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Effects of Ambient Air Pollution on Asthma-Related Emergency Department Visits within the Las Vegas Metropolitan Area

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EFFECTS OF AMBIENT AIR POLLUTION ON ASTHMA-RELATED

EMERGENCY DEPARTMENT VISITS WITHIN THE LAS VEGAS

METROPOLITAN AREA

By

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Abstract

The objective of this research was to evaluate the risk for asthma-related Emergency Department visits and their association with ambient air pollution within the Las Vegas metropolitan area. All data were aggregated by date and ZIP Code. The association was analyzed by applying the distributed lag non-linear model in an attempt to identify elevated concentrations of specific air pollutants as triggers and their delayed effects (lag days). Relative Risk (RR) and 95% confidence intervals were produced, while adjusting for socioeconomic status. This ecological population-based study analyzed daily asthma counts of Emergency Department visits from January 1st, 2009 to December 31st, 2014 (N= 109,550). The exposure-outcome analysis found that when PM_{10} reaches 265 µg/m^3, RR is greater than 1, between 0-2 days lag, dissipates, and peaks between 5-7 days lag. At initial exposure, PM_{10} had a RR of 2.83 (95% CI = 1.11, 7.20). At 7 days lag, PM_{10} reached a RR of 2.91 (95% CI= 1.21, 7.02), supporting that these associations present a non-linear lag effect. Understanding the adverse effects caused by elevated concentrations of criteria air pollutants, particularly when they exceed federal standards, and recognizing that a lag time exists, is a call to action for healthcare providers to educate their patients as to proper exposure prevention strategies and the development of tailored asthma management plans.

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Chapter 1. Introduction

Asthma is a chronic respiratory and lung disease induced by an inflammation and narrowing of the airways leading to bronchial hyper responsiveness and reversible airflow obstruction, and resulting in recurrent symptoms of shortness of breath, chest tightness, wheezing, and coughing (Alhassan et al., 2016; Centers for Disease Control and Prevention (CDC), 2017). Chronic lower respiratory diseases are the third leading cause of death in the United States (US), a significant health burden to both adults and children (CDC, 2016). In 2014, there were 2 million emergency department (ED) visits with asthma as the primary diagnosis – an 11.1% increase from 1.8 million in 2004, in spite of newer methods of detection, intervention and treatment (CDC, 2015; CDC, 2005). Current prevalence has been noted to be higher among some race and ethnic subgroups, such as Puerto Rican heritage (18.8%), black non-Hispanic (11.9%), and those under poverty level (12.4%) (Alhassan et al., 2016). "The strongest risk factors for developing asthma are a combination of genetic predisposition, obesity, viral respiratory infections, allergies, and environmental exposure to inhaled substances and particles that may provoke allergic reactions or irritate the airways," (World Health Organization (WHO), 2017).

The WHO defines ambient air pollution as "a contamination of the environment by any chemical, physical, or biological agent that modifies the

natural characteristics of the atmosphere," (WHO, 2017). Air pollutants of major public health concern include particulate matter (PM), inhalable particles with a diameter less than 10 aerodynamic micrometers (PM_{10}), or 2.5 aerodynamic micrometers ($PM_{2.5}$). PM is comprised of dust, dirt, soot, smoke and other particles and liquid droplets that are easily inhaled and that may lead to serious health problems. $PM_{2.5}$, also known as "fine particle" poses the greatest problem as it can penetrate deep into the lungs, possibly entering the bloodstream (Pope & Dockery, 2006). Other criteria air pollutants, like carbon monoxide (CO), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2), are also hazardous. The respiratory tract is most susceptible to air pollution as it is continually exposed to the ambient environment. O_3, NO_2, and $PM_{2.5}$ can induce airway inflammation, while O_3 and NO_2, can induce airway hyper-responsiveness – two characteristic features of asthma (Guarnieri & Balmes, 2014; Noyes et al., 2009).

Asthma affects people of every age, gender and race, but utilization of emergency department (ED) services is disproportionate. In Southern Nevada, for the years 2000-2008, Blacks and Hispanics had increased rates for hospitalization and ED use (Moonie, Segev, Shan, Pergola & Teramoto, 2015). Low income and minority populations showed increased rates for ED visits and decreased rates in access to primary care. This validates an existing health disparity among the uninsured or underinsured who rely on ED visits for treatment as opposed to

accessing a primary care physician for asthma care and management. This finding is consistent with a higher prevalence of asthma among those below the 100% poverty level. The longitudinal trend also revealed that children's asthma-related ED visits significantly increased over time (Moonie, Seggev, Shan, Pergola, & Teramoto, 2015). Among Nevadans, of whom 8.1% are asthmatic, lack of exercise correlates with an increased prevalence of asthma (Teramoto & Moonie, 2011), and "children with asthma have a greater risk of absenteeism associated with grade retention," (Moonie,Cross, Guillermo & Gupta, 2010). However, the effects of other risk factors, such as ambient air pollution, have not been assessed sufficiently for the Las Vegas Valley, where three fourths of Nevadans reside. According to the CDC, more than half of asthmatic children reported one or more attacks in 2016, and every year, 1 in 6 children with an asthma diagnosis has an ED visit (CDC, 2018). The average medical cost of asthma approximated $1,000 per child in 2012, not including indirect costs such as missed school days and parents' missed work days (CDC, 2018).

While changes in government policy would have the most significant impact, education, prevention and proper asthma management are individual-level interventions that mitigate effects to air pollution exposure. In 2013, only 51% of asthmatic children were given an action plan by their healthcare provider and only 46% were given advice on environmental control. In contrast, 76% were taught to

recognize early signs and symptoms of an attack and 80% were taught how to best respond (CDC, 2018). These numbers illustrate the gap between prevention and treatment. A better understanding of the adverse effects of air pollution at high concentrations as well as the duration of these effects can improve exposure reduction strategies as part of asthma action plans.

Chapter 2. Background

In metropolitan cities that have and continue to experience urbanization and expansion, such as Las Vegas, traffic-related air pollution (TRAP) is of significant health concern (Green et al., 2013). Oxidative stress, which occurs in severe asthmatic cases, has been associated with exposures to O_3, NO_2, and $PM_{2.5}$ (Guarnieri & Balmes, 2014). Over the years, research has suggested that ambient air pollutants aggravate pre-existing asthma, but not until recently, has the idea been explored that ambient air pollution causes new onset of asthma symptoms. Environmental exposures affect the development of lung function in early childhood and into adulthood, which is a major determinant of future respiratory health (Abelsohn & Stieb, 2011; Gauderman et al., 2004). "TRAP, particularly $PM_{2.5}$, negatively affects lung development with potential consequences for the development of asthma and chronic obstructive pulmonary disease," (Burbank, Sood, Kesic, Peden, & Hernandez, 2017; Dong et al., 2011; Q. Yu et al., 2017).

The southwest United States exemplifies the existing relationship between climate change and asthma. Global warming, combined with other weather conditions influenced by greater temperature variations caused by El Nino and La Nina, have increased forest fires (Barne et al., 2013). Wildfire smoke occasionally causes $PM_{2.5}$ levels to exceed far beyond National Ambient Air Quality Standards set by the US Environmental Protection Agency (Garfin, 2013). Climate change

continues to increase temperature extremes (i.e., heat waves) and impacts the
environmental distribution and biological effects of chemical toxins. By altering
temperature, air circulation patterns, precipitation, and salinity, global warming
will increase the risk of O_3 and particulate matter (Noyes et al., 2009). All of
these exposures have subsequent effects on respiratory health, specifically those
at risk for asthma.

