the five leading causes of death in King County: cancer, heart disease, Alzheimer's disease, unintentional injuries, and stroke. Many of the excess deaths may be preventable, given the elevated prevalence of physical inactivity, obesity, smoking, high blood pressure and potential exposure to airport-related pollution in some or all of the airport communities.

The number of excess deaths is the observed (actual) deaths minus the number of expected deaths. We also calculated the mortality ratio: the number of observed deaths divided by the expected deaths. A mortality ratio of greater than one means the number of observed deaths was higher than expected, and a ratio of less than one means observed deaths were less than expected.

Zone C, the airport community with the largest population, had the highest number of excess deaths (Table 14). Zone B had fewer estimated excess deaths and Zone A, with the smallest population size, had the lowest number of excess deaths.

In four out of the five leading causes, the number of deaths in airport communities exceeded the expected number of deaths. Cancer deaths were between 1.1 and 1.4 times higher than expected across the airport communities. Death from heart disease were 2.3 times higher in Zone A, 1.6 times higher in Zone B, and 1.3 times higher in Zone C. Unintentional injury deaths were between 1.2 and 1.8 times higher than expected. Stroke deaths were between 1.4 and 1.9 times higher than expected. Alzheimer's disease was the only cause of death for which observed deaths were not different from expected deaths.

Table 14

Excess deaths in airport communities[1]

5 leading causes of King County deaths, 2014-2018 average

Cause Of Death	Observed per year[2]	Expected per year[3]	Excess per year[4]	Mortality ratio[5]
Zone A: Less than 1 mile from airport				
Cancer	35	25	10	**1.4**
Diseases of heart	28	12	16	**2.3**
Alzheimer's disease	2	1	0	1.3
Accidents (Unintentional injuries)	12	7	5	**1.8**
Cerebrovascular diseases (stroke)	5	2	2	**1.9**
Zone B: 1 to less than 5 miles from airport				
Cancer	299	234	65	**1.3**
Diseases of heart	181	113	69	**1.6**
Alzheimer's disease	11	13	0	0.8
Accidents (Unintentional injuries)	82	58	24	**1.4**
Cerebrovascular diseases (stroke)	40	22	18	**1.8**
Zone C: 5 to 10 miles from airport				
Cancer	600	569	31	**1.1**
Diseases of heart	359	275	85	**1.3**
Alzheimer's disease	32	32	0	1.0
Accidents (Unintentional injuries)	170	138	32	**1.2**
Cerebrovascular diseases (stroke)	76	54	22	**1.4**

[1] Calculations exclude age 80 and older. See Appendix A for details.

[2] Observed per year is the number of actual deaths.

[3] Expected per year is the number of deaths that would have occured if the death rate was the same as the death rate in Balance of County. See Appendix A for details.

[4] Excess per year is the number of observed deaths minus the number of expected deaths. Numbers may not total due to rounding.

[5] Mortality ratio is the Zone's death rate divided by the death rate in Balance of County. See Appendix A for details.

Higher than Balance of County
Lower than Balance of County
Not different from Balance of County

7. Glioblastoma Cancer Incidence

Key points:

- During 2013-2017 combined, 400 new cases of glioblastoma were diagnosed in King County.
- The age-adjusted incidence rates of glioblastoma cancer for the airport communities did not differ from that of Balance of County.

Washington State Department of Health staff examined the incidence of glioblastoma cancer in King County by zone and found no differences in age-adjusted incidence rates. During 2013-2017 combined, 400 new cases of glioblastoma were diagnosed in King County. The age-adjusted incidence rate for Zone A was suppressed due to the low number of new cases but did not differ from that of Balance of County. The rates for Zone B and C also did not differ from Balance of County.

Table 15

Glioblastoma incidence
By zone, 2013 – 2017

Measure	Zone A	Zone B	Zone C	Balance of County	King County Overall
Age-adjusted incidence rate	^^	2.8	3.5	3.9	3.7
Confidence Interval for rate	^^	2.0 – 3.9	2.8 – 4.2	3.4 – 4.5	3.3 – 4.0
Average new cases per year	^^	8	23	48	80
Average population per year	^^	256,761	594,870	1,180,605	2,062,150

^^ Counts, rates and confidence intervals were not calculated if there are fewer than 10 new cases for the combined data from 2013-2017.

Note: Rates are per 100,000 and age-adjusted to the 2000 US Std Population (19 age groups) standard; rate confidence intervals are 95%. Invasive glioblastoma cancer is coded using International Classification of diseases for Oncology Third Edition (ICD-O-3) histology codes 9440-9442 with behavior code 3 (malignant); the airport communities and Balance of County areas were created using the 2010 census tracts.

Data Sources: Washington State Cancer Incidence data were from the Washington State Department of Health, Washington State Cancer Registry, released in March 2020. Population estimates were from the Washington State Office of Financial Management, released in January 2020.

III. WHAT POLLUTANTS RESULT FROM AIRPORT OPERATIONS AND WHAT ARE THE LIKELY HEALTH IMPACTS?

Report Requirement 2: Comprehensive literature review

> Proviso language: A comprehensive literature review concerning the community health effects of airport operations, including a strength-of-evidence analysis

A. SUMMARY

Airport operations result in noise and air pollution, which likely affect population health at common concentration levels. Noise pollution contributes to hypertension and heart disease and likely causes poor school performance among children. Air pollution impacts numerous organ systems (Table 16), and effects are especially pronounced for cardiovascular and respiratory outcomes. The air pollutants related to airport operations include particulate matter of various sizes, ozone, carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur oxides (SO_x), and other hazardous air pollutants. Fine particulate matter ($PM_{2.5}$) causes cardiovascular and respiratory problems, and likely causes cancer and central nervous system conditions, including dementia and neurodegeneration.

Existing research is less conclusive about larger and smaller particulate matter in comparison, though recent research demonstrates that exposure to both increases the risk of preterm births and respiratory concerns, among other issues. Ozone, NO_2, and SO_x cause short-term respiratory issues. NO_2 likely causes long-term cardiovascular problems, and CO causes short-term cardiovascular concerns and likely affects lung functioning. These pollutants are especially concerning for people with underlying respiratory or cardiovascular issues because they worsen existing conditions, though long-term exposures increase risk in the general population for developing problems. The hazardous air pollutants (HAPs) found at airports are known or suspected carcinogens and/or cause birth defects. Lead exposures are more common at Boeing Field, Renton, and Auburn airports than at SeaTac airport, because small planes with piston engines still use leaded gasoline. Lead causes central nervous system problems and is damaging even at low levels, thus is worth mentioning despite less prevalence at SeaTac airport.

Table 16

Health outcomes likely caused or caused by airport operations–related pollutants based on evidence to date		
Organ System	**Noise Pollution**	**Air Pollutants**
Cardiovascular	Causal	Causal
Respiratory	Not examined	Causal
Reproductive & fertility	Not examined	Causal
Birth outcomes	No causal evidence	Likely causal
Cancer	Not examined	Causal
Central nervous system	No causal evidence	Causal
Metabolic system	No causal evidence	No causal evidence

The type of exposure, amount of exposure, and length of exposure to pollutants can all impact risk. Vulnerable populations such as those with underlying heart conditions, heart disease, and asthma are more likely be affected by lower concentrations of air pollution and noise and more seriously affected

by higher levels of pollutants. Children and people with underlying health conditions such as asthma, other respiratory issues, and heart conditions are more vulnerable to pollutants. Risk of pollutant exposure is also related to socioeconomic status (SES); both individual SES and average SES of neighborhood independently impact health. People of color and people of lower SES face much higher health risks from air pollution and noise pollution compared to Hispanic/Latino populations and people of higher SES.[6] People of color and people of lower SES are also more likely to be exposed to higher levels of air pollution and noise pollution.

Multiple studies in the U.S. and other countries demonstrate higher noise levels related to airport operations and specifically aircraft.[7–9] Locally, a community-driven study of aviation noise demonstrated frequent noise pollution over FAA standards and well over World Health Organization (WHO) standards for environmental noise. Similarly, higher levels of air pollutants compared to ambient levels were found at several airports. Researchers with the University of Washington's Environmental and Occupational Health Sciences Department conducted the first study of UFP concentrations near the SeaTac airport during 2018–19 and found higher concentrations of ultra UFP (10–20 nanometers) below aircraft flight paths, with the highest concentrations associated with landings compared to departures. They concluded that communities underneath and downwind of the flight path are exposed to aircraft-related UFP concentrations.

B. LITERATURE REVIEW AND STRENGTH-OF-EVIDENCE ANALYSIS METHODS

This review of the literature examined the strength-of-evidence of research regarding airport-related air and noise pollution and the likelihood of harm to human health resulting from exposures. We reviewed the Environmental Protection Agency's most recent Integrated Science Assessment (ISA) per pollutant, because these documents represent consensus among EPA specialists and external subject matter experts. We also considered peer-reviewed literature and technical reports primarily published after the most recent ISA. Analysis is based on a review of 33 reports, including the ISAs; two letters by scientists involved in ISA production; and over 500 journal articles.

The strength-of-evidence analysis assessed the association between exposure and outcomes based on all relevant studies, given the quality of those studies. In other words, we drew conclusions about how strong the evidence to date was that various health outcomes are caused by air and noise pollution related to airport operations. Whenever possible, determinations draw directly on causality assessments in ISAs, technical reports, and systematic reviews, because they represent causal assessments made by subject matter experts. When ISAs were not available or older, such as for ultrafine particles and noise pollution, determinations relied more on systematic reviews and technical reports.

The strength-of-evidence criteria used for this analysis are based on the five-level causal framework that the EPA uses to assess air pollutant effects on health when conducting an ISA (Table 17).[10] A relationship is considered causal when multiple, high quality studies conducted by multiple researchers shows that exposure leads to the health outcome in question, the biological pathways of harm are supported by the evidence available, and alternative explanations have been ruled out. A relationship is considered likely causal when multiple, high-quality studies support that exposure leads to the effect, biological pathways are plausible but may be lacking some supporting evidence, and/or potential co-pollutant exposures have not yet been ruled out.

Table 17

Strength-of-Evidence Criteria Applied	
Causal	• Exposure is shown to lead to effect across multiple high-quality studies by multiple research groups. • Plausible alternative explanations for effect have been ruled out. • Biological pathways supported by evidence.
Likely Causal	• Exposure is shown to lead to effect across multiple high-quality studies, but uncertainties remain. • Plausible alternative explanations for effect have been ruled out but concerns about possible co-pollutant exposures may remain. • Biological pathways, while plausible, may be missing evidence.
Suggestive	• Exposure seems to lead to effect in at least one high-quality study, but alternative explanations for the effect, such as a third factor, cannot yet be ruled out. • May have a limited number of studies with modest effects or may have several studies with conflicting results.
Inadequate Evidence	It is unclear if exposure leads to effect or not, because of a lack of quality studies or because of significant inconsistency in results across studies.
Not Likely Causal	Exposure consistently does not appear to lead to effect across studies; adequate studies show no effect across different exposure levels and different populations.

The following review of the literature focuses on health impacts that are likely caused or caused by pollutants. Most information presented is from the ISAs; citations provide alternative and/or supplemental sources. Appendix C provides more details per pollutant, including health effects examined with findings suggestive of a relationship to the pollutant. Appendix D provides a visual description of health effects and pathways of harm for noise pollution, along with the five most common air pollutants associated with commercial airports.

C. LITERATURE REVIEW AND STRENGTH-OF-EVIDENCE ANALYSIS FINDINGS

The Seattle-Tacoma International Airport is the largest airport in Washington and in 2019 transported 51.8 million passengers and tons of cargo.[11] In 2018, the airport averaged 23 flights per hour per day, and from 6 a.m. through midnight typically experienced over 20 flights per hour.[12] Flights depart into the wind and land with the wind; at this airport, flights arrive and depart in a north-and-south pattern.[iii] For roughly 65% of the year, in the cooler months, winds come from the south; thus flights depart to the south and arrive from the north. Winds come from the north in the remaining months, so flights depart north and arrive from the south.

The airport's operations contribute to environmental pollution, with air and noise pollution having the largest impacts on community health. Most airport emissions come from airplanes, specifically the contrails formed from burning jet fuel. Planes idling or taxiing on runways increase emissions. Additional emissions result from vehicle traffic that are part of airport operations, some stationary sources within airports, and vehicle traffic going to and from the airport. The ground traffic pollutant with the greatest

[iii] For more information see https://www.portseattle.org/projects/flight-patterns.

impact is higher levels of black carbon within 500 meters of the source, though traffic-related air pollution can remain at elevated levels 1,500 feet from major roadways following rush hour.[13]

When planes taxi, take off, and land, air pollutants are blown downwind of the airplane in the contrails formed by engine exhaust. When planes are lower than 3,000 feet in the air, air pollutants travel downwind. When planes fly at cruising altitude above 3,000 feet, wind currents can carry pollution as far as 6,000 miles from the route. Pollution at these levels contributes to greenhouse gases, thus have a cumulative rather than direct impact on human health. Noise pollution, also called environmental noise, refers to elevated levels of unwanted sound in the environment that are disruptive or harmful for health and occur primarily as planes take off and land, though can also result from ground transportation.

This report focuses on airport-related impacts of noise and air pollution on communities, thus excludes occupational exposure to pollutants as well as water and soil pollution resulting from airport operations. Airport activities such as de-icing, washing, fuel spills, and other runoffs lead to water and soil pollution, which increases risks to ecosystems and impacts human health downstream when left untreated.[14] The review excludes the occupational health impacts of working at an airport, as well, to focus on airport operations–related impacts on general population health.

1. Noise Pollution Health Effects

Noise pollution refers to regular noise levels in the environment that are above levels identified as safe for human health. Road traffic noise and aircraft activity are the main sources of noise pollution related to airports. Aircraft noise is affected by the type of aircraft and engine, including its thrust, as well as environmental conditions influencing how sound travels, such as distance and weather.

Noise pollution impacts health by disrupting sleep and concentration and/or triggering stress and annoyance. Studies of self-reported sleep disruption and use of sedative medications confirm that noise pollution disrupts sleep.[15] Noise pollution activates the stress response in the body on a frequent basis, leading to chronic stress.[16] This can wear down various organ systems in the body, causing damage that can at a point become irreversible, which then increases risk for chronic diseases and early death.[17] Noise pollution can also disrupt sleep, which limits the body's recovery from stress. The body's stress response to noise can be, but does not have to be, triggered by conscious annoyance with noise levels. A stress response is also triggered automatically, even if noise levels are not consciously registered as annoying.[5] Chronic stress contributes to circulatory and cardiovascular health concerns. Very high levels of noise pollution can induce hearing loss, and non-auditory health impacts of noise pollution exposure include hypertension, cardiovascular problems, and reduced cognitive functioning (Table 18; see Figure D1 in Appendix D for pathways of harm).

School performance and cognition: Multiple studies found an association between children in schools exposed to higher noise pollution levels and poorer standardized test performance, reading comprehension and memory. Some of these controlled for other factors, including levels of air pollution, though many were cross-sectional.[8,18] A prospective longitudinal study conducted in Munich showed that students' poor short-term memory and reading cognition disappeared two years after the nearby airport was closed.[19] The mechanisms by which noise pollution affects cognition are not well identified. It could be that children are exposed to noise pollution at home, which impacts sleep and stress, and/or noise from aircrafts and other sources is chronically disruptive to classroom activity in schools highly exposed to noise pollution.[8,20]

Table 18

Health outcomes resulting from noise pollution *based on evidence to date*	
Health outcome	**Noise Pollution Impact**
Cardiovascular	Causal
Sleep disturbance	Causal
Annoyance	Causal
School performance	Likely causal

Cardiovascular problems: Long-term noise pollution can lead to chronic stress, inflammation, and oxidative stress, which in turn can lead to increased risk of hypertension[21,22] and heart disease.[23–26] These exposures also increase risk of stroke,[27] heart attack,[28] heart-related hospitalizations, and cardiovascular-related death.[29] The evidence is particularly strong that noise pollution contributes to ischemic heart disease.[23,24] The adverse health effect of noise pollution from aircraft has been validated locally. A retrospective cohort study confirmed that in Seattle, as in 88 other airport communities, airport-related noise exposure significantly increased the likelihood of hospital admissions for cardiovascular issues among people 65 years of age and older.[9]

Researchers have observed higher rates of diabetes and other metabolic outcomes in communities exposed to higher noise pollution, controlling for other factors,[21,30,31] though evidence is limited to a small number of studies. Other studies examined the presence of noise pollution and adult anxiety and depression symptoms and cognitive scores, but air pollution was not always controlled for and results were inconsistent.[8,32,33]

2. Air Pollution Health Effects

Air pollution from airport operations includes a variety of pollutants with varying quantities of emissions. The air pollutants of most concern related to aircraft and road traffic activity are:[34]

- Particulate matter (PM) classified by size as coarse PM, $PM_{2.5}$, and ultrafine PM (UFP);
- carbon monoxide (CO);
- nitrogen dioxide (NO_2);
- sulfur oxides (SO_x);
- ozone (O_3);
- lead (Pb); and
- some hazardous air pollutants (HAPs), also known as volatile organic compounds (VOCs).

The first six are criteria air pollutants, known to cause harm to human health and the environment and monitored and regulated by the Environmental Protection Agency (EPA) under the Clean Air Act, with the exception of UFP. Hazardous air pollutants are known or suspected to cause cancer or birth defects, though they tend to be less prevalent than most criteria pollutants. Following is a brief description of pollutant sources and evidence of harm to human health.

Although all air pollutants released by airport activity have the potential to cause harmful health effects at commonly observed concentrations, PM is a common pollutant that can have wide-ranging, serious health effects (Tables 18–19). Many argue that particulate matter is the greatest threat to human health among common air pollutants.[35,36] Particulate matter (PM) from combustion sources is a mixture of tiny solid and liquid particles, most less than 1 micrometer (μm) in diameter. They contain varying amounts of nitrogen oxides, elemental carbon, and many different hydrocarbons and more complex substances—including formaldehyde, acrolein, polycyclic aromatic hydrocarbons, and similar chemicals, as well as a variety of metals. Particle size influences how the particles move through the air; smaller particles can remain in the air longer and travel farther. Particle size also influences how particles move through the human body. Smaller particles are also more likely to remain in the lungs and move to the bloodstream, while the body more easily expels larger particles before they get to the lungs. Particulate matter emissions related to airports come primarily from aircraft exhaust and road vehicles but also from ground support vehicles and equipment. The PM resulting from ground operations is mostly black carbon (or soot).[37] PM from aircraft emissions is largely carbon-based UFP.[38]

Coarse particulate matter (diameters between 2.5 and 10 μm) related to airport activity is generally emitted by road traffic, ground support equipment, aircraft landing and takeoff, and wear on aircraft brakes and tires during landing. Auxiliary power units, construction, and application of solvents can also

contribute to coarse PM at airports. Because of its larger size, coarse PM is less buoyant and settles out of the air quicker than smaller particles do, thus it tends not to drift as far as smaller PM.

