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FINAL REPORT

ANALYSES OF HEALTH IMPACTS AND RISKS DUE TO UINTAH BASIN AIR QUALITY

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ANALYSES OF HEALTH IMPACTS AND RISKS DUE TO UINTAH BASIN AIR QUALITY

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Abstract

This report documents two different studies on health impacts and risks due to air quality in the Uintah Basin.

The first study is a statistical analysis exploring correlations between ozone concentrations and reported asthma cases in the Uintah Basin. Ozone is known to exacerbate asthma symptoms. We compared asthma cases at Ashley Regional Medical Center in Vernal, UT, to ozone concentrations at Ouray, UT, where high ozone concentrations have been measured in the winter. We could not find any definitive relationship or correlation between ozone concentration and asthma cases. Our inability to elucidate these relationships may be at least partially due to the small dataset available.

The second study applies EPA-recommended analyses to assess possible health risks due to toxic air contaminants (TAC) in the Uintah Basin. Such risks are categorized as acute (short term) or chronic (long term). The only acute risk identified in this study is that of ozone. Results for chronic risks are less conclusive because they rely on year-round measurements. (Since our primary concern has always been winter air quality, year-round measurements are not available.) We have determined that benzene, a compound known to pose both cancer and non-cancer chronic hazards, is likely to present low risk, but could present a marginal risk if it turns out that its average summer concentration is high. If the summer benzene concentration proves to be more than about 50% higher than the winter concentration, then we begin to cross over a threshold at which the EPA believes, “some sort of remediation is desirable.” Summertime measurements of benzene concentrations in the Basin, therefore, are called for.

Study 1. Winter Ozone and Asthma in the Uintah Basin

Introduction

Elevated surface ozone concentrations have been observed in the Uintah Basin since 2009. While ozone in the stratosphere protects us from the sun, "breathing air containing ozone can reduce lung function and increase respiratory symptoms, thereby aggravating asthma or other respiratory conditions" [Environmental Protection Agency, 2013]. Our objective here is to determine if high winter ozone concentrations are correlated with asthma-related hospital visits at Ashley Regional Medical Center.

A Utah Department of Health document, *Asthma in Utah: Burden Report 2012,* [Beck and Baxter, 2012] reports that Uintah and Duchesne counties have higher asthma emergency department visits and higher asthma hospitalization rates than the state average. This has led to a misconception that the two counties have higher *asthma rates* than the state average, when in fact the same report finds that Uintah and Duchesne counties are "similar to [the] state" average for the "prevalence of current asthma [… in] Utah adults." Closer inspection of the report indicates the most likely explanation for this discrepancy. Those counties with higher than average emergency department visits or higher than average hospitalization rates turn out to be predominantly rural, while the opposite is true of the more urban regions of the state. In other words, the Uintah Basin does not seem to be atypical in terms of total asthma cases, but it does seem to follow the well-known pattern that individuals in rural areas are more likely to seek medical treatment at a hospital emergency room than at a clinic. The Department of Health Burden Report does not specifically address ozone concentrations in the Basin, but it also does not find an asthma rate in the Basin significantly different from the state average.

High ozone concentrations are known to increase airway responsiveness to allergens in asthmatics [Kehrl et al., 1999]. Several studies have linked high ozone concentrations with increased asthma cases in hospitals [Silverman and Ito, 2010; Lin et al., 2008; Mar and Koenig, 2009; Babin et al., 2008; Strickland et al., 2010]. These studies were performed in areas where ozone levels are highest in the spring and summer.

Asthma is either caused or exacerbated by many factors, including genetics, diet, obesity, viral infections, allergen exposures, endotoxins, and atmospheric pollutants, including ozone. In fact, even exposure to non-immunological stimuli (strong smells, cold air, fog, smoke, exercise, dust, etc.) can exacerbate asthma symptoms in susceptible individuals [Sousa et al., 2013; Lenney, 2009; Corrigan, 2012]. Because there are so many potential triggers, isolating the effect of any one stimulus, such as ozone, is difficult except with large data sets. In essence, the ozone signal is swamped by the signals from all other stimuli. The various studies cited above were drawn either from large population distributions or long time series. As we show in this report, detecting ozone-asthma connections in small population centers such as the Uintah Basin is probably not possible, even when ozone concentrations exceed EPA standards.

A study connecting winter ozone concentrations and clinic visits for adverse respiratory-related effects was recently performed in Sublette County, Wyoming [Pride et al., 2013], which is the only other region known to have local wintertime ozone production leading to exceedances of EPA air quality standards. The study covered a 4-year period from 1/1/2008 to 12/31/2011, and found a 3% increase in clinic visits for every 10 ppb increase in ground-level ozone on the previous day. (The 95% confidence limits are 0%

and 7%.) The study was designed to compare the ozone exposure on some given day with that of the same days of the week within the same month. (In other words, the third Wednesday in February 2009 is compared with all other Wednesdays in February.) This allows the study to control for weekly and for seasonal trends. Cases of adverse respiratory effects were identified from electronic billing records generated by the only two clinics in the area, and were based on diagnostic codes corresponding to diagnoses of acute bronchitis, asthma, chronic obstructive pulmonary disease (COPD), pneumonia, upper respiratory tract infections, and "other respiratory." The complete study included about 13,000 clinic visits, distributed as follows: upper respiratory infections, 73.3%; COPD, 15.4%; asthma, 6.2%; pneumonia, 2.4%; acute bronchitis and "other," 1.4% each. Wyoming Department of Environmental Quality makes public announcements of ozone forecasts; these "ozone notification days" permit citizens to limit exposure and to voluntarily control emissions. Due to concerns that ozone forecasts would by themselves produce additional clinic visits, the authors performed follow-up analyses by eliminating notification days from the study. These follow-up calculations produced essentially the same result: a 3% uptick in next-day visits for every 10 ppb uptick in ozone concentration. Nevertheless, there are two obvious caveats to the study: (1) The study counts *visits*, not organic illnesses, and (2) the various diagnostic classifications include a broad spectrum of disease severity. It would be incorrect to say that the study finds a 3% increase in severe diseases like COPD or asthma.

Although there is much evidence that ozone affects respiratory outcomes in general, the relationship between ozone concentrations and health outcomes in the Uintah Basin is not well understood. For instance, cold weather may decrease exposure to winter ozone. Ozone levels are usually much lower indoors because ozone reacts quickly with surfaces such as furniture [World Health Organization, 2006; Geyh et al., 2000]. The effect of ambient ozone concentrations is weaker with a higher usage of air conditioning compared to open window ventilation [Geyh et al., 2000; Liu et al., 1997]. A heated building would probably provide similar protection. Outdoor ozone levels, therefore, may not accurately represent population exposure when cold weather forces individuals inside.