An exposure-response effect to ambient air pollution has been identified in
asthmatics as well as in healthy individuals. Exposure to elevated concentrations
of criteria air pollutants correlates with reduced air flow of the lungs, with the
most significant exposures being ozone and particulate matter (McDonnell,
Stewart, & Smith, 2007; Schelegle, Morales, Walby, Marion, & Allen, 2009). In
the literature, relationships between higher ozone levels and increased ED visits
and hospital admissions for asthma have been investigated. Inversely, improved
air quality correlates with a drop in ED visits as observed in both controlled
experimental exposure studies (chamber studies) and within the natural
environment (field studies) (Gauderman et al., 2015; Li, Wang, Kan, Xu, & Chen,
2010). More specifically, during the 2008 Olympic and Paralympic Games, when
the Chinese government placed restrictions on transportation and industrial
emissions in an effort to mitigate poor air quality and associated negative effects.
Li et. al reported a drop in the average of asthma-related outpatient visits from

12.5 -prior to government imposed restrictions, to 7.3 during the time of the games, after restrictions were in effect. (Li, Wang, Kan, Xu, & Chen, 2010).

Studies have suggested that the exposure-response relationship between air pollutants and respiratory disease is non-linear, meaning negative health outcomes (such as ED visits) do not increase in proportion to pollutants' level of concentration but rather increase more or less rapidly than a linear relationship would predict (Nasari et al., 2016; Rabinovitch, Silveira, Gelfand, & Strand, 2011; H. L. Yu & Chien, 2016). Comparing a linear fit and log-linear fit model to illustrate the association among ambient air particulate matter and asthma mediators, Rabinovitch et al. (2011) found such relationship to be the steepest at lower $PM_{2.5}$ exposure while tapering off at the highest levels of exposure, producing a curve and suggesting a nonlinear dose-response relationship. Nonlinear exposure-response curves with similar properties are found in recent literature that calls for future research to better understand the underlying mechanisms leading to nonlinear forms and statistical models that better estimate the "shape of the exposure-response function," (Nasari et al., 2016; Pope et al., 2009; Smith & Peel, 2010).

Associations between increased concentrations of criteria air pollutants and negative health effects on the respiratory system are present not only on days when concentrations peak at lag "0" (meaning effects were observed on the same

day of exposure), but some persons may experience a delayed effect, where clinical symptoms manifest days after initial exposure. There is some variability within the literature as to how many days lag before an increase in ED visits is observed. While much of the literature indicates an increase in ED visits occurs at peak exposure or within several days, fewer studies have explored delayed effects taking place at as long as 21 and 30 days (Liu,2016; Xu, 2013; Zhang, 2013). Because these studies are few, and delayed effects can be potentially observed past the 21 days, more studies evaluating a longer lag are needed. A recent study investigated the association between asthma hospital visits and O_3 concentration in Maricopa County, Arizona for the years 2007–2012, finding that the effect of higher O_3 levels on asthma-related hospital visits may persist after initial exposure (Mohamed, Goodin, Pope, Hubbard, & Levine, 2016), which is consistent with previous findings (Qiu et al., 2015; Q. Xu et al., 2016). For a more comprehensive analysis of air pollutant effects on asthma-related ED visits, we will consider multiple air pollutants and explore a lag period of at least 3 weeks.

Objective

The objective of this research is to evaluate the association between asthma-related ED visits and ambient air pollution within the Las Vegas, metropolitan area during the years 2009-2014. This study aims to identify the leading air pollutant in predicting asthma incidence. As a secondary aim, this

research hopes to establish how many days lag before a peak in asthma incidence occurs after a particular concentration of an air pollutant is observed. Currently, an existing lag time between exposure-response effects has not yet been established. Findings within this study may possibly contribute to the growing literature on the health effects of air pollution on asthma.

Research Questions

Question #1: Is the length of lag effect that must be considered for each air pollutant 7, 14, 21 or 28 days?

Question # 2: Based on the chosen length of lag, is a significant non-linear association between air pollutant concentration and ED visits observed?

Question # 3: Does incorporating additional air pollutants for a multivariate analysis render a better model in comparison to a univariate model?

Hypothesis

$H^1 0$: The length of lag, time between exposure and response effect, which must be considered for all pollutants is 21 days.

$H^1 a$: The length of lag effect that must be considered for all pollutants is not 21 days.

Expected outcome: Associations between criteria air pollutants and asthma will vary by type of pollutant quantitatively. Certain pollutants may have an exposure-response effect, where higher concentrations will increase the risk of ED visits but may take up to 21 days.

H^{2}0: Based on the chosen length of lag, a significant non-linear association between air pollutant and ED visits will not be observed.

H^{2}a: Based on the chosen length of lag, a significant non-linear association between air pollutant concentration and ED visits will be observed.

Expected outcome: While considering temporal dependency, it is expected that the association between air pollutant and ED visits will be non-linear as supported in the literature.

H^{3}0: Incorporating additional air pollutants for multivariate analysis does not render a model that is better fit to explore the association when compared to a univariate model.

H^{3}a: Incorporating additional air pollutants for multivariate analysis renders a model that is better fit to explore the association when compared to a univariate model.

Expected Outcome: It is expected that a multivariate analysis including additional

pollutant(s) wouldbe a better model to explore the association than a univariate

model, being that more predictors are being considered.

Chapter 3. Methods

Study Design

      This ecological population-based study takes place within the Las Vegas metropolitan area using aggregate count data. The time-series design uses daily counts of asthma ED visits from January 1st, 2009 to December 31st, 2014 as the dependent variable. Independent variables include air pollutants O_3, PM_{10}, $PM_{2.5}$ at 1 hour and 24 hours, and CO at 1 hour and 8 hours. The association was analyzed using the distributed lag non-linear model (DLNM) (Gasparrini, 2011) and controlled for socioeconomic status (SES), in other words, the effect that age, race, gender, household income and insurance coverage would have on ED visits in the calculation of RR with 95% confidence intervals (CIs) to determine significance.

Study Area

      Las Vegas is located in Nevada's Mojave Desert, within a basin, in the southern tip of Nevada as illustrated in Figure 1. It is surrounded by mountains on all sides with a rocky landscape, desert vegetation, wildlife and sits in a wide pass that leads to Lake Mead and Hoover Dam (McNamee, 2018). Las Vegas is the 28th most populated city in the US with an estimated 632,912 inhabitants (U.S. Census Bureau, 2016) and is the county seat for Clark County, which also includes the city of North Las Vegas, Henderson, and several unincorporated towns. As an agglomeration, this metropolitan area is known as Las Vegas

Valley. The population estimate is 2,155,664 inhabitants, of which 43.6% are

White Non-Hispanic, 30.9% are Hispanic or Latino, 12.2% are black or African-

American and 10.3% are Asian (U.S. Census Bureau, 2016). Clark County

continues to be the fastest growing metropolis of the US since the end of the last

century, with Las Vegas as an economic engine, making it a world-renowned

tourist destination with an established presence in commerce, international

business, entertainment, and urban development (McNamee, 2018).

