

Review

Historic and Modern Air Pollution Studies Conducted in Utah

Judy Ou ^{1,*}, Cheryl S. Pirozzi ², Benjamin D. Horne ^{3,4} , Heidi A. Hanson ^{1,5}, Anne C. Kirchhoff ^{1,6}, Logan E. Mitchell ⁷ , Nathan C. Coleman ⁸ and C. Arden Pope III ⁸ 

- ¹ Huntsman Cancer Institute, Cancer Control and Population Sciences, University of Utah, Salt Lake City, UT 84112, USA; heidi.hanson@hci.utah.edu (H.A.H.); Anne.Kirchhoff@hci.utah.edu (A.C.K.)
 - ² Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, University of Utah, Salt Lake City, UT 84132, USA; Cheryl.Pirozzi@hsc.utah.edu
 - ³ Intermountain Medical Center Heart Institute, Salt Lake City, UT 84107, USA; Benjamin.Horne@imail.org
 - ⁴ Division of Cardiovascular Medicine, Department of Medicine, Stanford University, Stanford, CA 94063, USA
 - ⁵ Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT 84132, USA
 - ⁶ Department of Pediatrics, University of Utah School of Medicine, Salt Lake City, UT 84132, USA
 - ⁷ Department of Atmospheric Sciences, University of Utah, Salt Lake City, UT 84112, USA; logan.mitchell@utah.edu
 - ⁸ Department of Economics, Brigham Young University, Provo, UT 84602, USA; 1nathancoleman@gmail.com (N.C.C.); cap3@byu.edu (C.A.P.III)
- * Correspondence: Judy.Ou@hci.utah.edu

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Abstract: Utah’s low-smoking population and high population density concentrated in mountain valleys, with intermittent industrial activity and frequent temperature inversions, have yielded unique opportunities to study air pollution. These studies have contributed to the understanding of the human health impacts of air pollution. The populated mountain valleys of Utah experience considerable variability in concentrations of ambient air pollution because of local emission sources that change over time and episodic atmospheric conditions that result in elevated concentrations of air pollution. Evidence from Utah studies indicates that air pollution, especially combustion-related fine particulate matter air pollution and ozone, contributes to various adverse health outcomes, including respiratory and cardiovascular morbidity and mortality and increased risk of lung cancer. The evidence suggests that air pollution may also contribute to risk of pre-term birth, pregnancy loss, school absences, and other adverse health outcomes.

Keywords: air pollution; mortality and morbidity; Utah; particulate matter; ozone

1. Introduction

The state of Utah has a long history of poor air quality that has provided substantial opportunities to study the health effects of air pollution. Most of Utah’s land mass includes sparsely populated mountains, deserts, and canyonlands with relatively clean, less polluted air. However, most Utahans live in urban areas located in valley basins surrounded by mountains. Approximately 80% of the population of Utah lives in a contiguous urban/suburban area called the Wasatch Front [1]. The Wasatch Front is a relatively narrow landmass—approximately 80 miles (130 km) long from north to south and 5 to 18 miles (8–29 km) wide—bordered on the east by the Wasatch Mountain Range. The relatively dense population of the Wasatch Front (compared to the rest of the state and most other areas in the Intermountain West) and related industrial, traffic, and other emission sources result in substantial levels of air pollution that can at times be the worst in the U.S. [2]. Furthermore, surrounding mountain

topography and interaction with meteorological conditions lead to considerable temporal variability in air pollution.

Changes in the operation of major industrial sources of air pollution in Utah—including the intermittent operation of a steel mill and copper smelter—have provided unique natural experiments or quasi-experimental conditions to explore health impacts of air pollution. Utah also consistently holds the nation's lowest smoking rate (9% compared to the national average of 17.1%) [3], which helps evaluate the health effects of air pollution with less potential for confounding from smoking.