Additionally, heat seems to worsen the effect of high ozone concentrations. The effects of ozone on respiratory admissions increase during warmer weather and ozone adds to mortality risk during heat waves [World Health Organization, 2008; Filleul et al., 2006]. A study in the Atlanta, Georgia, metropolitan area found the impact of ozone on pediatric asthma or wheeze in the warm season to be about twice that of the cold season [Strickland et al., 2010]. A deeper understanding of the effect of winter ozone on the population of the Uintah Basin is needed.

As reported below, we examined seasonal asthma trends in Vernal and found a statistically significant asthma maximum in September. Pediatric asthma cases occur during a 3-week period between about August 25 and September 15 at three times the annual rate. Local physicians are familiar with the maximum and attribute it to exposure to pollen [Anderson, 2013]. It has been suggested that similar maxima in other regions are attributable to upper respiratory viral infections [Johnston et al., 1996; Carlsen et al., 1984; Johnston et al., 2005; Grech et al., 2002]. (See Appendix for additional discussion of seasonal asthma maxima.) In any case, ozone concentrations are low during this same period, so that ozone can be ruled out as the cause of the September maximum. Furthermore, linear regression calculations failed to find any correlation between ozone and asthma in the Uintah Basin, with R^2 correlation coefficients of 0.02 or lower, see below.

Methods

Ashley Regional Medical Center in Vernal provided de-identified data for 424 asthma cases during a 3.5-year (1277-day) period between 1/1/2009 and 6/30/2012. The 424 cases fall in the following four categories: (1) patients presenting at the emergency room and then discharged, (2) patients presenting at the emergency room and then admitted to hospital, (3) direct admission to hospital by a pediatrician, (4) direct admission to hospital by a primary-care physician. The data do not include presentations to clinics that did not result in hospital admission. All age groups are represented, with 153 children's cases (ages 0 to 17) and 271 adult cases (ages 18 and above).

Measured ozone concentrations were available for Ouray, UT, starting 7/29/2009. Ouray is about 30 miles south of Vernal, but ozone data for Vernal is not available for the entire time period. Ozone concentrations at Ouray during ozone episodes are almost always higher than at Vernal, but both sites lie within the same airshed, are typically covered by the same cold air pool during wintertime inversions, and have concentrations that usually are highly correlated [Mansfield and Hall, 2013]. For this study, we use the daily maximum in the 8-hour running average concentration.

Data on exposure to cigarette smoke and pets were incomplete, so these effects were not analyzed.

We examined the data for seasonal trends in asthma and also performed several linear regression analyses. As explained below, the seasonal asthma trend is uncorrelated with the seasonal ozone trend, and the linear regressions show no correlation between asthma cases and ozone.

Seasonal Asthma Trends in the Uintah Basin

Ashley Regional Medical Center registered 153 pediatric asthma cases (ages 0 – 17) and 271 adult cases (ages 18+) over the 1277-day period of the study, for rates of 153/1277 = 0.12 cases/day for children, 271/1277 = 0.21 cases/day for adults, and an overall rate of 0.33 cases/day. Figure 1 shows the daily case rate for each calendar month. We note that for the month of September, the case rate climbs to almost 0.5 cases/day, and that adults and children both contribute to this September maximum. A less prominent maximum also occurs in March, but it appears to be due only to adults. A more detailed analysis (see Appendix) sheds additional light on the September maximum. As we show there, the pediatric case rate for one 3-week period increases to 0.33 cases/day, almost three times higher than the year-round average. Interestingly, the beginning of the 3-week period coincides almost exactly with the opening of school. Whether or not this is coincidental is not known, but correlations between ozone cases and the school calendar have been reported in other regions (see Appendix).

The Appendix shows the odds that this 3-week maximum has occurred entirely by chance are about 23 in one million, indicating that its occurrence is almost certainly non-random, i.e., that it has some underlying cause. As already mentioned, possibilities are allergens or respiratory infections, or both. The March maximum may also have an underlying cause, but it may also be a random fluctuation.

Figure 1 also shows monthly average ozone concentrations. These are largest in January, February, and March, and do not seem to be well correlated with the occurrence of asthma. The lack of correlation is seen in the scatter plot of Figure 2, which plots average monthly case rate against average monthly ozone concentration. With a correlation coefficient of only 0.014, we have to conclude that ozone concentration does not have a noticeable effect on asthma cases in the basin.