When inhaled, larger coarse PM generally settles in the upper respiratory tract (nose to bronchi), though some gets further and deposits in the lungs (See Figure D2 in Appendix D for an illustration of pathways). Short-term coarse PM exposure may lead to respiratory problems.[39] Days of higher coarse PM exposure are associated with more asthma attacks. Evidence is mixed for exacerbation of COPD symptoms, though coarse PM exposures may increase susceptibility to respiratory infections, and people with underlying respiratory illness may be more likely to experience respiratory death on days following high coarse PM levels. Short-term and long-term coarse PM exposure may also lead to cardiovascular problems.[76,80] Days of exposure to higher coarse PM levels may lead to small increases in blood pressure. People with underlying cardiovascular disease may experience worsened symptoms, changes in heartbeat, and increased risk of blood clots or heart attack and higher risk of cardiac-related death in the days following higher coarse PM exposure. Exposure over several years may lead to higher rates of heart disease, stroke, and pulmonary embolism.[40]

Fine Particulate Matter (diameters less than 2.5 µm)*:* Short- and long-term $PM_{2.5}$ exposures cause cardiovascular, respiratory, and cerebrovascular problems (Table 19 and Figure D3 in Appendix D). People with heart disease or hypertension are at higher risk of heart attack, stroke, and cardiac-related death following days of higher $PM_{2.5}$ levels.[41] People without underlying heart disease can experience increases in blood pressure and heart rate variability following days of higher $PM_{2.5}$ levels. Populations exposed to even moderate levels of $PM_{2.5}$ over many years develop higher rates of heart disease and hypertension. These populations experience more heart attacks, strokes, and cardiac-related deaths. Though the 2009 ISA and the 2019 ISA draft conclude that $PM_{2.5}$ *likely causes* respiratory effects, many experts have concluded the evidence is strong that both short- and long-term exposures *cause* respiratory effects.[42,43] During days of higher $PM_{2.5}$ levels, people with asthma are at higher risk of asthma attack. People with COPD and people with allergies are likely to experience worsened symptoms. People with underlying respiratory illness are more likely to experience respiratory death. Populations without underlying respiratory conditions are at increased risk of contracting respiratory infections on days following higher $PM_{2.5}$ levels. Young children exposed to moderate $PM_{2.5}$ over several years are more likely to develop asthma.[44] Their lungs may also not develop fully.[45] Populations exposed to moderate $PM_{2.5}$ over several years experience more respiratory infections and have higher rates of respiratory-related death.

Table 19

Health outcomes resulting from Fine Particulate Matter based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Causal	Causal
Respiratory	Causal	Causal
Nervous System	Suggestive evidence	Likely causal
Birth outcomes	Suggestive evidence	Likely causal

Long-term $PM_{2.5}$ exposure likely results in cancer and cancer-related deaths[46,47] as well as nervous system problems. Populations exposed to moderate $PM_{2.5}$ over several years have higher rates of lung cancer and lung cancer–related deaths. These populations aren't at higher risk of other types of cancer, but they are less likely to survive other types of cancer. Populations exposed to higher levels of $PM_{2.5}$ over several years have higher rates of dementia and cognitive decline among older adults.

Evidence is growing that $PM_{2.5}$ exposure increases risk of pre-term births. Researchers in Finland recently published findings from a population cohort study that controlled for pollutant exposures at home and relevant maternal characteristics like smoking.[3] They found that exposure to ambient NO, CO, SO_2, PM_{10}, and $PM_{2.5}$ during the third trimester increased risk of preterm birth; and exposure during week before pregnancy to $PM_{2.5}$ and PM_{10} and NO_2 increased risk of preterm birth. The short-term

exposure during the week before birth was associated with a 60–67% increase in the risk of preterm birth.[3]

Table 20

Health outcomes resulting from Ultrafine Pasrticulate Matter based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Suggestive evidence	Inadequate to date
Respiratory	Suggestive evidence	Inadequate to date
Nervous system	Suggestive evidence	Likely causal
Birth outcomes	Suggestive evidence	Suggestive evidence

Ultrafine particulate matter (UFP; diameters less than 0.1 µm) was difficult to measure in the past and is not regulated, thus infrequently monitored. As a result, scientific understanding of the health effects of UFP is in early stages but quickly evolving (See Figure D4 in Appendix D for an illustration of pathways of harm). UFP levels are not yet regulated by the EPA, so UFP emissions data is scarcely collected. Because $PM_{2.5}$, which includes UFP, dominates the health risks associated with aircraft-related air pollution and UFP's small size allows it to penetrate even deeper into the body, experts anticipate that UFP is particularly important for health. Long-term UFP exposure likely causes nervous system problems (Table 20). Longer periods of exposure to high UFP concentrations likely lead to stress response and inflammation throughout the brain. People exposed to high UFP levels for long periods can experience neurodegeneration and potentially develop Alzheimer's. Long-term exposure to high UFP may impact cognitive abilities and lead to more impulsive behavior. Some evidence suggests UFP may lead to respiratory, cardiovascular, and central nervous system problems, given that short-term exposures can trigger inflammation in the lungs and brain as well as a stress response.

Researchers in the United States examined effects of UFP exposure for a birth cohort that resided within 10 miles of the Los Angeles airport.[48] After controlling for maternal characteristics, exposure to traffic related pollution, and airport-related noise, results showed that exposure to aircraft-related UFPs was associated with preterm births and the odds of preterm births increased with greater exposure levels.[48]

Ozone (O_3) is a gas that forms from nitrous oxides and HAP reactions in the air, rather than being directly emitted from the airport. Ozone concentrations near airports tend to be lower than levels in surrounding urban areas.[36] The nitrogen dioxide emissions near airports react with ozone in the air, keeping ozone concentrations low. While ozone exposure is less of a concern close to airports, airport activities still contribute to higher regional ozone levels by emitting VOCs (volatile organic compounds) and NO_x.[36] VOCs are a subgroup that includes all HAPs of concern near airports. Aircraft emissions contribute, however, to global pollution by increasing regional ozone concentrations, thus impacting human health on a regional scale.

Table 21

Health outcomes resulting from Ozone based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Suggestive evidence	Suggestive evidence
Respiratory	Causal	Likely causal
Metabolic	Likely causal	Likely causal
Reproductive system	Suggestive evidence	Suggestive evidence

Ozone contributes to inflammation and oxidative stress (See Figure D5 in Appendix D). Short- and long-term exposure to ozone causes respiratory problems (Table 21). In the short-term, ozone can decrease lung functioning in young, healthy adults, which increases susceptibility to respiratory symptoms and infections and respiratory symptoms in general population. People with underlying respiratory issues are likely to suffer worse asthma, COPD, and allergies, leading to increased hospitalizations and greater risk of death. Long-term ozone exposure likely leads to development of asthma in children; increases susceptibility to respiratory infection, severe asthma, and development of allergies; and may lead to development of COPD. Some evidence suggests that short- and long-term ozone exposure may lead to cardiovascular effects, metabolic problems, and negative effect on cognition. Some findings show that ozone exposure

leads to impaired heart function, heart rate variability, inflammation or oxidative stress, and diabetes-related deaths, but results across all relevant studies are inconsistent. Some evidence shows an association with long-term exposure and blood pressure, hypertension, and cardiovascular mortality. Ozone exposures in first and second trimesters may lead to lower birth weight and pre-term birth, but alternative explanations have not been ruled out.

CO, NO_2, and SO_x can impact respiratory health in areas very close to emission sources, though airport activity emissions don't usually push regional concentrations above EPA standards. CO and NO_2 are released from the engines of planes, ground support vehicles and equipment, and road traffic.

Carbon monoxide (CO) causes inflammation and oxidative stress (Table 22; Figure D6 in Appendix D). The gas can also impact health by displacing oxygen in the bloodstream, which prevents organ systems in the body from receiving enough oxygen. Short-term CO exposure likely causes cardiovascular problems, specifically changes in heart rate due to lower oxygen in the blood. For people with underlying heart disease, exposures can worsen symptoms and trigger irregular heartbeat, which will increase risk of cardiac-related death.

Table 22

Health outcomes resulting from Carbon Monoxide based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Likely causal	Likely causal
Respiratory	Suggestive evidence	Inadequate evidence
Nervous system	Suggestive evidence	Suggestive evidence
Reproductive system	Suggestive evidence	Suggestive evidence

Nitrogen Dioxide (NO_2) causes inflammation and oxidative stress, primarily in the respiratory tract and lungs (Table 23; Figure D7 in Appendix D). Short-term NO_2 exposure causes respiratory problems by worsening asthma symptoms and triggering asthma attacks. Exposure may also contribute to respiratory symptoms and susceptibility to respiratory infections in the general population. Exposure may worsen symptoms for those with allergies or COPD. Exposure may increase risk for respiratory-related death for those with underlying respiratory conditions. Long-term NO_2 exposure likely increases risk of developing asthma. Some evidence suggests that short- and long-term NO_2 exposure may worsen underlying heart disease, increasing risk of heart attacks and cardiac-related death. Longer-term exposures may contribute to development of heart disease. Long-term NO_2 exposure may increase risk of lung cancer, as well as increased risk of developing insulin resistance and type 2 diabetes. Exposure during pregnancy may be associated with autism spectrum disorder. Longer term exposures may be associated with dementia, Parkinson's disease, and cognitive decline.

Table 23

Health outcomes resulting from Nitrogen Dioxide based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Suggestive evidence	Suggestive evidence
Respiratory	Causal	Likely causal
Nervous system	Inadequate evidence	Suggestive evidence
Reproductive system	Suggestive evidence	Inadequate evidence

Sulfur oxides cause inflammation and oxidative stress in the respiratory tract and lungs (Table 24; Figure D8 in Appendix D). Short-term SO_x exposure causes respiratory problems. The evidence is strongest for asthma exacerbation. Short-term exposures also may cause decreased lung function in people with underlying respiratory conditions. These impacts increase risk for respiratory death among people with respiratory conditions. Long-term SO_x exposure may lead to respiratory problems and contribute to asthma development as well as the severity of asthma in children. Exposure may also contribute to allergy development, increased susceptibility to respiratory infections, and respiratory-related death.

Table 24

Health outcomes resulting from Sulfur Oxides based on evidence to date		
Health outcome	**Short-term Exposure Impact**	**Long-term Exposure Impact**
Cardiovascular	Inadequate evidence	Inadequate evidence
Respiratory	Causal	Suggestive evidence
Nervous system	Inadequate evidence	Inadequate evidence
Reproductive system	Inadequate evidence	Inadequate evidence

Hazardous air pollutants (HAPs) include 187 air pollutants that the EPA knows or suspects to have serious health effects, such as cancer or birth defects. The greatest sources of HAPs related to airport activities are idling and taxiing aircrafts.[37] Road vehicles, ground support equipment, and stationary equipment like generators and AC units also contribute to airport HAPs. The EPA regulates HAPs by placing emissions standards on equipment (like vehicle engines) rather than monitoring air quality as is done for criteria air pollutants.

The Airport Cooperative Research Program (ACRP), authorized by Congress and sponsored by the Federal Aviation Administration to find solutions to issues airports face, concluded that the most important HAPs related to airport activities are:[49]

- formaldehyde;
- acrolein;
- 1,3-butadiene;
- naphthalene;
- benzene;
- acetaldehyde; and
- ethylbenzene.

Several other HAPs are emitted at airports, but the ACRP list prioritizes HAPs that have the highest emissions and are most likely to lead to serious health effects.[49] Formaldehyde, 1,3-butadiene, benzene; and acetaldehyde cause cancer while the remaining HAPs noted are likely to cause cancer. In the short term, most cause respiratory irritation, except benzene (see Appendix C for more details).

Lead (Pb) is both a HAP and a criteria air pollutant. Inhaled lead is absorbed from the lungs into the bloodstream, where it strongly inhibits nutrient absorption and is highly toxic for cell function in organ systems throughout the body, especially the brain. Lead also gets stored in the blood, bones, and tissues, where it can be released later, re-exposing the body many years after exposure. Lead is removed from vehicle fuel and commercial jets run on lead-free kerosene-based fuels, but many small piston-engine planes still use leaded-fuel. Piston-engine aircraft are the largest source of lead emissions in the U.S.[50] Lead emissions are primarily of concern near airports that serve small piston-engine planes (called “general aviation” airports). Airborne lead concentrations have been found higher than EPA standards over a half mile downwind of general aviation airports.[51] In addition to being inhaled, lead also settles out of the air into soils and dust, where it can be ingested by children.[52] Because lead does not break down in soils, children today also face exposure risk from high historical lead emissions related to aircraft, vehicle, and industrial activities.[53]

No safe threshold for lead exposure exists. The effects of even small amounts of lead are devastating for health. Lead exposure can cause a range of cognitive effects in children, such as lower IQ and issues with attention, impulse, executive function, hyperactivity, and school performance. Exposure also likely results in behavioral problems in children—internalizing problems such as depression and anxiety, and externalizing problems such as bullying and conduct disorders.[54] Motor function and hearing are also likely impacted by Pb exposures in children. Lead exposures in adults can cause hypertension and coronary heart disease,[55] and likely contributes to reduced immune system efficacy, depression and anxiety, and issues with cognitive function. Exposures can also cause delayed puberty and reduced reproductive function.

3. Who Is At Risk?

The type of exposure, amount of exposure, and length of exposure can all impact risk. Vulnerable populations such as those with underlying heart conditions, heart disease, and asthma are more likely affected by lower concentrations of air pollution and noise and more seriously affected by higher levels of pollutants. For example, on days with high air pollution concentrations, people with underlying conditions are more likely to suffer asthma attacks or heart attacks. Higher concentrations will also affect more people in the general population.

Risk of an adverse health outcome is not equal among everyone exposed to the same level of the same pollutant. Each person has their own unique mix of other exposures and individual characteristics, including stage of life or underlying health conditions, that shape their own risk or susceptibility. For example, children are especially susceptible to the effects of lead because they are more likely to ingest it, the same dose is larger relative to their body mass, and they are at a critical stage of development. Children also breathe two to three times faster than adults, so they may breathe in more pollutants. Older adults may be at increased risk for cancer-related health effects because they have had a longer time to accumulate more DNA damage. Noise sensitivity can vary by age, underlying medical/sleep conditions, work schedule, and noise insulation at home or work.

Underlying health conditions increase risk of more serious health effects. Someone with heart disease is at greater risk of more serious cardiac-related health effects from air and noise pollution exposures than someone without underlying heart health concerns. Beyond having an underlying health condition, increased activation of biological pathways (chronic stress, systemic inflammation, oxidative stress, DNA damage, and epigenetic changes) prior to noise or air pollution exposures increases risk of health effects from air and noise pollution exposures. These biological pathways can be activated by things like stressful life circumstances, restricted access to healthy foods, restricted opportunity for physical activity, unsafe working conditions, and inherited epigenetic changes related to these stressors.

The health risks of noise pollution and air pollution are not distributed evenly between communities near airports.[iv] Air pollutant exposures and noise levels vary across areas near airport activity (including flight paths and road traffic) over time, distance, local geography, and meteorological conditions. Risk of pollutant exposure is also related to socioeconomic status (SES) and race/ethnicity. Both individual SES and average SES of neighborhood independently impact health.[56] People of higher SES living in lower-SES neighborhoods have resources that may allow them to avoid or overcome health hazards from their neighborhood. For example, they may be able to insulate their homes to reduce their individual exposure to noise pollution. People of color are disproportionately represented among lower-SES groups in the U.S., and the systemic racism they face daily also adversely affects health.[57] These differences contribute to poorer health outcomes for people of lower SES, including higher rates of diabetes, heart disease, respiratory disease, and early death.

[iv] See for example https://fortress.wa.gov/doh/wtn/WTNIBL/.

People of color and people of lower SES face much higher health risks from air pollution and noise pollution compared to white people and people of higher SES.[6] They are more likely to live in neighborhoods with greater air and noise pollution exposures because these neighborhoods tend to have lower housing prices, and economic and racial segregation have resulted in discriminatory housing practices. Lower SES people are more likely to hold jobs where they are exposed to high noise levels, increasing baseline risk for effects from neighborhood noise pollution exposures. Many noise sensitivity factors, such as overnight shift work, sound insulation in buildings, and underlying medical or sleep conditions, are also likely to be more concentrated in lower-SES neighborhoods. People of color and people of lower SES are more likely to have underlying conditions like diabetes, heart disease, and respiratory conditions, and they are less likely to have access to appropriate health care. Epigenetic changes from exposures to previous generations may increase susceptibility to health effects from air pollution today.[58]

4. Are Communities Near Airports Exposed to Pollution Resulting from Airport Operations, and if So, How Much?

Not enough is known about people's exposure to airport-related noise and air pollution, likely because it can be difficult to assess and requires understanding the concentration of pollutants, duration of exposure, and in some cases infiltration of pollutants into biological systems. At an individual level, determining exposure requires either testing biologically to determine the level of pollutants in systems, or using personal, portable pollutant monitors to measure exposure as well as people's location when exposure occurred. Such studies are infrequent and time consuming; the more common approach is to use available information from existing ambient noise and air pollutant monitors and estimate people's exposure using residential addresses or similar data.

Noise Pollution

Multiple studies in the U.S. and other countries demonstrate higher noise levels related to airport operations and specifically aircraft.[7–9] Sound levels are measured in decibels (dB) based on human hearing, where 0 dB is barely audible and a food blender running 3 feet away is roughly 95 dB. A 10-unit increase in decibels is the same as roughly doubling the perceived sound level. Regulations for noise pollution usually apply to average noise levels over 24-hour periods (Ldn) with an adjustment made to lower the threshold for nighttime noise. An average noise level is limited however, and the maximum or peak sounds and how often they occur are important to health and have implications for lived experiences.[59]

Noise levels for the general public are no longer regulated at the federal level after the EPA's Office of Noise Abatement and Control was defunded in 1982. Prior to then, the EPA published guidelines advising that limiting outdoor Ldn to 55 dB and indoor Ldn to 45 dB was essential to protect people from activity interference and annoyance.[60] The Federal Aviation Administration (FAA) maintains that Ldn below 65 dB is compatible for schools and residential land use.[61] In 2018, the WHO revised guidelines for Europe to set aircraft noise thresholds at 45 dB during the day and 40 dB at night.[62] The WHO guidelines are health-based (i.e., recommendations based on protecting human health from exposure).

The WHO recommends that aircraft noise levels be reduced to 45 dB during the day and 40 dB at night because noise at higher than these levels can negatively impact sleep, children's learning, and health. For example, a cross-sectional study conducted in three nations of children in schools near airports found that reading performance declined with each increase of 5 dB of noise, after controlling for the level of school insulation and relevant family characteristics.[63]

The most recent completed study of local noise pollution in communities near the airport was the Beacon Hill Noise Measurement Project conducted in 2018.[64] Volunteer residents collected 24-hour periods of noise level information over 52 locations between April and September, as well as maximum

levels and how often they occurred. Of the 136 24-hour observation periods, over half of the 24-hour day-night average noise levels (Ldn) were over 65 dB. The maximum observed was 85.5 dB. Analyses of 1-second noise measurements across numerous days showed that noise levels exceeded 65 dB between 1% to 18% each hour between 3 a.m. and noon. A University of Washington undergraduate student collected observations to help assess noise levels related to aircraft versus ambient neighborhood noise and found that noise levels above 70 dB correlated with aircraft flying overhead. Noise levels without aircraft flying overhead were 45–55 dB. The student's observations of aircraft flights overhead aligned with noise levels recorded at a nearby Port of Seattle monitor.[v] University of Washington Seattle researchers, in partnership with El Centro de la Raza, are in the process of studying noise levels in the Beacon Hill community resulting from airplanes flying overhead.