Studies of particulate matter air pollution have been the primary focus in Utah. Monitoring of PM₁₀ (particles <10 µm in aerodynamic diameter) began at Utah monitoring sites in the mid to late 1980s, and regular monitoring of PM_{2.5} (particles <2.5 µm in aerodynamic diameter) began at some Utah monitoring sites in 1999. A growing number of studies have also investigated the effects of ozone (O₃) and nitrogen oxide (NO_x) pollutants. These pollutants are growing in relevance because Utah's four largest counties (Salt Lake, Utah, Davis, and Weber, which account for 75% of the population) are currently not compliant with federal national ambient air quality standards (NAAQS) for ozone. This problem is projected to worsen as emissions increase from a rapidly growing population and climate change threatens to increase ground-level ozone production [4,5]. Since 2009, Utah's population grew 14 percent to a current total of ≥3 million persons [6]. The population is anticipated to grow to 5.8 million in 2065, which represents a rate of change of 1.3 percent. This is nearly double the 0.7 percent growth seen nationally from 2016 to 2017 [7]. Utah has reduced air emissions by 38 percent during the past 15 years, but the winter temperature inversions still pose a major problem for the state, and summertime ozone emissions are emerging as a major public health concerns [6].

Early epidemiology studies of air pollution in Utah contributed to the 1971 U.S. Environmental Protection Agency (EPA) sponsored Community Health and Environmental Surveillance System (CHESS) studies. The CHESS studies in Utah were focused on the health effects of particulate matter and sulfur oxides, with a specific focus on sulfur dioxide and sulfates [8,9]. There was substantial controversy surrounding the CHESS studies, compromising the ability for the program to change public policy and move air pollution science forward.

Air pollution research in Utah resumed in the mid to late 1980s, including a unique natural experiment related to the intermittent operation of a major industrial source of pollution [10,11]; a series of panel studies on the link between air pollution, pulmonary function, and respiratory illness [12,13]; and early time-series studies of daily mortality [14,15]. These Utah-based studies were included in the Environmental Protection Agency's 1996 Air Quality Criteria for Particulate Matter Final Report, which laid the groundwork for fine particulate matter (PM_{2.5}) standards [16]. Today, Utah-based studies remain a part of a much larger and broader body of research that explores the health effects of air pollution. This article briefly summarizes the results of existing Utah-based studies of pollution's effects on multiple health outcomes including mortality, respiratory disease, cardiovascular disease, cancer survivorship, and birth outcomes (Table 1).

Table 1. Outline and review of air pollution and health studies from Utah.

Studies	Health End Points	Study Designs	Result	Summary of Results
Mortality, all Cause, Cardiovascular, Respiratory				
Archer 1990 [17]	Respiratory and lung cancer mortality	3-county ecologic	+	Mortality associated with higher levels of air pollution, especially combustion/industrial-source fine particles versus windblown fine particles.
Pope et al., 1992,1996,1999 [14,15,18]	All, cardiovascular, respiratory mortality	Daily time series	+	
Lyon et al., 1995 [19]	All, cardiovascular, respiratory mortality	Daily time series	0	
Styer et al., 1995 [20]	All mortality	Daily time series	0	
Ransom et al., 1995 [21]	All mortality and hospitalization	Natural experiment	+	
Pope et al., 2007 [22]	All mortality	Natural experiment	+	
Respiratory Illness/Function				
Love et al., 1982 [9]	Respiratory illness incidence and severity	Panel	0	Epidemiological and toxicological evidence that air pollution contributes to pulmonary inflammation, respiratory illness and disease and reduced pulmonary function.
Lutz 1983 [23]	Diagnosis of respiratory or cardiac illness	Episode study	+	
Pope 1989, 1991 [10,11]	Hospitalization for respiratory illness	Natural experiment	+	
Pope et al., 1991,1992 [12,13]	Respiratory symptoms/function in children	Panel/time series	+	
Pope and Kanner 1993 [24]	Pulmonary function effects for former smokers	Panel	+	
Ghio et al., 2001 [25]	Inflammatory lung injury in humans	Human in vivo	+	
Dye et al., 2001 [26]	Pulmonary toxicity in rats	Toxicology	+	
Watterson et al., 2007 [27]	Gene expression	Toxicology	+	
Beard et al., 2012 [28]	Emergency Department visits for asthma	Case-crossover	+	
Pirozzi et al., 2015a, 2015b [29,30]	Respiratory effects for former smokers	Observational/panel	+/0	
Horne et al., 2018 [31]	Respiratory infection	Case crossover	+	
Pirozzi et al., 2018 [32]	Pneumonia incidence	Case crossover	+	
Wagner et al., 2018, 2020 [33,34]	Aerobic, pulmonary function	Human experiment	0	
Cardiovascular Illness/Function				
Pope et al., 1999b, 1999c, 2004 [35–37]	Heart rate variability	Panel/human experiment	+/0	Evidence that air pollution contributes to risk of various cardiovascular events, impairs cardiac autonomic function, and contributes to inflammation and endothelial injury.
Pope et al., 2006, 2015 [38,39]	Ischemic heart disease events	Case crossover	+	
Pope et al., 2008 [40]	Heart failure, hospitalization	Case crossover	+	
O’Toole et al., 2010 [41]	Endothelial progenitor cells	Panel/human experiment	+	
Bunch et al., 2011 [42]	Atrial fibrillation hospitalization	Case crossover	0	
Pope et al., 2016 [43]	Endothelial injury and inflammation	Panel/human experiment	+	
Leiser et al., 2019 [44]	Cardiac hospital readmission/death	Medicare cohort	+	

Table 1. Cont.