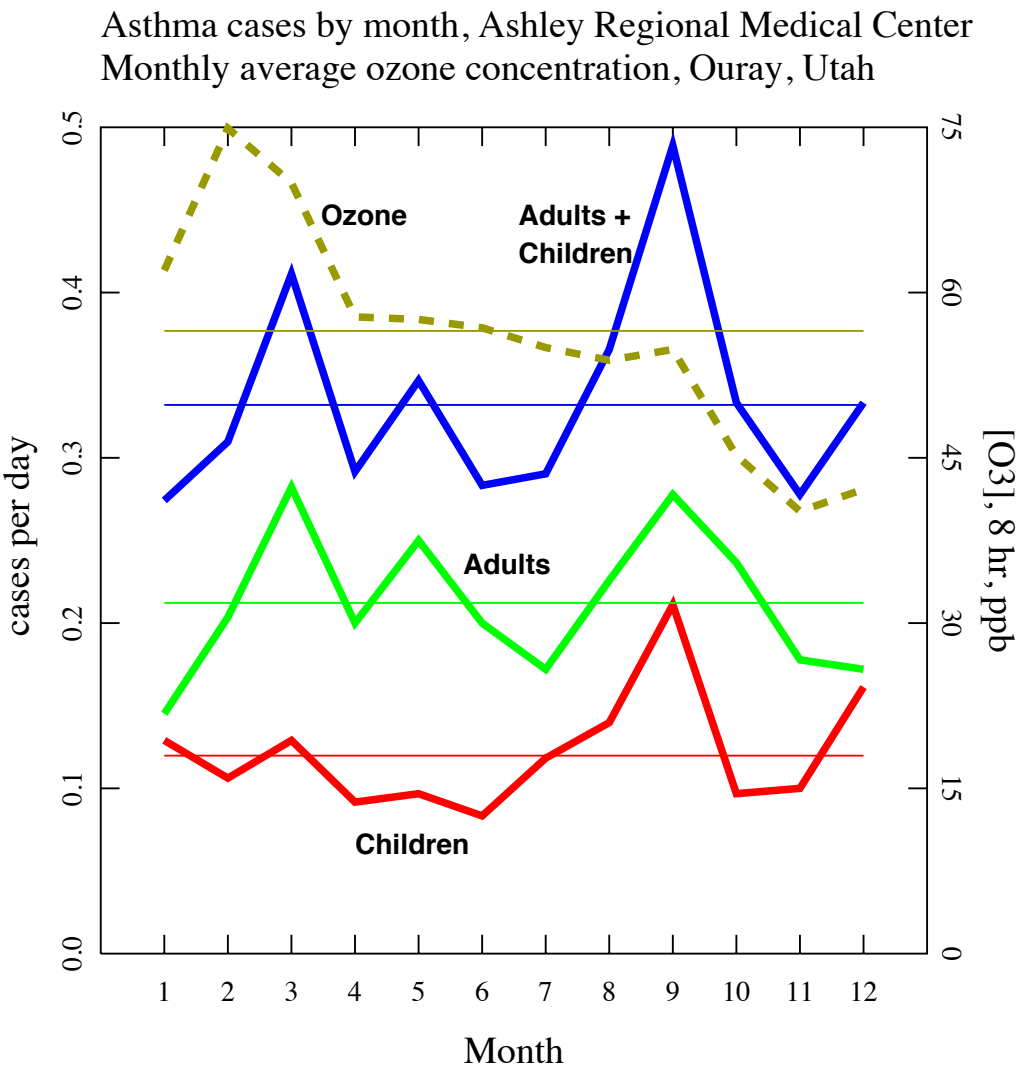

Figure 1. Monthly average asthma cases per day, in red, green, or blue for children, adults, and the entire population, respectively. Monthly average ozone concentrations are also shown. Horizontal lines are overall averages. A statistically significant asthma maximum occurs in September.

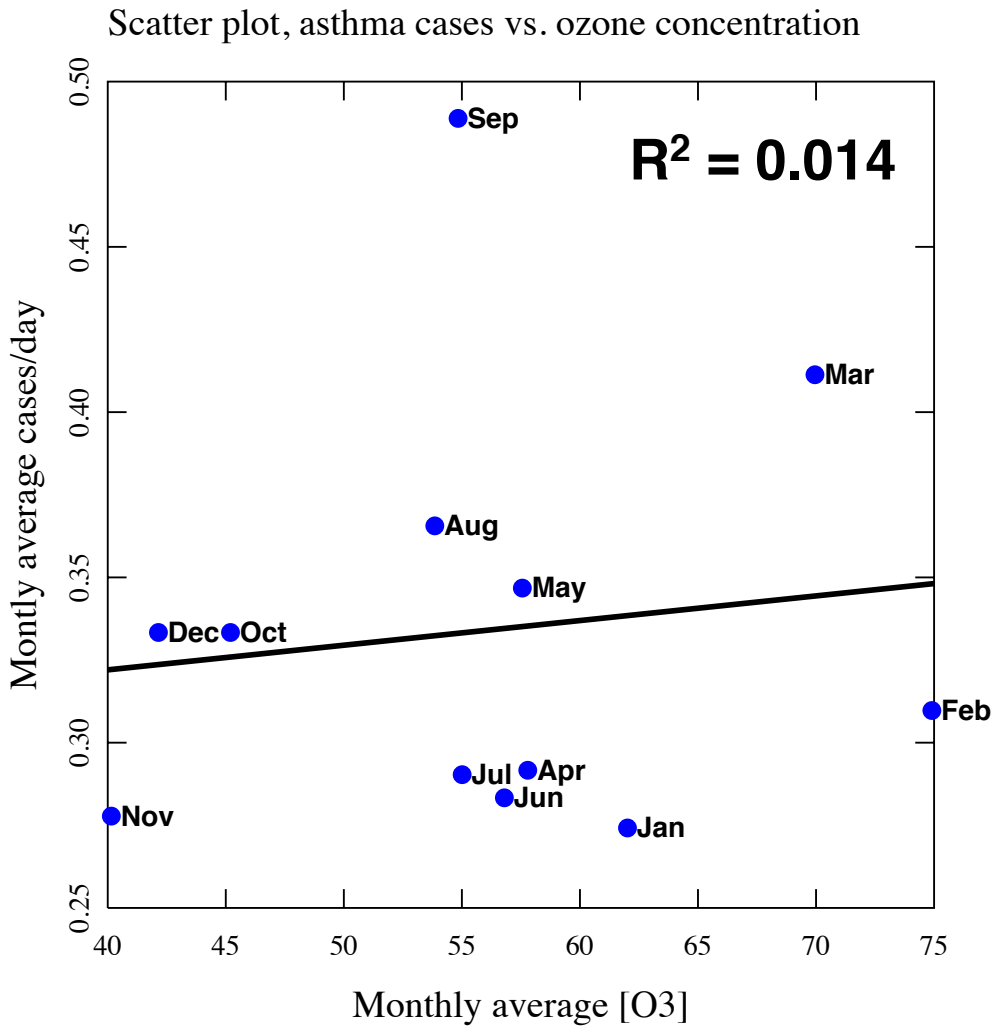

Figure 2. Scatter plot of monthly average case rate vs. monthly average ozone concentration. Also shown is the correlation line. The square of the correlation coefficient is 0.014.

Additional Evidence for Lack of Correlation between Ozone and Asthma

The distribution of ozone concentrations does not appear to depend on whether an asthma case has occurred (Figure 3). Ozone concentrations on days with an asthma case have a mean of 51 ppb and a standard deviation of 14.14 ppb. The mean for all of the concentrations for the period is 52 ppb and the standard deviation is 16 ppb, slightly higher than on days with asthma cases. An independent samples *t*-test for these means returned a *p*-value of 0.057, which indicates that the difference between the two means is only marginally significant. In other words, ozone concentrations on days with asthma cases do not appear to be different than ozone concentrations on days without asthma cases.

Winter ozone concentrations vary by year. For example, the 8-hour ozone concentration in Ouray rose above 75 ppb 42 times in 2010, but never in 2012. We compared the number of asthma cases by year,

focusing on only the first quarter of the year, when ozone concentrations are historically highest (Figure 4). A one-sample chi-square test confirmed that the variation in the number of asthma cases by year is insignificant (*p*-value of 0.601). One-sample chi-square tests also confirmed that asthma cases were not unequally distributed among the months of the year (*p*-value of 0.103) and among quarters of the year (*p*-value of 0.100). These tests suggest that asthma cases did not increase during the ozone season.