Figure 1. Las Vegas, Nevada
Source: Google Maps (2018)

Data Sources

Socioeconomic status variables for the Las Vegas Metropolitan Area were

extracted from PolicyMap, a geographic information system (GIS) tool that

facilitates data mapping in order to explore geospatial relationships between

social phenomena (www.policymap.com). PolicyMap data sets are supplied by both public and private sources. The demographic data used in this study (race, gender, age, household income and health insurance) was extracted from The American Community Survey where race and gender were percent calculations for all people in 2010, while values for age, household income and health insurance were the estimated typical values between 2011-2015.

Figure 2. Zip Code Map of Air Pollution Monitoring Stations in Las Vegas
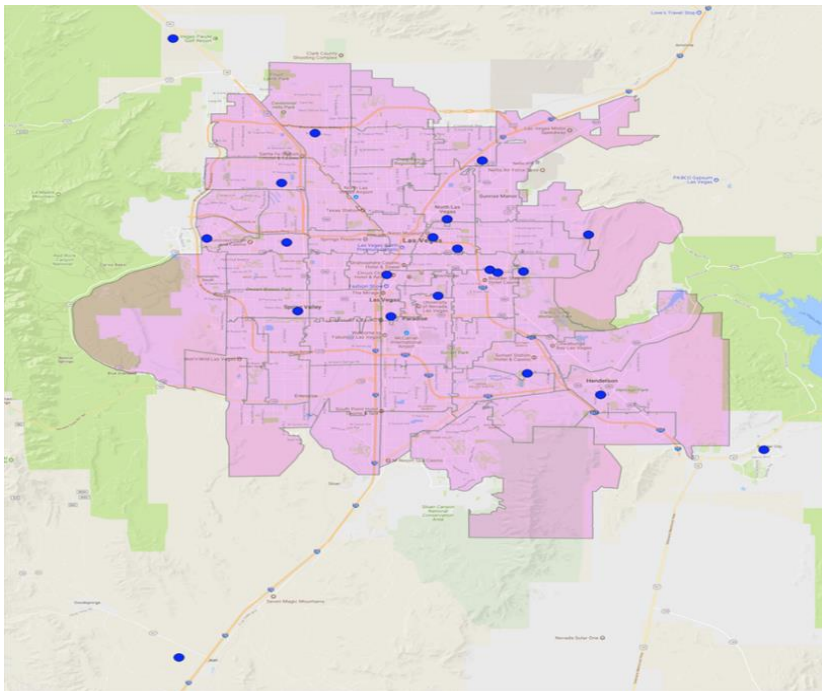
Air quality data were acquired from the Environmental Protection Agency (EPA) Air Quality System (AQS). The Clark County Air Quality Department

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(CCDAQ) operated an air quality monitoring network measuring hourly criteria air pollutants in the Las Vegas Valley. Figure 2 is a ZIP Code map showing the matrix of monitoring sites and data coverage. The blue dots represent the location of the 21 pollution monitoring stations, two of which were located within the same zip code and for which an average of the two daily measurements were used. After quality assurance, these data were reported to EPA AQS and made available to the public.

Daily asthma counts of ED visits from January 1st , 2009 through December 31st, 2014, were obtained from Center for Health Information Analysis (CHIA) at the University of Nevada, Las Vegas (UNLV). The State of Nevada, Department of Health and Human Services, Division of Healthcare Financing and Policy, contracts the services of CHIA "in the collection, and analysis of patient billings claims data from Nevada hospitals and Ambulatory Surgical Centers (ASC's) pursuant to NRS 449.485 and NAC 449.951-449.969," (www.chiaunlv.com). This mandates all Nevada hospitals and ASCs send CHIA every patient billing record that is produced. Daily values for asthma ED visits are aggregate count data. The original data are individual data where personal identifiers were de-identified and anonymized.

An IRB approval was not sought.

Theoretical Framework

The DLNM is a modelling framework that permits for the analysis of an exposure-response relationship of which effects may be delayed, also termed an *exposure lag-response association.* DLNM relaxes the linearity assumption and better incorporates the time dimension, recognizing a relationship across lags/ lag days as opposed to them being independent values (Chien, 2016; Gasparrini, 2011). Such flexibility accommodates the effect variations that simultaneously occur along the space of the predictor variable as well as in the lag dimension of occurrence. Combining the basis functions of these two dimensions (i.e., air pollutants and lag days) creates a cross-basis function, the foundation for the DLNM modelling framework.

Statistical Analysis

The DLNM incorporates exposure, confounders, and time, and adopts "a bi-dimensional perspective to represent associations which vary non-linearly along the space of the predictors and lags,"(Gasparrini, 2011). Attempting to compute the RR of ED visits, we assume that Y_{it} represents the count of ED visits at time t and location i, and follows a Poisson distribution by $Y_{it} \sim \text{Poisson}(\mu_{it})$, and we use the following model:

$$\text{Log}(\mu_{it}) = \alpha + \Sigma f(AP_{it}, lag=l) + f(t) + \Sigma\beta(SES)_i + \alpha i + \text{offset}$$

where μ_{it} is the expected value of Y_{it} and α is the intercept of the model. The function $f(AP_{it}, \text{lag}= l)$, is the cross-basis function of an air pollutant, which simultaneously describes the dependency among the space of the air pollutant and lag dimension, capturing both non-linear and delayed effects of air pollutants on ED visits. Lags will be explored from zero (present day) up to 28 days, and we may consider at most two air pollutants in the same model. The time smoother, $f(t)$, is a cubic spline for controlling temporal autocorrelations (Chien & Yu, 2014). Covariate vector $(SES)_i$, represents socioeconomic variables, and β is the corresponding coefficient vector. The random effect term, αi, is in place to explain remaining variations of asthma incidence by unobserved predictors. The offset is the logarithm of population (Chien & Yu, 2014).

For over-dispersed count data, a quasi-Poisson link function will be used rather than a Poisson link function in the DLNM. Quasi-Akaike Information Criterion (QAIC) estimates the quality of a model among various statistical

QAIC = −2(log-likelihood function) + 2(# of parameters) × (over dispersion coefficient)

models tested for the same set of data, providing a means for model selection. To compare full and reduced models, we will rely on QAIC values, as the least QAIC value will indicate which is the optimal model.

Statistical computing and graphics for this analysis are supported by R programming language. R 3.4.2 with packages "dlnm", "mgcv" (Mixed GAM

Computation Vehicle with automatic smoothness estimation) and "splines"

(function that describes smooth curves) will be used, facilitating the creation of

high level functions with multiple parameters. Three-dimensional (3D) graphical

representations and contour plots of overall and lag-specific effects will depict the

association.

Chapter 4. Results

Summary Statistics

Summary statistics for SES variables and criteria air pollutants are presented in Table 1. In 2010, it is estimated that 60.9% of the population was white, 50.2% were male and the average age was 37 years. Between 2011 and 2015, the estimated typical value of household income was $54,124.00 and 81.1% of the population had health insurance coverage. Mean values for all criteria air pollutants are under National Ambient Air Quality Standard (NAAQS) as set by the EPA, however the max values for O_3 (0.080 ppm), PM_{10} (267.00 ug/m^3) and 1hr $PM_{2.5}$ and 24hr $PM_{2.5}$ (78.75 ug/m^3 and 78.70 ug/m^3) are in excess from the recommended standard of 0.070 ppm for O_3, 150 ug/m^3 for PM_{10} and 35 ug/m^3 for 24hr $PM_{2.5}$. The total sample size is 109,550 as 6 years of daily measurements were taken for 50 ZIP Codes (2,191 days $\times$ 50 ZIP Codes). There were 21 air pollution monitoring stations, resulting in 29 (58%) ZIP Codes obtaining an imputed estimation from surrounding monitoring stations as they did not have a station of their own. A correlation matrix of air pollutants and asthma ED visits is shown in Table 2, indicating that O_3 is negatively correlated to 1hr CO (correlation coefficient = -0.75) and 8hr CO (correlation coefficient = -0.74), as well as a strong correlation between 1hr CO and 24hr $PM_{2.5}$ (correlation coefficient = 0.80). Naturally, the same air pollutant will be highly correlated to itself as observed at different time interval measurements for CO and $PM_{2.5}$.

