**BEFORE THE MINNESOTA OFFICE OF ADMINISTRATIVE HEARINGS**
600 North Robert Street
St. Paul, Minnesota 55101

**FOR THE MINNESOTA PUBLIC UTILITIES COMMISSION**
121 Seventh Place East Suite 350
St. Paul, Minnesota 55101-2147

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| In the Matter of the Further Investigation into Environmental and Socioeconomic Costs Under Minn. Stat. § 216B.2422, Subd. 3 | MPUC DOCKET NO. E-999/CI-14-643<br><br>OAH Docket No. 80-2500-31888 |

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# MINNESOTA LARGE INDUSTRIAL GROUP'S POST-HEARING BRIEF REGARDING PHASE II (CRITERIA-POLLUTANTS TRACK)

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## INTRODUCTION

The Clean Energy Organizations ("CEOs"), Xcel Energy, the Minnesota Department of Commerce (Division of Energy Resources) ("DOC"), and the Minnesota Pollution Control Agency ("MPCA" and jointly with the DOC the "Agencies") have each undertaken efforts to produce expert reports regarding their proposed revisions to existing values for $PM_{2.5}$, $SO_2$, and NOx for use in Minnesota resource planning and other resource-selection proceedings under Minn. Stat. § 216B.2422. To calculate the Criteria Pollutant damages as contained in their Criteria Pollutant testimony and reports, Drs. Desvousges, Muller, and Marshall each used a model to first estimate changes in ambient-air concentrations of Criteria Pollutants from various power-plant emissions. Second, they estimated the potential effects (damages) of those air quality changes. Damages considered included human health (premature mortality and morbidity), agriculture (crop production), materials (corrosion and soiling), and visibility, although Dr. Marshall considered only human-health damages.[1] Then Drs. Desvousges, Muller, and Marshall monetized those effects by estimating values for each type of environmental cost.[2]

The Minnesota Large Industrial Group ("MLIG"), an ad hoc coalition of large industrial energy consumers whose costs can constitute approximately 30% of their

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1 *See, e.g.,* Ex. 604 at 15:2-10; Ex. 808 at 4:1-16; Ex. 115 at 6:14-8:3; Tr. Vol. 6 at 188:2-4.

2 *See* Ex. 604 at 15:10-11; Ex. 808 at 4:1-16; Ex. 115 at 6:14-8:3.

overall cost of production,[3] is not offering affirmative values for the Criteria Pollutants $PM_{2.5}$, $SO_2$, or $NO_X$. Instead, MLIG offered rebuttal testimony calling into question the foundation of the testimony proffered by the CEOs, Xcel Energy, and the Agencies. To be sure, embedded in the potential health effects (damages) calculation of each of these experts was an assumed relationship for an increase in health effects, over and above a baseline, per 10 $\mu g/m^3$ change in the Criteria Pollutant. This increase in health effects was assumed to occur in a linear fashion irrespective of the baseline air concentration of the Criteria Pollutant.[4] The relationship between $PM_{2.5}$ air concentration and increased health-effect impacts made by the experts is key to the damage values calculated by Drs. Marshall, Muller, and Desvousges, and presented in summary form in their direct testimony.[5]

Dr. Desvousges — who holds three degrees in economics — relies for the human-health damages effects in part on studies that show an increased mortality risk of 26%

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[3] As the MLIG has noted with respect to the $CO_2$ phase, this proceeding is somewhat unique in that the large industrials also represent the economic interests of much smaller commercial ratepayers and regular households. While the DOC is a party to the proceeding, the Attorney General's Office as consumer advocate is not. And the Agencies have advocated very high damages values in both Phase I and this Phase without appropriate foundations. The MLIG thus remains troubled by what appears to be a disconnect between the Department's position in this docket and the ultimate rate impact that position could have if adopted by the Commission.

[4] *See, e.g.*, Ex. 609 at 44; Ex. 811 at 33 (Muller surrebuttal); Tr. Vol. 6 at 52:18-24 (Currie admission), 112:1-113:11 (Marshall) ("We used just one concentration-response function at a time. We don't have any different function for different parts of the country"); Tr. Vol. 7 at 141:24-142:3 (Desvousges) (used linear function); Tr. Vol. 8 at 44:10-45:2 (Muller) (same); Tr. Vol. 7 at 141:24-142:3.

[5] *See* Ex. 604 at 6, Table 1; Ex. 808 at 72, Table 11; Ex. 115 at 27, Table 1.

associated with an *increase* in ambient concentrations of 10 µg of $PM_{2.5}/m^3$.[6] Dr. Marshall — an associate professor of environmental engineering — takes an absolute approach and states that "[t]he consensus among epidemiological studies is that $PM_{2.5}$ exposure causes an increased likelihood of death and that there is no safe level of $PM_{2.5}$ concentrations; in other words, $PM_{2.5}$ causes increased rates of mortality even at the lowest observed levels."[7] Dr. Marshall relies on studies showing increases of mortality rates of 14% and 7.8% for every 10 µg of $PM_{2.5}/m^3$.[8] Dr. Muller — an associate professor of economics — relies on most of the same studies and comes to similar conclusions as Dr. Desvousges and Dr. Muller.[9]

The MLIG has shown that the ambient-air concentration of $PM_{2.5}$ in Minnesota and Wisconsin is below 12 $\mu g/m^3$ and has further shown, through both cross examination and through the affirmative epidemiological expert testimony of Dr. Roger O. McClellan, DVM, MMS, DSC, and the official findings of the United States Environmental Protection Agency ("EPA"), relying upon thousands of epidemiological studies and vetted by the EPA's Clean Air Scientific Advisory Committee ("CASAC"), that the assumed linear relationship for an increase in health effects over and above a baseline health risk is not valid below a 3-year average baseline air concentration of $PM_{2.5}$ of 12 $\mu g/m^3$. Accordingly, as applied in Minnesota, the conclusions reached by Dr. Muller,

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6 Ex. 604 at Schedule 2 at 30.

7 Ex. 115 at 21:15-17.

8 Ex. 115 at 21:18-22:5.

9 Ex. 808 at 39:12-40:8.

Dr. Marshall, and Dr. Desvousges are inadequate and invalid; each of these opinions is based on a positive correlation in Minnesota and Wisconsin based on national data, rather than local data, and none of the opinions consider either the uncertainties inherent in their methodologies or the lack of a linear health-damages relationship at the low $PM_{2.5}$ air concentrations present in Minnesota and Wisconsin.[10]

Ordinarily, this would not be the end of the matter, as both Dr. Muller and Dr. Desvousges calculated damages for the impact of $PM_{2.5}$, $SO_2$, and $NO_x$ on agriculture (crop production), materials (corrosion and soiling), and visibility.[11] Despite the existence of Dr. McClellan's October 30, 2015, rebuttal testimony and the advance notice that their experts' health-damages testimony could not survive scrutiny and lacked a proper foundation, none of the proponent parties has offered conflicting medical surrebuttal testimony, for example from Dr. Jacobs, a professor of epidemiology and community health at the School of Public Health of the University of Minnesota, although Dr. Jacobs offered detailed rebuttal testimony on other topics.[12] Nor have Drs. Muller or Desvousges broken out the portion of their damages calculations relating to

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10 Ex. 441 at 20-21; Ex. 443; Tr. Vol. 7 at 174:3-178:8.

11 *See, e.g.*, Ex. 604 (Desvousges Direct) at 5:1-11; Ex. 808 (Muller Direct) at 7, 38, 39, 59; Transcript ("Tr.") Vol. 7 at 45:24-46:12, 47:1-12 & 130:12-21 (Desvousges and Muller did consider impacts on agriculture, materials, and visibility). *But see* Tr. Vol. 6 at 188:2-4 (Dr. Marshall did not consider impacts on agriculture, materials, or visibility).

12 *See* Ex. 117 at 1-14.

agriculture, materials, or visibility.[13] There is accordingly no record evidence of those damages.

The CEOs, Xcel, and the Agencies, as the proponents of new values, had the burden of proof to establish those damages by a preponderance of the evidence. Because their health-damages calculations are invalid and because they have not separately set forth the remaining non-health damages, they have each failed to meet their burden of proof as required by the March 27, 2015, Burdens of Proof Order,[14] Minn. Rules Part 1400.7300, subp. 5, and *In re Quantification of Envtl. Costs Pursuant to Laws of Minn. 1993, Chapter 356, Section 3*, 578 N.W.2d 794, 801 (Minn. Ct. App. 1998). The Commission is accordingly left with no evidence to support any change in the current values for $PM_{2.5}$, $SO_2$, or NOx.

## ANALYSIS

### I. BURDEN OF PROOF

In 1993, the Legislature enacted Minn. Stat. § 216B.2422, subd. 3,[15] to require the Commission to "quantify and establish a range of environmental costs associated with each method of electricity generation." The statute requires utilities to use the values in Commission proceedings "in conjunction with other external factors, including

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13 *See* Ex. 604; Ex. 808; Ex. 115. Dr. Marshall has testified that damages relating to agriculture, materials, or visibility "don't contribute very much to the overall numbers." (Tr. Vol. 6 at 188:2-14.) Dr. Muller agreed that "most of the damage in terms of the monetized component is associated with human health effects." (Tr. Vol. 8 at 28:17-18.)

14 Order Regarding Burdens of Proof dated March 27, 2015 at 2 and 6.

15 1993 Minn. Laws Ch. 356, § 3.

socioeconomic costs, when evaluating and selecting resource options . . . ." The Commission established interim cost values in 1994, and final values in 1997, for Sulfur Dioxide ($SO_2$), Carbon Monoxide (CO), Carbon Dioxide ($CO_2$), Nitrogen Oxides ($NO_x$), Lead (Pb), and particulate matter less than 10 microns in diameter ($PM_{10}$).[16] In 2001, the Commission determined that the values should increase to account for inflation,[17] which has been done ever since.[18]

The CEOs filed a petition alleging that environmental cost values "are no longer supported by scientific evidence," and requested that the investigation be reopened.[19] After considering arguments for and against the petition, the Commission determined that the scientific evidentiary support for the existing values "had been reasonably called into question," and reopened its investigation.[20] On October 15, 2014, the Commission held

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16 *See In the Matter of Quantification of Environmental Costs Pursuant to Laws of Minnesota 1993, Chapter 356, Section 3*, Docket No. E-999/CI-93-583, Order Establishing Environmental Cost Values (January 3, 1997), and Order Affirming in Part and Modifying In Part Order Establishing Environmental Cost Values (July 2, 1997).