Studies	Health End Points	Study Designs	Result	Summary of Results
Cancer				
Lyon et al., 1981 [45]	Lung cancer incidence	Case-control	0	Evidence that radioactive fallout is associated with cancer mortality. Contributes to evidence that air pollution may be associated with cancer and cancer survival.
Archer 1990 [17]	Lung cancer mortality	3-county ecologic	+	
Blindauer et al., 1993 [46]	Lung cancer incidence	Natural experiment	0	
Ball et al., 2008 [47]	Lung, kidney, non-Hodgkin's	Ecological	+	
Ou et al., 2019 [48]	Respiratory health in cancer survivors	Case crossover	+	
Ou et al., 2020 [49]	Various types of cancer mortality	Cohort	+	
Birth Outcomes				
Parker et al., 2008 [50]	Pre-term birth	Natural experiment	+	Contributes to evidence of an association between air pollution and pre-term birth.
Mendola et al., 2019 [51]	Pre-term birth	Observational cohort	+	
Leiser et al., 2019 [52]	Pregnancy loss	Case crossover	0	
Other				
Ransom et al., 1992 [53]	School absences	Daily time series	+	Evidence that air pollution is associated with an increase in school absences and other health outcomes. Evidence for higher exposure amongst minority populations.
Zeft et al., 2009 [54]	Juvenile idiopathic arthritis	Case crossover	+	
Bakian et al., 2015 [55]	Suicide	Case crossover	+	
Hales et al., 2016 [56]	School absences	Natural experiment	+	
Youngquist et al., 2016 [57]	Emergency Medical Service calls	Case crossover	0	
Mullen et al., 2019 [58]	Racial/ethnic exposure disparity	Exposure modeling	+	
Collins et al., 2019 [59]	Racial/ethnic exposure disparity	Cross-sectional	+	

+ = positive association; 0 = no association; – = inverse association.

2. Mortality

Utah-based studies provide evidence of an association between air pollution and mortality. Archer evaluated longitudinal differences in mortality across three counties, contrasting death rates during periods of intermittent operation of a steel mill that was constructed during World War II in one of the counties (Utah County) [17]. This initial analysis was based on a simple ecological design that compared mortality in Utah County to two other study areas without a similar source of industrial pollution. It was estimated that 30 to 40% of respiratory cancer and nonmalignant respiratory disease deaths in one of these areas were associated with community air pollution emitted from the steel mill [17]. Additionally, analyses that treated the intermittent operations of a local steel mill [21] and the intermittent operation copper smelter [22] as natural experiments further observed that mortality was associated with fine-combustion and industrial-source particulate air pollution.

Several population-based, daily time-series studies that evaluated day-to-day changes in mortality counts with short-term (1–5 days) changes in air pollution have been conducted using Utah's Wasatch Front counties (Salt Lake, Utah, Davis, and Weber). The earliest study reported a 16% increase in mortality counts in Utah County associated with a 100 $\mu\text{g}/\text{m}^3$ increase in exposure to particulate matter air pollution, measured as PM_{10} over the previous 5 days, after controlling for time trends, seasonality, temperature, and relative humidity [14]. Pollution was most strongly associated with respiratory and cardiovascular deaths. Additional analyses of Utah County mortality data confirmed the PM–mortality association but questioned if the association was causal [19]. An extended analysis, however, demonstrated similar PM–mortality associations of (between 11–16% per 100 $\mu\text{g}/\text{m}^3$ of PM_{10}) using alternative synoptic weather modeling approaches to control for weather, suggesting that the observed PM–mortality associations were not the results of confounding by weather variables [15].