Air Pollution

Multiple studies document elevated levels of air pollutants, specifically particulate matter and black carbon, near airports and/or underneath flight pathways. Researchers found higher levels of air pollutants near Los Angeles airport,[65,66] LaGuardia in New York,[67] Logan airport in Boston,[68,69] the Ciampino airport in Rome,[70] and the Schiphol airport in the Netherlands.[71] In 2016, observations of pollutant levels at Los Angeles International Airport (LAX) and major freeways showed higher particulate concentrations, $PM_{2.5}$ concentrations, and black carbon concentrations per day near the airport compared to the freeways (11, 1.4, and 2.5 times higher respectively).[65] At that time, particulate number was considered a proxy for UFP because methods to reliably measure UFP were developed within the past few years. A 2014 study showed that particulate concentrations downwind of LAX were four times higher than ambient levels five miles from the airport and twice as high as ambient levels 10 miles away.[68] Another study of LAX with data collected in 2017 showed similar levels of UFP that were between 1.1 and 4.8 times higher than that of ambient concentrations. Higher levels of UFP were associated with airport landings, and these elevated levels of UFP corresponded to the highest noise levels from aircraft overhead.[72]

The Puget Sound Clean Air Agency monitors and reports on regional air quality. Its 2019 data summary noted that fine particle matter levels were within EPA standards throughout the year but at higher than recommended levels for human health using the Agency's definition (25 $\mu m/m^3$) during at least 22 days during winter.[73] The 2018 report noted higher than recommended levels of particle matter and ozone during the year.[74]

Estimates reported by SeaTac airport for 2016 show that most airport-related air pollutant emissions that are regulated result from aircraft engines (Table 25). Lead levels are lower than the emissions estimates shown below; 2014 EPA data estimate 0.02 tons of lead emissions from SeaTac airport.[75] In comparison, King County airport activities resulted in 1.53 tons of lead emissions in 2014; 93% of these emissions came from Auburn and Renton municipal airports, Boeing Field, and Crest Airpark. Lead emissions may be higher at local airports other than SeaTac because the use of airplane fuel containing lead is higher.[vi] Typical ground transportation emissions (from people driving to and from the airport) tend to contribute CO, NO_x, VOCs, and PM emissions on a scale similar to aircraft emission sources in immediate proximity to the roadway.[36]

[v] For Port of Seattle noise monitoring information, see https://www.portseattle.org/page/aircraft-noise-monitoring-system.

[vi] For more information about aircraft fuels containing lead, see the Federal Aviation Administrations' 2019 Fact Sheet – —Leaded Aviation Fuel and the Environment at https://www.faa.gov/news/fact_sheets/news_story.cfm?newsId=14754.

Table 25

Criteria Pollutant Emissions Inventory, 2016 Seattle-Tacoma International Airport						
Emission Source	**NO_x**	**VOC**	**CO**	**SO_X**	**PM_{10}**	**$PM_{2.5}$**
Aircraft Engines	1,775	261	1,455	162	13	13
Auxiliary Power Unit	40	3	33	5	5	5
Ground Support Equipment	370	94	2,769	19	25	25
Stationary Sources	18	1	12	0	1	1
Parking	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Ground Transportation	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Total	2,267	379	4,841	190	48	47
n.a.=not available Source: Landrum & Brown using the FAA's AEDT Version 2c Service Pack 2, Sept 2017.						

As mentioned previously, UFP is not regulated and therefore not systematically monitored, though understanding UFP is critical for communities near airports because aircraft exhaust emissions are largely carbon-based UFP. The first study measuring UFP emissions from aircraft traffic near SeaTac Airport was completed in 2019 and is reviewed in section IV of this report.[76]

PM from airplane exhaust is likely present indoors as well as outdoors. A recent study of air pollutant concentrations outdoors and indoor in a residence representative of neighborhood housing structures and under Boston Logan International Airport's flight paths demonstrated that outdoor pollutants penetrated indoors within minutes, indoor and outdoor concentrations were similar (suggesting that penetration is substantial), concentrations were greater during landings than takeoffs, and indoor concentrations were higher than those reported by area outdoor monitoring stations.[77]

Because it is not possible to conduct randomized experiments to study airport-related air pollutant exposures and confounders such as noise levels are common, modeling is used to examine the impact of exposure on population health. For example, researchers recently modeled the number of children in schools near seven California airports who were exposed to airport-related air pollutants by calculating when children in school were downwind of the airport.[78] They estimated that 8.7% of children were exposed to airport-related air pollutants for an hour or more per day. A greater proportion of economically disadvantaged children (10.6%) were exposed an hour or more per day. One estimate of the number of deaths attributable to aircraft emissions at cruising altitude was 8,000 per year but did not factor in emissions from takeoffs and landings.[79] A more recent estimate attributed roughly 16,000 deaths per year globally to aircraft emissions and showed that CO and NO_2, along with contrails, were significant contributors to reduced air quality and should be targeted for reduction.[80] An FAA-sponsored study estimated 160 deaths annually from aircraft $PM_{2.5}$ emissions during takeoff and landing between the 325 U.S. airports studied from 2005 to 2006.[81] The same study estimated that SeaTac aircraft $PM_{2.5}$ emissions during landing and takeoff made up approximately 0.25% of all $PM_{2.5}$ emissions in the Seattle-Tacoma region and 0.87% of all $PM_{2.5}$ from mobile sources in the region.[81] The most recent local estimate is from a 2017 study of 66 major airports, which estimated roughly 0.7 deaths annually to SeaTac emissions and 0.1 deaths annually from Boeing Field emissions, primarily from $PM_{2.5}$ and pollutants that form $PM_{2.5}$.[82]

Many of the health effects reviewed may take place at exposure levels below the EPA standards, especially the effects of $PM_{2.5}$,[83–85] A few recent studies have estimated over 100,000 early deaths per year in the US from $PM_{2.5}$.[83,86] A cohort study of U.S. veterans estimated risk of $PM_{2.5}$ exposure based on county-level measurements and modeled the relationship to cause-specific death rates; the authors found that 99% of the estimated burden of death from nonaccidental causes was associated with

exposures under the EPA standards.[87] A number of researchers argue that the $PM_{2.5}$ standard should be much lower than it is.[85,88]

The EPA standards and monitoring process is not designed to protect neighborhoods closest to emission sources. The EPA regulates ambient concentrations of most of the air pollutants reviewed, and local concentrations are likely higher than regulation levels. Apart from lead emissions, EPA monitors are generally not intended to monitor local areas of highest concentrations; the monitors tend to be located where they can measure background concentrations.[89] Even when those monitored concentrations are within EPA standards, communities closer to sources of these air pollutants will be exposed to higher concentrations than what the monitors reflect. As mentioned previously, these communities tend to have greater populations of people of color and higher rates of poverty.[90] For example, a 2010 study measuring street-level pollutants in Seattle's International District found much poorer air quality than that measured by the nearest air quality monitoring station a mile away.[91]

5. What Do We Know about Mitigating Airport Related Pollutants?

Evidence about what works in mitigating airport-related pollutants is also somewhat limited. Most of the relevant research explores the effectiveness of filtration systems, airport operation designs, alternative fuels, and use of green space. Recent findings are summarized below.

Building seals and effective filtration systems will prevent exposure to pollutants and disruptive noise. Because people spend most of their time indoors, most of their exposure to particulate matter and other air pollutants happens indoors. Removal of particulates/air pollutants indoors is dependent on the quality of heating, ventilating, and air-conditioning (HVAC) systems. HVAC filtration standards are based on the ability to remove particulates by size, represented by a minimum efficiency removal value (MERV). This current standard does not address the ability to filter out UFP.[92] A 2014 analysis of HVAC filtration systems suggests that $PM_{2.5}$ and UFPM removal efficiency increases with higher MERV; the percentage of particulate matter removed was a median of 71% for $PM_{2.5}$ for MERV14, and was near 100% for HEPA filters.[93] The ASHRAE filtration standard for schools following the COVID-19 outbreak is MERV13, which is expected to remove, on average, a minimum of 75% of particles having a size of 0.3–1.0 µm (UFPs).[94] Ensuring that buildings have high efficiency filtration systems will help reduce exposure to air pollutants.[95–97] One estimate suggests that use of high-efficiency filters will reduce premature mortality by up to 2.5% and increase life expectancy by 0.02–1.6 months.[98] Building construction, specifically window type and heating system, was related to the levels of indoor pollutants relative to outdoor levels of pollutants in one study.[99]

Having high-efficiency filtration systems and sealed buildings may not be enough, however, to reduce people's exposure. A six-week, randomized, crossover study of 23 low-income homes in urban areas (Boston and Chelsea, MA) showed that high-efficiency particulate air (HEPA) filtration reduced particulate number concentrations by 50–85% but did not reduce inflammation in people, perhaps because of longer-term exposures (carry-over effects) and/or residents opening windows.[100] Researchers in Denmark found in an experimental study that human behavior had as large an impact on indoor PM concentrations as did a particulate filtration system.[101] Similarly, one study showed that using personal air monitors increased awareness of where air pollution occurs and its effects, but providing personal air monitors was insufficient for changing people's behaviors to minimize exposure.[102]

Airport departure and arrival operation changes can reduce pollution. Airport departure designs that minimize the time planes idle or taxi, use steeper takeoff trajectories, and optimize engine thrust can help reduce emissions and noise pollution. Holding at the gate rather than having planes taxi can reduce emissions.[103] Research conducted at the Detroit, MI airport showed that holding planes at the gate for 25 minutes or so to reduce congestion reduced emissions by 35–38% relative to no gate holds.[104] Taking off at 81% thrust instead of full thrust reduced emissions in one study.[105] Local data has been used to plan and test departure and arrival patterns to reduce noise pollution.[106]

Alternative biofuels can also reduce emissions, as long as thermodynamic efficiency of engine combustion remains the same or improves. One experiment showed that blending biofuel with conventional fuel reduced the amount of particulate emissions during flight by 50–70%.[107] Scientists recently developed an approach to convert plant waste (specifically the cellulose) into plane fuel with higher heat values than conventional plane fuel.[108] Higher volumetric heat values (referred to as higher-density fuel) mean that the same volume of fuel gives the plane more range, which then is likely to reduce CO_2 emissions.

Green space, specifically coniferous tree coverage, is associated with lower concentrations of particulate matter, though the mechanisms and effects are complex and appear to depend on factors including leaf structure, scale, and region, among other considerations.[109] For example, a study in Finland found only slight or insignificant effects of trees on concentrations of particulates, NO_2, and VOCs.[110] A recent study of five cities in China showed that more tree coverage and grass coverage is correlated with lower $PM_{2.5}$ concentrations in neighborhood green spaces greater than 200 m^2 but not necessarily smaller areas.[111] In that study, total green space and tree coverage had a stronger relationship with lower $PM_{2.5}$ concentrations than did grass coverage. PM accumulates on the surface or hairs and in the wax of leaves, thus the ability to capture PM deposits depends on the type of tree leaf. Leaves with more hairs on top and lower specific areas (leaf area/leaf mass; i.e., smaller, thicker leaves) capture more PM than do hairless leaves.[112,113] In a few studies, coniferous trees captured more PM than broadleaf trees.[112,114–116] Green spaces did not reduce gaseous air pollutants at the same levels they reduced PM in at least one study,[117] and analyses of urban forests in Florida showed marginal reductions in CO_2 emissions.[109]

IV. WHAT WERE FINDINGS FROM THE UNIVERSITY OF WASHINGTON PUBLIC HEALTH STUDY OF ULTRA-FINE PARTICULATES?

Report Requirement 3: Findings of the University of Washington School of Public Health study

> Proviso language: The findings of the University of Washington School of Public Health school of public health study on ultrafine particulate matter at the airport and surrounding areas

A. SUMMARY

Researchers with University of Washington's Environmental and Occupational Health Sciences Department conducted the first study of UFP concentrations near SeaTac airport during 2018–19.[118] They observed amounts of UFP, CO_2, and black carbon 10 miles south and north of the airport using fixed and mobile monitors in 2019. They found that both air and ground traffic increased UFP concentrations, though the size distributions of UFP and concentrations of black carbon differed by source. Monitors below air traffic flight paths showed higher concentrations of ultra UFP (10–20 nanometers), with the highest concentrations associated with landings compared to departures. Monitors near I-5 and SR 99 South showed higher concentrations of larger UFP and black carbon. The fixed site monitors showed higher concentrations of black carbon and UFP in fall and winter compared to spring and summer months. Findings support the conclusion that communities underneath and downwind of the flight path are exposed to aircraft-related UFP concentrations.

B. EXCERPT FROM EXECUTIVE SUMMARY OF MOBILE OBSERVATIONS OF ULTRAFINE PARTICLES STUDY REPORT[12]

The Mobile ObserVations of Ultrafine Particles Study (MOV-UP) is a two-year project funded by Washington State to analyze potential air quality impacts of ultrafine particles from aircraft traffic for communities near and underneath Seattle-Tacoma International Airport (SeaTac) flight paths. The study assessed ultrafine particle concentrations (UFPs) within 10 miles of the airport in the directions of

aircraft flight. The University of Washington research team that led the study designed the project to investigate the implications of aircraft traffic at SeaTac by (1) assessing the concentrations of UFPs in areas surrounding and directly impacted by aircraft traffic; (2) distinguishing and comparing UFP concentrations attributable to aircraft-related and other sources; and (3) coordinating with local governments, and sharing results and soliciting feedback from community stakeholders. Over the course of four seasons, we conducted both fixed-site and mobile sampling schemes to collect time-resolved measures of UFP, carbon dioxide (CO_2), and black carbon (BC) concentrations, and UFP size distributions.

This study primarily found that UFPs derive from both roadway traffic and aircraft sources, with the highest UFP counts found nearest major roadways (Interstate 5). Total concentrations of UFP alone (10–1,000 nm) did not distinguish roadway and aircraft features.

However, key differences exist in the particle size distribution and the black carbon concentration for roadway and aircraft features. These differences can help distinguish between the spatial impact of roadway traffic and aircraft UFP emissions using a combination of mobile monitoring and standard statistical methods.

Fixed-site monitoring confirms that aircraft landing activity is associated with a large fraction of particles in the range of 10–20 nm (ultra UFP). Mobile-derived fuel-based emissions factors (# ultra UFP/kgFuel) are consistent with differences in emissions between aircraft and roadway vehicles. The MOV-UP study findings demonstrate two clear and consistent spatial features of ultrafine particles independently associated with vehicle traffic and aircraft emissions.

V. RECOMMENDATIONS TO ADDRESS HEALTH ISSUES

Report requirement 4: Recommendations to address health issues

Any recommendations to address health issues related to the impact of the airport on the community

To address the health disparities of airport communities, we recommend the following:

- Implement focused efforts to address the health disparities of airport communities, including mitigating the health impacts of airport operations
    - Ensure participatory community engagement (particularly from Black/African American, Hispanic/Latino, and other communities of color who are disproportionately impacted) in plans and efforts to address the health of airport communities.
    - Increase culturally and linguistically appropriate prevention and disease management efforts in airport communities to address health disparities with emphasis on:
        - Preventing and treating chronic conditions (such as cancer, heart disease, diabetes, and chronic lower respiratory diseases) and unintentional and intentional injuries.
        - Ensuring healthy births, including improving access to culturally and linguistically appropriate prenatal and postnatal care.
    - Ensure that children and young adults are thriving by providing quality education and equitable opportunities for learning.
    - Increase awareness of the risks associated with airport-related air and noise pollution and the ways to prevent or mitigate the risks (e.g., appropriately sealed and ventilated residences and buildings).

- Continue development and implementation of strategies to mitigate airport-related air and noise pollution. These include:
    - Working with community residents and residential property owners to support healthy housing conditions (e.g., adequate ventilation and effective sealing of residences) through mitigation packages.
    - Ensuring a safe physical environment of childcare settings, schools, community centers, and long-term care facilities to protect vulnerable populations (e.g., adequate ventilation and effective sealing of buildings).
    - Continuing to improve airport operations to reduce noise and emissions on neighboring communities through technological and operational controls and through voluntary or regulatory policies aimed at reducing airport-related emissions.
    - Promoting healthy habitats by creating green spaces, specifically coniferous tree coverage, to capture particulate matter, thereby reducing people's exposure.
- Expand the systematic monitoring of pollutants (both outdoor and indoor exposures), including the implementation of new technologies to improve measurement of exposures indoors and outdoors. Understanding how pollutants penetrate not only residences but also schools, childcare, and long-term health facilities is critical to reducing people's exposure.
    - To better understand noise pollution, systematically measure the maximum or peak noise levels resulting from airport operations and the occurrence with which noise levels are above health-based standards.
    - Increase the number of fixed and mobile air quality measurements to create a network of regular monitoring to understand the spatiotemporal distribution of air pollution in the environment.[119]
    - Measure noise and air pollution in the communities proximal to the airport, since regional monitoring data can be lower than levels experienced closer to airport operations.
- Support further research to address gaps in knowledge, especially concerning levels of pollutant exposure resulting from airport operations, indoors and outside, and the precise mechanisms and degree of harm. Areas of future research include, but are not limited to:
    - The extent to which outside pollutants infiltrate indoor settings, i.e., residences, childcare settings, community centers, and long-term care facilities;
    - The impact of sound exposure levels on human health and quality of life, especially child and adult cognition, and the pathways through which noise pollution affects health; and
    - The mechanisms and nature of ultrafine particle (UFP) effects on human health.

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Appendices

Appendix A: Community Health Profile Methods
Appendix B: Community Health Profile Additional Data
Appendix C: Strength-of-Evidence Analysis Methods and Annotations
Appendix D: Strength-of-Evidence Pathway Trees for Noise Pollution and Air Pollutants Common to Airport Operations

Appendix A. Community Health Profile Methods

Appendix A. Community Health Profile Methods

How Zones Were Defined for Analysis

The airport communities included Zone A (less than 1 mile from the Seattle-Tacoma International Airport [SeaTac]), Zone B (1 to less than 5 miles from the airport) and Zone C (5 to 10 miles from the airport), to meet the requirements of the proviso. Balance of County, the area within King County more than 10 miles from the airport, is the non-overlapping comparison area. The zones overlap the census tract areas with the greatest average number of flights per day based on 2018 SeaTac flight data (Figure A1). Appendix B provides King County average rates and confidence intervals.

Datasets consisted of individuals' health events (e.g., birth risk factors, deaths) or survey responses (e.g., Behavioral Risk Factor Surveillance survey). Depending on the data source, either census tract or ZIP code of residence of the individual was usually the smallest geographic unit available. For these datasets, a census tract or ZIP codes was considered in a Zone if 50% or more of its area lay in the Zone. Accordingly, King County tracts or ZIPs that lay outside 10-mile radius—that is, outside of the outer boundary of Zone C—were assigned to Balance of County. Census tract and ZIP code overlay with Zone were calculated using geocoding packages in R (see Table A2 for zip codes by zone).[1–5]

We did not have residential information from students who took part in the Healthy Youth Survey (HYS) or for students in datasets from the Office of the Superintendent of Public Instruction (OSPI). For these sources, data was available by school building. To create Zone aggregates:

1. A file with the latitude and longitude of each King County public school was downloaded from the Washington Geospatial Open Data Portal.[6]
2. The latitude and longitude of each school were assigned to the appropriate Zone or Balance of County using geocoding software.
3. All HYS respondents or OSPI table counts from the school were assigned to the Zone in which the building was located.