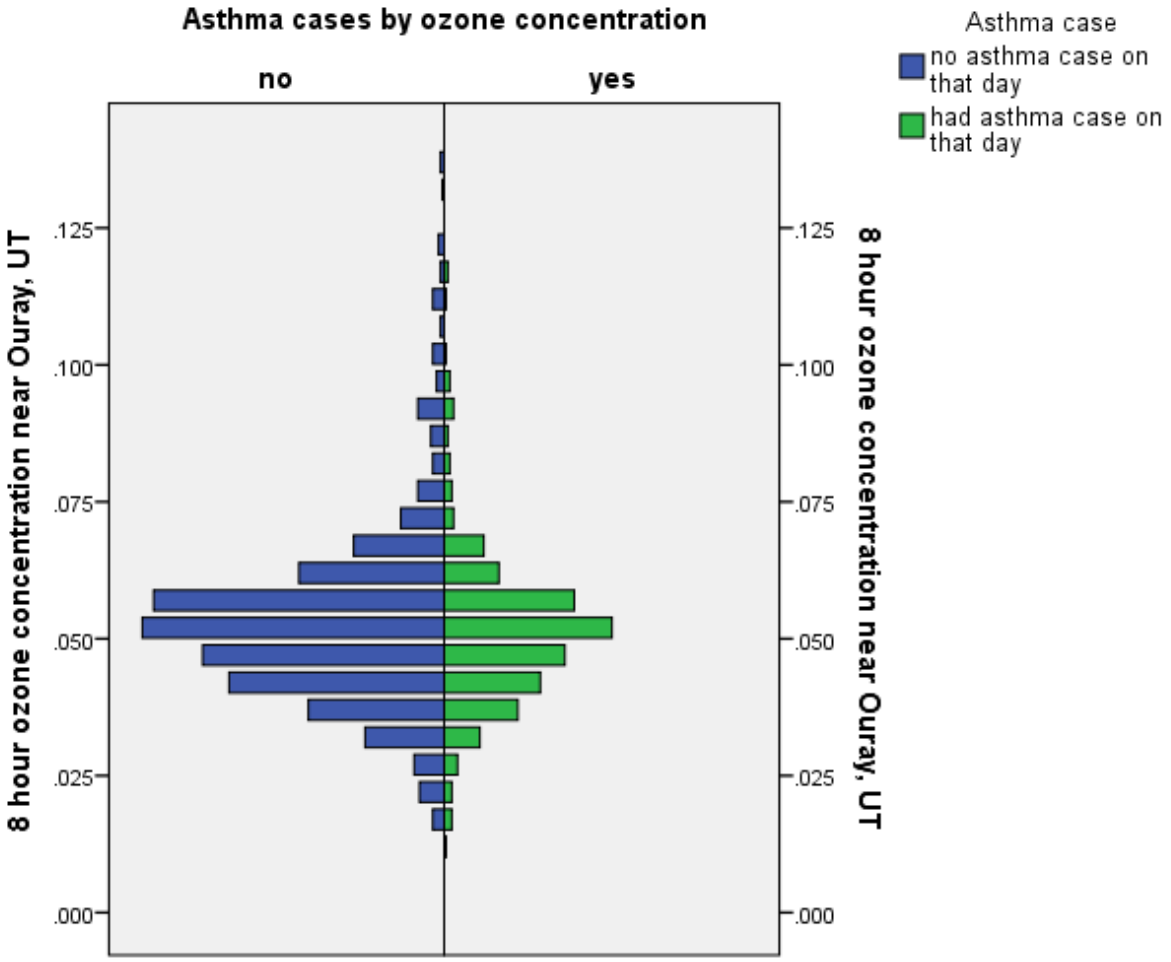

Figure 3. The bar chart on the left shows the distribution of ozone concentrations on days with no asthma cases, that on the right for days with asthma cases. Statistical tests verify that the two distributions are equivalent.

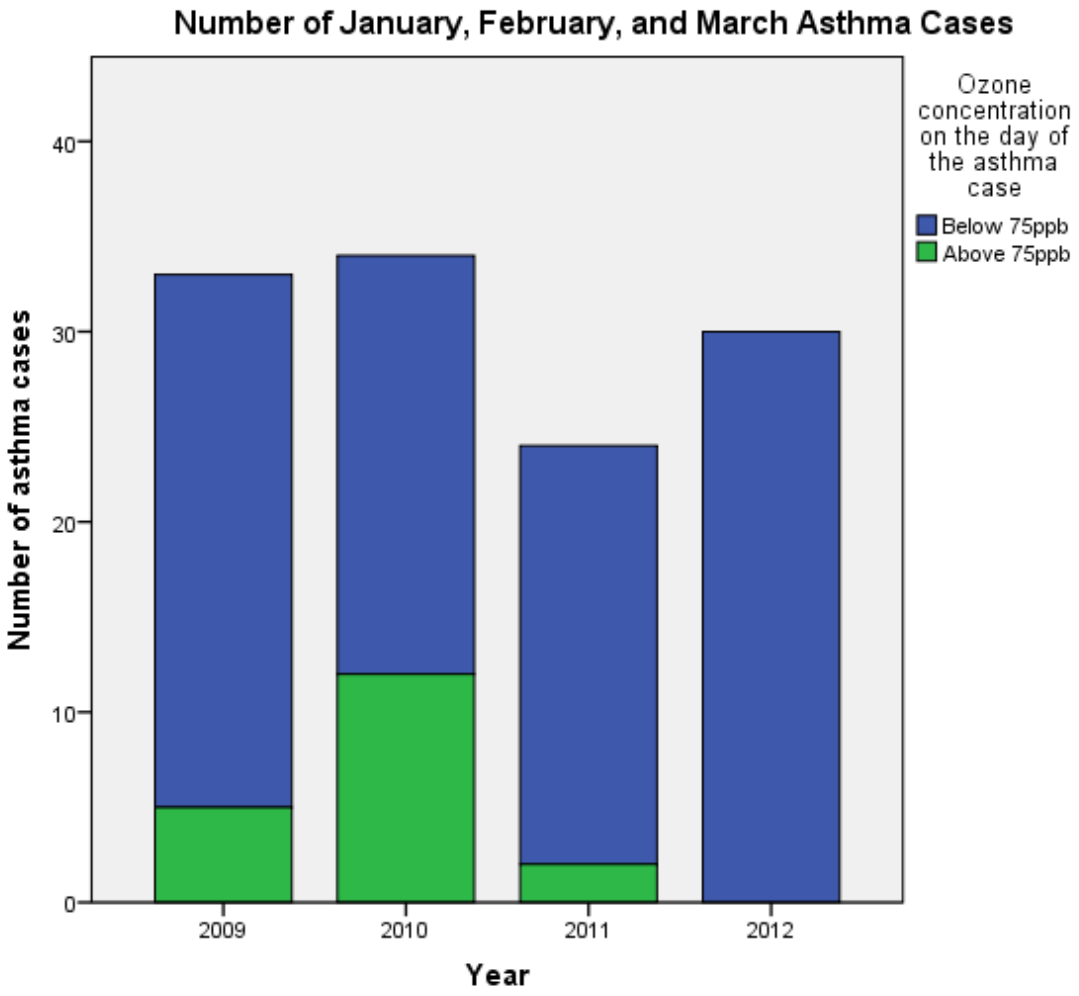

Figure 4. The number of asthma cases during the first quarter of each year is statistically the same in both good and bad ozone years.