A daily time-series analysis of mortality counts and air pollution in a neighboring Wasatch Front county (Salt Lake County) did not find evidence of an association between mortality and PM_{10} [20]. A more comprehensive population-based daily time-series mortality study was conducted using the populations from all three primary metropolitan areas of the Wasatch Front including the following: the Ogden area (Weber County), the Salt Lake City area (Salt Lake and Davis Counties), and the Provo/Orem area (Utah County) [18]. The Salt Lake City area experienced many more high PM episodes dominated by windblown dust. When the pollution data were screened to exclude windblown dust episodes (based on clearing index screening), comparable PM_{10} –mortality associations were observed across the Wasatch Front metropolitan areas (between 0.8–1.6% change in mortality per 10 $\mu\text{g}/\text{m}^3$) [18]. It was concluded that stagnant air pollution episodes with higher concentrations of combustion-source and industrial-source fine particles were more strongly associated with elevated mortality (as opposed to windblown dust episodes with higher coarse, crustal derived particles).

3. Lung Disease and Respiratory Health Outcomes

3.1. Human Health Outcomes

Multiple Utah-based studies reported that air pollution is associated with adverse respiratory health outcomes. As noted above, population-based daily time-series mortality studies observe that short-term increases in pollution are associated with increased respiratory mortality counts [14,15,18]. Short-term increases in PM_{10} and $\text{PM}_{2.5}$ during winter inversions are associated with significant increases in the risk estimates for outpatient visits, emergency department visits, and hospital admissions for respiratory disease in multiple counties [10,11,23,28]. These studies found that there were nearly twice as many respiratory hospital admissions for children during the period the steel mill was operating compared to when the steel mill was not operating [10,11]. One study found that for months when PM_{10} was over 50 $\mu\text{g}/\text{m}^3$, the average annual standard at the time, hospital admissions increased by 89% for children and 47% for adults [10].

Respiratory infections are a particular concern as acute lower respiratory infections, bronchitis, and pneumonia have significant associations with short-term increases in particulate matter

pollution [31,32]. Pediatric populations are particularly vulnerable to respiratory infections associated with short-term increases in PM_{2.5}. One study found that the odds ratio for acute lower respiratory infection in young children (0–2 years of age) is 1.15 (95% CI: 1.12–1.19) per 10 µg/m³ PM_{2.5}, with a lag period of up to 28 days [31]. The associations are somewhat larger for pneumonia, with odds ratios ranging from 1.35–1.50 [32]. Pediatric populations exposed to high levels of PM_{2.5} also report increased use of asthma medication, coughing, and increases in reported symptoms of respiratory disease [12,13].

3.2. Lung Function and Performance

Utah-based studies provide evidence that air pollution is associated with reduced lung function in susceptible populations. Studies of 16 healthy adults exposed to PM_{2.5} below the federal 24 h health standard found no negative effects on respiratory function or aerobic performance after 20 min of heavy exercise [33,34], but multiple studies reported significant effects on lung function among children and adults with preexisting chronic obstructive pulmonary disease (COPD) [12,13,24]. Among fourth and fifth grade elementary students, 150 µg/m³ increases in PM₁₀ were associated with a 3–6% decline in lung function [12]. In a cohort of fifth and sixth graders, short-term increases in PM₁₀ were associated with declines in peak expiratory flow (PEF), irrespective of exhibited symptoms [13]. Among adult smokers with COPD, an increase of 100 µg/m³ in PM₁₀ was significantly associated with a 2% decrease in forced expiratory volume [24]. For adult COPD patients, respiratory symptoms significantly increased after days with increased PM_{2.5} [29].

3.3. Biomarkers

Multiple laboratory and human biomarker studies support local inflammation as the primary mechanism by which particulate matter and ozone pollution exert adverse effects on the respiratory system. An *in vitro* study reported that human bronchial epithelial cells (BEAS-2B) exposed to PM_{2.5} found in Cache Valley significantly upregulated genes activating receptors to interleukins 1 and 6 (IL-1R1 and IL-6R), IL-6 and phosphorylated STAT3 protein release, indicating activation of the IL-6/gp130/STAT3 signaling pathway [27]. The study also reported slight cytotoxicity of the Cache valley PM_{2.5} [27]. An *in vivo* study examined the effect of PM_{2.5} particulates collected during the period of intermittent steel mill operation installed in the trachea of Sprague–Dawley rats. Rats exposed to PM_{2.5} collected during steel mill operation expressed significant pulmonary injury and neutrophilic inflammation, which was suggested to be due to metals contained in the particulate matter [26].