Table 1. Summary Statistics for Air Pollutants and SES. Abbreviations: Carbon monoxide (CO); Ozone (O_3); Particulate matter with inhalable particles < 10 aerodynamic micrometers (PM_{10}); and Particulate matter with inhalable particles < 2.5 aerodynamic micrometers ($PM_{2.5}$). *Denotes percent taken of all people in 2010. **Denotes estimated typical values between 2011-2015.

	Mean	SD	Min	Q1	Median	Q3	Max
SES							
White% *	60.90	12.33	28.92	53.75	59.39	71.61	85.30
Male% *	50.24	2.06	46.48	49.21	49.72	50.32	59.11
Age **	37.22	5.63	27.00	34.00	37.00	41.00	63.00
Household ** Income	54,124.00	16,315.36	22,392	42,145	53,082	65,410	88,899
Health ** Insurance%	81.14	8.31	62.7	73.73	83.02	87.63	93.44
Pollutant							
Daily CO (ppm) based on 1hr data	0.55	0.29	0.00	0.33	0.48	0.72	3.10
Daily CO (ppm) based on 8hr data	0.56	0.30	0.00	0.33	0.47	0.72	3.11
Daily O_3 (ppm)	0.03	0.01	0.00	0.02	0.03	0.04	0.08
Daily PM_{10} (ug/m^3)	22.37	8.32	0.00	17.00	21.91	26.84	267.00
Daily $PM_{2.5}$ (ug/m^3) based on 1hr data	8.21	2.64	0.00	6.31	7.77	9.58	78.75
24hr $PM_{2.5}$ (ug/m^3) based on 24hr data	8.25	2.48	0.00	6.71	7.89	9.34	78.70

Table 2. Correlation Matrix of Criteria Air Pollutants and Asthma ED count. Abbreviations: Carbon monoxide (CO); Ozone (O_3); Particulate matter with inhalable particles < 10 aerodynamic micrometers (PM_{10}); and Particulate matter with inhalable particles < 2.5 aerodynamic micrometers ($PM_{2.5}$). (*) Denotes highly correlated variables.

	1hr CO	8hr CO	O_3	PM_{10}	1hr $PM_{2.5}$	24hr $PM_{2.5}$	Asthma ED
1hr CO	1.00	**0.97***	**-0.75***	0.04	0.21	0.26	-0.09
8hr CO		1.00	**-0.74***	0.04	0.21	0.25	-0.08
O_3			1.00	0.04	-0.11	-0.12	-0.03
PM_{10}				1.00	0.43	0.44	-0.05
1hr $PM_{2.5}$					1.00	**0.80***	0.03
24hr $PM_{2.5}$						1.00	0.03
Asthma ED							1.00

Time series plots in Figure 3 show a clear seasonality in the temporal

variation of asthma ED visits and individual criteria air pollutants. The study area

had 0.75 daily asthma-related ED visits (SD = 1.23). Higher counts for ED visits

Figure 3. Temporal Variation of Asthma ED Visits and Individual Criteria Air Pollutants, 2009-2014.
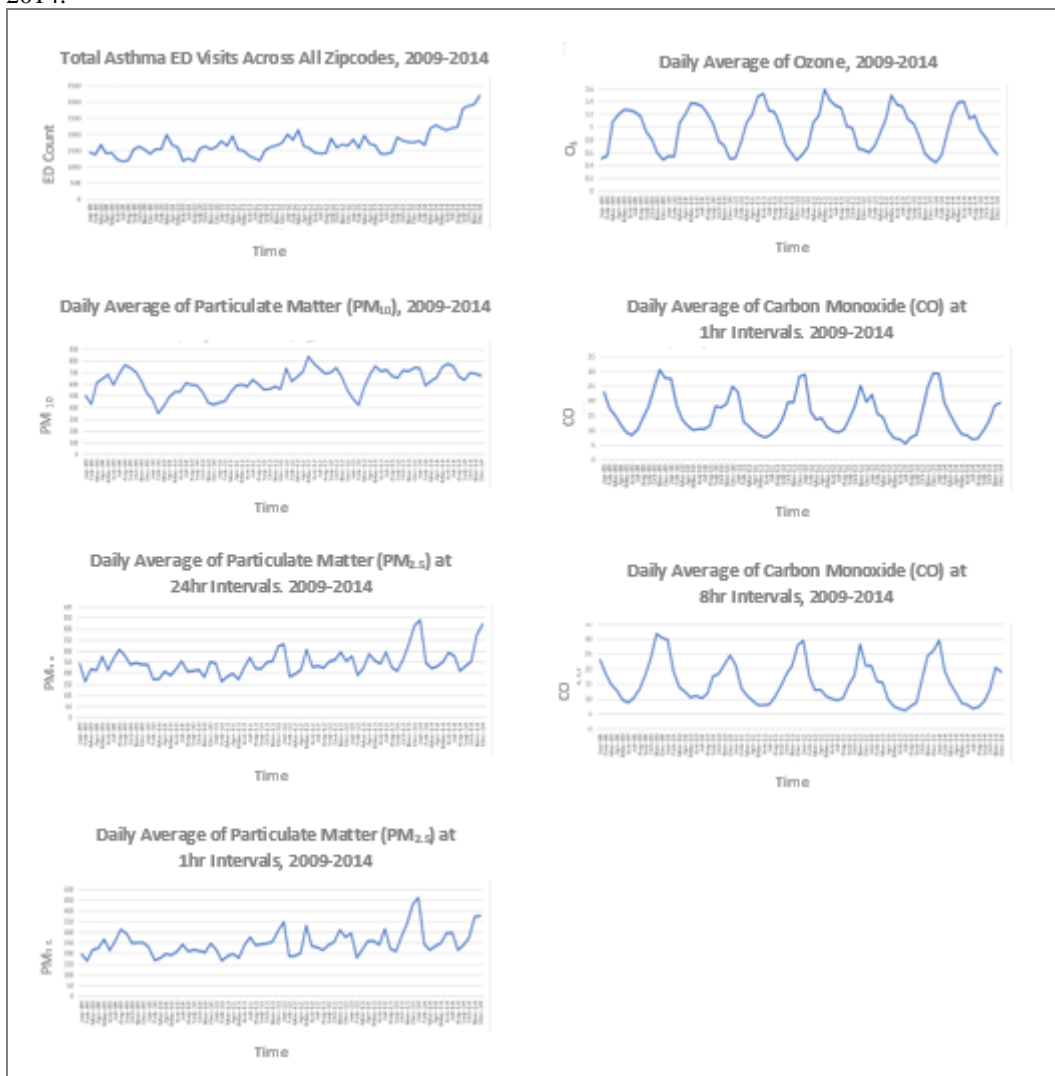
seem to have occurred in spring and in winter. Both asthma ED visits and individual criteria air pollutants show a steady incline as of February and peak in spring when criteria air pollutants are likely to exceed ambient air quality standards.