17 *See* Order Updating Externality Values and Authorizing Comment Periods on $CO_2$, $PM_{2.5}$, and Application of Externality Values to Power Purchases (May 3, 2001).

18 *See, e.g.*, Notice of Updated Environmental Externalities Values, PUC Docket Nos. E-999/CI-93-583 and E-999/CI-00-1636, May 27, 2015.

19 *See* October 9, 2013, Memorandum in Support of Clean Energy Organizations' Motion to Update Externality Values for Use in Resource Decisions in Commission Docket No. E-999/CI- 93-583 at 18-19.

20 Order Reopening Investigation and Convening Stakeholder Group to Provide Recommendations for Contested Case Proceeding, Docket No. E-999/CI-00-1636 (February 10, 2014).

that it would investigate the appropriate cost values for $PM_{2.5}$, $SO_2$, $NO_x$, and $CO_2$.[21]

On March 27, 2015, Administrative Law Judge Schlatter ruled that "no special burden of proof attaches to proceedings under Minn. Stat. § 216B.2422, and that any party advocating a position must support that position by a preponderance of the evidence."[22] Accordingly, "[a] party or parties proposing that the Commission adopt a new environmental cost value … bears the burden of showing, by a preponderance of the evidence, that the value being proposed is reasonable and the best available measure of the environmental cost …"[23] Conversely, "[a] party opposing a particular proposal need *only* demonstrate that the proponent of proposed value cannot meet the preponderance requirement, because the proponent's evidence is flawed, or the proposal is impracticable."[24] "Practicable" has been defined by the Commission in its January 3, 1997, Order Establishing Environmental Cost Values, to mean "feasible" or "capable of being accomplished."[25] "If the weight of the evidence is evenly balanced, for and against, the *opponent* has met its burden because the proponent will not have achieved the required preponderance of the evidence."[26]

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21 Notice and Order for Hearing at 4-5.

22 Order Regarding Burdens of Proof dated March 27, 2015 at 5 (*citing* Minn. Rules Part 1400.7300, subp. 5).

23 Order Regarding Burdens of Proof dated March 27, 2015 at 2, ¶ 1.

24 *Id.* at 6 (emphasis added).

25 Order Establishing Environmental Cost Values dated January 3, 1997, at 10-11.

26 Order Regarding Burdens of Proof dated March 27, 2015 at 6 (emphasis added).

## II. MINNESOTA AND WISCONSIN'S AMBIENT AIR HAS LOW $PM_{2.5}$ CONCENTRATIONS

It is undisputed in this proceeding that the overall air quality in Minnesota has improved significantly since the 1990s.[27] Emissions of PM, $SO_2$, and $NO_x$ from point sources located in Minnesota have decreased dramatically since the Original Study in 1995 due to voluntary reductions, increased regulation, and improved pollution control technology on point sources. As a result, ambient concentrations of PM, $SO_2$, and $NO_x$ have all declined and air quality has significantly improved in Minnesota because of the reduced point source emissions.[28] In fact, the Minnesota Pollution Control Agency has reported that aggregate emissions of $NO_X$, $SO_2$, and $PM_{2.5}$ in Minnesota decreased by more than 35% from 1997 to 2012.[29]

According to the MPCA,

> Over the last 20 years, as a result of controls put in place under the Clean Air Act, annual air pollution emissions in Minnesota have decreased by nearly 40 percent. Among all sources, the greatest emission reductions have been achieved by power plants, with emissions falling by nearly 70 percent between 1990 and 2013.

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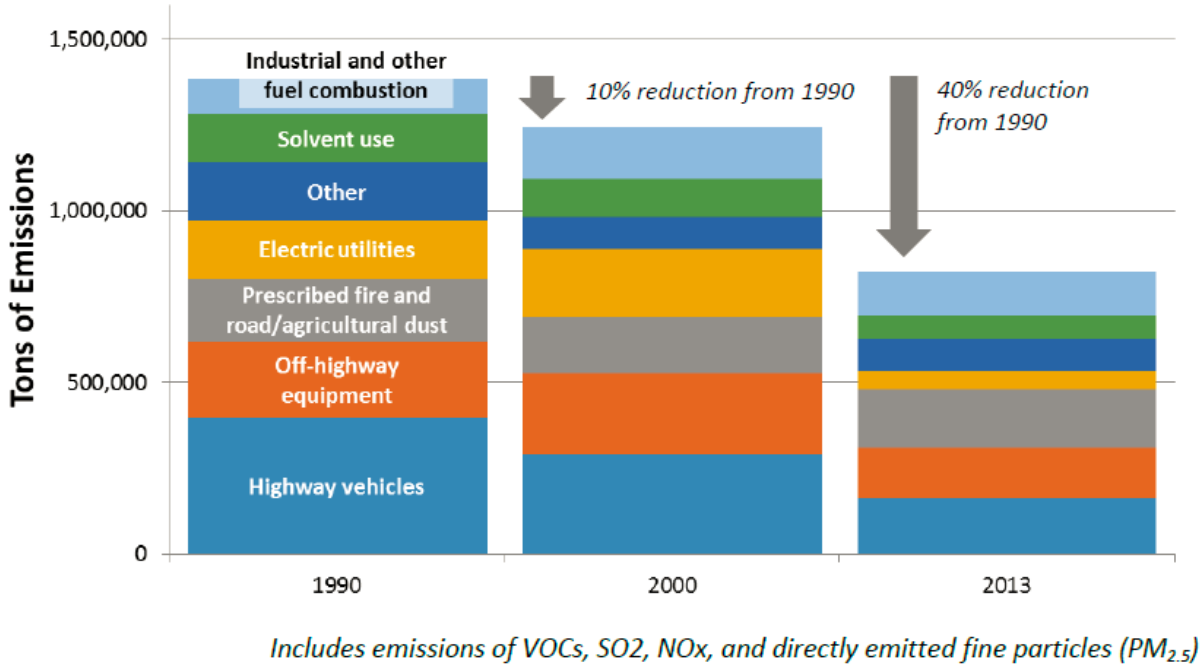
27 *See, e.g.*, Ex. 604 at 11:18-24.

28 *See, e.g.*, Ex. 604 at 11:18-24.

29 *Id.* at 12, Fig. 1 & n.6 (containing link to the MPCA report).

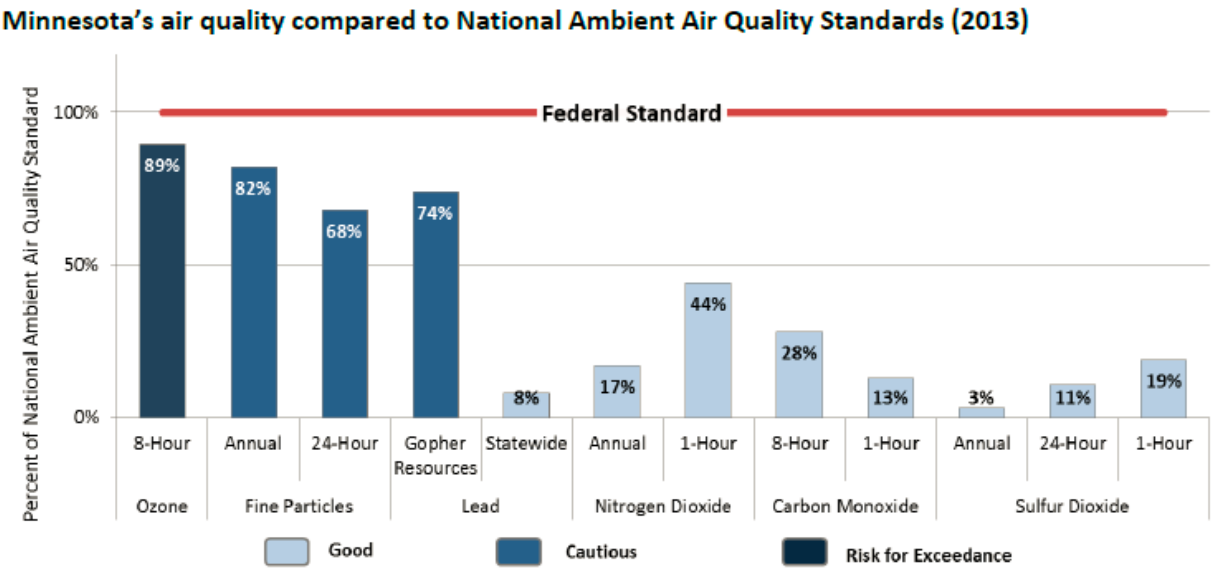
**Trends in air pollution emissions by source category, 1990-2013**[iv]




*Includes emissions of VOCs, SO2, NOx, and directly emitted fine particles ($PM_{2.5}$)*

(2015 MPCA Report to Minnesota Legislature at 7, cited in Ex. 604 at 12, n.6.) Again according to the MPCA,

> **Objective: Minnesota's air is better than air quality standards**
>
> The federal Clean Air Act requires the EPA to set National Ambient Air Quality Standards for pollutan that are considered harmful to public health, and the environment. The EPA set standards for six common air pollutants — ozone, fine particles, lead, nitrogen dioxide, carbon monoxide, and sulfur dioxide. Every five years, the EPA is required to review the science related to the environmental and health impacts associated with these pollutants. If the body of scientific research indicates an existin standard is not protective, the EPA is required to strengthen the standard.
>
> One of the MPCA's clean air goals is that Minnesota's air is better than air quality standards. To asse its progress toward meeting this goal, the MPCA monitors air pollution across the state and compare the results to these standards. In 2013, monitoring results in all areas of the state were better than a quality standards.




(2015 MPCA Report to Minnesota Legislature at 14, cited in Ex. 604 at 12, n.6.)

Dr. McClellan has shown that the $PM_{2.5}$ (weighted mean) annual values at monitoring sites in Wisconsin and Minnesota are remarkably similar and that all those $PM_{2.5}$ values, except for the extended Chicago-Metropolitan area (shown within the Wisconsin data), are less than 12 $\mu g/m^3$, the annual $PM_{2.5}$ National Ambient Air Quality Standard ("NAAQS") set by the EPA. (Ex. 441 at App. 2 at 6; Ex. 443[30] at numbered page 2; Ex. 453 (NAAQS standards).) The Chicago-Metropolitan area $PM_{2.5}$ concentration in 2014 was only 12.1 $\mu g/m^3$ (it was lower in other years). (Ex. 443 at numbered pages 3 & 10 (12.1 in 2014); and pages 6 and 8 (lower in 2012 and 2013).) In some Minnesota communities, such as, for example, Bemidji, Brainerd, and Marshall, the

30 Dr. Desvousges fully endorsed the contents of Ex. 443, (Tr. 7 at 93:1-10), while Dr. Marshall did "not have any reason to doubt the accuracy of the table[s]" in Ex. 443. (Tr. Vol. 6 at 96:23-99:14.)

measured concentrations of $PM_{2.5}$ in 2014 are less than half of the $PM_{2.5}$ NAAQS.[31] Specifically, Tables 1 through 6 below, taken from Ex. 443 (ellipses added), show tabular data from the EPA website for all the criteria pollutants measured at monitoring sites in Wisconsin and Minnesota during 2012, 2013, and 2014, including $PM_{2.5}$.