A human *in vivo* study installed aqueous extracts of PM collected during intermittent steel mill operation over a 3-year period inside the lungs of 24 nonsmoking healthy volunteers. Subjects administered the extracts of PM from filters taken while the steel mill was in operation had significantly high levels of neutrophil infiltration and elevated concentrations of fibronectin and α -1-antitrypsin, indicating inflammatory lung injury [25]. In a separate study, human exhaled breath condensate was collected from former smokers with moderate to severe COPD on days with PM_{2.5} that was considered “clean” and on days with higher PM_{2.5} pollution during winter inversions [29]. High PM_{2.5} levels were associated with increases in nitrite plus nitrate (NO_x), a biomarker of oxidative stress in COPD patients (mean of 3.16 difference between polluted and clean days), but not former smokers without COPD. Ozone was also examined as a potential pulmonary inflammatory agent among individuals with COPD. High ozone was associated with increased NO_x and thus oxidative stress and pulmonary inflammation in both COPD patients (8.7 vs. 28.6 on clean versus polluted days) and persons without COPD (7.6 vs. 28.5), with no difference between the groups [30].

4. Cardiovascular Disease

Several Utah-based studies have found an association between air pollution and cardiovascular health. As noted above, population-based daily time-series mortality studies have observed that short-term increases in pollution are associated with increased cardiovascular mortality [14,15,18]. Case-crossover studies of patients drawn from a large cardiac catheterization registry who lived in the Wasatch Front

area of Utah observed that a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ air pollution was associated with a 4.5% (95% CI: 1.1–8.0) increased risk of acute ischemic coronary events (unstable angina and myocardial infarction) [38]. The elevated risk was primarily observed among patients with angiographically demonstrated underlying coronary artery disease. An additional similar case-crossover study provided further evidence that, for patients living on Utah's Wasatch Front, a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ exposures contribute to the triggering of acute coronary events (OR 1.06, 95% CI: 1.02–1.11), especially ST-segment elevation myocardial infarction (OR 1.15, 95% CI: 1.03–1.29) [39]. Case-crossover studies of hospitalizations further observed that air pollution was associated with heart failure hospitalizations (13.1% increase per 10 $\mu\text{g}/\text{m}^3$, 95% CI: 1.3–26.2) [40] but not with hospitalizations for atrial fibrillations [42]. A cohort study examining the risk of hospital readmission and death after cardiovascular events found that an increase of 10 $\mu\text{g}/\text{m}^3$ in $\text{PM}_{2.5}$ led to a 25–30% increased risk of readmission [44].

Utah studies have explored pathophysiological pathways that link exposure to air pollution with cardiovascular disease. $\text{PM}_{2.5}$ air pollution in Utah has been associated with changes in cardiac autonomic function as measured by measures of heart rate variability [35–37], blood markers of inflammation [37,43], decreasing circulating levels of endothelial progenitor cells [41], and vascular/endothelial injury [43]. A recent study conducted in Utah County analyzed blood drawn from panels of healthy, nonsmoking young adults. The timing of multiple blood draws took advantage of frequent persistent temperature inversion episodes, allowing for blood draws at times with varying levels of exposure to $\text{PM}_{2.5}$ pollution [43]. Increased air pollution exposure was associated with elevated immune cells, a systemic increase in inflammatory and antiangiogenic cytokines with suppression of proangiogenic growth factors. Additionally, elevated air pollution exposure was associated with increased circulating endothelial microparticles, indicating endothelial cell apoptosis and vascular injury [43].

5. Cancer

Although air pollution is currently classified by the International Agency for Research on Cancer as a known carcinogen and extensive research support its association with incident cancer (especially lung cancer) and cancer mortality [60], published studies in Utah on the topic are limited and inconclusive. A cluster investigation around point sources of pollution reported no significant increase in the incidence of cancers among residents near a coke oven in a steel mill, but did report a slight increase in the number of excess lung cancers near the coke ovens [45]. In a separate study, mortality from respiratory cancers in a low-smoking county with a steel mill was estimated to be 38% higher than mortality in a neighboring low-smoking county without a steel mill [17]. In a case-control study that adjusted for smoking, no consistent difference was found in the rates of lung cancer incidence between Utah county and several other counties [46]. A cluster study of lung, kidney, and non-Hodgkin lymphomas found elevations in the number of kidney (Risk Ratio (RR) range: 0.50–3.17) and lung cancers (RR range: 1.02–1.51) around Hill Air Force Base, with the authors attributing the elevated risk of cancers to water contamination from the Air Force base rather than potential air pollutants from the base or other emission sources [47].