Unstable Rates

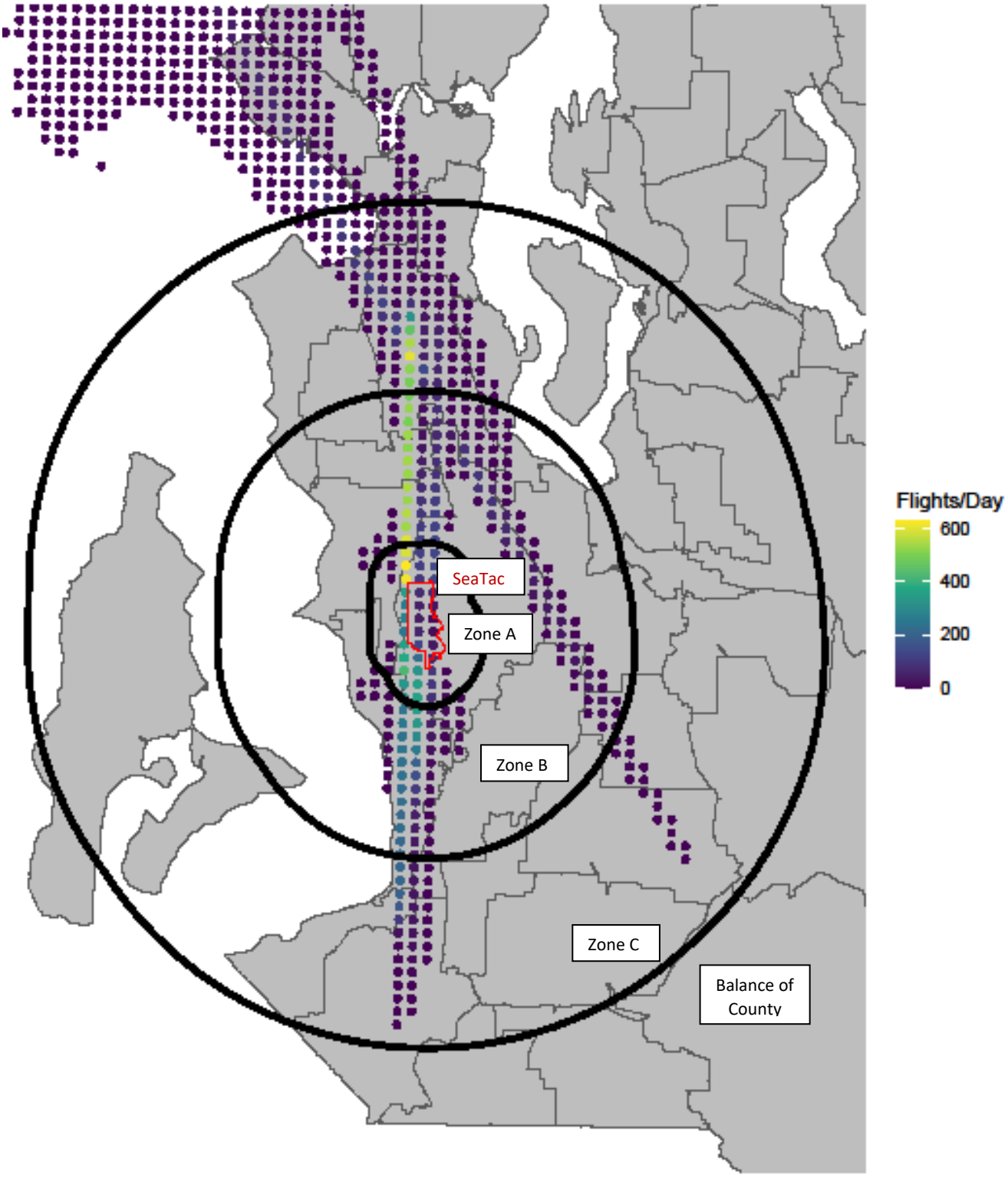
We flagged rates as unstable in data tables if the relative standard error—the rate standard error divided by the rate—was >30%. This is the guideline followed by the Public Health – Seattle & King County's (PHSKC) Assessment, Policy Development and Evaluation unit.

Data Suppression for Confidentiality

Use of some datasets in the report was governed by data sharing agreements that required the user to suppress rates to protect confidentiality. When data was suppressed, the table cell was noted with the "^^" symbol. The details of cell suppression requirements are as follows:

- The HYS data sharing agreement states: "Generally, if there are at least 3 schools and 3 school districts at a geographical level for which data are being reported, the schools and school districts are not identifiable."[7] Zone A did not meet this requirement; thus results for Zone A were suppressed for HYS indicators.
- The Washington State Department of Health (DOH) small numbers standard requires that rates and other information based on numerators between 1 and 9 be suppressed for certain population data.[8] This applied to measures derived from datasets for births, deaths, and hospitalizations.
- Hospitalization data included hospitalizations of Washington residents in Oregon for complete counts. Oregon data suppression rules require data to be suppressed if numerators are less than 10.

Figure A1
SeaTac Average Flights per Day in 2018 and Community Health Profile Zones



Leading Causes of Death

We identified leading causes by ranking cause of death for each of seven race groups in King County. We included a cause if it was among the 10 leading causes for any race. In the Leading Causes of Death table, the causes are listed in the order of their rank in King County as a whole. For instance, after all cause mortality, cancer was listed next because it has the most deaths of any specific cause of death in King County. Leading causes of death by race/ethnicity for King County can be found in PHSKC's Community Health Indicators report online.[9]

Excess Deaths

We adapted our method from a CDC report,[10] though we made some changes to work with local data. As in the CDC report, deaths in people age 80 and older were omitted from all calculations. We used 79 as the upper age boundary for potentially preventable deaths, since it is close to the King County average life expectancy (81.7 years, 2014–18 average).

We calculated age-standardized mortality ratios (SMRs) and observed and expected deaths using established methods.[11,12] To standardize by age, for each cause of death, we calculated observed and expected deaths for eight 10-year age groups (0–9, 10–19, … 70–79) for the Zones and Balance of County. We summed observed and expected deaths over age groups to arrive at the total observed and expected counts. The SMR was the total observed divided by the total expected.

If the lower limit of the SMR confidence interval (CI) was greater than 1, the SMR and excess deaths were significantly elevated. The choice of CI calculation method depended on the number of observed deaths. If there were fewer than 100 deaths, we calculated a CI for observed deaths using the Poisson distribution. The SMR's lower CI was the lower CI of the observed number divided by expected. If there were 100 or more deaths, we calculated a CI for the SMR following DOH assessment guidelines.[13] A negative number of excess deaths was set to 0.

Data Analysis

Data management, analysis, and table production were conducted with R.[1,4,14] DOH's Community Health Assessment Tool (CHAT) was used to provide grouped geographic units for Zone calculations, and provided counts, populations, rates, standard errors, and confidence intervals used in calculations for births, deaths, and hospitalizations.

Hospitalization and death rates were age-adjusted (except where noted) per 100,000 population. Rates for birth risk factors, educational attainment, high school graduation, poverty and foreign born, and measures derived from the Healthy Youth Survey and the Behavioral Risk Factor Surveillance survey were given in percentages and not age-adjusted. All counts were average yearly counts. For counts from surveys, we rounded to the nearest hundred to account for survey error. Otherwise, average yearly counts were rounded to the nearest integer. For some measures, counts could not be accurately calculated and were noted as "N/A."

In measures from the Healthy Youth Survey, to assess the potential confounding effect of grade in comparisons between a zone and Balance of County, we stratified by grade. Because the stratified comparisons were consistent with unstratified comparisons, the unstratified rates and comparisons are presented in the profiles.

Differences were considered statistically significant when 95% confidence intervals for rates did not overlap. We grouped data from 2014 to 2018 together to stabilize rates if these years were available. Otherwise, we grouped other recently available years (Table A1).

Table A1 Measures by Data Source and Years of Analysis		
Measure	**Source**	**Years analyzed**
Population estimates by age group	Washington State Office of Financial Management, Forecasting Division, single year intercensal estimates 2001–19, Community Health Assessment Tool (CHAT), March 2020	2014–18
Population estimates by race		
Near poverty and poverty	American Community Survey, https://www.census.gov/programs-surveys/acs/, Table B17024	2014–18
Adult educational attainment	American Community Survey, https://www.census.gov/programs-surveys/acs/, Table B15003	2014–18
Foreign born	American Community Survey, https://www.census.gov/programs-surveys/acs/, Table B05002	2014–18
Uninsured (age 19–64)	American Community Survey, https://www.census.gov/programs-surveys/acs/, Table B27001	2014–18
Met 3rd grade reading standard	Office of the Superintendent of Public Instruction public use tables, https://data.wa.gov/Education/Report-Card-Assessment-Data-2018-19-School-Year/5y3z-mgxd, accessed 8/2020.	2018–19
High-school graduation rate		
Adequate prenatalprenatal care	Washington State Department of Health, Center for Health Statistics, Birth Certificate Data, 1990–2018, Community Health Assessment Tool (CHAT), October 2019	2014–18
Premature births		
Low birth weight		
Child overall health status	King County Best Starts for Kids Health Survey	2017, 2019
Child did not receive needed health care		
Children with a caregiver with inadequate sleep		
Dental checkup: none in the last year	Washington State Healthy Youth Survey	2016, 2018
Current cigarette smoking		
Current e-cigarette or vape pen use		
Met physical activity recommendations		
Obese		
Depression (school age)		
Health status fair/poor	Washington State Department of Health, Center for Health Statistics, Behavioral Risk Factor Surveillance System, supported in part by the Centers for Disease Control and Prevention, Cooperative Agreement U58/DP006066-05 (2019)	2014–18
Could not see doctor due to cost		
Current smoker		
High blood pressure		
Obese		
No physical activity in leisure time		
Inadequate sleep		2013, 2014,

Table A1 Measures by Data Source and Years of Analysis		
Measure	**Source**	**Years analyzed**
		2016, 2018
Has asthma now (prevalence)	Washington State Department of Health, Center for Health Statistics, Behavioral Risk Factor Surveillance System, supported in part by the Centers for Disease Control and Prevention, Cooperative Agreement U58/DP006066-05 (2019)	2014–18
Ever had stroke		
Ever had heart attack		
COPD (prevalence)		
Diabetes (prevalence)		
Depression (adults)		
Asthma, child and adult (hospitalization)	Washington Hospital Discharge Data, Comprehensive Hospitalization Abstract Reporting System (CHARS) 1987–2018. Washington State Department of Health, Center for Health Statistics, Community Health Assessment Tool (CHAT), July 2019	2016–18
Cerebrovascular disease (stroke hospitalization)		
COPD (hospitalization)		
Diabetes (hospitalization)		
Diseases of the heart		
Life expectancy	Washington State Department of Health, Center for Health Statistics, Death Certificate Data, 1990–2018, Community Health Assessment Tool (CHAT), October 2019	2014–18
Leading causes of death		
Leading causes of cancer death		
Leading causes of unintentional injury death		
Excess and potentially preventable deaths		

Table A2 Zip Codes by King County Zone				
Zone A	**Zone B**	**Zone C**	**Balance of County**	
98148	98032	98002	98004	98107
98158*	98198	98003	98005	98109
	98055	98001	98007	98112
	98057	98006	98008	98115
	98166	98070	98010	98117
	98188	98030	98014	98119
	98146	98031	98024	98121
	98168	98040	98027	98122
	98178	98056	98029	98125
		98058	98033	98199
		98059	98034	98155
		98104	98038	98177
		98126	98039	98195
		98106	98042	98224
		98108	98045	98288

Table A2 Zip Codes by King County Zone				
Zone A	**Zone B**	**Zone C**	**Balance of County**	
		98116	98051	98011
		98118	98052	98028
		98134	98053	98077
		98136	98065	98072
		98144	98074	98022
		98023	98075	98019
			98101	98047
			98102	98092
			98103	98133
			98105	

* ZIP code 98158 is for SeaTac Airport and has 0 residential population.

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Appendix B. Community Health Profile Additional Data

Appendix B. Community Health Profile Additional Data

Table B1 Population Estimates by Race/Ethnicity and Age Group by Zone, Balance of County and King County, 2014–18 annual average										
Categorical Group	Zone A		Zone B		Zone C		Balance of County		King County	
Total	30319	100%	260163	100%	605546	100%	1207782	100%	2103810	100%
Age										
Under 18	6944	23%	60202	23%	138221	23%	235712	20%	441079	21%
18 to 24	2922	10%	22250	9%	49405	8%	111731	9%	186308	9%
25 to 44	9744	32%	79124	30%	178513	29%	409925	34%	677307	32%
45 to 64	7064	23%	65759	25%	159739	26%	306420	25%	538981	26%
65 and over	3645	12%	32828	13%	79668	13%	143994	12%	260134	12%
Race/Ethnicity										
American Indian/Alaska Native	280	1%	2003	1%	3530	1%	7573	1%	13386	1%
Asian	4095	14%	50289	19%	117565	19%	172659	14%	344609	16%
Black or African American	5064	17%	34887	13%	57406	9%	36574	3%	133930	6%
Hispanic/Latinx	6871	23%	46111	18%	67244	11%	88627	7%	208854	10%
Multiple race	1358	4%	12752	5%	29778	5%	51902	4%	95790	5%
Native Hawaiian/Pacific Islander	1113	4%	4986	2%	6815	1%	4356	0%	17270	1%
White	11538	38%	109134	42%	323208	53%	846092	70%	1289971	61%

Table B2[1]
Measures by Area by Zone, Balance of County, and King County

Indicator	Zone A				Zone B				Zone C				Balance of County			King County		
	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI
Percent in households living at less than 200% of the federal poverty level by age																		
Total population	37.2%	11,500	34.4%	39.9%	31.7%	84,100	30.8%	32.7%	24.4%	148,800	23.8%	25.0%	16.1%	15.8%	16.4%	20.7%	20.5%	21.0%
Under 18 years	51.1%	3,800	44.2%	57.9%	46.7%	27,800	44.1%	49.3%	33.5%	44,200	31.9%	35.2%	14.6%	13.8%	15.4%	25.2%	24.5%	26.0%
Educational attainment, age 25 or older																		
Associate degree or more+	28.1%	6,100	25.7%	30.5%	38.6%	71,700	37.7%	39.5%	51.4%	222,800	50.7%	52.0%	68.7%	68.2%	69.2%	59.6%	59.2%	60.0%
Child educational success																		
High school graduation rate	80.7%	NA	74.4%	85.8%	72.3%	NA	70.3%	74.3%	81.0%	NA	80.1%	81.9%	87.7%	87.1%	88.4%	82.7%	82.2%	83.2%
Met 3rd grade reading standard	43.4%	NA	38.0%	49.0%	39.0%	NA	37.1%	41.0%	54.6%	NA	53.4%	55.8%	73.3%	72.5%	74.1%	62.9%	62.3%	63.6%
Foreign born																		
Foreign born	35.9%	11,500	33.1%	38.7%	27.1%	72,600	26.2%	28.1%	23.9%	147,500	23.3%	24.4%	20.5%	20.1%	20.8%	22.5%	22.3%	22.7%
Birth risk factors																		
Early and adequate prenatal care	63.9%	300	60.7%	67.2%	68.8%	2367	67.6%	70.0%	71.1%	5042	70.2%	71.9%	75.0%	74.4%	75.7%	72.6%	72.1%	73.1%
Low birth weight (singleton)	5.8%	28	4.9%	6.8%	6.1%	217	5.8%	6.5%	5.5%	400	5.3%	5.8%	4.7%	4.5%	4.8%	5.2%	5.1%	5.3%
Premature births (singleton)	9.8%	48	8.6%	11.1%	9.4%	334	9.0%	9.9%	8.2%	593	7.9%	8.5%	6.6%	6.4%	6.8%	7.6%	7.4%	7.7%
Children: Overall health status, access to care, and risk factors																		
Child overall health status excellent or very good	88.4%	NA	82.4%	94.4%	82.9%	NA	79.4%	86.4%	86.5%	NA	84.3%	88.6%	92.1%	90.9%	93.4%	88.9%	87.9%	90.0%
Child did not receive needed health care	6.1%	NA	1.6%	10.6%	6.1%	NA	3.8%	8.5%	3.8%	NA	2.7%	4.8%	2.9%	2.1%	3.6%	3.7%	3.1%	4.3%
No dental checkup in last year	^^	^^	^^	^^	19.2%	NA	16.8%	21.7%	16.9%	NA	15.6%	18.2%	11.8%	11.1%	12.6%	14.5%	13.7%	15.2%
Current cigarette smoking	^^	^^	^^	^^	5.2%	NA	4.0%	6.8%	5.2%	NA	4.5%	5.9%	4.4%	4.0%	5.0%	4.8%	4.4%	5.2%

[1] ^^: Data suppressed to meet confidentiality standard.
NA: Estimated counts were not available for these measures.