Table 1:

**Air Quality Statistics Report**

**Geographic Area:** Minnesota
**Summary:** by CBSA
**Year:** 2012
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Brainerd, MN | . | . | . | . | 0.07 | 0.062 | . | . | . | 16 | 4.6 | . | . | . |
| Duluth, MN-WI | 1.6 | 1 | . | . | 0.08 | 0.063 | . | . | . | 22 | 6.3 | 50 | 21 | 0.01 |
| Fargo, ND-MN | 0.6 | 0.4 | 34 | 5 | 0.07 | 0.063 | 4 | 1 | 0 | 23 | 7.5 | 92 | 21 | . |
| La Crosse, WI-MN | . | . | . | . | 0.08 | 0.069 | . | . | . | 22 | 8.2 | . | . | . |
| Marshall, MN | . | . | . | . | 0.07 | 0.067 | . | . | . | 20 | 7.3 | . | . | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 3.4 | 1.5 | 57 | 11 | 0.08 | 0.068 | 16 | 4 | 1 | 34 | 10.4 | 70 | 25 | 0.11 |
| Red Wing, MN | . | . | . | . | 0.07 | 0.065 | . | . | . | . | . | . | . | . |
| Rochester, MN | . | . | . | . | 0.08 | 0.069 | . | . | . | 19 | 7.8 | . | . | . |
| St. Cloud, MN | . | . | . | . | 0.07 | 0.064 | . | . | . | 20 | 8.4 | . | . | 0.01 |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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Readers are cautioned not to rank order geographic areas based on AirData reports. Air pollution levels measured at a particular monitoring site are not necessarily representative of the air quality for an entire county or urban area.

This report is based on monitor-level summary statistics. Air quality standards for some pollutants (PM2.5 and Pb) allow for combining data from multiple monitors into a site-level summary statistic that can be compared to the standard. In those cases, the site-level statistics may differ from the monitor-level statistics upon which this report is based.

Source: U.S. EPA AirData <http://www.epa.gov/airdata>
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[31] Ex. 443 at numbered page 3.

Table 2:

## Air Quality Statistics Report

**Geographic Area:** Minnesota
**Summary:** by CBSA
**Year:** 2013
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Brainerd, MN | . | . | . | . | 0.07 | 0.06 | . | . | . | 15 | 4 | . | . | . |
| Duluth, MN-WI | 4.6 | 1.5 | . | . | 0.07 | 0.059 | . | . | . | 19 | 7.7 | 59 | 24 | 0 |
| Fargo, ND-MN | 0.8 | 0.3 | 36 | 4 | 0.07 | 0.059 | 4 | 1 | 0 | 18 | 7.2 | 62 | 16 | . |
| La Crosse, WI-MN | . | . | . | . | 0.07 | 0.061 | . | . | . | 18 | 8.3 | . | . | . |
| Marshall, MN | . | . | . | . | 0.07 | 0.066 | . | . | . | 21 | 7.3 | . | . | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 3.3 | 2.5 | 45 | 13 | 0.08 | 0.067 | 15 | 15 | 1 | 23 | 10.2 | 70 | 27 | 0.11 |
| Red Wing, MN | . | . | . | . | 0.07 | 0.062 | . | . | . | . | . | . | . | . |
| Rochester, MN | . | . | . | . | 0.07 | 0.064 | . | . | . | 21 | 8.7 | . | . | . |
| St. Cloud, MN | . | . | . | . | 0.07 | 0.061 | . | . | . | 22 | 6.4 | . | . | 0 |

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Table 3:

## Air Quality Statistics Report

**Geographic Area:** Minnesota
**Summary:** by CBSA
**Year:** 2014
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Bemidji, MN | . | . | . | . | . | . | . | . | . | 16 | 5.1 | . | . | . |
| Brainerd, MN | . | . | . | . | 0.07 | 0.057 | . | . | . | 18 | 5.1 | . | . | . |
| Duluth, MN-WI | 1.3 | 0.8 | . | . | 0.07 | 0.057 | . | . | . | 18 | 8.4 | 86 | 28 | 0 |
| Fargo, ND-MN | 0.5 | 0.3 | 34 | 4 | 0.07 | 0.059 | 3 | 1 | 0 | 17 | 6.7 | 72 | 16 | . |
| La Crosse, WI-MN | . | . | . | . | 0.07 | 0.063 | . | . | . | 22 | 8.1 | . | . | . |
| Marshall, MN | . | . | . | . | 0.07 | 0.062 | . | . | . | 17 | 5.9 | . | . | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 2.7 | 1.6 | 50 | 16 | 0.08 | 0.064 | 12 | 5 | 1 | 29 | 10.3 | 76 | 25 | 0.12 |
| Red Wing, MN | . | . | . | . | 0.07 | 0.063 | . | . | . | . | . | . | . | . |
| Rochester, MN | . | . | . | . | 0.07 | 0.062 | 2 | 1 | 1 | 20 | 8.2 | . | . | . |
| St. Cloud, MN | . | . | . | . | 0.07 | 0.062 | . | . | . | 19 | 6.2 | . | . | . |
| Winona, MN | . | . | . | . | . | . | . | . | . | 22 | 8.3 | . | . | . |

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Table 4a:

## Air Quality Statistics Report

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2012
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Appleton, WI | . | . | . | . | 0.09 | 0.077 | . | . | . | 26 | 8.6 | . | . | . |
| Baraboo, WI | . | . | . | . | 0.09 | 0.073 | . | . | . | 23 | 10.1 | 39 | 12 | . |
| Beaver Dam, WI | 0.6 | 0.4 | . | . | 0.09 | 0.078 | 6 | 2 | 0 | 26 | 8.9 | 44 | 15 | . |
| Chicago-Naperville-Joliet, IL-IN-WI | 4 | 1.9 | 70 | 22 | 0.12 | 0.093 | 108 | 22 | 3 | 31 | 11.5 | 153 | 31 | 0.13 |
| Duluth, MN-WI | 1.6 | 1 | . | . | 0.08 | 0.063 | . | . | . | 22 | 6.3 | 50 | 21 | 0.01 |
| Eau Claire, WI | . | . | . | . | 0.08 | 0.068 | . | . | . | 23 | 8.1 | 40 | 17 | . |
| Fond du Lac, WI | . | . | . | . | 0.1 | 0.079 | . | . | . | . | . | . | . | . |
| Green Bay, WI | . | . | . | . | 0.11 | 0.086 | 72 | 14 | 2 | 28 | 9.6 | . | . | . |
| Janesville, WI | . | . | . | . | 0.09 | 0.08 | . | . | . | . | . | . | . | . |
| La Crosse, WI-MN | . | . | . | . | 0.08 | 0.069 | . | . | . | 22 | 8.2 | . | . | . |
| Madison, WI | . | . | . | . | 0.09 | 0.074 | . | . | . | 27 | 9.4 | 36 | 16 | . |
| Manitowoc, WI | . | . | 9 | 2 | 0.1 | 0.088 | . | . | . | . | . | . | . | . |
| Milwaukee-Waukesha-West Allis, WI | . | . | 45 | 12 | 0.11 | 0.093 | 21 | 6 | 1 | 30 | 10.9 | 47 | 23 | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 3.4 | 1.5 | 57 | 11 | 0.08 | 0.068 | 16 | 4 | 1 | 34 | 10.4 | 70 | 25 | 0.11 |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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Source: U.S. EPA AirData <http://www.epa.gov/airdata>
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Table 4b:

## Air Quality Statistics Report

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2012
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Platteville, WI | . | . | . | . | . | . | . | . | . | 22 | 9.1 | . | . | . |
| Racine, WI | . | . | . | . | 0.11 | 0.09 | . | . | . | . | . | . | . | . |
| Sheboygan, WI | . | . | . | . | 0.11 | 0.093 | . | . | . | . | . | . | . | 0.1 |
| Watertown-Fort Atkinson, WI | . | . | . | . | 0.09 | 0.078 | . | . | . | . | . | . | . | . |
| Wausau, WI | . | . | . | . | 0.08 | 0.069 | . | . | . | . | . | . | . | . |
| Whitewater, WI | . | . | . | . | 0.1 | 0.077 | . | . | . | . | . | . | . | . |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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This report is based on monitor-level summary statistics. Air quality standards for some pollutants (PM2.5 and Pb) allow for combining data from multiple monitors into a site-level summary statistic that can be compared to the standard. In those cases, the site-level statistics may differ from the monitor-level statistics upon which this report is based.