Figure 5 shows a scatter plot giving the number of cases on any one day as a function of the ozone concentration on that day. In this case, the linear regression correlation coefficient is extremely small (0.003), indicating lack of correlation.

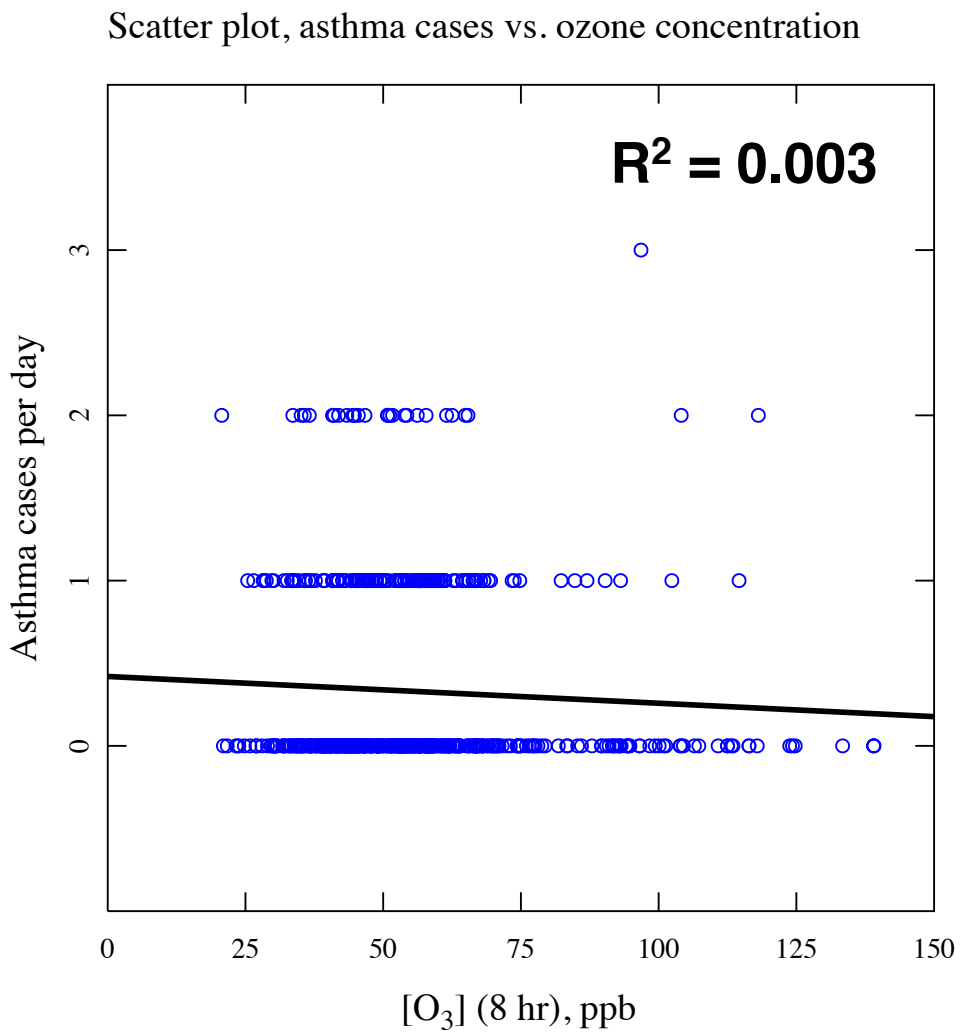

Figure 5. Asthma cases per day vs. ozone concentration. The linear regression correlation line is also shown. The squared correlation coefficient is only 0.003.

Conclusions

We were unable to elucidate a relationship between ambient ozone concentrations and asthma-related hospital visits or admissions. Scientific studies have clearly documented that this relationship exists, but other factors, such as other triggers for asthma symptoms and less ozone exposure because of cold winter temperatures, have obscured the effects of ozone on asthma in these data. Scientific studies capable of detecting the effects of ozone on asthma apparently require large data sets in order to see the relatively small ozone signal. Ozone does not appear to be among the dominant factors causing asthma symptoms in the Uintah Basin.

The data do demonstrate a statistically significant asthma maximum in the Vernal area in September that is not related to ozone concentration, and which coincides, perhaps coincidentally, with the opening of the school year.

Future studies should include more types of respiratory cases, such as COPD, in addition to asthma. A study that examines the relationship between high ozone concentrations and lung volume in children or adults could also provide more information.

Acknowledgements

We are grateful for the generous financial support of the Uintah Impact Mitigation Special Service District and the Utah Science, Technology, and Research Initiative. We are also grateful to Arnita Perry and Debbie Spafford at Ashley Regional Medical Center for providing us with the asthma data.

Study 2: Health Risk Assessments of Uintah Basin Air Quality

Results from Sublette County, Wyoming, and Motivation of our Study

In response to public concerns over the health impacts of ozone and toxic air contaminants (TAC) in the Upper Green River Basin of Sublette County, Wyoming, the Sublette County Commissioners, the Wyoming Department of Health, and the Wyoming Department of Environmental Quality contracted with Sierra Research, Inc., to conduct a health risk assessment for the county [Walther, 2011]. From February 2009 to March 2010, Sierra monitored the ambient concentrations of 51 TAC and ozone. In regards to the health impacts of ozone, they concluded, "Because the highest measured concentration was less than the [NAAQS] standard [of 75 ppb], it can be concluded that ozone levels […] were low enough to avoid any direct health impacts (i.e., NAAQS are set to protect public health, including the most sensitive subpopulations of infants, the elderly and the ill, with a large margin of safety)" [Walther, 2011]. However, as luck would have it, the Wyoming study coincided with a low ozone season, and as is well known, Sublette County has experienced higher ozone concentrations in other winters. As for the other 51 TAC, the report concluded, "The estimated health impacts of the 51 TAC monitored in the study are not high enough to suggest a need for a more refined health risk assessment of the TAC in the ambient air in and near Sublette County" [Walther, 2011].

As part of the Uintah Basin air quality study, we have accumulated seasonal concentration data on a number of volatile organic compounds (VOC). The list of compounds we chose to measure was based on ozone reactivity, not on direct toxicity to humans. Nevertheless, our list contains known carcinogens, and so we thought it would be informative to submit our concentration data to an analysis similar to that performed in Wyoming. This is not intended to be an in-depth analysis of the toxicity of contaminants in the Uintah Basin airshed, but rather a preliminary assessment to determine if further study is warranted.