Table B2[1]
Measures by Area by Zone, Balance of County, and King County

Indicator	Zone A				Zone B				Zone C				Balance of County			King County		
	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI
Current e-cigarette or vape pen use	^^	^^	^^	^^	11.3%	NA	9.1%	13.9%	12.8%	NA	11.5%	14.2%	14.4%	13.0%	16.0%	13.5%	12.6%	14.5%
Obese	^^	^^	^^	^^	15.2%	NA	13.3%	17.2%	12.2%	NA	11.0%	13.5%	7.5%	7.0%	8.1%	10.1%	9.4%	10.8%
Met physical activity recommendations	^^	^^	^^	^^	18.2%	NA	16.3%	20.2%	19.6%	NA	18.4%	20.7%	21.3%	20.4%	22.2%	20.3%	19.6%	20.9%
Adults: Overall health status, access to care, and risk factors																		
Health status fair/poor	25.5%	6000	13.7%	42.4%	15.7%	31500	13.4%	18.3%	14.9%	69500	13.4%	16.5%	10.0%	9.1%	10.9%	12.0%	11.3%	12.7%
Uninsured (age 19–64)	14.3%	2800	12.3%	16.4%	13.8%	23200	13.0%	14.6%	5.8%	48400	5.6%	6.0%	7.8%	7.6%	7.9%	7.8%	7.6%	7.9%
Could not see doctor due to cost	26.3%	6100	14.3%	43.3%	14.1%	28200	11.8%	16.8%	11.3%	53000	10.0%	12.9%	9.8%	8.9%	10.7%	10.8%	10.1%	11.6%
Current smoker (adult)	8.2%	1900	2.6%	23.4%	17.7%	35400	15.0%	20.8%	12.1%	56700	10.7%	13.7%	9.5%	8.6%	10.5%	11.1%	10.4%	11.9%
High blood pressure	27.3%	6400	13.5%	47.3%	29.3%	58500	26.0%	32.7%	28.3%	132300	26.3%	30.5%	23.6%	22.3%	24.9%	25.4%	24.4%	26.5%
Obese	44.3%	10300	28.8%	61.0%	29.2%	58300	26.0%	32.5%	24.4%	113900	22.6%	26.2%	19.1%	18.0%	20.2%	21.5%	20.6%	22.5%
No physical activity in leisure time	31.7%	7400	18.4%	48.9%	20.5%	41000	17.7%	23.6%	17.5%	81600	15.7%	19.3%	12.1%	11.2%	13.1%	14.6%	13.7%	15.4%
Inadequate sleep	33.5%	7800	18.9%	52.1%	34.5%	69000	30.7%	38.6%	35.0%	163700	32.5%	37.6%	27.0%	25.5%	28.6%	30.1%	28.9%	31.3%
Child with a caregiver with inadequate sleep	28.9%	1600	15.1%	42.7%	35.1%	12700	29.6%	40.5%	30.1%	22900	26.5%	33.6%	27.3%	24.2%	30.5%	29.3%	27.1%	31.5%
Chronic conditions: prevalence, and hospitalization																		
Prevalence																		
Has asthma now	5.6%	1300	1.8%	16.1%	8.2%	16400	6.5%	10.3%	10.0%	46600	8.7%	11.4%	8.3%	7.6%	9.2%	8.7%	8.1%	9.4%
Ever had stroke	2.8%	600	0.6%	11.3%	3.4%	6900	2.3%	5.2%	3.1%	14600	2.4%	4.1%	1.9%	1.6%	2.3%	2.4%	2.0%	2.7%
Ever had heart attack	2.7%	600	0.9%	7.8%	3.6%	7100	2.5%	5.0%	3.4%	16000	2.7%	4.4%	2.5%	2.2%	3.0%	2.9%	2.5%	3.3%
Chronic obstructive pulmonary disease	2.9%	700	0.7%	11.6%	4.8%	9600	3.7%	6.3%	4.1%	19300	3.3%	5.1%	3.3%	2.9%	3.9%	3.7%	3.3%	4.1%

Table B2[1]
Measures by Area by Zone, Balance of County, and King County

	Zone A				Zone B				Zone C				Balance of County			King County		
Indicator	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI
Diabetes	12%	2800	6%	22%	10%	19800	8%	12%	8%	38200	7%	9%	6%	6%	7%	7%	7%	8%
Depression (school-age)	^^	^^	^^	^^	34.4%	NA	32.8%	36.1%	33.5%	NA	32.2%	34.9%	29.4%	28.4%	30.4%	31.4%	30.6%	32.1%
Depression (adults)	11.3%	2600	5.1%	23.3%	20.5%	40900	17.9%	23.3%	22.2%	103600	20.4%	24.0%	21.5%	20.4%	22.8%	21.3%	20.4%	22.2%
Hospitalization (rate/100,000)																		
Asthma (adult)	^^	^^	^^	^^	28.4	^^	24.16	33.14	28.51	^^	25.9	31.4	16.6	15.1	18.1	21.6	20.4	22.9
Asthma (child)	^^	^^	^^	^^	100.2	^^	85.5	116.6	86.9	^^	78.5	95.9	71.3	65.3	77.9	82.2	77.4	87.2
Cerebrovascular diseases (stroke)	304.8	17	240.5	382.6	270.3	413	258.5	282.5	241.9	981	235.0	248.9	213.0	208.0	218.1	228.1	224.4	232.0
COPD	81.7	5	51.2	125.9	59.4	94	54.1	65.2	68.3	286	64.8	72.1	52.3	49.9	54.8	58.3	56.4	60.2
Diabetes total	179.7	10	133.0	239.5	133.2	202	125.0	141.9	102.5	414	98.0	107.1	70.7	67.9	73.5	NA	NA	NA
Diseases of the heart	876.5	48	764.5	1001.6	600.4	918	582.8	618.4	520.5	2134	510.4	530.6	463.3	456.0	470.8	498.1	492.6	503.7
Life expectancy and leading causes of death																		
Life expectancy (in years)	77.9	77.1	78.6	79.4	79.2	79.7	81.2	81.1	81.4	82.9	82.8	83.0	81.7	81.6	81.8	77.9	77.1	78.6
Cause of death																		
All	808.6	248	763.0	856.5	723.4	1901	708.6	738.3	639.1	3950	630.1	648.3	572.2	566.0	578.5	621.4	616.5	626.3
Cancer	158.7	48	138.8	181.0	160.9	428	154.0	168.0	138.6	878	134.5	142.9	135.3	132.3	138.4	140.6	138.3	143.0
Diseases of heart	187.7	59	166.3	211.5	142.9	374	136.4	149.6	126.6	783	122.6	130.7	115.3	112.6	118.2	124.4	122.3	126.6
Alzheimer's disease	52.2	17	41.3	65.5	38.1	96	34.7	41.7	43.7	259	41.4	46.2	47.8	46.0	49.6	45.6	44.3	47.0
Accidents (unintentional injuries)	45.8	14	35.7	58.3	39.1	106	35.8	42.7	35.7	222	33.6	37.9	30.4	29.0	31.9	34.9	33.7	36.0
Cerebrovascular diseases (stroke)	32.7	11	24.5	43.3	34.9	90	31.7	38.3	34.5	209	32.5	36.7	28.8	27.4	30.3	31.6	30.5	32.7
Chronic lower respiratory disease	38.5	10	28.4	51.1	34.9	90	31.7	38.4	27.6	168	25.8	29.6	22.5	21.2	23.8	25.9	24.9	27.0
Diabetes mellitus	33.9	10	25.2	45.2	26.3	70	23.5	29.2	22.0	137	20.4	23.8	14.4	13.4	15.5	18.7	17.8	19.5
Intentional self-harm (suicide)	9.8	3	5.4	16.9	13.1	35	11.2	15.2	11.8	75	10.6	13.1	11.8	10.9	12.7	12.1	11.5	12.8

Table B2[1]
Measures by Area by Zone, Balance of County, and King County

Indicator	Zone A				Zone B				Zone C				Balance of County			King County		
	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Number	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI	Rate	Lower 95% CI	Upper 95% CI
Chronic liver disease and cirrhosis	18.8	6	12.5	27.7	13.1	37	11.2	15.2	10.2	69	9.2	11.4	7.9	7.2	8.6	9.6	9.0	10.2
Influenza and pneumonia	14.3	4	8.8	22.3	13.5	35	11.5	15.7	10.0	61	8.9	11.3	8.6	7.9	9.5	9.9	9.3	10.6
Essential hypertension and hypertensive renal disease	^^	^^	^^	^^	8.7	22	7.1	10.5	6.6	41	5.7	7.6	4.6	4.0	5.2	5.8	5.3	6.2
Nephritis, nephrotic syndrome, and nephrosis	7.0	2	3.4	13.3	9.1	24	7.5	11.0	7.0	42	6.0	8.0	4.1	3.6	4.7	5.7	5.3	6.2
Septicemia	6.7	2	3.2	12.8	7.9	20	6.4	9.6	4.0	24	3.3	4.8	1.2	0.9	1.5	3.0	2.7	3.3
Assault (homicide)	^^	^^	^^	^^	4.0	11	3.0	5.3	3.2	17	2.5	3.9	1.8	1.5	2.2	2.6	2.3	3.0
Certain conditions originating in the perinatal period	^^	^^	^^	^^	8.7	22	7.1	10.5	6.6	41	5.7	7.6	4.6	4.0	5.2	5.8	5.3	6.2
Leading causes of cancer death																		
Lung cancer	36.1	11	26.8	48.0	36.5	96	33.2	40.0	29.9	188	28.0	31.9	27.7	26.3	29.2	29.8	28.7	30.9
Colorectal cancer	10.8	3	5.9	18.4	14.8	40	12.8	17.1	11.0	70	9.8	12.2	11.2	10.4	12.1	11.6	11.0	12.3
Breast cancer (female)	20.5	3	11.5	34.3	20.0	28	16.8	23.7	19.4	68	17.3	21.6	18.1	16.6	19.6	18.8	17.7	20.0
Leading causes of unintentional injury death																		
Poisoning	20.1	6	13.6	29.0	14.8	41	12.8	17.0	12.4	80	11.2	13.7	10.2	9.5	11.1	12.5	11.8	13.1
Falls	8.5	3	4.6	14.9	10.4	27	8.7	12.4	11.1	68	9.9	12.4	11.1	10.2	12.0	11.1	10.5	11.8
Motor Vehicle-Traffic	8.4	3	4.5	15.0	7.1	19	5.7	8.7	5.5	34	4.7	6.5	4.2	3.7	4.8	5.2	4.8	5.7

Table B3
Potentially preventable deaths in airport communities

Cause of Death by Zone	*Observed**			Expected*	Excess*	*SMR*		
	Observed	Lower 95% CI	Upper 95% CI			SMR	Lower 95% CI	Upper 95% CI
Zone A								
Cancer	175	150.0	202.9	126.0	49.0	1.4	1.2	1.6
Diseases of heart	140	117.8	165.2	60.6	79.4	2.3	1.9	2.7
Alzheimer's disease	9	4.1	17.1	7.0	2.0	1.3	0.6	2.5
Accidents (Unintentional injuries)	60	45.8	77.2	33.2	26.8	1.8	1.4	2.3
Cerebrovascular diseases (stroke)	23	14.6	34.5	12.0	11.0	1.9	1.2	2.9
Zone B								
Cancer	1494	1419.2	1571.7	1168.8	325.2	1.3	1.2	1.3
Diseases of heart	907	848.9	968.0	563.5	343.5	1.6	1.5	1.7
Alzheimer's disease	55	41.4	71.6	65.2	-10.2	0.8	0.6	1.1
Accidents (Unintentional injuries)	409	370.3	450.6	291.2	117.8	1.4	1.3	1.5
Cerebrovascular diseases (stroke)	199	172.3	228.7	111.3	87.7	1.8	1.5	2.1
Zone C								
Cancer	3000	2893.6	3109.3	2845.9	154.1	1.1	1.0	1.1
Diseases of heart	1797	1714.9	1882.1	1374.4	422.6	1.3	1.2	1.4
Alzheimer's disease	159	135.2	185.7	160.0	-1.0	1	0.8	1.2
Accidents (Unintentional injuries)	849	792.8	908.1	687.6	161.4	1.2	1.2	1.3
Cerebrovascular diseases (stroke)	381	343.7	421.2	271.4	109.6	1.4	1.3	1.6

*Observed, expected and excess deaths are totaled for the 5-year period so the reader can see how significance method was chosen (see Appendix A for details).

Appendix C. Strength-of-Evidence Analysis Methods and Annotations

Appendix C. Strength-of-Evidence Analysis Methods and Annotations

Strength-of-evidence ratings presented in this report align with the five-level causal framework that the EPA uses in the Integrated Science Assessment (ISA) reports developed to assess air pollutant effects on health.[1] The scale summarizes how strongly the scientific evidence supports the claim that an exposure like noise pollution can cause a given set of health outcomes such as cardiac effects. The outcomes under consideration are grouped into categories such as "cardiovascular outcomes" or "birth outcomes" to help summarize the vast number of outcomes that have been studied. These reflect categories used in the systematic reviews and ISAs reviewed for this report. In the case of air pollution exposures, most of the outcome groups are further separated according to short- or long-term exposures. In the ISA documents the EPA makes explicit causal judgements on the five-level scale for the same groups of outcomes we present in this review. We focused on specific outcomes; associations with mortality more generally can be found in ISA reports. A paraphrased summary of the five levels is provided in Table C1; color classifications correspond to strength-of-evidence pathway trees in Appendix D.

Table C1
Strength-of-Evidence Criteria Applied

Causal	Exposure is shown to lead to effect across multiple high-quality studies by multiple research groups. Plausible alternative explanations for effect have been ruled out. Biological pathways supported by evidence.
Likely Causal	Exposure is shown to lead to effect across multiple high-quality studies, but uncertainties remain. Plausible alternative explanations for effect have been ruled out, but concerns about possible co-pollutant exposures may remain. Biological pathways, while plausible, may be missing evidence.
Suggestive	Exposure seems to lead to effect in at least one high-quality study, but alternative explanations for the effect, such as a third factor, cannot yet be ruled out. May have limited number of studies with modest effects or may have several studies with conflicting results.
Inadequate Evidence	It is unclear if exposure leads to effect or not, because of a lack of quality studies or because of significant inconsistency in results across studies.
Not Likely Causal	Exposure consistently does not appear to lead to effect across studies. Have adequate studies that show no effect across different exposure levels and different populations.

In these criteria, the exposure levels considered are those that would be plausible based on recent monitored levels in community settings. High-quality studies are studies that are peer-reviewed and account for potential causes of bias or flaws in design, measurement, or analysis that could impact results, thus identifying an effect while ruling out possible alternative causes.

A. Noise Pollution Strength-of-Evidence Findings

Annoyance is a pathway because it can amplify the effects of noise pollution on health, as well as a health outcome because annoyance impacts quality of life and mental health. Not everyone will experience annoyance at the same level of noise, but annoyance can add to the body's stress response and increase the disruption that noise causes in daily activities like sleeping, often leading to larger health effects for people who experience more annoyance. Likewise, school performance is discussed as a health outcome because it impacts quality of life, mental health, and lifelong health trajectories for children.

The strength-of-evidence determinations below are based primarily on systematic reviews of scientific studies. Several studies examined aircraft noise specifically, and the strength-of-evidence analysis is based on studies of traffic noise and general environmental noise as well. Support for noise pollution's

relationship to cardiovascular problems and poorer school performance is reviewed in the body of the report. Following are additional details regarding linkages to annoyance, sleep disturbance, birth outcomes, metabolic system, and nervous system concerns.

Noise pollution exposure CAUSES sleep disturbance.[2–4] Studies of self-reported sleep disruption and use of sedative medications confirm that noise pollution disrupts sleep.[4] Numerous studies show an association between chronic sleep disturbance and long-term health concerns, including obesity, diabetes, and hypertension, though more research is needed to understand the specific pathways and mechanisms.[5] Some studies have questioned whether noise-induced disruptions merely replace natural sleep disruptions, leaving the effects of these disturbances on health unclear. In the short term, however, sleep disturbance can impact productivity, stress levels, and immune function.[6] Sleep can also be disrupted by noise pollution in ways that impact quality of sleep without causing waking.[2,6] Sleep disturbances also contribute to annoyance and chronic stress and may impact metabolic functions.[2,7]

Noise pollution exposure CAUSES annoyance.[2,8] Some populations are more likely to experience noise-related annoyance. Annoyance can amplify the stress response caused by noise, leading to stronger effects on health. Annoyance can also be disruptive for mental health and social relationships.

Evidence **SUGGESTS** that long-term noise pollution exposures may have metabolic and neurological effects. Researchers have observed higher rates of diabetes and other metabolic outcomes in communities exposed to higher noise pollution, controlling for other factors,[2,9,10] though evidence is limited to a small number of studies. Researchers hypothesize that the effects of noise pollution on stress response and sleep disturbance can impact metabolic functions and insulin sensitivity.[2,11,12] Similarly, noise pollution may contribute to poorer neurological outcomes because of sleep disturbance and stress response.[2,11,13] Researchers have examined the effects of noise pollution-induced sleep disturbance on child mental and emotional health, hyperactivity, and conduct issues at school.[2,14,15] Other studies have examined adult anxiety and depression symptoms and cognitive scores.[16] However, air pollution has not been adequately controlled for in many studies, and some results have been inconsistent.

Evidence is **INADEQUATE** for long-term noise pollution exposure and concerning birth outcomes. Some studies of occupational noise exposures have shown higher risk for low birth weight and preterm births, but there have been few studies on residential noise pollution exposures and pregnancy outcomes.[17] It is hypothesized that the stress response induced by chronic noise pollution exposures contributes to inflammation and oxidative stress, which can have small effects on fetal growth and potential for earlier delivery.[18,19] A 2019 analysis of several studies found a small effect on birth weight, but did not find evidence of effects on other birth outcomes.[20] Recent analyses of a London birth cohort found a higher risk of preterm birth with noise levels over 65 dB when air pollutants were included in the model, but not independently.[21] More rigorous studies are needed to better understand effects on reproduction and birth outcomes.

B. Air Pollution Strength-of-Evidence Findings

1. Particulate Matter

Particulate matter (PM) from combustion sources is a mixture of tiny solid and liquid particles. Most are less than 1 micrometer in diameter. They contain varying amounts of nitrogen oxides, elemental carbon, and many different hydrocarbons and more complex substances—including formaldehyde, acrolein, polycyclic aromatic hydrocarbons, and similar chemicals, as well as a variety of metals. Particle size influences how the particles move through the air—smaller particles can remain in the air longer and travel farther. Particle size also influences how the particles move through the human body. Particulate matter includes many compounds, and is defined based on the size of the particles:

- $PM_{10–2.5}$ refers to particles with diameters between 2.5 and 10 µm (considered "coarse" particles).
- $PM_{2.5}$ refers to particles with diameters less than 2.5µm (including both ultrafine and fine particles). These particles are small enough to find their way deep into the lungs and move into other systems of the body.
- Ultrafine PM (UFPM) includes only the smallest particles (diameters less than 0.1µm or micrometer). Like $PM_{2.5,}$ UFPM can penetrate cells and thus penetrate bodily systems. These particles are small enough to find their way deep into the lungs and move into other systems of the body.

The strength-of-evidence determinations for coarse, fine, and ultrafine PM are based on the 2019 ISA on Particulate Matter from the EPA, expert review letters that preceded finalization of the 2019 PM ISA,[18,19,22] and systematic reviews and technical reports published since 2010. The following information is from the 2019 PM ISA, unless citations indicate different or additional sources.

2. Coarse Particulate Matter

Evidence **SUGGESTS** that short-term coarse PM exposure may lead to respiratory problems.[23] Days of higher coarse PM exposure are associated with more asthma attacks. Evidence is mixed for exacerbation of COPD symptoms. Coarse PM exposures may increase susceptibility to respiratory infections, and people with underlying respiratory illness may be more likely to experience respiratory death on days following high coarse PM levels.

Evidence **SUGGESTS** that short-term and long-term coarse PM exposure may lead to cardiovascular problems.[18,19,22] Days of higher coarse PM levels may lead to small increases in blood pressure. People with underlying cardiovascular disease may experience worsened symptoms, changes in heartbeat, and increased risk of blood clots or heart attack. Populations may experience higher rates of cardiac-related death in the days following higher coarse PM exposure. Populations exposed to coarse PM over several years may have higher rates of cardiac-related death. Exposure over several years may lead to higher rates of heart disease, stroke, and pulmonary embolism.[24]

Evidence **SUGGESTS** that long-term coarse PM exposure may lead to metabolic and central nervous system problems and cancer.[25] Populations exposed to coarse PM over several years may have higher rates of type 2 diabetes. Exposure over several years may lead to more issues related to higher blood glucose levels and insulin resistance.[26] Populations exposed to coarse PM over longer periods may have higher rates of anxiety or depression[27] and cognitive decline in adults. Some studies have shown increases in lung cancer in regions of higher exposure and two studies have found higher rates of breast and liver cancers in higher-exposure areas. Exposure may alter gene expression in the brain, which can lead to brain tumors, though only a few animal studies have examined this outcome.[27]

Evidence **SUGGESTS** that coarse PM exposure has an impact on birth outcomes. Exposure is associated with preterm birth[27,28] and lower birth weight, but all other explanations have not been ruled out. There is mixed evidence from a small number of studies for effects on preeclampsia and some birth defects. Exposure could contribute to infant death from respiratory causes, but this has been examined in only two studies.