Question #1: Is the length of lag effect that must be considered for each air pollutant 7, 14, 21 or 28 days?

Lag effect was explored at 7, 14, 21 and 28 days lag for all pollutants and determined by least QAIC values as shown in Table 3. All pollutants rendered the least QAIC value at 7 days lag, rejecting $H^1 0$, which hypothesized 21 days as the lag effect to be considered for all pollutants. Three degrees of freedom *(df)* for lag space were adopted from the literature (Wang & Lin, 2014; Z. Xu et al., 2013) while 3*df*, 4*df* and 5*df* were explored for all pollutants and determined by QAIC values as shown in Table 4. The optimal degrees of freedom for all pollutants was 3*df* and of all pollutants, PM_{10}, had the least QAIC indicating PM_{10} is the best univariate model to explore the association.

Question # 2: Based on the chosen length of lag, is there a significant non-linear lag effect?

In the univariate model, Figure 4 shows the distributed lag non-linear model results of pollutant PM_{10} effect (2009-2014). Figure 4(a) demonstrates the effect

of PM_{10} concentration change on the risk for ED visits, and suggests that a drastic

increase in risk occurred when PM_{10} exceeded $200\,\mu g/m^3$ at both day of initial

exposure (lag 0) and at 7 days lag. In figure 4(b), the RR reached its maximum

Table 3. Model selection for days lag by Quasi-Akaike information criterion (QAIC). Value are set at 7, 14, 21 and 28 days lag across all pollutants. Abbreviations: Carbon monoxide (CO); Ozone (O_3); Particulate matter with inhalable particles < 10 aerodynamic micrometers (PM_{10}); and Particulate matter with inhalable particles < 2.5 aerodynamic micrometers ($PM_{2.5}$). * Denotes the least QAIC value per pollutant.

| Air Pollutant	7 days lag	14 days lag	21 days lag	28 days lag
1hr CO (ppm)	2684443*	2684594	2684926	2685283
8hr CO (ppm)	2684393*	2684520	2684941	2685337
O_3 (ppm)	2683778*	2683943	2684357	2684821
PM_{10} ($\mu g/m^3$)	2683038*	2683212	2683747	2684318
1hr $PM_{2.5}$ ($\mu g/m^3$)	2684502*	2684703	2685084	2685366
24hr $PM_{2.5}$ ($\mu g/m^3$)	2684683*	2684947	2685390	2685770

Table 4. Model selection for degrees of freedom by Quasi-Akaike information criterion (QAIC). Degrees of freedom (df) in the basis function of lag are set at 3 and the (df) in the basis function of air pollutants are tested at 3df, 4df and 5df across all pollutants. Abbreviations: Carbon monoxide (CO); Ozone (O_3); Particulate matter with inhalable particles < 10 aerodynamic micrometers (PM_{10}); and Particulate matter with inhalable particles < 2.5 aerodynamic micrometers ($PM_{2.5}$). *Denotes the model with the least QAIC value.

| Air Pollutant	3df	4df	5df
1hr CO (ppm)	2684443	2684808	2685178
8hr CO (ppm)	2684393	2684736	2685116
O_3 (ppm)	2683778	2684190	2684573
PM_{10} ($\mu g/m^3$)	2683038*	2683440	2683851
1hr $PM_{2.5}$ ($\mu g/m^3$)	2684502	2684907	2685283
24hr $PM_{2.5}$ ($\mu g/m^3$)	2684683	2685049	2685367

when PM_{10} concentrations were > 250 ($\mu g/m^3$), as observed in red during initial

exposure (lag 0), which gradually dissipates by 2 days lag and gradually increases

as of 5 days lag, reaching a similar maximum at 7 days lag. Figure 4(c)

demonstrates that when PM_{10} reached 265 $\mu g/m^3$, the RR was greater than 1

between 0-2 days lag and again between 5-7 days lag. At initial exposure, PM_{10}

had a RR of 2.83 (95%CI= 1.11, 7.20). At 7 days lag, PM_{10} reached a RR of 2.91

(95% CI= 1.21, 7.02), meaning that PM_{10} concentrations of 265 $\mu g/m^3$, are

Figure 4. Univariate analysis: 3D graph, contour plot and slice plot illustrating relative risk (RR) of asthma-related ED visits at lagged days along pollutant concentration, where the reference levels are PM_{10} at mean value, 22.37($\mu g/m^3$) and present day (lag 0). **(a)** 3D plot depicts variation of RR of asthma related ED visits along with pollutant concentration and lagged day; **(b)** The contour plot shows hot spots of RR of asthma related ED visits with pollutant concentration along the x-axis and lag days along y-axis. **(c)** Exposure-outcome analysis (x-axis indicates days lagged, y-axis indicates the RR outcome. The grey zone indicates the 95% confidence interval of estimated RR under the specific exposure-outcome value.
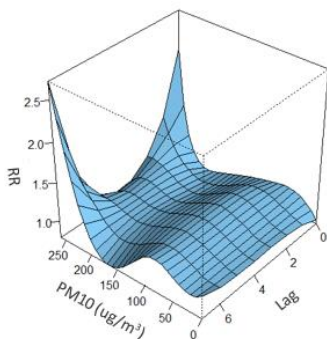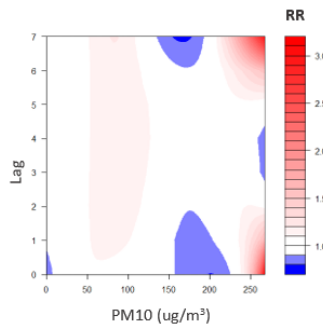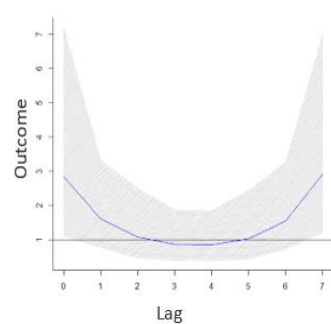
a. 3D Graph (PM_{10})   *b. Contour Plot* (PM_{10})   *c. Slice Plot* (PM_{10})

associated with a more than doubled risk for an asthma related ED visit at both

initial exposure and at 7 days lag. These results support H^2a in that a significant

non-linear association between air pollutant concentration and ED visits will be

observed.

Question #3: Does incorporating additional air pollutants for a multivariate

analysis render a better model in comparison to a univariate model?

Table 5 shows the 29 models considered for multivariate analysis. It was

determined that the model with 2 cross-basis functions, including PM_{10} and 1hr

$PM_{2.5,}$ was the optimal multivariate model as it rendered the lowest QAIC value.

However, compared to the univariate model, the multivariate model had a higher

QAIC value, supporting H^30, in that incorporating additional air pollutants for

multivariate analysis does not render a model that is better fit to explore the

association. For comparison, distributed lag non-linear models for the multivariate

model were plotted and are shown in Figure 5 for predictions of PM_{10}, while

predictions of 1hr $PM_{2.5}$ are shown in Figure 6. Based on QAIC values, the

alternative multivariate model with 2 cross-basis functions included pollutants

PM_{10} and O_3, see Supplement Figure 1 in Appendix for PM_{10} prediction results.