To date, most research on the topic of air pollutants and cancer focused on cancer incidence or mortality in a general population. Researchers in Utah were the first to investigate the effect of air pollution on the pulmonary health and mortality of cancer survivors after diagnosis. Cancer itself and the long-term toxic effects of cancer therapies on cancer survivors may increase their susceptibility to health events and mortality associated with air pollution. In a statewide case-crossover study of childhood cancer survivors, $\text{PM}_{2.5}$ was associated with significant increases in the risk for respiratory hospitalization and emergency room visits or hospitalization for respiratory infection. The risk for respiratory events was significantly higher among childhood cancer survivors than population comparisons without a cancer history (OR 1.84, 95% CI: 1.13–3.00 per 10 $\mu\text{g}/\text{m}^3$) [48]. $\text{PM}_{2.5}$ exposure from diagnosis through 5 and 10 years after diagnosis was also associated with all cause and cancer mortality among pediatric, adolescent, and young adult survivors (AYA) with certain diagnoses [49].

Pediatric patients diagnosed at age 14 years or younger with lymphoma (1.34, 95% CI: 1.06–1.68) or central nervous system (CNS) tumors (1.27, 95% CI: 1.05–1.52) had a significant increase in their risk for cancer mortality associated with a 5 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ exposure within 10 years from diagnosis. Among AYAs diagnosed from age 15 to 39 years, $\text{PM}_{2.5}$ was associated with all cause and cancer mortality among survivors with central nervous system tumors (1.20, 95% CI: 1.04–1.38), breast cancer (1.16, 95% CI: 0.97–1.39), and colorectal cancer (1.23, 95% CI: 1.00–52) within 10 years of diagnosis.

6. Birth Outcomes

Human epidemiologic studies in Utah of the association between air pollutant exposure and birth outcomes are rare. Two studies reported significant associations between increased exposure to $\text{PM}_{2.5}$ and risk of preterm births [50,51]. The sources of $\text{PM}_{2.5}$ and pollutants studies varied between the studies. An earlier study implemented a quasi-experimental design to examine the effect of a steel mill closure on preterm births and birth weight [50]. Utah mothers who were pregnant around the time of the steel mill closure were less likely to have a preterm birth than mothers who were pregnant before or after the closure (RR 0.95, 95% CI: 0.77–1.18). Reducing exposure during the second trimester appeared to have the highest effects in reducing preterm births. No effects on birth weight were observed, but the authors acknowledged the small sample size found in the study and their lack of exact exposure estimates for each mother.

The latter study examined the association of preterm births and air pollution while accounting for the composition of the pollution among mothers with two consecutive pregnancies [51]. This study reported that high exposure to sulfur dioxides, ozone, nitrogen oxides, carbon monoxides, and particles $<10 \mu\text{g m}^{-3}$ had a positive association with second pregnancy preterm births (range of 17–43% increase in risk). Only one Utah-based study examined the associations of $\text{PM}_{2.5}$ and nitrogen dioxide with spontaneous pregnancy loss [52]. The authors reported a significant 16% increase in the odds of spontaneous pregnancy loss associated with a 10 ppb increase in 7-day levels of nitrogen dioxide, and positive but non-significant associations with 3- and 7-day averages of $\text{PM}_{2.5}$ [52].

7. Other Outcomes

Air pollution appears has additional wide-ranging effects on society that may affect public use of emergency services, education, and mental health. Utah-based studies report significant associations between short-term $\text{PM}_{2.5}$ exposure and emergency services calls for diabetic symptoms, but no significant associations with CV or respiratory symptoms [57]. School absences may also be associated with PM_{10} and $\text{PM}_{2.5}$, with effects varying by the lag periods of interest and scope of the study. A single-county study found a significant association between a 28-day moving average of PM_{10} equal to 100 $\mu\text{g}/\text{m}^3$ with a 2% increase in the rate of school absences in one school district and an elementary school in Utah county [53]. A study of school districts in multiple Utah counties recently reported a similar finding that a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ was associated with a 1.7% increase in daily elementary school absences. These findings were robust even after controlling for structural factors such as seasonal trends across school years, day-of-week effects, holiday effects, and weather [56].