Source: U.S. EPA AirData <http://www.epa.gov/airdata>
Generated: November 18, 2015

Table 5a

## Air Quality Statistics Report

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2013
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Appleton, WI | . | . | . | . | 0.08 | 0.067 | . | . | . | 22 | 8 | . | . | . |
| Baraboo, WI | . | . | . | . | 0.07 | 0.063 | . | . | . | 16 | 7.1 | 31 | 11 | . |
| Beaver Dam, WI | 0.9 | 0.3 | . | . | 0.08 | 0.067 | 9 | 2 | 0 | 18 | 7.9 | 35 | 14 | . |
| Chicago-Naperville-Joliet, IL-IN-WI | 2.7 | 1.4 | 64 | 21 | 0.09 | 0.075 | 73 | 17 | 4 | 27 | 11.3 | 121 | 39 | 0.1 |
| Duluth, MN-WI | 4.6 | 1.5 | . | . | 0.07 | 0.059 | . | . | . | 19 | 7.7 | 59 | 24 | 0 |
| Eau Claire, WI | . | . | . | . | 0.07 | 0.06 | . | . | . | 20 | 7.3 | 52 | 19 | . |
| Fond du Lac, WI | . | . | . | . | 0.08 | 0.065 | . | . | . | . | . | . | . | . |
| Green Bay, WI | . | . | . | . | 0.08 | 0.068 | 76 | 13 | 2 | 22 | 7.7 | . | . | . |
| Janesville, WI | . | . | . | . | 0.07 | 0.067 | . | . | . | . | . | . | . | . |
| La Crosse, WI-MN | . | . | . | . | 0.07 | 0.061 | . | . | . | 18 | 8.3 | . | . | . |
| Madison, WI | . | . | . | . | 0.07 | 0.067 | 8 | 4 | 1 | 23 | 9.3 | 29 | 16 | . |
| Manitowoc, WI | . | . | 9 | 2 | 0.09 | 0.073 | . | . | . | . | . | . | . | . |
| Milwaukee-Waukesha-West Allis, WI | . | . | 50 | 10 | 0.09 | 0.07 | 23 | 5 | 1 | 25 | 10 | 38 | 17 | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 3.3 | 2.5 | 45 | 13 | 0.08 | 0.067 | 15 | 15 | 1 | 23 | 10.2 | 70 | 27 | 0.11 |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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Source: U.S. EPA AirData <http://www.epa.gov/airdata>
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Table 5b

**Air Quality Statistics Report**

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2013
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Platteville, WI | . | . | . | . | . | . | . | . | . | 19 | 8.9 | . | . | . |
| Racine, WI | . | . | . | . | 0.08 | 0.066 | . | . | . | . | . | . | . | . |
| Sheboygan, WI | . | . | . | . | 0.09 | 0.078 | . | . | . | . | . | . | . | 0.11 |
| Watertown-Fort Atkinson, WI | . | . | . | . | 0.08 | 0.069 | . | . | . | . | . | . | . | . |
| Wausau, WI | . | . | . | . | 0.07 | 0.063 | . | . | . | . | . | . | . | . |
| Whitewater, WI | . | . | . | . | 0.07 | 0.067 | . | . | . | . | . | . | . | . |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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Source: U.S. EPA AirData <http://www.epa.gov/airdata>
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Table 6a:

## Air Quality Statistics Report

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2014
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Appleton, WI | . | . | . | . | 0.08 | 0.07 | . | . | . | 2 | 8.6 | . | . | . |
| Baraboo, WI | . | . | . | . | 0.08 | 0.064 | . | . | . | 2 | 7.8 | 28 | 10 | . |
| Beaver Dam, WI | 0.4 | 0.4 | . | . | 0.08 | 0.071 | 6 | 4 | 0 | 2 | 8.5 | 30 | 12 | . |
| Chicago-Naperville-Joliet, IL-IN-WI | 5.3 | 1.9 | 67 | 21 | 0.09 | 0.076 | 53 | 12 | 2 | 3 | 12.1 | 93 | 46 | 0.15 |
| Duluth, MN-WI | 1.3 | 0.8 | . | . | 0.07 | 0.057 | . | . | . | 1 | 8.4 | 86 | 28 | 0 |
| Eau Claire, WI | . | . | . | . | 0.07 | 0.061 | . | . | . | 2 | 8.2 | . | . | . |
| Fond du Lac, WI | . | . | . | . | 0.08 | 0.067 | . | . | . | . | . | . | . | . |
| Green Bay, WI | . | . | . | . | 0.08 | 0.066 | 79 | 16 | 3 | 2 | 9.1 | . | . | . |
| Janesville, WI | . | . | . | . | 0.08 | 0.072 | . | . | . | . | . | . | . | . |
| La Crosse, WI-MN | . | . | . | . | 0.07 | 0.063 | . | . | . | 2 | 8.1 | . | . | . |
| Madison, WI | . | . | . | . | 0.08 | 0.069 | 10 | 4 | 1 | 2 | 9.3 | 41 | 18 | . |
| Manitowoc, WI | . | . | 6 | 1 | 0.07 | 0.066 | . | . | . | . | . | . | . | . |
| Milwaukee-Waukesha-West Allis, WI | 1.2 | 0.7 | 53 | 16 | 0.09 | 0.074 | 27 | 8 | 1 | 30 | 10.5 | 53 | 19 | . |
| Minneapolis-St. Paul-Bloomington, MN-WI | 2.7 | 1.6 | 50 | 16 | 0.08 | 0.064 | 12 | 5 | 1 | 29 | 10.3 | 76 | 25 | 0.12 |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#co

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Table 6b:

**Air Quality Statistics Report**

**Geographic Area:** Wisconsin
**Summary:** by CBSA
**Year:** 2014
**Exceptional Events:** Excluded (if any)
Statistics in red are above the level of the respective air quality standard

| CBSA | CO 1-hr 2nd Max | CO 8-hr 2nd Max | NO2 98th %ile | NO2 Ann. Mean | O3 1-hr 2nd Max | O3 8-hr 4th Max | SO2 99th %ile | SO2 24-hr 2nd Max | SO2 Ann. Mean | PM2.5 98th %ile | PM2.5 Wtd. Mean | PM10 24-hr 2nd Max | PM10 Annual Mean | Lead Max 3-mo Avg |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Platteville, WI | . | . | . | . | . | . | . | . | . | 2 | 8.1 | . | . | . |
| Sheboygan, WI | . | . | . | . | 0.08 | 0.072 | . | . | . | . | . | . | . | 0.09 |
| Watertown-Fort Atkinson, WI | . | . | . | . | 0.08 | 0.071 | . | . | . | . | . | . | . | . |
| Wausau, WI | . | . | . | . | 0.07 | 0.064 | . | . | . | . | . | . | . | . |
| Whitewater, WI | . | . | . | . | 0.09 | 0.073 | . | . | . | . | . | . | . | . |

Get detailed information about this report, including column descriptions, at http://www.epa.gov/airquality/airdata/ad_about_reports.html#con

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Source: U.S. EPA AirData <http://www.epa.gov/airdata>
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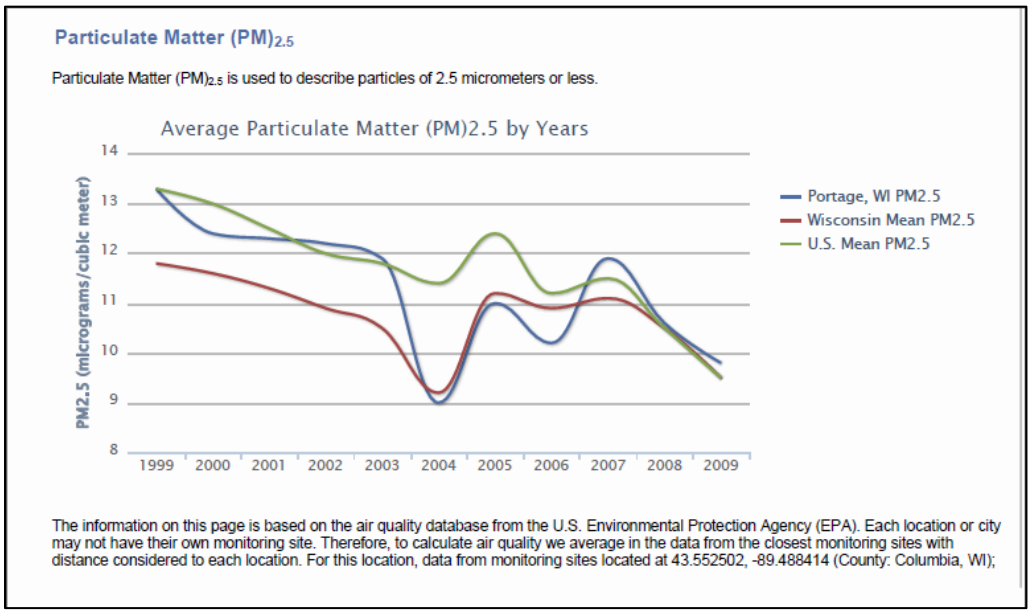
Figures 1 through 5 below are plots of the average $PM_{2.5}$ air concentrations from 1999 to 2009 for Portage, Wisconsin, and four Minnesota communities (Tracy, Marshall, Rochester, and Minneapolis).[32] The figures illustrate continuous reductions in the $PM_{2.5}$ concentrations in ambient air in all five communities over the decade.[33] Dr. McClellan noted in Ex. 443 with respect to these Figures that "[t]he similarity of the changes on a state-wide basis for Wisconsin and Minnesota and for Portage, Wisconsin, and the four

32 Ex. 443 at numbered page 11.

33 *Id.*

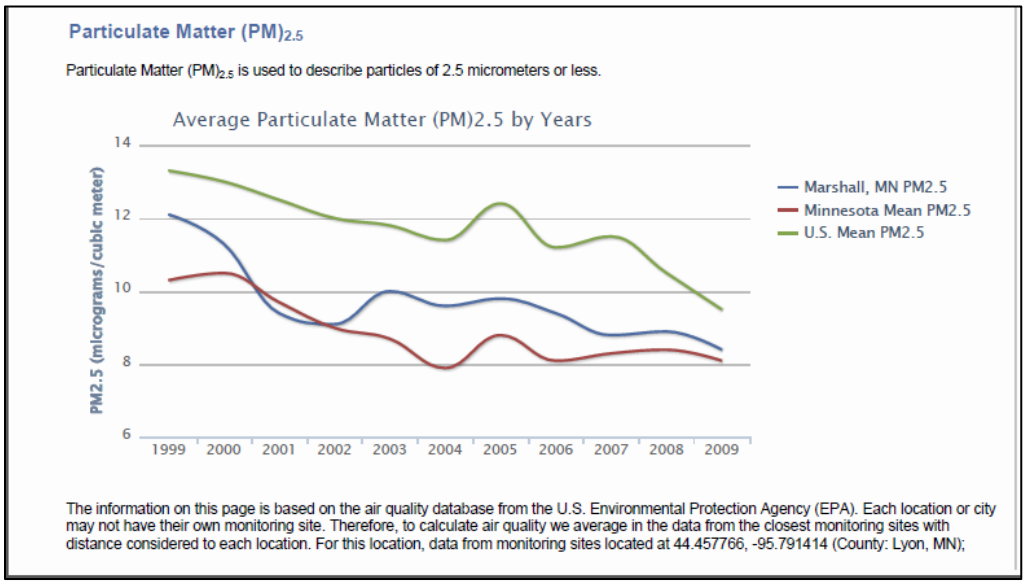
Minnesota communities is noteworthy. For example, in 2009, the mean $PM_{2.5}$ concentration value for Wisconsin communities was 9.5 $\mu g/m^3$ and for Minnesota communities it was 8.1 $\mu g/m^3$. The $PM_{2.5}$ concentration for Portage, Wisconsin, was 9.8 $\mu g/m^3$ and for Tracy, Marshall, Rochester, and Minneapolis (provided by www.USA.com), it was 8.7, 8.4, 9.8, and 10.1 $\mu g/m^3$, respectively."[34]