Figure 5(a) demonstrates the variation of the RR along with 1hr PM_{10}

concentration and lag. Figure 5(b) presents a hot spot at initial exposure between

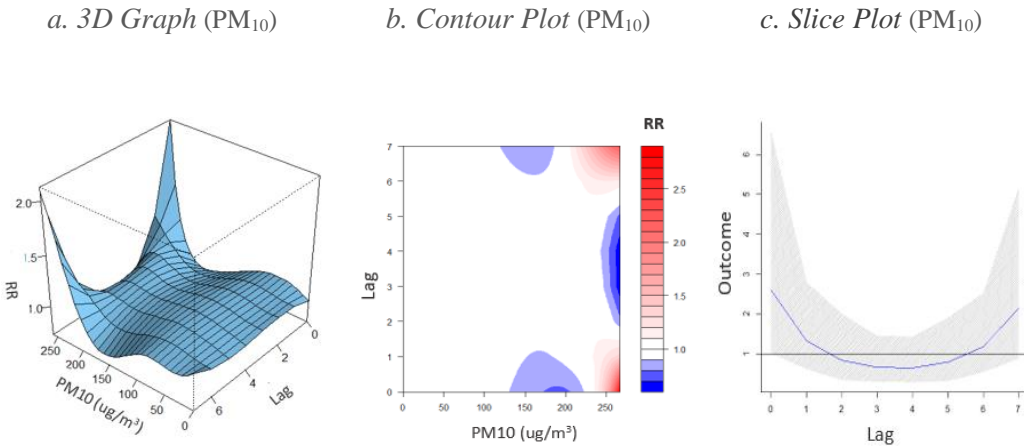lag 0-1, when 1hr PM_{10} levels exceeded 250 $\mu g/m^3$, and another between lag 6

and 7. The corresponding 265 μg/m^3 slice plot, figure 5(c), indicates the RR was

greater than 1 as of initial exposure and up until 1.5 days lag with a RR of 2.60

(95%CI= 1.01, 6.55) and again after 5 days lag, reaching a RR of 2.14 (95%

Table 5. Model selection by Quasi-Akaike information criterion of 2,3 and 4 cross-basis functions. Abbreviations: Carbon monoxide (CO); Ozone (O$_3$); Particulate matter with inhalable particles < 10 aerodynamic micrometers (PM$_{10}$); and Particulate matter with inhalable particles < 2.5 aerodynamic micrometers (PM$_{2.5}$). *Denotes the lowest QAIC value.

2 cross-basis functions	QAIC	3 cross-basis functions	QAIC
1hr CO + O$_3$	2685396	1hr CO + O$_3$ + PM$_{10}$	2685670
1hr CO + PM$_{10}$	2684665	1hr CO + O$_3$ + 1hr PM$_{2.5}$	2687390
1hr CO + 1hr PM$_{2.5}$	2686227	1hr CO + O$_3$ + 24hr PM$_{2.5}$	2687353
1hr CO + 24hr PM$_{2.5}$	2686450	1hr CO + PM$_{10}$ + 1hr PM$_{2.5}$	2685873
8hr CO + O$_3$	2685331	1hr CO + PM$_{10}$ + 24hr PM$_{2.5}$	2686481
8hr CO + PM$_{10}$	2684617	8hr CO + O$_3$ + PM$_{10}$	2685630
8hr CO + 1hr PM$_{2.5}$	2686190	8hr CO + O$_3$+ 1hr PM$_{2.5}$	2687332
8hr CO + 24hr PM$_{2.5}$	2686400	8hr CO + O$_3$ + 24hr PM$_{2.5}$	2687290
O$_3$ + PM$_{10}$	2684614	8hr CO + PM$_{10}$ + 1hr PM$_{2.5}$	2685836
O$_3$ + 1hr PM$_{2.5}$	2685660	8hr CO + PM$_{10}$ + 24hr PM$_{2.5}$	2686446
O$_3$ + 24hr PM$_{2.5}$	2685823	O$_3$ + PM$_{10}$ + 1hr PM$_{2.5}$	2685309
PM$_{10}$ + 1hr PM$_{2.5}$	**2684519***	O$_3$ + PM$_{10}$ + 24hr PM$_{2.5}$	2685768
PM$_{10}$ + 24hr PM$_{2.5}$	2685009		
4 cross-basis functions	**QAIC**		
1hr CO + O$_3$ + PM$_{10}$ + 1hr PM$_{2.5}$	2687334		
1hr CO + O$_3$ + PM$_{10}$ + 24hr PM$_{2.5}$	2687707		
8hr CO + O$_3$ + PM$_{10}$ + 1hr PM$_{2.5}$	2687296		
8hr CO + O$_3$ + PM$_{10}$ + 24hr PM$_{2.5}$	2687665		

CI=0.89, 5.14). Figure 6(a) demonstrates the variation of RR along 1hr $PM_{2.5}$

concentration and lag. The contour plot, figure 6(b), shows a hot spot as of initial

exposure which extends through 3 days lag, dissipates, and returns at 7 days lag

Figure 5. Multivariate Analysis Predicting PM_{10}: 3D graph, contour plot and slice plot illustrating relative risk (RR) of asthma-related ED visits at lagged days along pollutant concentration, where reference levels are PM_{10} at mean value, 22.37µg/m³ and present day (lag 0). **(a)** 3D plot depicts variation of RR of asthma related ED visits along with pollutant concentration and lagged day; **(b)** The contour plot shows hot spots of RR of asthma related ED visits with pollutant concentration along the x-axis and lag days along y-axis. **(c)** Exposure-outcome analysis (x-axis indicates days lagged, y-axis indicates the RR outcome. The grey zone indicates the 95% confidence interval of estimated RR under the specific exposure-outcome value.

a. 3D Graph (PM_{10})   *b. Contour Plot* (PM_{10})   *c. Slice Plot* (PM_{10})

when 1hr $PM_{2.5}$ levels surpass 50 µg/m³. The corresponding slice plot, figure 6(c),

indicates the RR was greater than 1 between 0-3 days lag, having a maximum RR

of 1.24 (95% CI= 0.62, 2.47) at 1 day lag and a RR of 1.20 (95%CI= 0.46, 3.15)

at 7 days lag, however, the range for confidence intervals falls below the value of

1, rendering this result insignificant.

Figure 6. Multivariate Analysis Predicting 1hr PM$_{2.5}$: 3D graph, contour plot and slice plot illustrating relative risk (RR) asthma-related ED visits at lagged days along pollutant concentration, where reference levels are 1hr PM$_{2.5}$ at mean value, 22.37μg/m^3 and present day (lag 0). **(a)** 3D plot depicts variation of RR of asthma related ED visits along with pollutant concentration and lagged day; **(b)** The contour plot shows hot spots of RR of asthma related ED visits with pollutant concentration along the x-axis and lag day**s** along y-axis. **(c)** Exposure-outcome analysis (x-axis indicates days lagged, y-axis indicates the RR outcome). The grey zone indicates the 95% confidence interval of estimated RR under the specific exposure-outcome value.