Evidence is **INADEQUATE** for coarse PM exposure and reproduction and fertility effects, and birth outcomes. Exposure could relate to infertility, endometriosis, and reduced birth rates, but only a couple of studies have examined any of these outcomes. Exposure is associated with preterm birth[21,27] and lower birth weight,[18,19] but all other explanations have not been ruled out. There is mixed evidence from

a small number of studies for effects on preeclampsia and some birth defects. Exposure could contribute to infant death from respiratory causes, but this has been examined in only two studies.

Evidence is **INADEQUATE** for long-term coarse PM exposure and respiratory problems. Exposure over several years may lead to poorer lung function, asthma development, and susceptibility to respiratory infections among children. However, other explanations have not been ruled out.

Evidence is **INADEQUATE** for short-term coarse PM exposure and central nervous system effects. Short-term exposure may trigger stress response, leading to higher levels of stress-related chemicals in the brain, though only a single study has examined this outcome.

3. Fine Particulate Matter

Short- and long-term $PM_{2.5}$ exposures CAUSE cardiovascular and cerebrovascular problems. People with heart disease or hypertension are at higher risk of heart attack, stroke, and cardiac-related death following days of higher $PM_{2.5}$ levels.[28] People without underlying heart disease can experience increases in blood pressure and heart rate variability following days of higher $PM_{2.5}$ levels. Populations exposed to even moderate levels of $PM_{2.5}$ over many years develop higher rates of heart disease and hypertension. These populations experience more heart attacks, strokes, and cardiac-related deaths.

Short- and long-term $PM_{2.5}$ exposures CAUSE respiratory problems. While the 2009 and 2019 ISAs conclude that $PM_{2.5}$ *likely causes* respiratory effects, many experts have concluded the evidence is strong that both short- and long-term exposures *cause* respiratory effects.[18,19] During days of higher $PM_{2.5}$ levels, people with asthma are at higher risk of asthma attack. People with COPD and people with allergies are likely to experience worsened symptoms. People with underlying respiratory illness are more likely to experience respiratory death. Populations without underlying respiratory conditions are at increased risk of contracting respiratory infections on days following higher $PM_{2.5}$ levels. Populations exposed to moderate $PM_{2.5}$ over several years are likely to experience more respiratory infections and have higher rates of respiratory-related death.

Long-term $PM_{2.5}$ exposure LIKELY CAUSES cancer and cancer-related death. Populations exposed to moderate $PM_{2.5}$ over several years have higher rates of lung cancer and lung cancer–related deaths. These populations aren't at higher risk of other types of cancer, but they are less likely to survive other types of cancer.

Long-term $PM_{2.5}$ exposure LIKELY CAUSES nervous system problems. Populations exposed to higher levels of $PM_{2.5}$ over several years have higher rates of dementia and cognitive decline among older adults.[29]

Long-term $PM_{2.5}$ exposure LIKELY CAUSES birth outcome concerns. Long-term $PM_{2.5}$ exposure is related to higher rates of preeclampsia and gestational diabetes among pregnant people, lower birth weights and more preterm births,[21,27] and some birth defects, as well as higher occurrences of fetal death and stillbirth, and infant death (death before first birthday).

Evidence **SUGGESTS** that long-term $PM_{2.5}$ exposure may lead to reproduction and fertility problems. Exposure is shown in some studies to result in negative effects on sperm and eggs, changes in ovulation, and erectile dysfunction in the general population, but existing studies are limited, and for effects on sperm one study found no effect. Exposure to higher levels while trying to conceive has resulted in reduced ability to conceive among people undergoing *in vitro* fertilization.

Evidence **SUGGESTS** that short-term $PM_{2.5}$ exposure may lead to nervous system problems. People with Parkinson's disease may experience an aggravation of symptoms due to days of higher $PM_{2.5}$ levels. People may experience higher levels of cortisol related to stress on days following higher $PM_{2.5}$ levels.

Evidence **SUGGESTS** that short- and long-term $PM_{2.5}$ exposure may lead to metabolic problems. Following days of higher $PM_{2.5}$ levels, populations with diabetes and metabolic disease are likely to experience worsened symptoms and require hospitalizations. On these days, populations without underlying diabetes or metabolic disease are likely to have increases in blood sugar and insulin levels. Populations exposed to moderate levels of $PM_{2.5}$ over several years are likely to develop higher rates of metabolic syndrome and type 2 diabetes and suffer higher rates of metabolic- and diabetes-related deaths.

4. Ultrafine Particulate Matter

Evidence SUGGESTS that long-term UFP exposure may result in nervous system problems. Longer periods of exposure to high UFP concentrations likely lead to stress response and inflammation throughout the brain. People exposed to high UFP levels for long periods can experience neurodegeneration and potentially develop Alzheimer's. Long-term exposure to high UFP may impact cognitive abilities and lead to more impulsive behavior.

Evidence **SUGGESTS** that short-term UFP exposure may lead to respiratory problems. Days of higher UFP levels may lead to poorer lung function and worse symptoms among people with asthma. People with asthma may also experience more respiratory infections. People with COPD may experience worse symptoms. People with underlying respiratory disease may be at higher risk of death on days of higher UFP levels.

Evidence **SUGGESTS** that short-term UFP exposure may lead to cardiovascular problems. Strongest evidence: People with underlying heart disease may experience more heart-rate variability and susceptibility to blood clotting in the days following higher UFP exposure. Other evidence: People with underlying heart disease may experience worse symptoms of heart disease and stroke in days following high UFP exposure. General population may experience increases in blood pressure in days following high UFP exposure.

Evidence **SUGGESTS** that short-term UFP exposure may lead to central nervous system problems. Short-term exposures to higher UFP levels can trigger stress response and inflammation in the brain.

Evidence **SUGGESTS** that long-term UFP exposure may lead to adverse birth outcomes. Two recent population-based cohort studies found UFP exposure was related to preterm births.[27,30] A limited number of studies in the U.S. produced mixed results in associating exposure to higher UFP levels with low birth weight and preterm birth.

Evidence is **INADEQUATE** for long-term UFP exposure and effects on reproduction and fertility. Exposure to higher UFP in utero may contribute to higher testosterone, though this has not been shown to affect sperm count or quality. Evidence is limited to two studies.

Evidence is **INADEQUATE** for long-term UFP exposure and respiratory and cardiovascular problems. Exposure to UFP over several years could impact asthma development and respiratory death, but the evidence is limited to just a couple of studies. Similarly, exposure to UFP over several years could impact heart function and lead to plaque in arteries, but evidence linking long-term UFP exposure and cardiovascular problems is limited to just a couple of studies.

Evidence is **INADEQUATE** for long-term UFP exposure and metabolic problems. Short-term exposure to higher UFP may contribute to an increase in blood sugar, but the evidence is limited to a single study. Long-term exposure to higher UFP may contribute to an increase in fasting blood sugar and average blood sugar, but the evidence is limited to a single study.

Exposure to higher, long-term UFP levels has been shown to lead to genotoxicity and oxidative stress, which can lead to cancer, but only one study has examined cancer as an outcome (breast cancer) and no effect was seen.

5. Ozone

The following causal judgements are based on the EPA's 2019 Ozone ISA and accompanying public comments from former independent review panel members,[31] as well as systematic reviews[32] and technical reports published since 2012. The following information is from the 2019 Ozone ISA,[33] unless citations indicate different or additional sources.

Short-term ozone exposure CAUSES respiratory problems. Multiple studies show that short-term exposure can cause decreased lung function in young, healthy adults and lead to increased susceptibility to respiratory infections and respiratory symptoms in the general population. It can also worsen symptoms of asthma, COPD, and allergies, leading to increased hospitalizations as well as death in people with underlying respiratory issues.

Long-term ozone exposure LIKELY CAUSES respiratory problems. Exposure likely leads to development of asthma in children and increases the severity of asthma. It can lead to more susceptibility to respiratory infection, development of allergies, and development of COPD. Some studies link long-term exposure to respiratory-related deaths, but this evidence is inconsistent.

Short-term ozone exposure LIKELY CAUSES metabolic problems. Studies link exposure to higher blood sugar, higher insulin levels, and other metabolic-related changes.

Evidence **SUGGESTS** that short- and long-term ozone exposure may lead to cardiovascular effects and metabolic problems. Some findings show that ozone exposure leads to impaired heart function, heart rate variability, inflammation, or oxidative stress, and diabetes-related deaths, but results across all relevant studies are inconsistent. Some evidence shows an association with long-term exposure and blood pressure, hypertension, and cardiovascular mortality. To date, little evidence shows a link to heart disease, heart attack, heart failure, or stroke.

Evidence **SUGGESTS** that long-term ozone exposure may lead to fertility and reproductive effects, as well as birth outcomes. A limited number of studies show effects on sperm quality, but alternative explanations have not been ruled out. Exposures in first and second trimesters may lead to lower birth weight and preterm birth,[21] but alternative explanations have not been ruled out.

Evidence **SUGGESTS** that short- and long-term ozone exposure may lead to nervous system effects. Short-term exposure appears to increase depressive symptoms, but evidence is still limited. Stronger evidence exists for long-term exposure effects on cognition. Long-term exposure also could affect depression, neurodegenerative disease, and autism spectrum disorder (ASD), but the evidence is still very limited, especially for ASD.

Evidence is **INADEQUATE** for long-term ozone exposure and cancer. Exposure may contribute to DNA damage, which can lead to cancer, but the evidence is still limited to only a few studies. Some evidence exists for connections with lung cancer, but the populations studied were not representative of general population. Evidence does not point to association with other cancers.

6. Carbon Monoxide

The causal judgements below are based primarily on the EPA's 2010 Carbon Monoxide ISA.[34] Systematic reviews and technical reports published since 2010 were also reviewed for changes in the evidence.

Short-term CO exposure LIKELY CAUSES cardiovascular problems, specifically changes in heart rate due to lower oxygen in the blood.[35] For people with underlying heart disease, exposures can worsen symptoms and trigger irregular heartbeat, which increases the risk of cardiac-related death.

Evidence **SUGGESTS** that short-term CO exposure may lead to respiratory effects. Exposure may worsen symptoms of asthma and COPD in affected populations. Exposure may have small impacts on lung function in the general population.

Evidence **SUGGESTS** that short-term CO exposure may lead to nervous system effects. Exposures causing inflammation in the brain may lead to an increase in depression symptoms.

Evidence **SUGGESTS** that long-term CO exposure may lead to nervous system effects, specifically increased risk of Parkinson's disease and dementia.

Evidence **SUGGESTS** that long-term CO exposure may lead to effects on pregnancy and birth outcomes. Exposures may increase risk of low birth weight, preterm birth, stillbirth, heart-related birth defects, and infant mortality. Exposure during early pregnancy may be connected to development of autism spectrum disorder as well as increased risk of gestational diabetes.

Evidence is **INADEQUATE** for long-term CO exposure effects on respiratory and cardiovascular functioning. Only a few studies examined CO's impact on allergy and asthma development and severity, and alternative explanations were not ruled out when some impact on lung functioning was observed. Similarly, only a few studies have examined CO exposure associations with heart attack and strokes.

7. Nitrogen Dioxide

The following causal judgements are based on the EPA's 2016 oxides of nitrogen ISA,[36] unless cited otherwise. The 2016 ISA did not assess evidence for effects on the central nervous system, and metabolic effects were not assessed separately from cardiovascular effects. Causal determinations for effects on the metabolic and central nervous systems are instead based primarily on systematic reviews and technical reports published since 2015.[31,37–39]

Short-term NO_2 exposure CAUSES respiratory problems. Exposure to NO_2 can worsen asthma symptoms and trigger asthma attacks. Exposure may also contribute to respiratory symptoms and susceptibility to respiratory infections in the general population. Exposure may worsen symptoms for those with allergies or COPD. Exposure may increase risk for respiratory-related death for those with underlying respiratory conditions.

Long-term NO_2 exposure LIKELY CAUSES respiratory problems. Exposure likely increases risk of developing asthma.

Evidence **SUGGESTS** that short- and long-term NO_2 exposure may lead to cardiovascular effects.[35] Short-term exposures may worsen underlying heart disease. This can increase risk for heart attacks and cardiac-related death. Longer-term exposures may contribute to development of heart disease, which can increase risk of heart attacks and cardiac-related death.

Evidence **SUGGESTS** that long-term NO_2 exposure may lead to cancer, specifically lung cancer. Other cancers (brain, breast, cervical, prostate, bladder, and leukemia) may also be associated with exposure, but the evidence is very limited.

Evidence **SUGGESTS** that long-term NO_2 exposure may lead to metabolic effects,[40,41] specifically, increased risk of developing insulin resistance and type 2 diabetes.

Evidence **SUGGESTS** that long-term NO_2 exposure may lead to nervous system effects.[31] Exposure during pregnancy may be associated with autism spectrum disorder.[42] Longer-term exposures may be associated with dementia,[43] Parkinson's disease,[44,45] and cognitive decline.

Evidence **SUGGESTS** that long-term NO_2 exposure may affect pregnancy and birth outcomes. Exposure during early pregnancy may slightly increase risk for gestational diabetes.

Evidence is **INADEQUATE** for short-term NO_2 exposure and metabolic effects;[46] the number of studies is extremely limited.

Evidence is **INADEQUATE** for short-term NO_2 exposure and nervous system effects,[47] though a few studies linked short-term exposures to inflammation in the brain causing depression symptoms.[25]

Evidence is **INADEQUATE** for long-term NO_2 exposure and effects on reproduction and fertility. Exposures may impact sperm count and quality through inflammation and oxidative stress, but the evidence is limited and mixed.

8. Sulfur Oxides

The causal judgments for sulfur oxides are primarily based on the 2017 ISA on sulfur oxides from the EPA[48] unless otherwise noted with a citation.

Short-term SO_x exposure CAUSES respiratory problems. The evidence is strongest for asthma exacerbation. Short-term exposures also may cause decreased lung function in people with underlying respiratory conditions. These impacts increase risk for respiratory death among people with respiratory conditions.

Evidence **SUGGESTS** that long-term SO_x exposure may lead to respiratory problems. Exposure may contribute to asthma development and severity of asthma in children. Exposure may also contribute to allergy development, increased susceptibility to respiratory infections, and respiratory-related death.

Evidence is **INADEQUATE** for SO_x exposure and cancer. A few studies point to potential effects on risk of lung cancer and risk of death among people with bladder cancer. Overall, evidence is inconsistent and major uncertainties remain.

Evidence is **INADEQUATE** for short-term SO_x exposure and cardiovascular effects. Exposures could contribute to aggravated heart disease or heart failure, heart attack risk, and cardiac-related death. Some evidence is mixed and major alternative explanations have not been ruled out.

Evidence is **INADEQUATE** for long-term SO_x exposure and cardiovascular effects. Long-term exposures could contribute to risk of heart disease, heart attack, stroke, and cardiac-related death, but evidence has been inconsistent.

Evidence is **INADEQUATE** for SO_x exposure and nervous system effects.[47] Short- and long-term exposures may have contributed to depression symptoms in a few studies, but the evidence is very limited.[25]

Evidence is **INADEQUATE** for SO_x exposure and pregnancy and birth outcomes. There is some evidence that exposure may increase risk of preterm birth, but major uncertainties remain. Exposure may contribute to lower birth weights, gestational diabetes, pregnancy loss/fetal death, birth defects, and infant death, but evidence is inconsistent and alternative explanations have not been ruled out.

Evidence is **INADEQUATE** for SO_x exposure and reproduction and fertility effects. A few studies point to potential effects on sperm quality and reduced conception, but major uncertainties remain.

9. Hazardous Air Pollutants

Hazardous air pollutants (HAPs) include 187 air pollutants that the EPA knows or suspects to have serious health effects such as cancer or birth defects. The EPA regulates HAPs by placing emissions standards on equipment (like vehicle engines) rather than monitoring air quality as is done for criteria air pollutants. The greatest sources of HAPs related to airport activities are idling and taxiing aircraft.[49] Road vehicles, ground support equipment, and stationary equipment like generators and AC units also contribute to airport HAPs. The Airport Cooperative Research Program (ACRP) has concluded that the most important HAPs related to airport activities are:[50]

- formaldehyde;
- acrolein;
- 1,3-butadiene;
- naphthalene;
- benzene;
- acetaldehyde; and
- ethylbenzene.

Several other HAPs and potential HAPs are emitted at airports and can have health effects, but the ACRP list prioritizes HAPs that have the highest emissions and are most likely to lead to serious health effects.[50]

Airports report quantities of volatile organic compound (VOC) emissions because VOCs can react in the air to form ozone. Many VOCs are also HAPs because they can affect human health even if they don't form ozone. Aircraft emissions contain some of the most toxic HAPs—formaldehyde, acrolein, and 1,3-butadiene—compared to other airport-related emission sources.[50] One national study estimated that from 2005 to 2006, before the addition of the third runway, SeaTac aircraft VOC emissions during landing and takeoff made up approximately 0.28% of all VOC emissions in the Seattle-Tacoma region.[51]

The impact of airport HAPs emissions on air quality in nearby neighborhoods remains unclear and seems to vary between airports. A study of O'Hare International Airport in Chicago measured higher formaldehyde and acetaldehyde levels at sites near the airport compared to sites in other areas of the city and came to the same conclusion.[52] The ACRP notes that although HAP concentrations return to background levels very quickly in areas around airports, airport activities still contribute to regional ambient concentrations.[50] A 2008 study in Rhode Island found elevated HAP concentrations in areas nearest T. F. Green Airport.[53]

Table C2 summarizes the health effects of the seven HAPs of most concern related to airport activity. They are ordered by how important ACRP concluded they were in relation to airport activities and community health. The health effects listed in the table are based on reviews by EPA and the Centers for Disease Control and Prevention (CDC), along with other systematic reviews of ambient HAP exposures.[54–66] Much of the evidence is lab studies of exposed animals and some epidemiological studies of effects in workers who were chronically exposed to HAPs in their occupation. Formaldehyde has the largest evidence base, which includes many epidemiological studies of observed effects in humans. We did not find major studies of health effects from HAP exposures for communities near airports to help inform causal judgements.