An emerging body of research reported significant associations between suicide completion and increases in specific air pollutants. In Utah, interquartile-range increases in 3-day cumulative averages of nitrogen dioxide and lag-day increases in $\text{PM}_{2.5}$ were associated with an increased suicide risk (OR 1.20, 95% CI: 1.04–1.39 and OR 1.05, 95% CI: 1.01,1.10, respectively). Exposure to nitrogen dioxide during the spring and fall and exposure to $\text{PM}_{2.5}$ during the spring were reported as having significant associations with suicide [55].

Although air pollution has systemic inflammatory effects, few studies in Utah have examined its association with arthritic disease. A single study of preschool aged children living in Utah's Wasatch Front reported significant associations between 14-day increases in $\text{PM}_{2.5}$ concentrations and a significant elevation in the risk of juvenile idiopathic arthritis (JIA) (RR 1.60, 95% CI: 1.00–2.54). The risk was higher in males than females and in patients with systemic onset JIA [54].

Inequities in exposure to air pollutants by racial and ethnic groups have also been observed in Utah [58,59]. A study of air pollution exposure in Salt Lake City estimated that schools with higher proportions of racial/ethnic minority students were consistently exposed to more PM_{2.5} pollution (Hispanics and RRs: 1.02–1.12; non-Hispanic minorities and RRs: 1.01–1.04) [58]. Another study of Salt Lake City residents reported inequalities in air pollution exposure across differences in race, ethnicity, and religion (negative association between PM_{2.5} concentration and White percentage, $p \leq 0.001$) [59].

8. Summary and Conclusions

Utah-based air pollution studies have made important contributions to understanding the health effects of air pollution. As would be expected, the evidence suggests that Utahans experience similar health effects of air pollution as observed elsewhere [61,62]. Figure 1 summarizes key health effects of air pollution based on evidence from the overall scientific literature, with the effects that have been observed in Utah-based studies highlighted [63,64]. Figure 2 is a photograph of the pollution in Utah Valley around the local steel mill, the health effects of which have been examined in multiple studies. The majority of research in Utah has been focused on particulate matter’s effects on human health outcomes. The particulate matter measures in past studies largely originated from industrial emissions, wood burning, and mobile transportation sources. Although the steel mill featured in several Utah-based studies closed in the early 2000s and Utah has seen improvements in air quality since the 1990s, Utah has seen a regular annual increase in emissions of particulate matter pollution from wildfires that are increasing in frequency and severity as a result of warmer and drier conditions due to climate change [65]. Particulate matter from these wildfires originates from fires in Utah and in states across the west coast. The composition of the particulate matter from these wildfires likely differs from the prior particulate matter studied, the health effects of which have yet to be determined.