a. 3D Graph (1hr PM$_{2.5}$)   *b. Contour Plot* (1hr PM$_{2.5}$)   *c. Slice Plot* (1hr PM$_{2.5}$)
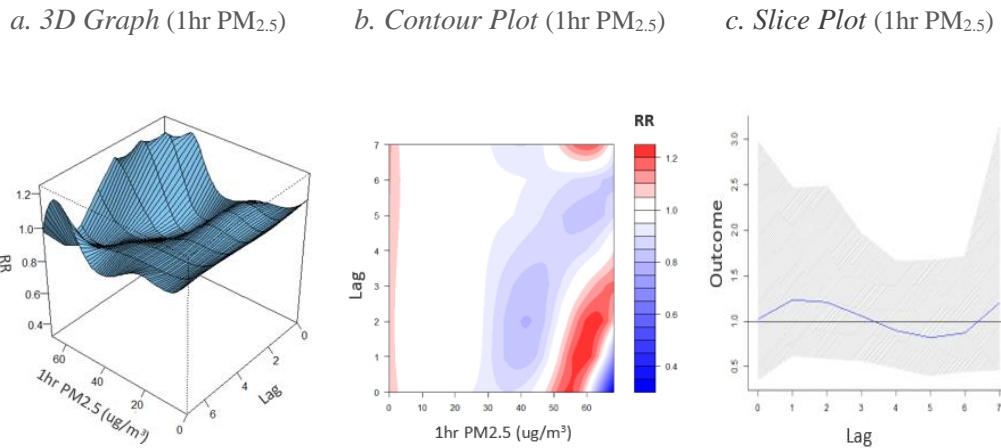
Chapter 5. Discussion

This study aimed to explore the association between critical air pollutants and asthma ED visits while considering the temporal dependency between exposure and outcome. The analysis determined, by means of QAIC values, that 7 days was the appropriate duration of lag effect to be considered for all pollutants. Based on this lag, a significant non-linear lag effect was found. When additional pollutants were incorporated in the analysis, they did not enhance the model to better explore the association.

Using QAIC values, it was determined that 7 days was the appropriate duration of lag effect to be considered as each pollutant rendered their smallest QAIC at 7 days lag, with PM_{10} being the best univariate model to explore the association. There are few studies that have used DLNM to study the association between criteria air pollutants and asthma related health outcomes. One study explored the delayed effects of PM_{10} on asthma ED visits up to 21 days lag and similar to our study, reported significant findings at lag 0 (Xu, 2013), while another explored the delayed effects of PM_{10} on primary healthcare visits with 15 days lag and observed a statistically significant increase in the number of asthma primary healthcare visits through day 15 (Taj, 2016). Difference in results may be attributed to the difference in outcome variables. Among studies that use ED visits as outcome, most significant findings occur within a relatively shorter lag time, if not at immediate exposure. In contrast, our study had significant findings

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at 7 days lag as well, refuting the notion that severe asthma attacks among those susceptible, occur at immediate exposure.

Based on the chosen length of lag, this study revealed non-linear behavior when analyzing the associations between PM_{10} and ED visits of asthma, which concords with similar studies where concentration-response curves suggest non-linear relations between PM exposures and respiratory disease outcomes (Ge et al., 2018; Guarnieri & Balmes, 2014; Nasari et al., 2016; Pope et al., 2009; Smith & Peel, 2010; H. L. Yu & Chien, 2016). The drastic increase in risk that occurred when PM_{10} exceeded 200 $\mu g/m^3$ at lag 0, is consistent with most studies in the literature whose findings report asthma related ED visits to occur in those most fragile at initial exposure (Ge et al., 2018; Q. Xu et al., 2016). Such concentrations of PM_{10} are well over the 150 $\mu g/m^3$ limit indicated by the EPA's National Ambient Air Quality Standard. When PM_{10} concentrations were at 150 $\mu g/m^3$, our model showed a decrease in level of risk as RR approximated the value of "1" for all days lag. In our study, this pollutant's effect showed to gradually dissipate after initial exposure, then gradually increased to reach a similar maximum at 7 days lag, finding which supports that there are others at similar risk of asthma-related ED visit outcomes up to 7 days later, as found in a similar study with $PM_{2.5}$ (Chien, Chen, & Yu, 2018). This emphasizes the need for susceptible individuals and healthcare practitioners to better understand the long-term impact of criteria air pollutants in order to implement best health

practices. Although this study shows PM_{10} exposure is associated with the risk of ED visits due to asthma, only 14 days exceeded NAAQS for PM_{10}, reason why the overall health impact within the Las Vegas metropolitan area may not necessarily be considered significant. Nonetheless, with further urbanization and expansion in the Las Vegas metropolitan area, extreme temperatures due to global warming and a continuance of forest fires in California, the number of days per year that exceed NAAQS may be on the rise.

Asthma-related ED visits are not only a key indicator of poor asthma management but also of populations at risk for future asthma attacks. Elevated concentrations of criteria air pollutants have a public impact on the community. Negative health outcomes such as asthma-related ED visits burden the healthcare system and have serious cost implications, especially when uninsured and underinsured individuals rely on ED visits for asthma treatment. Those most susceptible, particularly children, would benefit most from improved air quality considering the negative effects poor air quality has on the development of lung function, a major determinant of life long respiratory health. Changes in government policy / further EPA restrictions would have the most significant impact as a reduction in the concentrations of criteria air pollutants may reduce ED visits.

It was expected that including additional pollutant(s) to the original model would render a model better fit to represent and explore the association. Studies comparing univariate models to multivariate models specifically for the association between $PM_{2.5}$ -PM_{10} and asthma ED visits were not found in the literature. In theory, multivariate models are more complex, aiming to integrate additional contributing factors and multiple parameters that render a better explanation of the association than do simpler univariate models (Gasparrini, 2011; Lowe et al.; 2018). However, the QAIC value indicated the univariate PM_{10} model better minimized the trade-off function between number of parameters and goodness of fit than did the PM_{10} - 1hr $PM_{2.5}$ multivariate model in our study. This could be due to limitations in our baseline model or collinearity as additional pollutants are integrated.

To the author's knowledge, this is the first study to explore the effects of criteria air pollutants on ED visits using DLNM within the Las Vegas metropolitan area. DLNM allows for imputed air pollution data to consider regional variation. Lagged and non-linear effects were explored using advanced statistical methods to better explain exposure-response relationships of which effects may be delayed. Compared to similar studies, this study looked at various pollutants for a more comprehensive analysis. The results of this study contribute to the growing literature on the health effects of air pollution on asthma, being that existing lag time between exposure-response effects have not yet been

established. The findings could help medical providers and those most susceptible better understand the lag effects of high concentrations of pollutants so that appropriate asthma management plans can be developed at the individual level.

Several limitations should be acknowledged. First, the count for ED visits obtained from CHIA consisted only of Nevada hospitals, which excludes urgent care centers and consequently, a higher asthma count which may impact the association our study aimed to explore. Second, with only 21 monitoring stations, 58% of ZIP codes received imputed data, being that the dlnm is a modelling framework that depends on variability, its predicting power may have been limited. In terms of measurement error, monitoring stations may not exactly represent the real individual inhalation measurement. Third, this study did not control for environmental factors that could potentially confound the effect of PM_{10} on ED visits. Initially, daily weather data from 11 stations was collected, but because the imputed weather data in each ZIP code was limited, weather variables were not considered in the models. With the use of aggregate data, and thus aggregate exposure, potential confounders at the individual level (comorbidities, repeat visits) were not controlled for. The possibility of integrating other statistical software/programs was considered but not incorporated, in part because of its complexity, timing and uncertainty as to its impact on the model's predicting power. Individual-level confounding variables, (ie., smoking), only have valid data by county level, and not zip code level. In an

attempt to explain remaining variations of asthma incidence by unobserved

predictors, (i.e., weather conditions, or individual risk factors such as smoking),

a random-effect intercept was included in the models.