Table C2 Health Effects of Hazardous Air Pollutants Most Prevalent in Airport Operations				
HAP	EPA Classification	WHO Classification	Cancer Effects	Non-Cancer Effects
Formaldehyde	Probable carcinogen	Known carcinogen	Sinonasal and nasopharyngeal cancers, leukemia, possibly lung cancer	Short-term: respiratory and eye irritation Long-term: possible effects on white blood cell counts
Acrolein	Not classifiable	Not classifiable	No or inadequate information–	Short-term: respiratory and eye irritation
1,3-Butadiene	Known carcinogen	Known carcinogen	Leukemia and lymphatic cancers	Short-term: respiratory and eye irritation Long-term: possible cardiovascular diseases, blood disorders
Naphthalene	Probable carcinogen	Probable carcinogen	Lung and laryngeal cancers	Long-term: possible anemia, liver damage, brain damage, cataracts
Benzene	Known carcinogen	Known carcinogen	Leukemia	Short-term: drowsiness, dizziness, rapid heart rate, headaches, etc. Long-term: blood disorders, reproductive effects, damage to immune system
Acetaldehyde	Probable carcinogen	Known carcinogen	Nasal & laryngeal tumors in animal studies	Short-term: respiratory irritation Long-term: possible developmental effects
Ethylbenzene	Not classifiable	Possible carcinogen	No or inadequate information	Short-term: respiratory and eye irritation, dizziness at higher doses Long-term: possible hearing and kidney effects

The EPA and WHO classifications are based on each agency's confidence that the pollutant causes cancer based on the scientific evidence.[58] In a few cases, the WHO has made a stronger classification than the EPA. Formaldehyde dominates the cancer risks of HAPs.[67] Over half of cancer cases nationwide that are attributed to outdoor HAP exposure are attributed to chronic formaldehyde exposure.[68] Acetaldehyde, benzene, naphthalene, and 1,3-butadiene are also significant carcinogens.

There is little to no evidence that acrolein and ethylbenzene cause cancer, but they are included in the list of HAPs of most concern because they are toxic even in low concentrations. This is especially true for acrolein, which dominates the non-cancer risks of HAPs.[67,68] Short-term exposures to several HAPs cause severe respiratory irritation.[57,59,69]

- **Long-term formaldehyde exposure can CAUSE cancer.** When chronically inhaled, formaldehyde can cause inflammation, oxidative stress, and genotoxicity leading to cancer. For formaldehyde these cancers are especially concentrated in the nose and throat, but there is also evidence for leukemia and possibly lung cancer as well. While the EPA has listed formaldehyde as a possible carcinogen, the National Academy of Science has agreed with the WHO and formally concluded that inhaled formaldehyde is a known carcinogen.[69]

- **Short-term formaldehyde exposure CAUSES respiratory and eye irritation.** Formaldehyde may also lead to asthma exacerbation, though evidence is mixed.
- Evidence **SUGGESTS** that long-term formaldehyde exposure has diverse effects in blood. These include changes in white blood cell counts and other immune response–related effects. The evidence for an association is strong, but a wide range of effects have been observed and some studies have found opposing results.[69]

Acrolein:[55,70]

- Evidence is **INADEQUATE** to link acrolein exposure and cancer.
- **Short-term acrolein exposure CAUSES respiratory and eye irritation.** As a highly reactive compound, acrolein has intensely irritating effects, even at low concentrations. Generally, people with underlying respiratory conditions may experience more severe respiratory symptoms from irritating exposures like acrolein, but there are few human studies to confirm this specifically for acrolein.[71]

1,3-Butadiene:[60,72]

- **Long-term 1,3-butadiene exposure can CAUSE cancer.** When chronically inhaled, 1,3-butadiene can cause inflammation, oxidative stress, and genotoxicity leading to cancer. Unlike several other HAPs, 1,3-butadiene has more systemic effects in the body, rather than local effects focused on the nose and throat where inhalation occurs. 1,3-butadiene is linked to leukemia and lymphatic cancers.
- **Short-term 1,3-butadiene exposure CAUSES respiratory and eye irritation.**
- Evidence **SUGGESTS** that long-term 1,3-butadiene exposure may increase risk of cardiovascular disease and blood disorders. This is likely related to inflammation from chronic exposure and resulting oxidative stress.

Naphthalene:[61]

- **Long-term naphthalene exposure is a LIKELY CAUSE of cancer.** Naphthalene exposure is linked to laryngeal cancer (occurring in the larynx, or upper part of the throat) and lung cancer.
- Evidence **SUGGESTS** that long-term naphthalene exposure may be linked to several serious non-cancer effects. Long-term naphthalene exposure has been observed to cause several serious effects in animals (including anemia, cataracts, neurological damage, and liver damage), but these effects have not been confirmed in humans.

Benzene:[64,65]

- **Long-term benzene exposure can CAUSE cancer.** Benzene passes from the lungs to the bloodstream when inhaled. It can sometimes be stored in bone marrow and disrupt blood production and immune response, potentially leading to leukemia over time.
- **Short-term benzene exposure CAUSES drowsiness, dizziness, rapid heart rate, headaches, and similar symptoms.**
- Evidence **SUGGESTS** that long-term benzene exposure is linked to blood disorders like anemia, damage to the immune system, and effects on menstruation and birth outcomes. These effects were observed in animal lab studies and some effects were observed in occupational studies.

Acetaldehyde:[56]

- **Long-term acetaldehyde exposure can CAUSE cancer.** The WHO and EPA differ on whether acetaldehyde causes or probably causes cancer. Animal lab studies observed nasal and laryngeal tumors from acetaldehyde exposures. Studies in humans have been very limited.
- **Short-term acetaldehyde exposure CAUSES respiratory and eye irritation.**
- Evidence is **INADEQUATE** to link long-term exposures and developmental effects. Acetaldehyde exposure was linked to serious developmental effects in a lab animal study, but the exposure route was injection rather than inhalation.

Ethylbenzene:[63,66]

- Evidence **SUGGESTS** that long-term ethylbenzene exposure may be linked to cancer. Evidence has been strong in animal studies, but a single occupational study in humans did not observe any effects.
- **Short-term ethylbenzene exposure CAUSES respiratory and eye irritation and—at higher doses—dizziness.**
- Evidence from animal exposure studies **SUGGESTS** that long-term ethylbenzene exposure (even at lower concentrations) may be linked to damage to the inner ear and kidneys. These effects have not been confirmed in humans.

While the potential health effects of HAPs shown above are serious, the scale of airport emissions and related residential exposure are important for understanding the risk posed by airport-related HAPs. A 2008 FAA-sponsored study found that deaths from airport-related $PM_{2.5}$ were 100 to 200 times larger than the cancer impacts from HAPs.[49,73] These estimates don't include non-cancer effects of HAPs, but the non-cancer effects also do not approach the magnitude of effects from $PM_{2.5}$.

10. Lead

Lead (Pb) is both a HAP and a criteria air pollutant. Inhaled lead is absorbed from the lungs into the bloodstream, where it strongly inhibits nutrient absorption and is highly toxic for cell function in organ systems throughout the body, especially the brain. Lead also gets stored in the blood, bones, and tissues, where it can be released later, re-exposing the body many years after exposure.

Because lead is now removed from gasoline, lead emissions from automobiles are no longer a concern. Commercial jets run on lead-free, kerosene-based fuels, but many small piston-engine planes still use leaded fuel. Piston-engine aircrafts are the largest source of lead emissions in the U.S.[74] Airborne lead concentrations have been found higher than EPA standards over a half mile downwind of general aviation airports.[49] The causal judgments for lead described here and in the report are based primarily on the EPA's 2013 lead ISA.[74] Lead emissions from commercial airports are a few orders of magnitude smaller than other emissions and health effects are reviewed in the main report.

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Appendix D. Strength-of-Evidence Pathway Trees for Noise Pollution and Air Pollutants Common to Airport Operations