**Figure 1: Portage Wisconsin (1999 through 2009):**

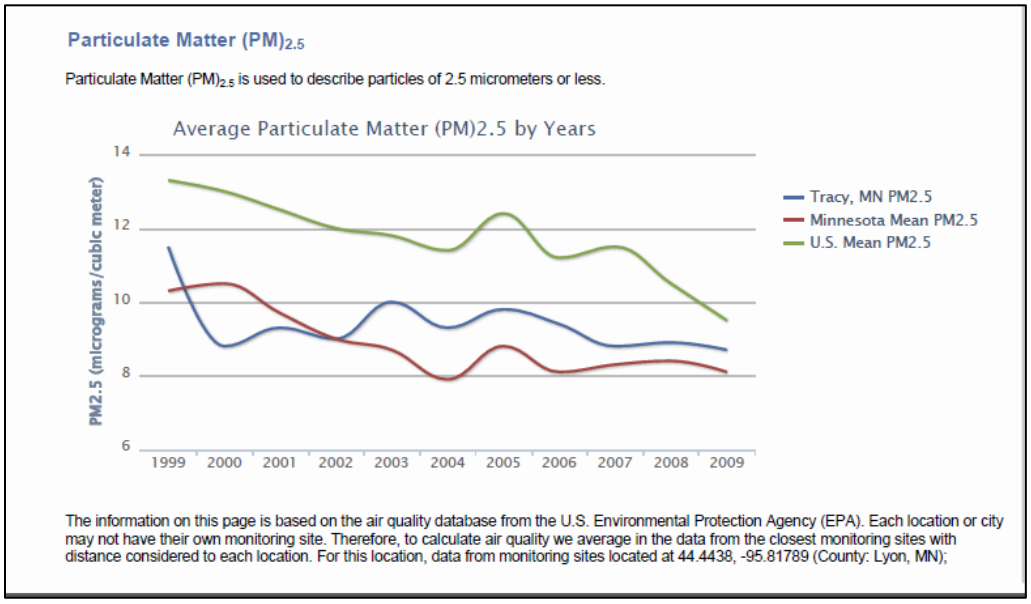



34 Ex. 443 at numbered page 11.

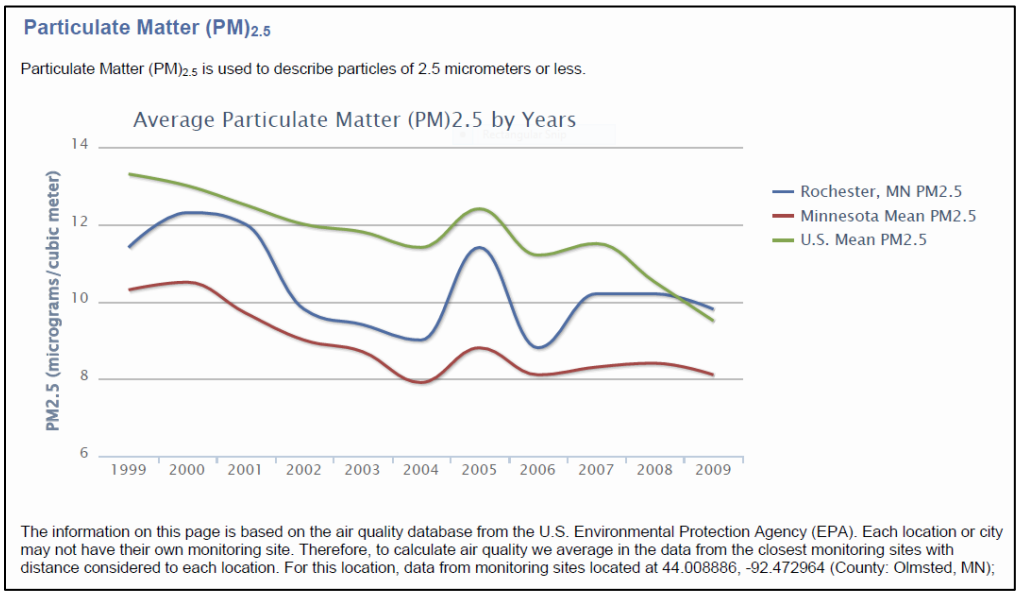
## Figure 2: Marshall, Minnesota (1999 through 2009):



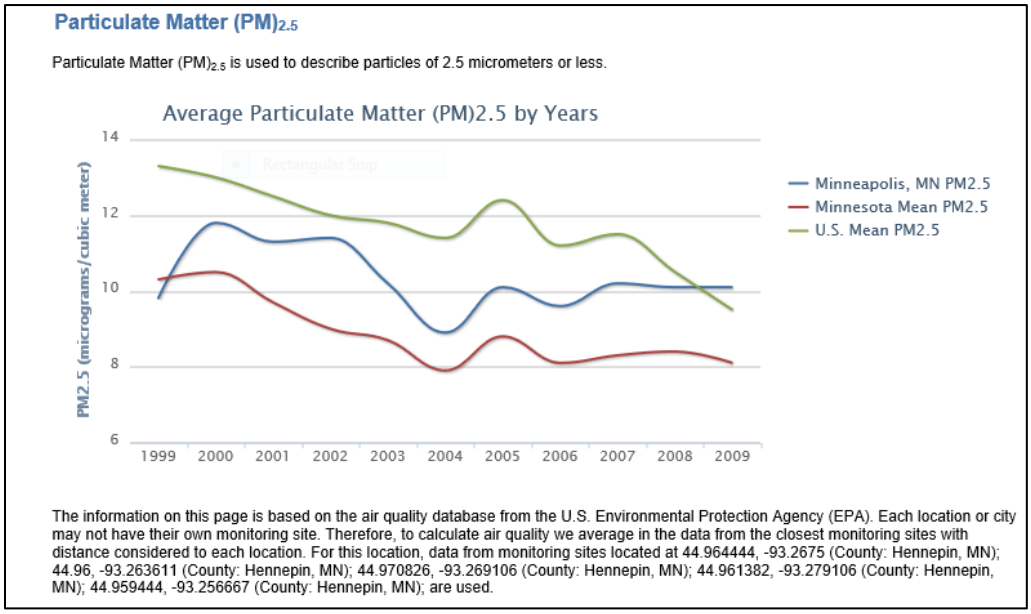

## Figure 3: Tracy, Minnesota (1999 through 2009):



## Figure 4: Rochester, Minnesota (1999-2009):




## Figure 5: Minneapolis, Minnesota (1999-2009):




Based on this data, Dr. McClellan testified that "[t]he air quality in Portage, WI is

not remarkably different than that found in most communities across Minnesota,"[35] and that "it is important to recognize that all of the measured concentrations of $PM_{2.5}$ in Minnesota are below the Annual NAAQS standard for $PM_{2.5}$ – 12 µg/m$^3$."[36]

## III. NO RELIABLE SHOWING CAN BE MADE OF HUMAN-HEALTH IMPACT AT AMBIENT-AIR CONCENTRATIONS OF $PM_{2.5}$ BELOW 12 µg/m$^3$

Although damages numbers have been set in this proceeding for $PM_{2.5}$, $SO_2$, and NOx, those damages in reality all turn around calculations for $PM_{2.5}$, because the gasses $SO_2$ and NOx turn into solid or liquid "secondary $PM_{2.5}$" after being emitted into the environment as the result of chemical reactions.[37] Dr. Marshall clarified that "[t]he way in which we calculate those involves not the direct inhalation of $SO_2$ as $SO_2$."[38] After determining the dispersion patterns of the primary $PM_{2.5}$ and the formation and dispersion of the secondary $PM_{2.5}$, health impacts and other damages calculations are then made.[39] Those health impacts underlying damages calculations have been subjected to scrutiny in thousands of studies.[40] Doctors Desvousges, Marshall, and Muller relied on only a very

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35 Ex. 443 at numbered p. 14; Ex. 441 at App. 2 at 6.

36 Ex. 441 at App. 2 at 6.

37 Tr. Vol. 7 at 7:22-8:19 (Marshall) ($SO_2$). *See also* Tr. Vol. 7 at 135:22-137:4; 146:22-147:2 (Muller) ($SO_2$ and NOx).

38 *Id.*

39 Ex. 115 at 7:15-16; Tr. Vol. 6 at 45:16-46:7.

40 Tr. Vol. 7 at 86:10-14 (Desvousges); 176:12-19 (McClellan). *See also* Ex. 444 (Federal Register / Vol. 78, No. 10 / Tuesday, January 15, 2013 (Air Quality Designations for the 2012 Primary Annual Fine Particle ($PM_{2.5}$) National Ambient Air Quality Standards (NAAQS); Final Rule)) at 3097 ("a substantial amount of new research has been conducted since the close of the science assessment in the (continued)

few of those studies, and each used a linear concentration-response function to calculate the damages, regardless of the ambient-air concentrations at issue in this proceeding.[41] None of them considered whether their linear damages model was in fact valid across the spectrum of ambient-air concentrations at issue in this proceeding. In fact, use of a linear damages model is not valid.

Dr. McClellan credibly testified, without any conflicting testimony or evidence, that in areas in Minnesota and Wisconsin where the annual mean averaged $PM_{2.5}$ exposure over 3 years is at 12 $\mu g/m^3$ or below there is no medical or other scientific basis for projecting mortality related to current or projected levels of $PM_{2.5}$.[42] Further, Dr. McClellan testified that it is important to recognize that for downwind areas that may exceed that exposure level, any calculated increase in mortality attributable will be extraordinarily small related to the baseline mortality, as shown in the findings of Lepeule et al (2012).[43] The primary (or health) damages conclusions reached by Drs. Muller, Marshall, and Desvousges were all based on national concentration-response

(continued)

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last review of the PM2.5 NAAQS (U.S. EPA, 2004), with important new information coming from epidemiological studies, in particular. This body of evidence includes hundreds of new epidemiological studies conducted in many countries around the world.").

41 *See, e.g.*, Ex. 609 at 44; Ex. 811 at 33 (Muller surrebuttal); Tr. Vol. 6 at 52:18-24 (Currie admission), 112:1-113:11 (Marshall) ("We used just one concentration-response function at a time. We don't have any different function for different parts of the country"); Tr. Vol. 7 at 141:24-142:3 (Desvousges) (used linear function); Tr. Vol. 8 at 44:10-45:2 (Muller) (same).