Outcomes from this study may prompt further research so we may better

understand the behavior of such associations. Future research may include

incorporating a spatial function as part of the model in order to consider spatial

heterogeneity and to establish spatial vulnerability (variation of ED visits that

isn't explained by non-spatial predictors) as the spatial variation of ED visits is

not entirely attributed solely by criteria air pollutant exposure. Including a spatial

function would consider geographic data as an independent variable in the model.

In conclusion, the study shows that within the Las Vegas metropolitan

area, PM_{10} exposure is associated with the risk of ED visits due to asthma, and

PM_{10} has a nonlinear lagged effect for asthma occurrences. Understanding the

existence of a lag time is imperative for healthcare providers to emphasize and

educate their patients as to proper prevention and asthma management practices.

Appendix

SF1. Alternative Multivariate Analysis Predicting PM_{10}: 3D graph, contour plot and slice plot illustrating relative risk (RR) of asthma-related ED visits at lagged days along pollutant concentration, where reference levels are PM_{10} at mean value, $22.37\mu g/m^3$ and present day (lag 0). **(a)** 3D plot depicts variation of RR of asthma related ED visits along with pollutant concentration and lagged day; **(b)** The contour plot shows hot spots of RR of asthma related ED visits with pollutant concentration along the x-axis and lag days along y-axis. **(c)** Exposure-outcome analysis (x-axis indicates days lagged, y-axis indicates the RR outcome. The grey zone indicates the 95% confidence interval of estimated RR under the specific exposure-outcome value. At initial exposure, lag 0, RR is 2.59 (95%CI= 1.03, 6.55); Lag 7, RR is insignificant at 2.14(95%CI= 0.89, 5.14)
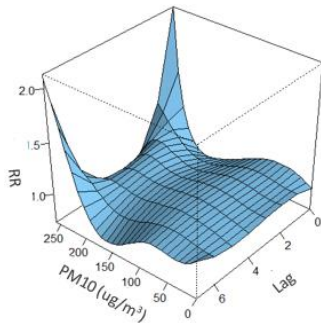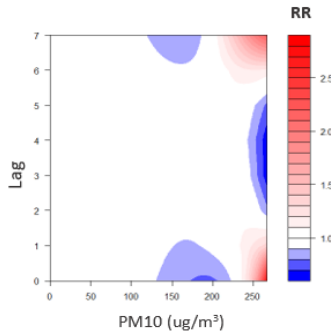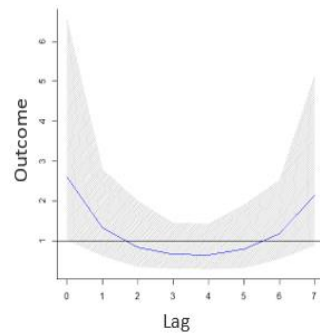
a. 3D Graph (PM_{10})   *b. Contour Plot* (PM_{10})   *c. Slice Plot* (PM_{10})

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Curriculum Vitae

LOIREN MONARDES

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Professional Summary

I am a driven individual with attention to detail and problem solving skills who brings a positive attitude to collaborative efforts. I am a public health advocate who looks forward to exchanging ideas and knowledge with peers and community partners in order to bridge needs to care/resources.

Relevant Coursework

Applied Linear Models	Biostatistical Methods	Research Methods
Epidemiology	Chronic Diseases	Infectious Diseases
Immunology	Virology	Molecular Cell Biology

Skills

- Database Research, Literature Review, Surveys, Data Compilation and Data Evaluation
- Proficient in Microsoft Office, specifically Word and Excel. Basic SPSS, QuickBooks
- CDC Training and experience working BSL-2
- Knowledge of the principles, theories and practices of public health microbiology, knowledge of standard laboratory diagnostic equipment and complex testing procedures; ability to make judgements in selecting the most pertinent guidelines, interpreting precedents, and adapting standard formulas, methods or procedures to fit facts and conditions
- Community Outreach / Community Engagement

Education

Master of Public Health – Biostatistics &Epidemiology

University of Nevada Las Vegas

GPA 3.82 Expected Completion Fall 2018

Bachelor of Science, Microbiology

University of Texas at El Paso

GPA 3.09; Major 3.48

Professional Experience

Nevada Institute for Children's Research & Policy   04/2017 to 05/2018

Bilingual Research Assistant

- Coordinated and collaborated with project partners to collect and track data from investigations, surveys and other sources, and used data to support the evaluation of the effectiveness of public health programs.
- Established rapport with Spanish speaking members of our community in a manner that is culturally sensitive and facilitates data collection.
- Child Death Review – compile information from multidisciplinary agencies, create summaries that highlight risk factors to illness or injury and enter/report data that is consistent with the clinical data repository for Fatality Review and Prevention.

Southern Nevada Health District   05/2017 to 10/2017

Data Collection Specialist II

- Performed research and data collection on overdose and prevention within the Las Vegas community. Managed data collection, analysis and reported as to community partners' use of HIV/HCV message.
- Developed resource guides to link Persons who inject drugs (PWID) to HIV/HCV testing and treatment. Assisted in the development and delivery of prevention strategies training for the HIV/HCV/PWID recovery/treatment community.
- Assisted in the planning of Southern Nevada Harm Reduction Alliance (SNHRA) events in a coordinated effort with community partners/government entities. Participated in community outreach events, presenting educational information as well as resource guides aimed at linkage to care.

Providence Memorial Hospital, El Paso, Texas   06/2010 to 12/2010

Med Tech II

- Performed routine and complex laboratory testing and analysis following established protocols and procedures.
- Received all incoming specimens for diagnostic testing, read plates for 120+ patients daily and performed testing for antibiotic resistance/susceptibility.
- Interpreted and reported results in a timely manner via data management system, telecommunication and interfacing with doctors and nurses.

City of El Paso, Texas - Department of Public Health   12/2009 to 06/2010

Microbiologist

- Performed complex and routine clinical procedures in the testing of Influenza A and B using real time PCR. Analyzed and interpreted results, and subtype for H1N1 according to CDC protocols.
- Provided daily reports and weekly statistics to the City of El Paso Epidemiology Department. Collaborated with hospital, laboratories and public health officials.

Scherr & Legate PLLC, El Paso, Texas   05/2007 to 12/2009
Discovery Department Lead

- Prepared and answered Discovery.
- Responsible for partner attorney's exhibit books, compilation and summary of evidence.

Law Offices of Michael J. Gopin, El Paso, Texas   06/2004 to 04/2006

Legal Secretary

- Liaison between Spanish speaking clients, their attorney and co-counsel, healthcare providers, insurance carriers, adjusters, government agencies and other stakeholders.
- Prepared content specific case files including related documentation of contracts, releases, and insurance forms. Prepared case summaries and expense reports.

Training

Introduction to the Incident Command System ICS-100, ICS for Single Resources and Initial Action Incidents ICS-200, National Incident Management System IS-00700.a, National Response Framework IS-00800.b.

Collaborative Institutional Training Initiative: Authorship, Collaborative Research, Conflicts of Interest, Data Management, Mentoring, Peer Review and Research Misconduct.

Fundamentals of HIV Prevention Counseling, TopSafe, Client Centered Counseling, Determine & CTR forms Training

Screening, Brief Intervention and Referral to Treatment (SBIRT) for Substance Use Disorders