The concentration data that we use for this study were taken at two different sites in the Uintah Basin. The Horsepool site is in Wonsits Valley, central Uintah County, in a region of intense natural gas production, with data collection occurring between December 2012 and March 2013. The Roosevelt site is in the city of Roosevelt, eastern Duchesne County, near Constitution Park, with data collection from mid-December 2012 to the end of February 2013.

Preliminary Screening

Health effects of inhaled compounds are classified in three different ways: chronic cancer, chronic non-cancer, and acute. Chronic risks are those associated with long-term exposure and whose health effects are considered to be cumulative over a lifetime. Acute risks are those associated with a short-term (less than an hour or a day) exposure [EPA-904-B-06-001, 2010]. Following EPA recommendations [EPA-904-B-06-001, 2010], a preliminary screen of all of the detected compounds is performed by comparing the largest daily average concentration encountered in the dataset with a screening concentration. Each compound can be screened in this way for chronic cancer risk, chronic non-cancer risk, and acute risk. Appropriate screening factors have never been determined for all possible compounds, and we can only include compounds with known toxicities. Furthermore, this first analysis is only intended to be a

preliminary screening. Compounds that fail to pass are not necessarily posing a threat; rather the procedure serves to identify compounds that might require further attention.

We first discuss chronic non-cancer effects. For each compound, a screening value is obtained, and the compound is assumed to fail the screening if the maximum daily average concentration exceeds the screening value. The chronic non-cancer screening value is derived from its reference concentration (RfC), which is defined at the EPA/IRIS (Integrated Risk Information System) website as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime" [EPA/IRIS]. Chronic non-cancer screening values are taken to be 10% of the RfC both by the Wyoming study [Walther 2011] and in recommendations from EPA, [EPA-904-B-06-001, 2010] for essentially two reasons: first, in the interest of being more conservative, and second, to correct for synergistic hazards that might occur when individuals are exposed to mixtures rather than pure compounds. The screening values we use come from one or the other of those two documents.

Table 1 compares the maximum daily average concentration at Horsepool and Roosevelt of compounds for which a chronic non-cancer screening value is available. Compounds that fail the screening (i.e., those for which the maximum daily average exceeds the screening value) are highlighted in red.

Table 1. Preliminary non-cancer chronic screening of compounds in the air of the Uintah Basin. All concentrations are in $\mu g/m^3$. Red ink indicates compounds that do not pass the screening. (N-hexane was not measured at Horsepool.)

Compound	Maximum Daily Average Concentration		Non-Cancer Chronic Screening Value (10% Of Rfc)
	Horsepool	Roosevelt	
benzene	19.2	27.0	3
cyclohexane	31.2	43.3	600
ethylbenzene	4.59	3.04	100
isopropylbenzene (cumene)	22.4	39.7	40
m,p-xylenes	3.07	13.3	10
o-xylene	3.13	2.73	10
n-hexane	-----	131	70
Styrene	20.3	41.8	100
Toluene	26.6	32.1	40

We next present the results of the chronic cancer screening. Again the procedure calls for us to compare the maximum daily average concentration against a screening value. Screening values for chronic cancer risks were selected either from the Wyoming or the EPA document, and protocols for their definition are also given in the EPA document [Walther, 2011; EPA-904-B-06-001, 2010]. Only two of our compounds have tabulated cancer screening values. The results are shown in Table 2, and all compounds that do not pass this initial screening are again highlighted in red.

Table 2. Preliminary cancer screening of compounds. All concentrations are in µg/m^3. Red ink indicates compounds that do not pass the screening.

Compound	Maximum Daily Average Concentration		Cancer Screening Value
	Horsepool	Roosevelt	
benzene	19.2	27.0	0.13
ethylbenzene	4.59	3.04	0.4

Finally, we discuss the results of the acute screening study. Sources that we consulted for acute screening values were the Wyoming and EPA documents already mentioned [Walther, 2011; EPA-904-B-06-001, 2010], the California Office of Environmental Health Hazard Assessment, [California OEHHA, 2013] and the EPA Acute Exposure Guidelines [EPA AEGL, 2013]. In the case of screening for acute risks, many more screening values are available. However, the procedure is essentially the same, compounds do not pass the screening if the maximum daily average concentration is greater than the screening value. Only one compound, ozone, failed the acute screening test.

In summary, five compounds, benzene, ethylbenzene, m-xylene, o-xylene, and n-hexane, are deemed to have failed the initial screening for chronic concerns. Only one compound, ozone, failed the initial screening for acute hazards. We reiterate that at this level of screening these compounds do not necessarily constitute a health risk. The purpose of the preliminary screening is to identify compounds for greater scrutiny. For example, in the case of the non-cancer chronic test, a compound fails if the *highest* daily average in the dataset is larger than 10% of a concentration which is "likely to be without deleterious effects" even when inhaled over a lifetime.

Additional Screening

Following EPA guidance [EPA-904-B-06-001, 2010], additional screening calculations are called for to characterize both the excess cancer risk (defined as the amount of risk from some specified source over and above the risk presented by all other sources) and the non-cancer chronic risk from all the compounds that failed to pass the preliminary screening.

Assessment of the chronic risk requires year-round averages of pollutant concentrations, but this presents a challenge for us, since measurements were only made in winter. We worked an estimation approach that gives the annual average concentrations of the compounds of interest (next section) listed in the following table. However, the estimation procedure may be inaccurate. We will use these estimates in the following calculations, and then point out afterwards the effects of any errors.

Table 3. Estimates of the average annual concentration of five compounds in the Uintah Basin (counting m- and p-xylene as separate compounds).

Compound	Estimated Annual Concentration µg/m^3
benzene	3.5
ethylbenzene	0.43
m,p-xylenes	2.0
n-hexane	17

Assessment of the chronic cancer risk is obtained by dividing the annual average concentration by the screening value for those compounds for which a cancer screening value is available:

BENZENE: $\frac{3.5}{0.13} = 27$

ETHYLBENZENE: $\frac{0.43}{0.4} = 1.1$

The accumulated cancer risk is the sum of the above ≈ 28, which is, interestingly, the same value obtained for Sublette County [Walther, 2011, p. 39]. Obviously, the cancer risk is dominated by benzene. Because of the way in which the chronic screening values are determined, this represents a lifetime excess cancer risk of 28 individuals in a population of one million. Quoting EPA:

> The level of total cancer risk that is of concern is a matter of personal, community, and regulatory judgment. In general, the USEPA considers excess cancer risks that are below about 1 chance in 1,000,000 (1×10^{-6} or 1E-06) to be so small as to be negligible, and risks above 1E-04 [100 in 1,000,000] to be sufficiently large that some sort of remediation is desirable. Excess cancer risks that range between 1E-06 and 1E-04 are generally considered to be acceptable … although this is evaluated on a case-by-case basis and EPA may determine that risks lower than 1E-04 [100 in 1,000,000] are not sufficiently protective and warrant remedial action. [EPA Region 8, 2013]

The 100/1,000,000 threshold is reached for benzene at an annual average concentration of 13 $\mu g/m^3$, that for ethylbenzene at a concentration of 40 $\mu g/m^3$.