42 Ex. 441 at 21:3-4 & Ex. 441 at App. 2 at 9.

43 Ex. 441 at App. 2 at 9.

data, rather than local data.[44] Additionally, they each failed to consider the community-exposure level, rendering their methodology and opinions invalid at $PM_{2.5}$ ambient-air exposure levels below 12 μg/$m^3$.[45] Dr. McClellan further testified that the assumptions and corresponding calculations underlying the ranges proposed by the Agencies, CEOs, and Xcel Energy are too speculative and lack evidentiary support.[46]

### A. There is no proximate cause between exposure to $PM_{2.5}$ at ambient-air concentrations below 12 μg/$m^3$ and human-health impact

Based on all of the available epidemiological evidence, both Dr. McClellan and the United States Environmental Protection Agency hold that there is no proximate cause between human exposure to $PM_{2.5}$ at ambient-air concentrations below 12 μg/$m^3$ and health damages. The California Air Resources Board agrees as well, and no contrary evidence has been introduced. Because Minnesota and Wisconsin have ambient-air concentrations well below 12 μg/$m^3$, no damages can be shown from the very small increase in $PM_{2.5}$ exposures calculated by Doctors Muller, Desvousges, and Marshall, as summarized in Table 2 of Dr. Desvousges' surrebuttal report (Ex. 609) at 43 (ellipsis added):

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44 *Id.* at 10.

45 *Id.*

46 Ex. 441 at 20-21.

**Table 2:** Comparison of baseline ambient $PM_{2.5}$ concentrations from epidemiology studies with predicted concentration changes from air models[a]

| | Concentration of $PM_{2.5}$ ug/$m^3$ |
|---|---|
| Baseline from epidemiology studies (Table 3.2.1.2)[b] | 16 |
| Range in concentration from the epidemiology studies (Table 3.2.1.2)[b] | 8 - 23 |
| Average change in concentration from the epidemiology studies (Table 3.2.1.2)[b] | 10 |
| Average change in concentration from CAMx within 100 mile radius of MN | 0.0000198 |
| Average change in concentration from AP2 within 100 mile radius of MN | 0.0000205 |
| Average change in concentration from InMap within 100 mile radius of MN | 0.0000323 |
| Average change in concentration from AP2 beyond 100 mile radius of MN | 0.00000298 |
| Average change in concentration from InMap beyond 100 mile radius of MN | 0.000000643 |

[a]Sherco model is used for comparison.
[b]From Direct Testimony of William H. Desvousges, Schedule 2

In fact, the health impact from such changes cannot be measured,[47] and the studies are not designed to evaluate such small impacts. As Dr. Desvousges testified in his surrebuttal,

> When health studies are conducted to evaluate the impacts of these pollutants [$PM_{2.5}$] on humans, the average ambient concentrations for the study areas are on the order of magnitude of 16 µg/$m^3$ and the marginal change in risk

[47] Tr. Vol. 8 at 33:25-34:7 (Muller).

> presented is based on a marginal change in ambient PM2.5 concentrations of 10 µg/m3. As shown in Table 2 below [omitted here], the values from the epidemiological literature are multiple orders higher than the values predicted by the air dispersion models in this case. These health studies are not designed to determine the impacts of a 0.00001 µg/$m^3$ change in ambient concentrations, as is being discussed here, but rather they are conducted in order to determine what impacts a representative actual ambient concentration of pollution has on human health.[48]

In fact, as Dr. Desvousges volunteered, "none of the air dispersion models in this proceeding, including the CAMx model [used by him], include any type of measure of the dispersion (uncertainty) around the predicted changes in concentrations."[49] "The externality concentrations modeled here are very small, with significant digits represented out to the hundred thousandth decimal point and beyond with a great deal of uncertainty surrounding the results. When these very small values of changes in ambient concentrations are being used in damage calculations, it is assumed that those values are statistically different than zero, when there is no information to support that conclusion."[50]

1. According to Dr. McClellan, there is no proximate cause between exposure to $PM_{2.5}$ at ambient-air concentrations below 12 µg/$m^3$ and human-health impact

Each of Drs. Muller, Desvousges, and Marshall was questioned about causation. In their written testimony, Dr. Marshall and Dr. Muller used the derivations of the term

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48 Ex. 609 at 42-43.

49 *Id.* at 44.

50 Ex. 609 at 44 (*citing Reassessing the Human Health Benefits from Cleaner Air*, Louis Anthony Cox, Jr., *Risk Analysis*, Vol. 32, No. 5, 2012).

"causation," rather than "correlation" or "association." Dr. Marshall was unable to support the use of that term,[51] whereas Dr. Muller correctly volunteered that all of the studies that he relied upon (the same studies relied upon by Dr. Marshall) use not the terms "cause" or "causation," but rather "correlation" or "association."[52] In fact, causation is a higher scientific threshold compared to correlation or association.[53]

Dr. Muller cogently testified that there are types of correlation:

> [R]andom variables can be positively correlated, in which case high values of one variable occur with high values of another variable, and low values of variable happen with low values of another variable. Positive correlation, two things move together. There's negative correlation where you have high values of one variable tend to occur with low values of another. And then there's uncorrelated, right, where the variables are not related at all.[54]

Drs. Desvousges and Muller both testified that to show causation resulting from exposure to $PM_{2.5}$, a showing is required (1) that the presence of particulate matter preceded premature mortality or damage; (2) that exposure and damages move in the same direction, like correlation or association; and (3) that all other explanations on premature

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51 Tr. Vol. 6 at 65:10-69:19 (referring questions regarding causation to Dr. Jacobs at Tr. Vol. 6 at 69:3-8).

52 Tr. Vol. 8 at 17:16-25 (Muller). *See also* Ex. 117 (Jacobs rebuttal) at Schedule 2 (Krewski report (no mention of causation)) & Schedule 3 (Lapeule report (no mention of causation)).

53 Tr. Vol. 7 at 79:12-80:13 (Desvousges); Tr. Vol. 6 at 63:23-25 (Marshall) (causation relates in part to strength of evidence).

54 Tr. Vol. 8 at 59:14-25.

mortality have been ruled out.[55] This, the witnesses—economics experts each—could not do: Dr. Marshall admitted that "there are lots of things that cause premature mortality," and that he had not ruled out causes other than $PM_{2.5}$ exposure,[56] while Dr. Desvousges testified that as far as he knew, no study has shown the three causation factors for $PM_{2.5}$ exposure at ambient-air concentrations below 12 $\mu g/m^3$.[57] Dr. Muller admitted that he used the term "cause" in the sense of "correlation" or "association," rather than in an epidemiological sense.[58]

In his testimony, Dr. McClellan challenges the methodology followed and data relied upon by Drs. Desvousges, Marshall, and Muller with respect to their calculation of mortality and morbidity damages at low $PM_{2.5}$ ambient-air concentrations. Dr. McClellan opined that based on current $PM_{2.5}$ ambient-air concentrations in Minnesota and communities in Wisconsin, "it is my opinion, with a reasonable degree of medical certainty, that there is no medical or other scientific basis for projecting mortality attributable to $PM_{2.5}$" in Minnesota and western Wisconsin.[59] Dr. McClellan thus challenges an "essential input to their [Drs. Desvousges, Marshall, and Muller] mathematical calculations of increased morbidity and mortality and associated monetized

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55 *Id.* at 11-21 (Muller); Tr. Vol. 7 at 80:14-21 (Desvousges).

56 Tr. Vol. 6 at 75:21-76:6.

57 Tr. Vol. 7 at 80:22-81:1.

58 Tr. Vol. 8 at 17:19-18:4.

59 *See, e.g.,* Ex. 441 at App. 2 at 10 (McClellan rebuttal).

damages."[60]

Dr. McClellan is very knowledgeable about the scientific information available regarding the Criteria Pollutants and the process by which scientific information on the pollutants is obtained, reviewed, integrated, and used to establish NAAQS. His knowledge is based on his professional education and experience in comparative medicine, toxicology, aerosol science, and risk assessment. Since the mid-1970s, he has been actively involved in the EPA advisory process, reviewing the scientific evidence that informs the policy judgments leading to the EPA Administrator's promulgation of NAAQS for each of the Criteria Pollutants. His involvement has included participation on and chairing of EPA's Clean Air Scientific Advisory Committee (CASAC). Dr. McClellan is a Diplomat, by examination, of the American Board of Toxicology and the American Board of Veterinary Toxicology.[61] He is also a Fellow of the Academy of Toxicological Sciences, the American Association for Aerosol Research, the Society for Risk Analysis, the Health Physics Society, and the American Association for the Advancement of Science.[62] He has been elected to membership in the Institute of Medicine (now the National Academy of Medicine) of the National Academy of Science based on his contributions to improving human health.[63] He has received numerous awards from professional societies and other organizations for his service and scientific

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60 *Id.* at 20-21.

61 Ex. 441 at 3 & App. 1.

62 Ex. 441 at 3 & App. 1.

63 *Id.*

contributions, including an Honorary Doctor of Science degree in 2005 by the Ohio State University for his contributions to comparative medicine and the science undergirding improved air quality, and has been invited on 19 occasions to testify before U.S. Congressional committees.[64] In short, Dr. McClellan is extraordinarily well qualified to opine about the damages to the human population from exposure to the Criteria Pollutants $PM_{2.5}$, $SO_2$, and $NO_X$.

It is undisputed that "[t]he human health effects associated with exposure to sufficiently high concentrations of $PM_{2.5}$ are significant and include premature mortality, aggravation of respiratory and cardiovascular disease and development of chronic respiratory disease. In addition, there are welfare effects associated with elevated $PM_{2.5}$ levels."[65] However, "[i]t is important to recognize that, by and large, the diseases associated with air pollution are not uniquely attributed to air pollution. Rather, the largest portion of the diseases of interest is attributed to other factors."[66] Dr. McClellan opined, "with a reasonable degree of medical certainty, that the three damage reports prepared by Dr. Desvousges, Dr. Marshall, and Dr. Muller all fail to provide an adequate scientific basis for their mathematical exercises." While their math is not wrong, they have taken an overly simplistic approach to modeling this link between ambient-air and health:

> They all assume a linear association between any incremental

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64 Ex. 441 at 4, 8-10 & App. 1.

65 Tr. Vol. 7 at 172:3-9 (McClellan).

66 *Id.* at 172:9-14.