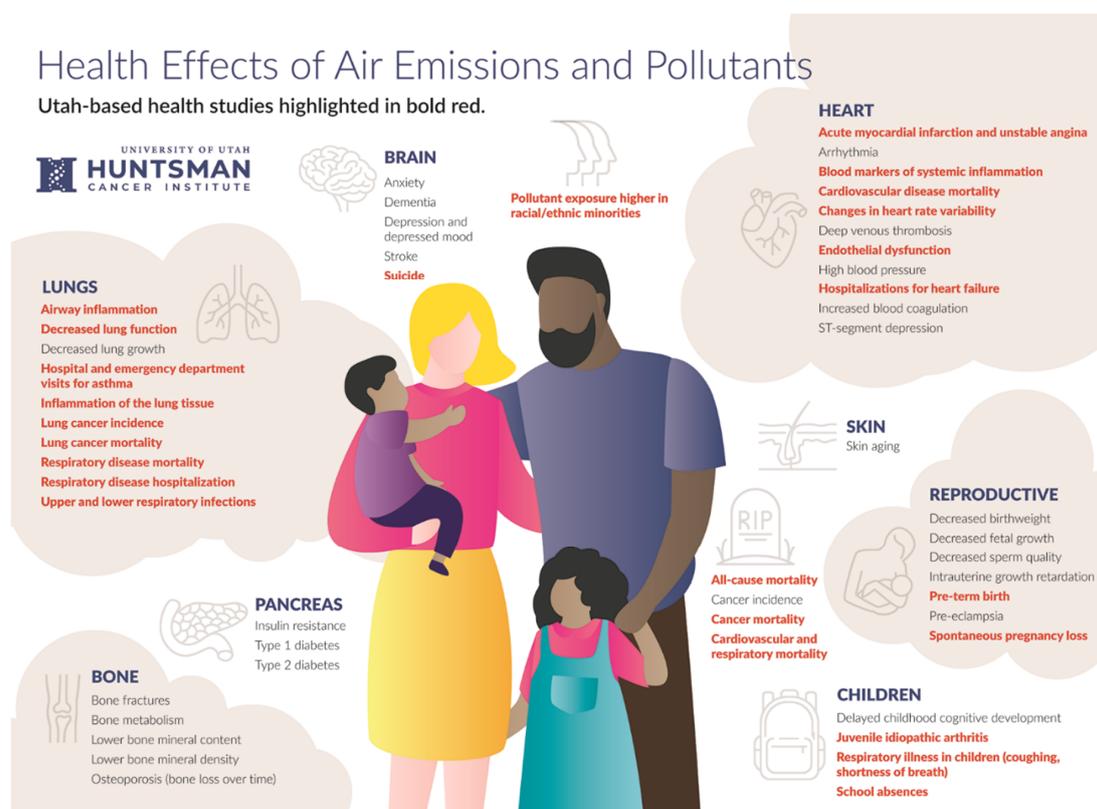


Figure 1. Health effects of air pollution (attached). Adapted from Thurston et al. 2017 [63] and The Utah Roadmap 2020 [64].



Figure 2. Photograph of Geneva Steel, Utah Valley, 1989 (PM_{10} (particles $<10\ \mu\text{m}$ in aerodynamic diameter) approximately $150\ \mu\text{g m}^{-3}$).

Ozone and nitrogen oxides are air pollutants present in Utah, but their health effects on the Utah population are understudied relative to the research on particulate matter pollution. In addition, Utah is one of the nation's largest emitters of toxic air emissions [66], primarily due to a large copper mine located west of the Wasatch Front. Few studies in Utah have examined how exposures to air toxics influence health outcomes such as cancer incidence or cancer mortality. Future directions for air pollution studies in Utah include more human health studies that examine associations between health and wildfire smoke, ozone, nitrogen oxides, air toxics, and the results of multi-pollutant exposures. Furthermore, as new low-cost and mobile air quality measurements are deployed, studies could focus on the spatial pattern of health effects at smaller spatial scales than counties.

Utah has experienced rapid population growth in recent decades largely along the already densely populated Wasatch Front. The majority of Utahans utilize cars as their primary means of transportation. Governmental policies that can increase the availability and use of public transportation and city zoning laws that can reduce the number of residences located near mobile and point sources of pollution are not uniformly implemented across the Wasatch Front. Consequently, air pollution emissions in Utah from mobile sources and chronic exposure to mobile emissions may increase as the population continues to grow unless public policy can accelerate the adoption of low- or zero-emission technologies [64].

Utah-based studies and many others across the world report a substantial number adverse health effects due to air pollution exposure. Despite decades of research, the debate about air pollution and its health implications continues. One of the primary arguments against the findings of air pollution studies is the role of causation in human epidemiologic studies. Opponents of air pollution policies argue that human epidemiologic studies leave too much uncertainty around whether air pollution truly has a casual effect on the adverse health outcomes documented in the literature. Because air pollution exposure is widespread, finding counterfactual populations to demonstrate causality in air pollution studies can be challenging. Utah is home to the nation's only population database, the Utah

Population Database (UPDB), which can create longitudinal residential histories for all Utahans from first residence or birth in the state to death or emigration from the state by linking driver license, vital records, voter registration, and marriage and divorce records to personal identifying information. Data about air pollution exposure at the address level, all medical history and cancer diagnoses for Utah residents, vital status, and family history of disease for all persons living in the state are also available through the UPDB. Future studies on the topic of health outcomes in Utah can address questions regarding causality in epidemiologic studies by leveraging the long-term follow up, comprehensive capture of confounders like smoking, longitudinal residential history, and matching capabilities of the Utah Population Database.

Utah's contribution to the literature on the health impacts of air pollution will continue to grow. Future topics of relevance include studies of governmental policies addressing air pollution exposure; inequities in these exposures; health studies that incorporate air toxics and ozone and nitrogen oxides; understanding how air pollution contributes to COVID-19; effects of wildfires exacerbated by climate change; source apportionment studies and their associated health effects; and sub-county gradients in air pollution. The unique pollution patterns, data resources, and scientific capacity in Utah can be leveraged to address knowledge gaps that remain in this field.

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