To assess the non-cancer chronic risk, each annual average concentration is divided by its chronic RfC value (defined above) to obtain its chronic health hazard index:

BENZENE: $\frac{3.5}{30} = 0.12$

ETHYLBENZENE: $\frac{0.43}{1000} = 0.00043$

m,p-XYLENES: $\frac{2.0}{100} = 0.020$

n-HEXANE: $\frac{17}{700} = 0.024$

The total chronic health hazard index is determined by summing all of the above:

$$0.12 + 0.00043 + 0.020 + 0.024 \approx 0.16.$$

This index is also dominated by benzene. A total chronic health hazard index below 1.0 is considered to be less than significant [Walther, 2011].

In summary, our estimate of the cancer risk due to the VOC compounds measured in the air of the Uintah Basin is 28/1,000,000. Our estimate of the non-cancer chronic health hazard is 0.16. The acceptability thresholds established by EPA for these risks are 100/1,000,000 and 1.0, respectively. Benzene is the dominant contributor to both of these risks. The only compound identified to pose an acute risk to health in the Uintah Basin is ozone.

Estimates of the Annual Concentrations of Benzene and Additional VOC

Our measurements of VOC concentrations in the Uintah Basin only occurred in wintertime, while the computations of health risks given above require annual averages. Because of the obvious differences in meteorology, we expect summer and winter VOC concentrations to be considerably different.

Wintertime concentrations of VOC are correlated with the presence of thermal inversions because inversions indicate a stable, dense layer of air adjacent to the ground surface. The strength of a thermal inversion is quantified by the so-called lapse rate, which measures the rate of temperature rise or fall with increasing altitude. We have already estimated the lapse rate in the Uintah Basin on any given day for the last 60 years from the distribution of surface temperatures measured on that day. By comparing lapse rates and benzene concentrations on those days for which the pollutants have been measured, we find that concentrations are high when the atmosphere is inverted, and we were able to derive a mathematical formula to estimate the pollutant concentration on any given day from the lapse rate data. We then applied that formula over all 60 years of lapse rate data, permitting us to estimate pollutant concentration on any given day, summer or winter. These values were then averaged to obtain a year-round average for benzene of 3.5 $\mu g/m^3$.

In emissions scenarios such as these, we find that concentrations of any two compounds obey proportionalities, e.g., the [butane]/[benzene] ratio is relatively constant from day to day. These proportionalities were obtained from the wintertime measurements, and applied to the annual averages, to obtain all of the other average concentrations given in Table 3.

The average benzene concentration in mid-January in Roosevelt is 8.5 $\mu g/m^3$. The mid-July value obtained by this approximation technique is much lower at 2.0 $\mu g/m^3$, and the annual average, as cited above, is 3.5 $\mu g/m^3$. The technique obviously predicts that summer concentrations are lower, but it essentially equates the concentration on a non-inverted day in January to the concentration in July, which is, in fact, an untenable approximation. One could argue that concentrations in summer should be lower because the atmosphere is usually better mixed. On the other hand, one could argue that emissions of gases evaporating from sources such as tank batteries and evaporation ponds are higher in summer because of the elevated temperature. In the Wyoming study, the maximum observed concentrations almost always occurred in the summer [Walther, 2011, see for example Tables E-1 to E-12]. Such maxima may be the result of spikes when an emission plume from some source passes over

the monitor, so they do not necessarily imply that the average summer concentration is greater than that of the winter. But then, there are also emission plumes blowing around in the winter. Based on these results, we believe it is prudent to recognize that we do not have good estimates of the year-round average concentrations of benzene or other VOC, and that it is difficult to know for certain if the summer concentrations are higher or lower than the winter ones.

If the annual average concentration of benzene turns out to be the same as the January value of 8.5 $\mu g/m^3$, then the cancer risk calculated above rises to nearly 70/1,000,000, while the non-cancer chronic health hazard rises to nearly 0.4. If the summer average turns out to be about 50% higher than the winter average, then we start to enter the domain in which the EPA believes, "Some sort of remediation is desirable." We believe, therefore, that monitoring of VOC in summer as well as in winter is called for, so that we are better able to assess the risks associated with benzene inhalation.

Conclusions

We have assessed both acute (short term) and chronic (long term) health risks. The only acute risk identified is that of ozone. The fact that ozone makes the list is no surprise, but it is reassuring that no other acute hazard was found. Our results for chronic health risks are inconclusive because a full assessment requires annual measurements, while for now only winter measurements have occurred. The only possible chronic hazard appears to be benzene (a carcinogen), but it should be of concern only if its typical summertime concentration is somewhat larger than that of winter. We suggest that summertime measurements of benzene be performed to clarify this question.

Acknowledgements

We are grateful for financial support for this project from the Uintah Impact Mitigation Special Service District and the Utah Science Technology and Research Initiative.

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APPENDIX: Seasonal Trends in Asthma and Correlations with the School Year

Figure 1 indicates that asthma cases in the Uintah Basin follow seasonal trends. Such trends are of interest because they help indicate the most active asthma triggers. Since ozone is not high in September, it is probably not a dominant asthma trigger in the Uintah Basin.

Similar seasonal trends in asthma cases appear throughout the world. Table 4 summarizes the results of 14 studies from around the world. The seasonal trends do not seem to be identical from one region to the next: some have narrow maxima, others broad. While the month in which the maximum occurs also varies, almost invariably the studies describe a maximum occurring sometime in autumn or winter. Of particular interest is the study by Johnston, et al., [2005] examining pediatric asthma cases in the province of Ontario, Canada. The maximum they describe is narrow, spanning only three weeks, and sharp (see their Figure 1). So sharp, in fact, that they use the phrase "September epidemic."

It is also interesting that some of the studies mention that seasonal asthma trends correlate with the local scholastic calendar [Johnston et al., 1996; Grech et al., 2002; Johnston et al., 2005]. It is not difficult to see how seasonal trends could arise from asthma triggers such as pollen or spores whose release follows an annual pattern. More difficult to explain are these correlations with the school year. Viral infections of the upper respiratory tract have been proposed, under the assumption that the classroom brings children together and facilitates the spread of viruses [Johnston et al., 1996; Carlsen et al., 1984; Johnston et al., 2005; Grech et al., 2002]. Viral infections can also explain how adults are able to participate in any school-caused maximum: The children bring the virus home and infect the rest of the household. Other explanations are the lower use of control medications, and that allergens are found at higher levels in the school environment than at home [Johnston et al., 2005]. Of course, correlation is not causation, and at least in some cases it could just be that some other trigger happens to coincide with the opening of school.