> increase in the ambient concentrations of the pollutant and increased health risks. Moreover, Dr. Marshall and Dr. Muller assume the statistical association represents a causal link. And I say a causal link between any increase in the pollutant and increased disease regardless of the baseline of PM in the ambient air. Dr. Marshall was unable to explain that causation is different from mere mathematical association and that it requires ruling out other explanations of premature mortality. Responding to their criticism of my opinion, I note that no study has shown the presence of particulate matter preceding premature mortality and correlation in the absence of other explanations for mortality at PM concentrations below 12 $\mu g/m^3$, as would be required to show causation at those concentrations.[67]

In other words, "in [Dr. McClellan's] opinion the medical evidence [shows] that $PM_{2.5}$ at annual concentrations on the order of 12 micrograms [μg/m3] and lower do[es] not have associated identifiable medical effects.[68]

Dr. McClellan's opinion is supported by Dr. Desvousges, who volunteered that

> the results of these health studies are considered linear, that it, with every increment or decrements of change in ambient concentrations, there is a presumed corresponding change in health damages. However, *this has not been researched*. That is, even though health studies are conducted on, for example, ambient concentrations of 12 $\mu g/m^3$, it is presumed that a 0.00001 $\mu g/m^3$ change in concentration will have a risk associated with it by using a scalar multiple based on the impacts seen at the 12 $\mu g/m^3$ level. However, *this linear relationship has not been evaluated at very low concentration levels* and thus this assumption is conservative and may over estimate impacts. EPA has evaluated impacts, however, and has determined that levels of PM below 12 $\mu g/m^3$ is

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67 Tr. Vol. 7 at 174:11-175:3 (McClellan).

68 Tr. Vol. 7 at 193:10-22 (McClellan in response to question from T. DeBleeckere).

> protective of human health and these are the levels at which the NAAQS have been set.[69]

An understanding of the potential hazard of any airborne pollutant requires an evaluation of the science extending from (a) emissions from particular sources, (b) transport and potential transformations in the atmosphere, (c) exposure of receptor populations, (d) the uptake and translocation of the inhaled material by individuals, (e) mechanisms of detoxification, damage and repairs, and (f) the occurrence of disease over and above that occurring naturally or from other causative factors.[70] In this context it is critical to keep in mind that there are substantial differences in ambient (*i.e.*, outdoor) air across the United States and around the world, that the population throughout life breathes a complex mixture of gases and particulate matter, and that what is in the air varies considerably between our homes, schools, work places and other environs we may live and work in over a lifetime.[71]

Dr. McClellan testified that the use of linear air concentration-response models implies that the calculated damage values are applicable to all emissions irrespective of the air quality in a particular area, which is incorrect.[72] Instead, data from the American Cancer Society study as reflected in Figure 5 in Appendix 2 of Dr. McClellan's rebuttal testimony shows that a statistically significant effect is not observed below approximately

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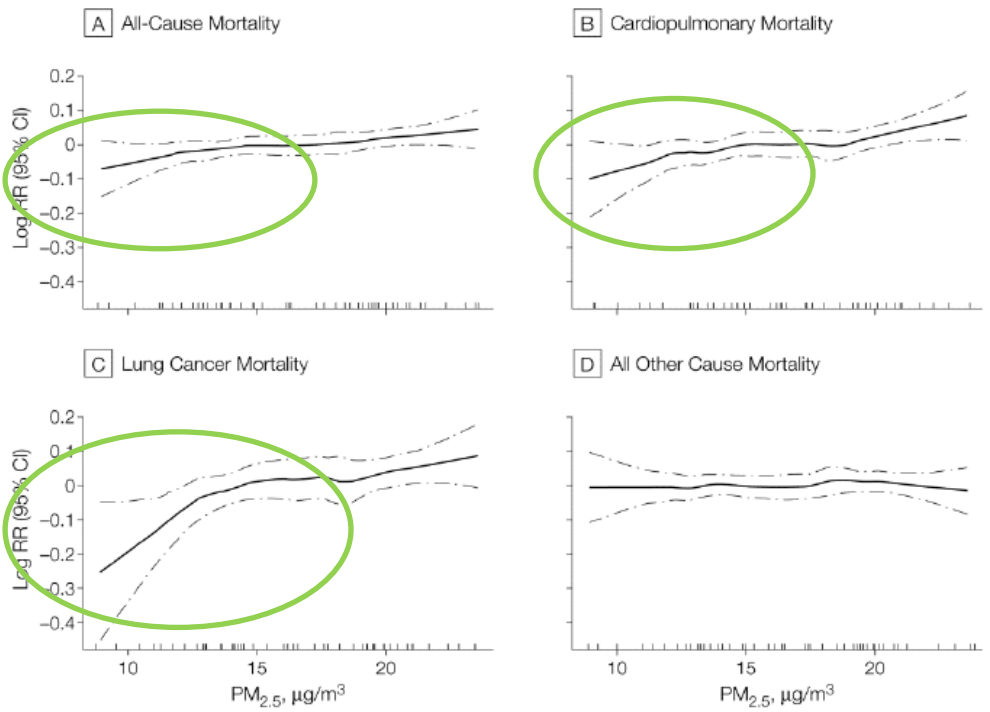
69 Ex. 609 at 44 (emphasis added).

70 Ex. 441 at App. 2 at 3.

71 *Id.*

72 Ex. 441 at 21 & App. 2 at 7-10.

13.5 µg/m$^3$ for all-cause mortality, nor below 13.8 µg/m$^3$ for cardiopulmonary and lung-cancer mortality, or 13.2 µg/m$^3$ for all-other-cause mortality,[73] with the central tendency for each trending below 0 toward the lower exposure end of the spectrum and even the upper confidence bound for lung-cancer mortality trending below 0 at that point (ellipses added):[74]




Vertical lines along x-axes indicate rug or frequency plot of mean fine particulate pollution; $PM_{2.5}$, mean fine particles measuring less than 2.5 µm in diameter; RR, relative risk; and CI, confidence interval.

Figure 5. Non-parametric smoothed exposure-response relationship. Vertical lines along the x-axes indicate a rug or frequency plot of mean fine particulate pollution. CI, confidence interval; $PM_{2.5}$, fine particles measuring less than 2.5 µm in diameter; RR, relative risk.

73 *See* Ex. 441, App. 2 at 16.

74 Ex. 441 at App. 2 at 8, 16.

If one were to take this data as true, then exposure would be protective of health.[75] In other words, one should be more exposed, because it's good for health. A more realistic interpretation of the data is that it is simply unreliable at lower exposure levels.[76] Dr. McClellan testified that according to these studies, relied upon by Drs. Muller, Marshall, and Desvousges, there is no medical evidence of any excess deaths associated with these low ambient concentrations of $PM_{2.5}$, such that in areas in Minnesota and Wisconsin that have mean annual $PM_{2.5}$ ambient-air concentrations averaged over 3 years of 12 $\mu g/m^3$ or below, there is no medical or other scientific basis for projecting mortality related to current or projected levels of $PM_{2.5}$.[77]

Lepeule *et al* (2012) reported that a small signal of adverse health effects was still present with a linear concentration-response function for all-cause mortality for $PM_{2.5}$ down to 8 $\mu g/m^3$ when using a linear model.[78] Addressing this concern, Dr. McClellan testified that the lowest air concentrations measured in linear concentration-response functions by definition dictates the lowest level of linearity, and that it is important to recognize that the increased risk is actually dominated by the measurements and population of the *dirtiest* cities.[79] It should further be noted that the 3-year average mean

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75 Tr. Vol. 8 at 146:12-148:8 (Muller); Tr. Vol. 7 at 204:23-206:22 (McClellan).

76 Tr. Vol. 7 at 204:23-206:22 (McClellan).

77 Ex. 441 at 21:3-4; Ex. 441 at App. 2 at 9.

78 Ex. 441 at App. 2 at 7.

79 *Id.*

concentration of $PM_{2.5}$ in the ambient air across the American Cancer Society cohort was 14 μg/$m^3$, well above that of Minnesota and Wisconsin.[80]

Of much more relevance to the issue before the Commission here as it relates to exposure to low concentrations of $PM_{2.5}$ is the Greven study. Dr. McClellan testified that Greven *et al.* (2011) conducted a large retrospective cohort study of Medicare enrollees, linking ambient levels of $PM_{2.5}$ to mortality data by monitor site during the period 2000-2006 and that Greven in this seminal paper reported an increase in the national life expectancy for reductions in the yearly average $PM_{2.5}$, but that the observation is based on national trends in $PM_{2.5}$ and mortality and that Greven calls attention to confounding by other variables trending on the national level.[81] Dr. McClellan noted that Greven observed major differences across the United States using sophisticated spatial modeling techniques, which included a local coefficient $ß_1$ that measures the association between local trends in $PM_{2.5}$ and mortality and a global coefficient $ß_2$ that measures the association between the $PM_{2.5}$ national trend and the national trend in mortality.[82] Greven found estimates of the local coefficient $ß_1$ to be approximately zero and non-significant nationally and in all three regions of the United States (East, Center and

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80 Ex. 441 at App. 2 at Attachment 2 (McClellan, *Hazard and risk: assessment and management* (book chapter)) at 78. Dr. Desvousges meanwhile testified that the average mean ambient-air $PM_{2.5}$ concentration of the studies relied upon was 16 μg/$m^3$. (Tr. Vol. 7 at 106:18-22.)