Figure D1. Noise Pollution Strength-of-Evidence Pathway Tree

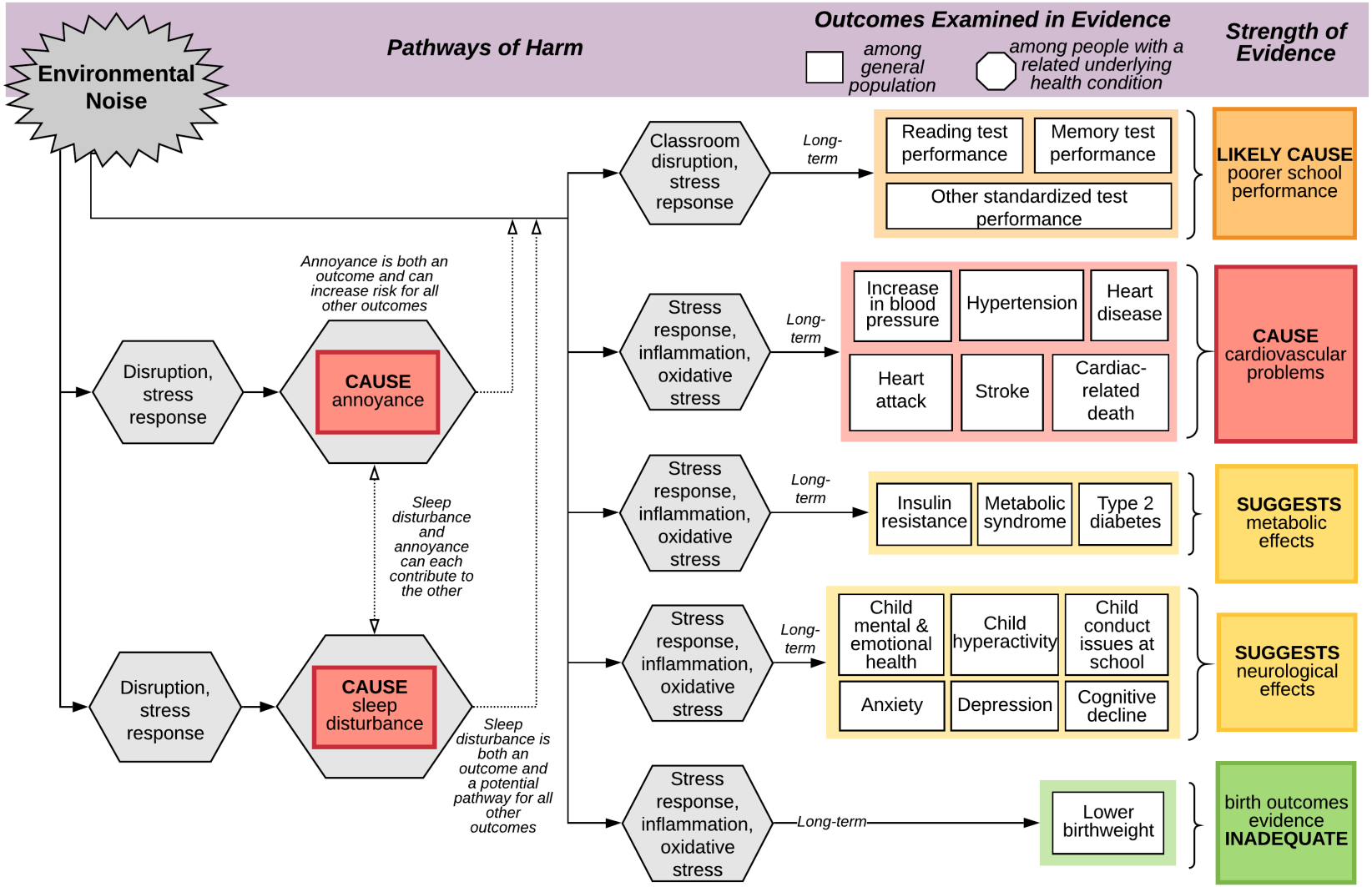

Figure D2. Coarse Particulate Matter Strength-of-Evidence Pathway Tree

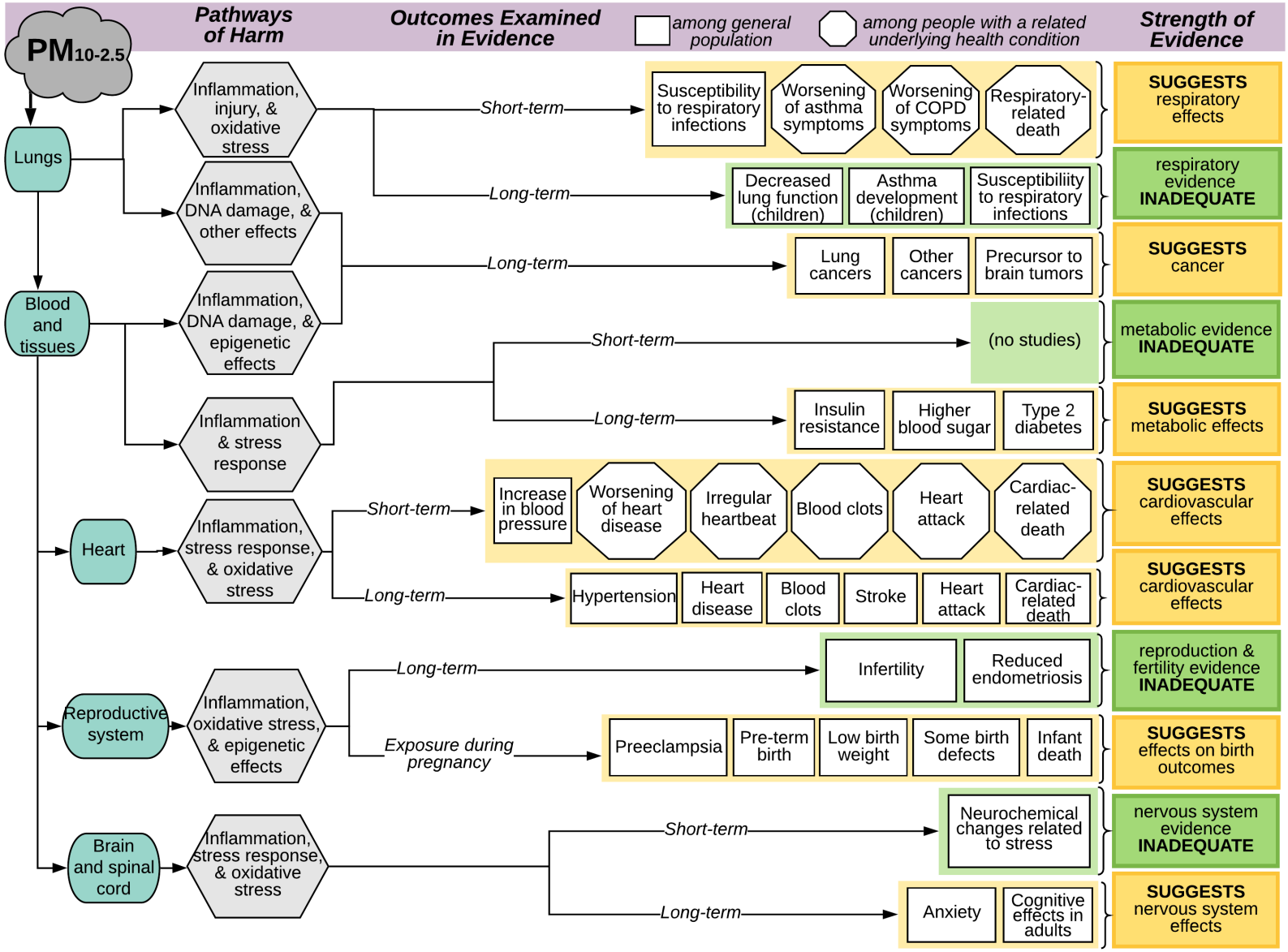

Figure D3. Fine Particulate Matter Strength-of-Evidence Pathway Tree

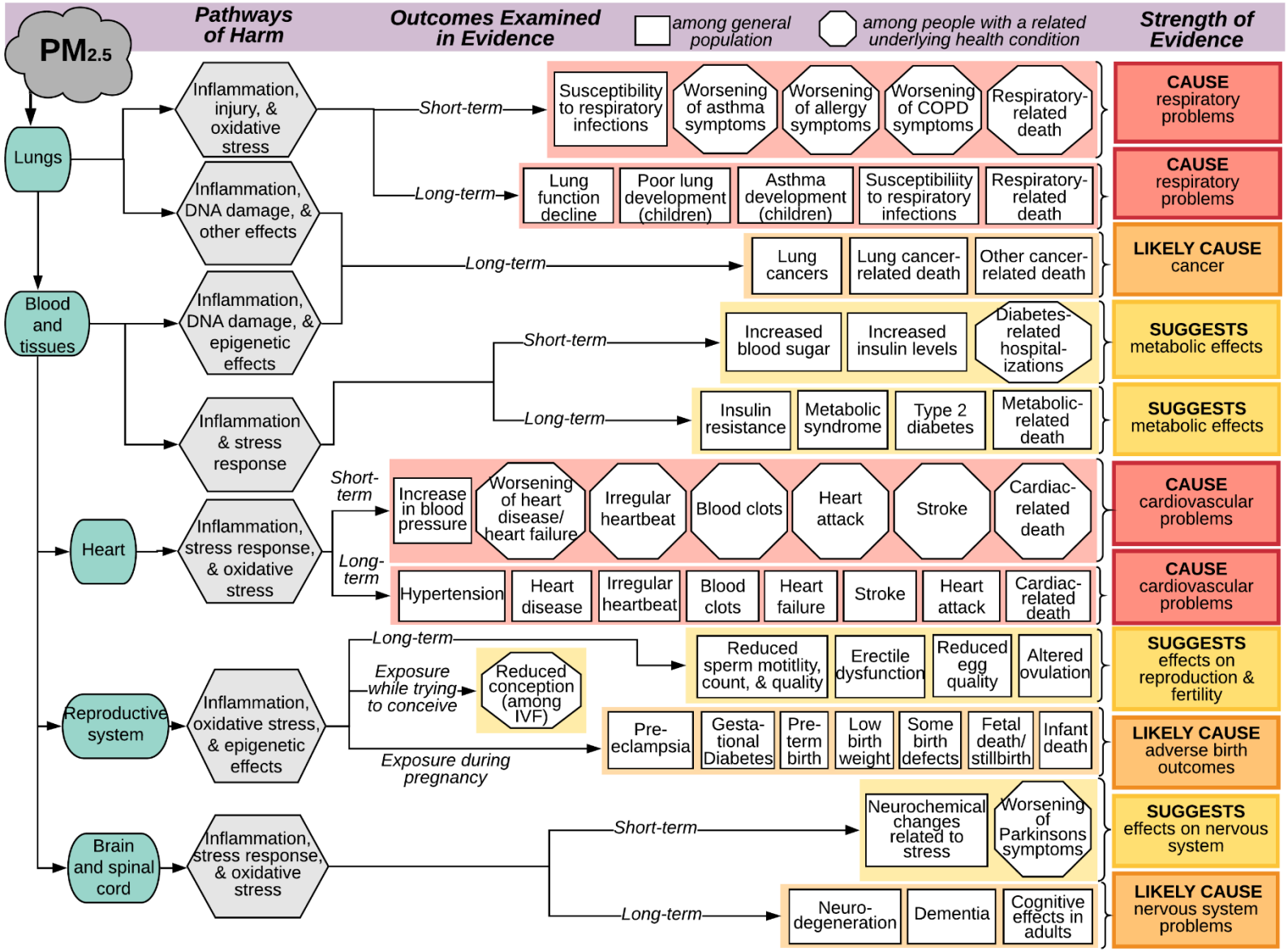

Figure D4. Ultrafine Particulate Matter Strength-of-Evidence Pathway Tree

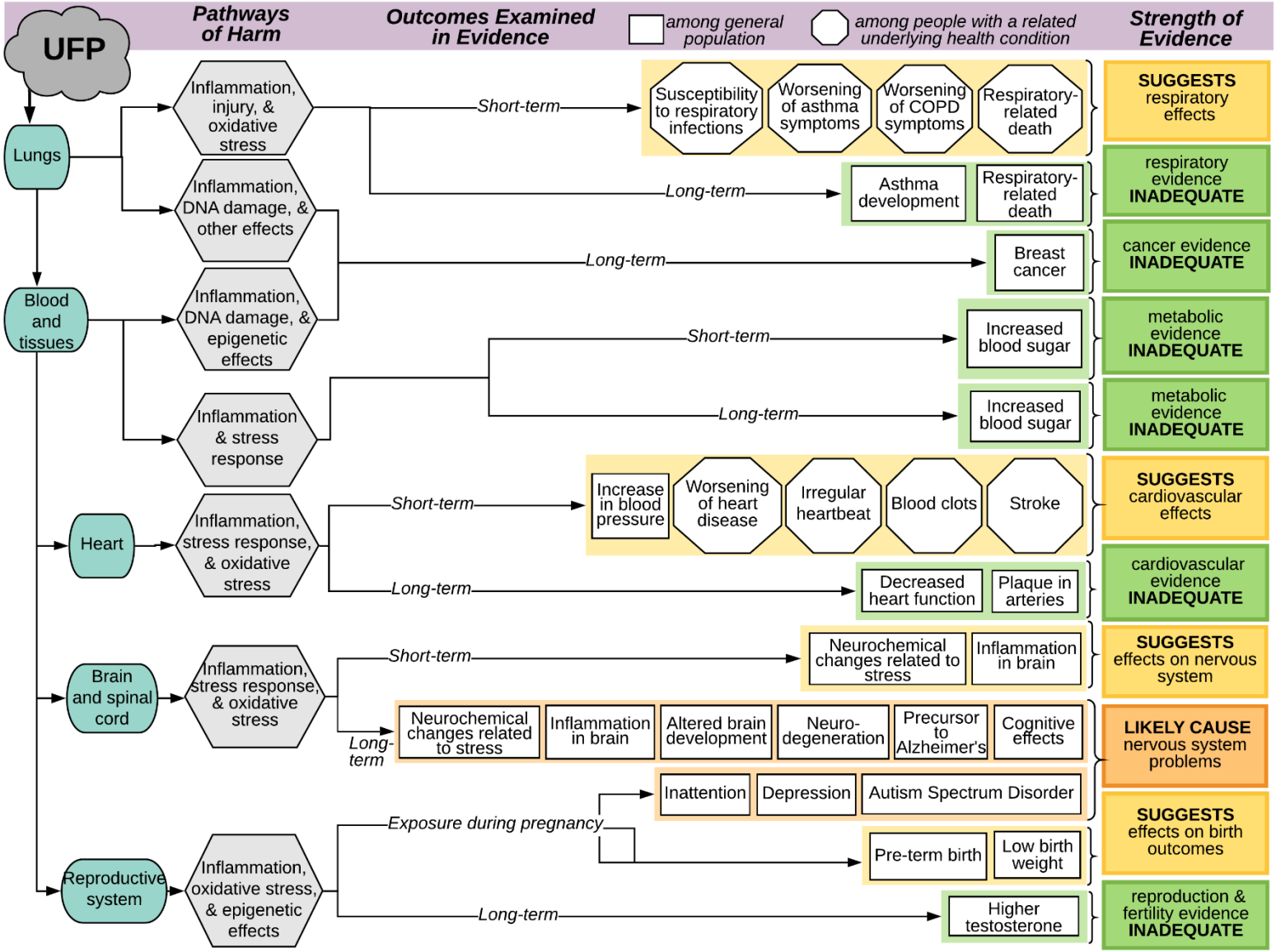

Figure D5. Ozone Strength-of-Evidence Pathway Tree

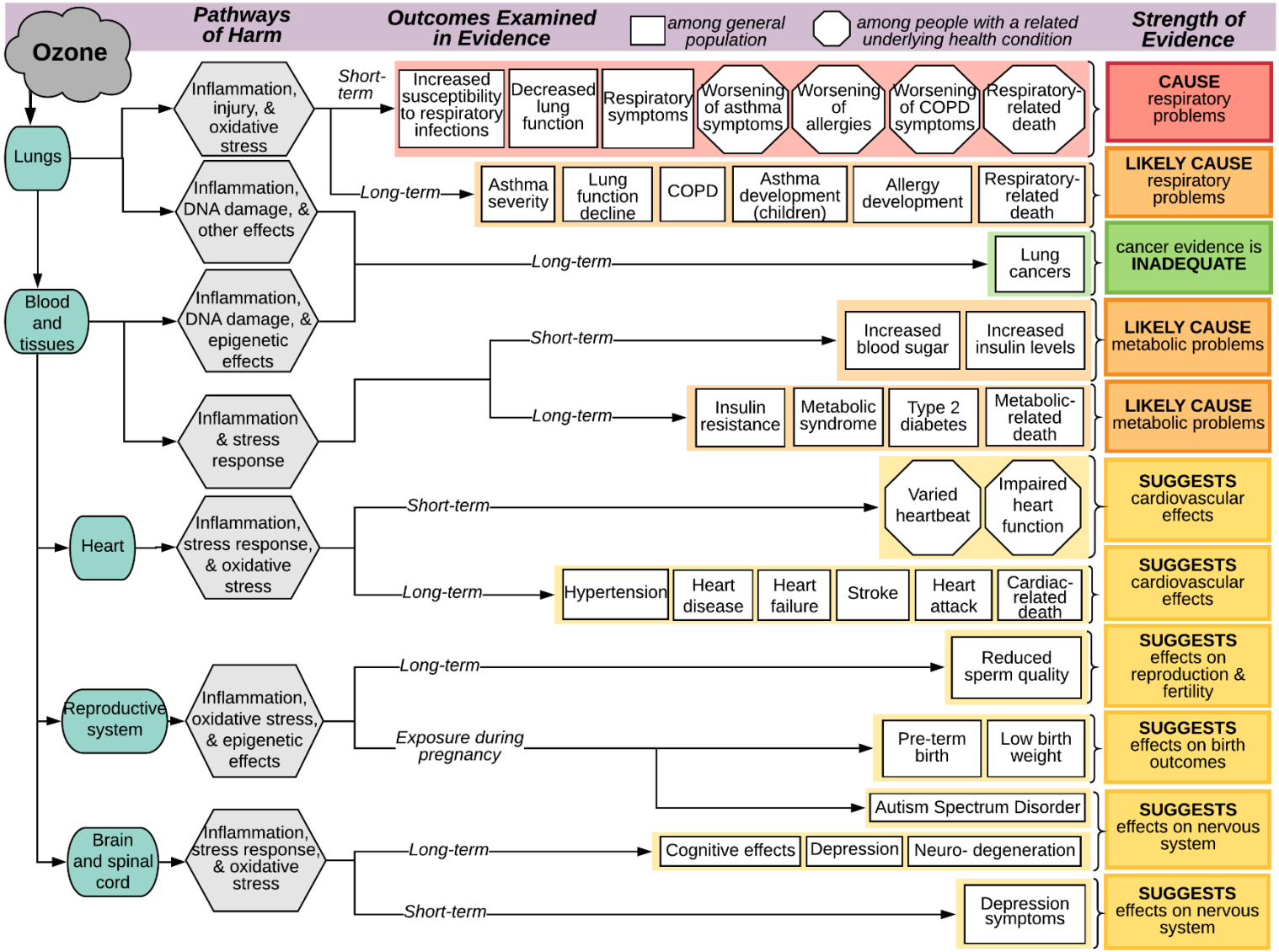

Figure D6. Carbon Monoxide Strength-of-Evidence Pathway Tree

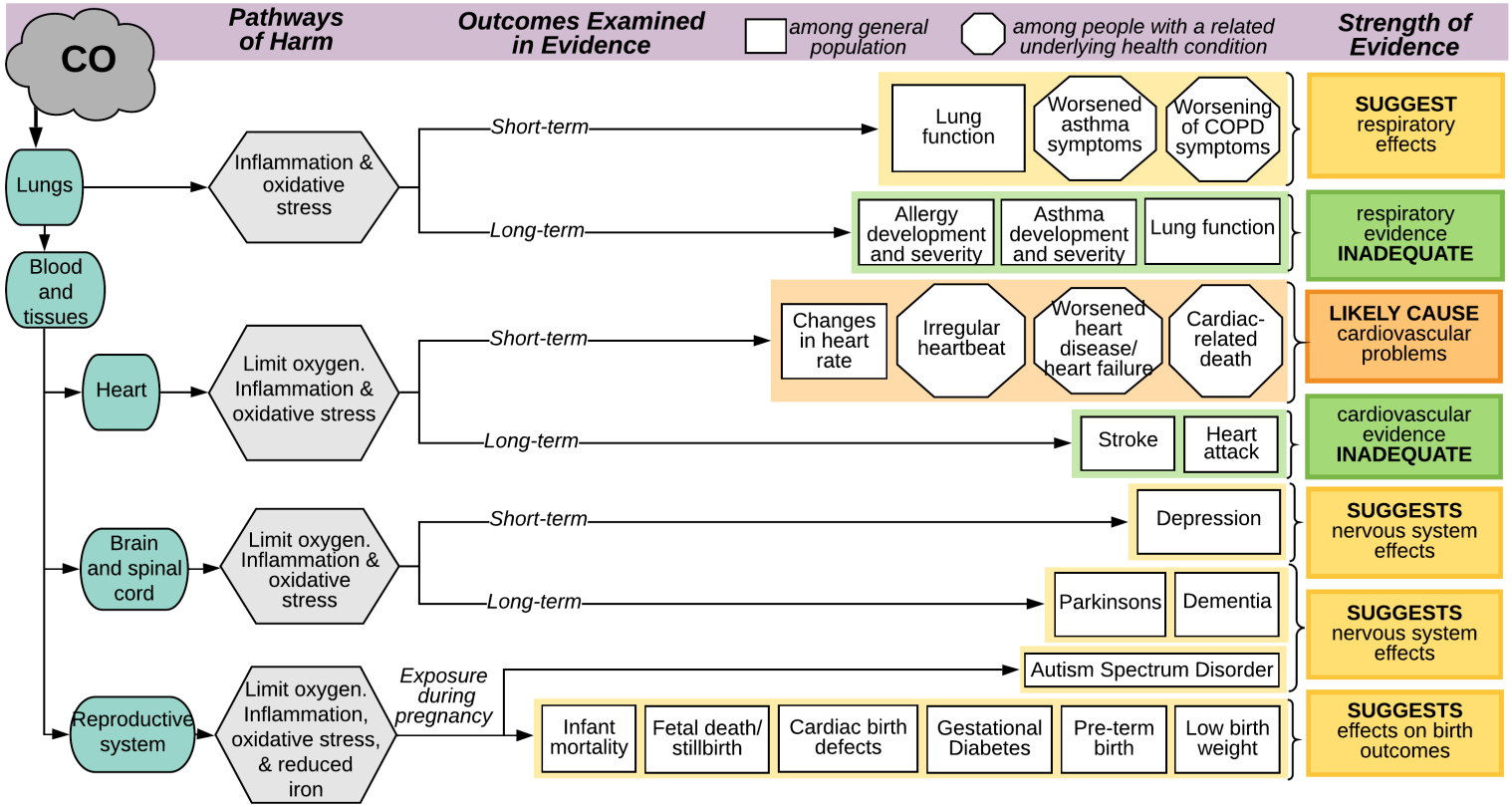

Figure D7. Nitrogen Dioxide Strength-of-Evidence Pathway Tree

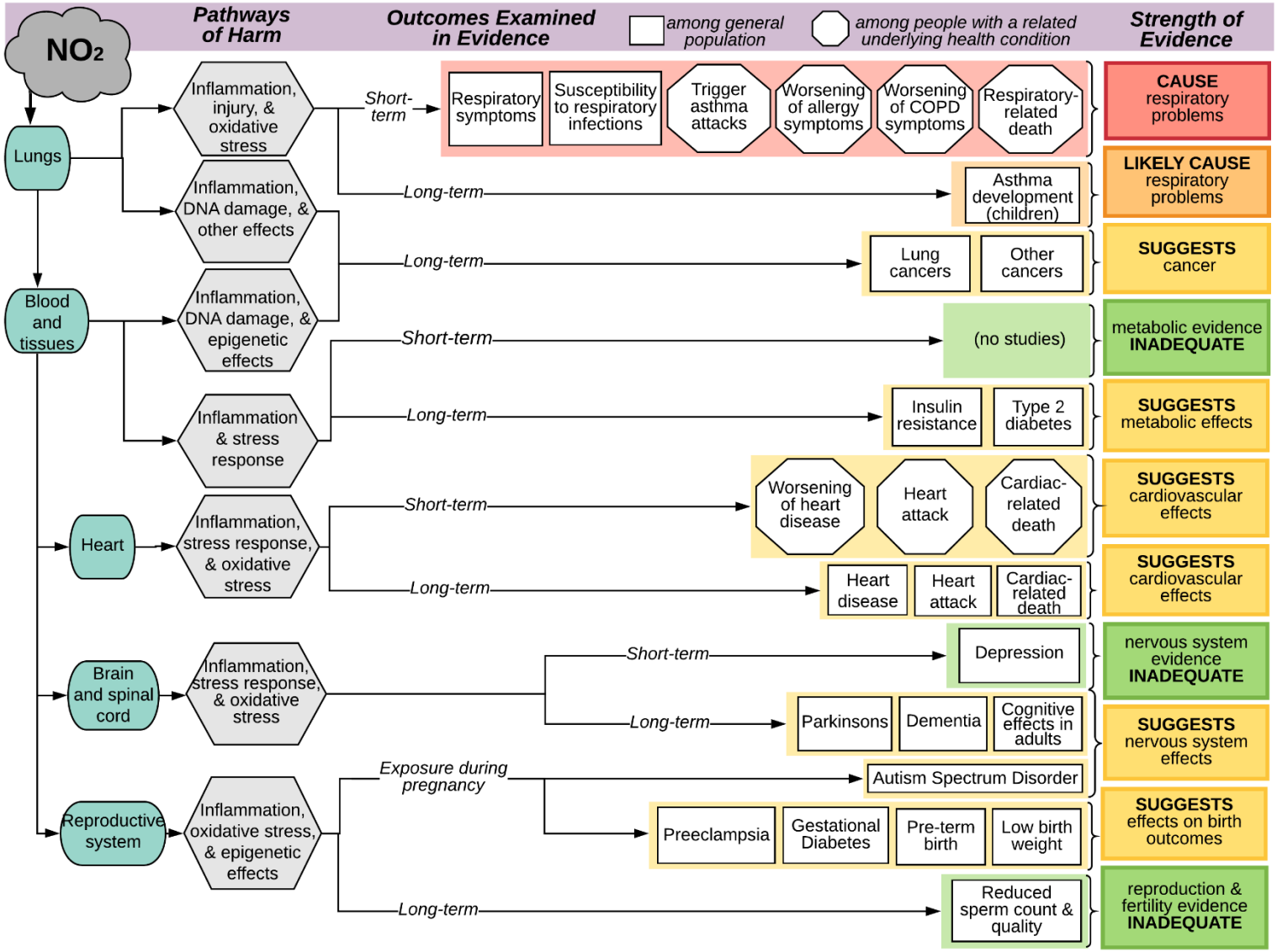
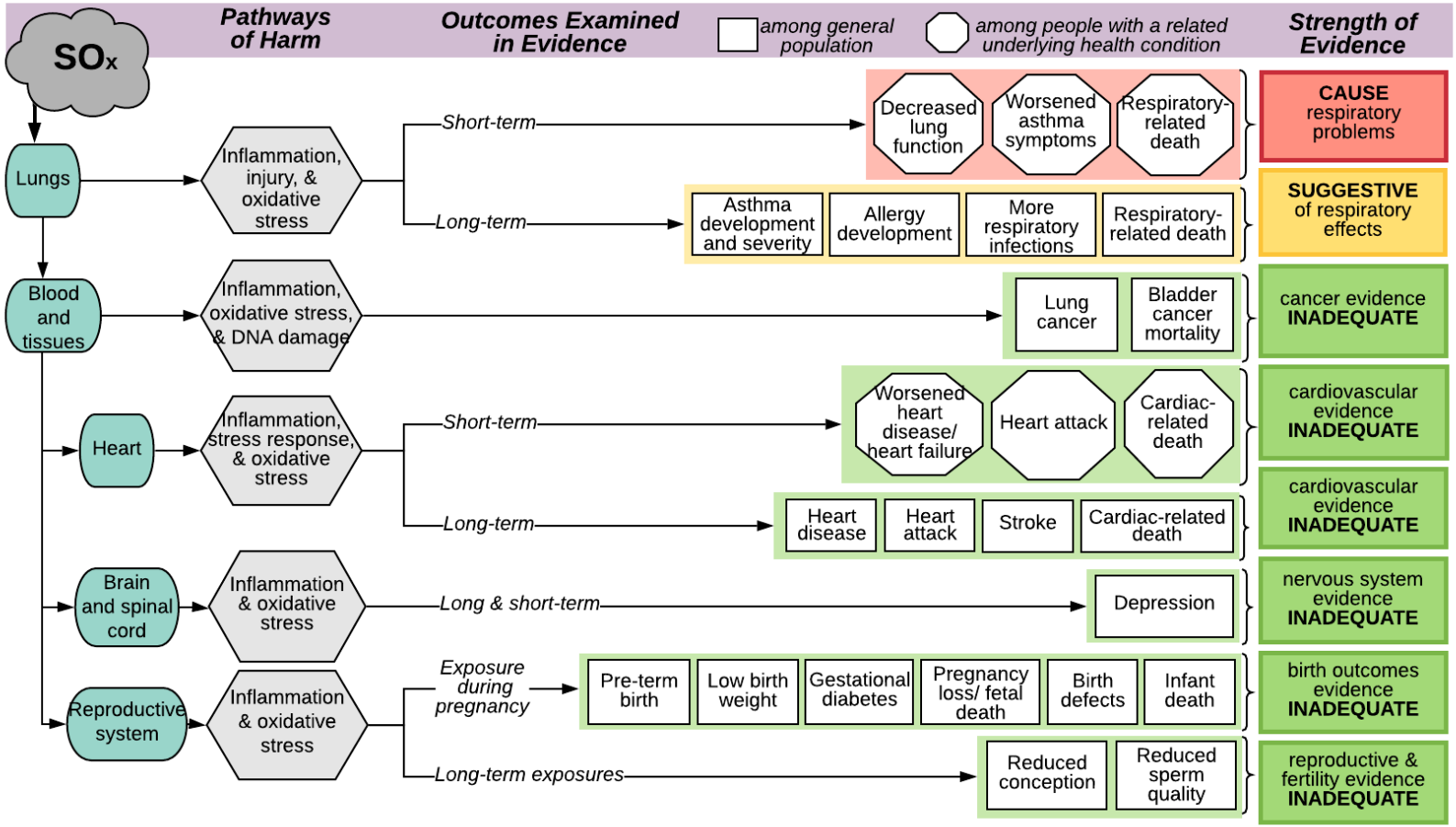
Figure D8. Sulfur Oxide Strength-of-Evidence Pathway Tree