In any case, a school-year correlation appears in the Vernal asthma data. Figure 6 shows the number of cumulative pediatric asthma cases relative to the first day of school. In other words, day 0 on the horizontal axis is the opening day of school, while the vertical axis displays the total number of cases seen since the first day of school, counting backwards on the days prior to day 0. (Over the three fall seasons covered by this study, day 0 in the Uintah School District has been the fourth Wednesday in August.) The average slope of the curve indicates the case rate. For 90 days prior to the first day of school, and for about 70 days between day +21 and day +90, the case rate is in the vicinity of 0.11 cases/day, essentially the same as the overall rate. However, between day 0 and day +20, i.e., for the first three weeks of school, the case rate is about 3 times higher, around 0.33 cases/day.

As mentioned, this could be a random occurrence. If it were random, it would mean that 21 cases happened to fall in one particular set of 63 days (a 3-week period during each of three different years) out of a total of 153 cases in 1277 days. The probability that 21 or more cases would fall in any one set of 63 days when the overall rate is 153 cases in 1277 days, assuming that all cases occur randomly is

$$\sum_{n=21}^{153} \left(\frac{153!}{n!(153-n)!} \right) q^n \left(1-q\right)^{153-n} \cong 22.87 \times 10^{-6}$$

where q = 63/1277. In other words, the odds that this 3-week maximum has occurred by chance are about 23 in one million.

Table 4. Summary of 14 studies that report seasonal asthma trends.

Author(s) & *Region*	Description of Seasonal Trends	Ages Studied
Johnston, et al. (1996) *Southampton, England*	"Admissions for asthma are more frequent during periods of school attendance."	All
Pendergraft, et al. *USA (all regions)*	Peak from October through February.	≥ 5
Strickland, et al. *Atlanta, GA (metro)*	Admissions November through April are 21% higher than May through October.	5-17
Han, et al. *South Taiwan*	"a peak […] in winter and a nadir […] in summer."	6-15
Johnston, et al. (2005) *Ontario, Canada*	"Asthma exacerbation […] occur[s] globally after school returns." Strong spike in pediatric hospital treatments during September is termed "September epidemic."	5-15
Crighton, et al. *Ontario, Canada*	"A small peak in hospitalisations in May, a trough in July, and a large peak between September and November."	All
Harju, et al. *Finland*	Primary peak in October, secondary in May.	< 15
Weiss *USA*	Peak from September to November.	5-34
Fleming, et al *England and Wales*	"Particularly high rates of admission occurred in September."	0-14
Grech, et al *Malta*	Peak in January and trough in August.	Pediatric* and adult
Kimbell-Dunn, et al *New Zealand*	Peak in autumn (April-June), sharp decrease in summer (January).	5-44
Monteil, et al. *Trinidad*	"Highest in the last quarter of the year; lowest in July and August."	All
Carlsen, et al. *Norway*	"More attacks [occur…] during spring and autumn."	2-18
Khot, et al. *England and Wales*	Highest in September, also high in June, July, and October.	4-14

* "In school-aged children, the end of school in June was associated with a sharp (91%) drop in admissions, and restarting school in October was associated with an even sharper (165%) rise" [Grech et al, 2002].

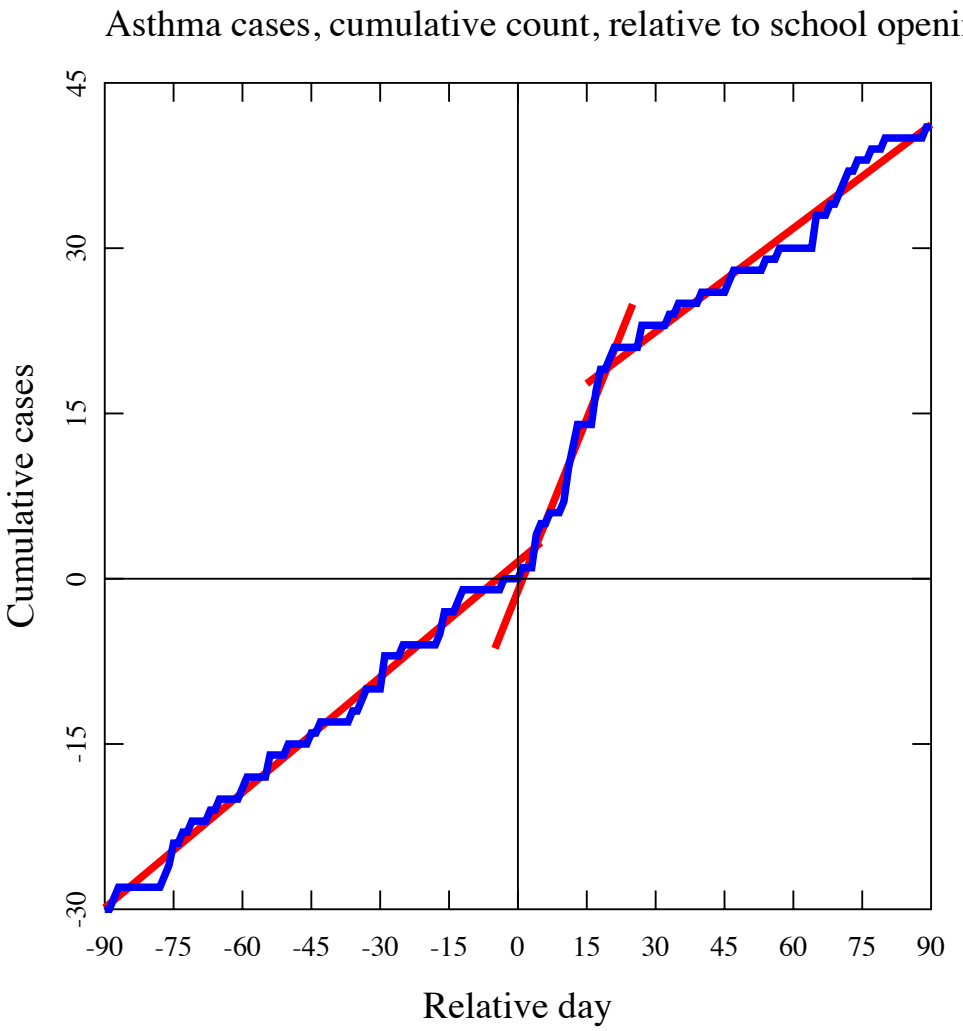

Figure 6. Over a period of three weeks starting from the opening day of school, pediatric asthma cases in the Vernal area occur at about three times the usual rate. We show that the effect is not due to chance.