81 Ex. 441 at App. 2 at 8.

82 *Id.*

West).[83] Estimates of $ß_1$ indicate that after adjusting for the association between national trends in mortality and $PM_{2.5}$, there is no significant association between an increase in the local yearly average $PM_{2.5}$ concentrations and the risk of dying in a given month.[84] Dr. McClellan testified that this important finding is illustrated graphically in Figure 6 (ellipses added),[85] and "clearly applicable to the Minnesota case, since the data base used includes populations with monitored ambient-air concentrations of PM2.5 substantially in excess of those measured in Minnesota":[86]

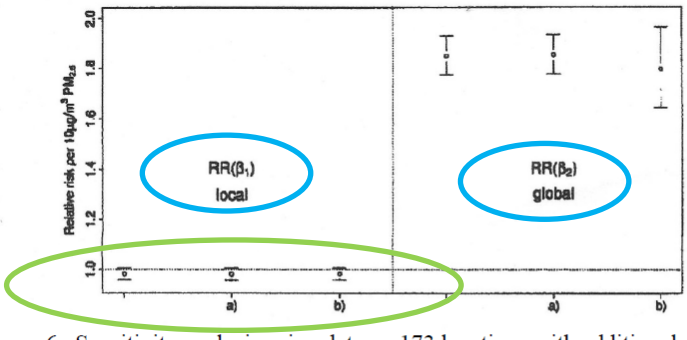


Figure 6. Sensitivity analysis using data on 173 locations with additional variables from the BRFSS-SMART survey. The left-most estimate shows estimate $ß_1$ and $ß^2$ from model (3) for this subset of the data. a) indicates the analysis including additional variables on the level of the monitor's county; the proportion of current smokers and of nonwhites, and the mean income and body mass index. b) gives the results for the same analysis allowing separate coefficients for the four variables' global and local trends [Greven, Dominici and Zeger, 2011]

Enlarging the relevant area shows clearly that the mean value for local sensitivity to $PM_{2.5}$ is below 1.0, and that even the statistical bands of uncertainty are almost entirely below 1.0:

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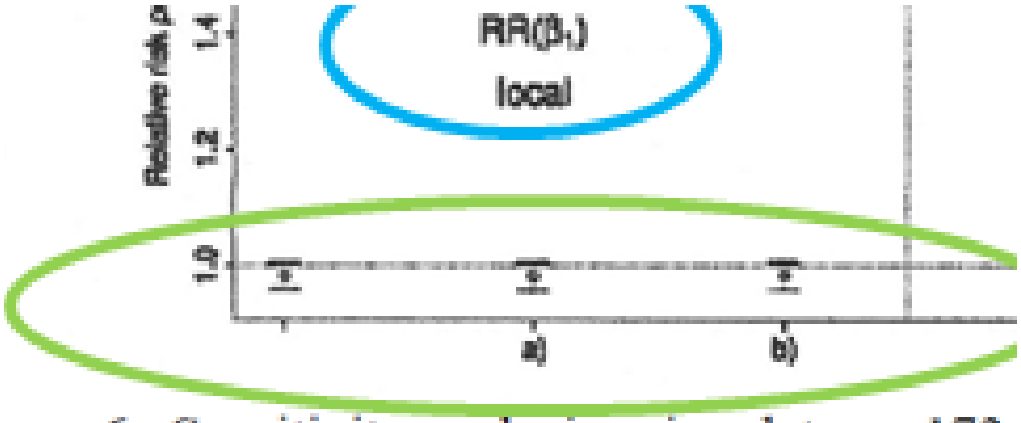
83 Ex. 441 at App. 2 at 8.

84 *Id.*

85 Ex. 441 at Appendix 2 at 8, 17.

86 Ex. 441 at App. 2 at 8-9.




gure 6. Sensitivity analysis using data on 173

In other words, there is no statistical or medical evidence of an association between exposure to $PM_{2.5}$ and adverse health effects in the Greven study at the local level.[87] Dr. McClellan further opined, based on the findings of Lepeule *et al*. (2012), that for downwind areas where mean annual $PM_{2.5}$ ambient-air concentrations averaged over 3 years are in excess of 12 $\mu g/m^3$, any calculated increase in mortality attributable will be extraordinarily small related to the baseline mortality.[88] Dr. McClellan accordingly concluded, with a reasonable degree of medical certainty, that the primary (or health) damages conclusions reached by Drs. Muller, Marshall, and Desvousges are invalid, based as they were on national concentration-response data, rather than local data, and because they were based on linear air concentration-response models that were applied to all emissions irrespective of the air quality in a particular area and without considering

87 Ex. 441 at App. 2 at 8.

88 *Id.*

the community-exposure level.[89]

2. The U.S. EPA agrees that there is no proximate cause between exposure to $PM_{2.5}$ at ambient-air concentrations below 12 $\mu g/m^3$ and human-health impact

Dr. McClellan's epidemiological opinion is supported not only by a lack of impeaching data, cross-examination, and absence of contrary testimony, but is in fact affirmatively supported by a vast amount of research undertaken pursuant to the United States Clean Air Act by the EPA, which issued a formal final updated rule when it set new NAAQS for Particulate Matter as announced in the January 15, 2013, Federal Register.[90]

By law, the primary NAAQS "shall be ambient air quality standards the attainment and maintenance of which in the judgment of the [EPA] Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."[91] In the new NAAQS rules, the EPA made revisions to the suite of standards for particulate matter ("PM") "to provide requisite protection of public health and welfare and to make corresponding revisions to the data handling conventions for PM and to the ambient air monitoring, reporting, and network design requirements,"

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89 *See, e.g.*, Ex. 441 at 21; Tr. Vol. 7 at 177:18-178:8. Additionally, Dr. Desvousges admitted that if the assumption of a linear relationship between $PM_{2.5}$ exposure and human-health effects were incorrect from a medical perspective, it would affect his analysis. (Tr. Vol. 7 at 84:7-85:6.)

90 Ex. 444A (Federal Register / Vol. 78, No. 10 / Tuesday, January 15, 2013 (pp. 3087-3167 and 3265) (Air Quality Designations for the 2012 Primary Annual Fine Particle ($PM_{2.5}$) National Ambient Air Quality Standards (NAAQS); Final Rule)).

91 42 U.S.C. § 7409(b)(1).

"based on its review of the air quality criteria and the national ambient air quality standards (NAAQS) for particulate matter."[92] Among other changes, the EPA revised the annual primary (health-based) standards for $PM_{2.5}$ by lowering the level to 12.0 $\mu g/m^3$ "so as to provide increased protection against health effects associated with long- and short-term exposures (including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease), and to retain the 24-hour $PM_{2.5}$ standard at a level of 35 $\mu g/m^3$."[93]

Sections 108 and 109 of the Clean Air Act, 42 U.S.C. §§ 7408 and 7409, govern the establishment, review, and revision, as appropriate, of the NAAQS to protect public health and welfare. The Clean Air Act requires periodic review of the air quality criteria—the science upon which the standards are based—and the standards themselves.[94] The final rule announced in the January 15, 2013, Federal Register was made pursuant to these statutory requirements.[95] Between 2007 and 2011, the EPA prepared draft and final Integrated Science Assessments, Risk and Exposure Assessments, and Policy Assessments.[96] Multiple drafts of all of these documents were subject to review by the public and were peer reviewed by CASAC, the independent

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92 *See* 42 U.S.C. § 7409(d); Federal Register / Vol. 78, No. 10 / Tuesday, January 15, 2013 (pp. 3086-3287, available in full at https://www.gpo.gov/fdsys/pkg/FR-2013-01-15/pdf/2012-30946.pdf) ("2013 NAAQS Fed. Reg.") at 3086.

93 2013 NAAQS Fed. Reg. at 3086

94 Ex. 444A at 3088.

95 *Id.*

96 *Id.*

scientific review committee established pursuant to 42 U.S.C. § 7409(d)(2)(A).[97] The EPA proposed revisions to the primary and secondary PM NAAQS on June 29, 2012.[98] The final rulemaking announced in the January 15, 2013, Federal Register was the final step in the review process.[99]

The EPA announced that "[t]his action provides increased protection for children, older adults, persons with pre-existing heart and lung disease, and other at-risk populations against an array of $PM_{2.5}$-related adverse health effects that include premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease. The EPA also is eliminating spatial averaging provisions as part of the form of the annual standard to avoid potential disproportionate impacts on at-risk populations."[100]

In preparing the 2013 PM NAAQS, the Administrator of the EPA recognized that the Clean Air Act "requires her to reach a public health policy judgment as to what standards would be requisite—neither more nor less stringent than necessary—to protect public health with an adequate margin of safety, based on scientific evidence and technical assessments that have inherent uncertainties and limitations. This judgment requires making reasoned decisions as to what weight to place on various types of

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97 Ex. 444A at 3088, 3090 (independent review function performed by CASAC since early 1980's).

98 *See* 77 Federal Register 38890.

99 Ex. 444A at 3088.

100 Ex. 444A at 3088.

evidence and assessments, and on the related uncertainties and limitations. Thus, in selecting the final standards, *the Administrator is seeking not only to prevent fine particle concentrations that have been demonstrated to be harmful but also to prevent lower fine particle concentrations that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree*."[101] In other words, the judgment exercised by the Administrator was exercised with a thorough eye on public safety.

In addition to previously-considered or existing epidemiological studies, the EPA considered "hundreds of new epidemiological studies conducted in many countries around the world."[102] Not surprisingly, the EPA "placed greater weight on U.S. and Canadian studies using $PM_{2.5}$ measurements, since studies conducted in other countries may reflect different demographic and air pollution characteristics."[103] The newly available research studies as well as the earlier body of scientific evidence presented and assessed in the Integrated Science Assessment underwent intensive scrutiny through multiple layers of peer review and opportunities for public review and comment.[104] In developing the final rule, the EPA drew upon "an integrative synthesis of the entire body of evidence concerning exposure to ambient fine particles and a broad range of health endpoints," "focusing on those health endpoints for which the Integrated Science Assessment concludes that there is a *causal or likely causal relationship* with long- or

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101 Ex. 444A at 3097 (emphasis added).

102 *Id.*

103 *Id.*

104 *Id.*

short-term $PM_{2.5}$ exposures."[105] (*See e.g.*, Ex. 444A at 3131-3133, 3135.) But the EPA also considered health endpoints for which the Integrated Science Assessment concluded there was evidence *suggestive of a causal relationship* with long-term $PM_{2.5}$ exposures.[106] (*See e.g.*, Ex. 444A at 3131-3133, 3135.) The EPA further drew upon "a quantitative risk assessment based upon the scientific evidence described and assessed in the Integrated Science Assessment."[107] These analyses also underwent "intensive scrutiny through multiple layers of peer review and multiple opportunities for public review and comment."[108] It should be noted that while Dr. Marshall provided extensive testimony in this case, he proved at the evidentiary hearing to be wholly unfamiliar with the NAAQS rule, the scientific information underlying the rule, and the process used by the EPA to arrive at the rule; an astonishing feat for one who would hold himself out to be an expert in this case.[109]

Dr. Desvousges testified (and showed) that he was familiar with the EPA's 2013 PM Final Rule and agreed that the rigor of the EPA review made the studies upon which the EPA relied in issuing that Final Rule "the most reliable source of scientific

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105 Ex. 444A at 3097 (emphasis in original).

106 *Id.* (emphasis in original).

107 *Id.*

108 *Id.*

109 *See, e.g.*, Tr. Vol. 6 at 58 (denying 12 $\mu g/m^3$ NAAQS in effect), 72-73, 101-102; Tr. Vol. 7 at 43 (admitting 12 $\mu g/m^3$ NAAQS in effect based on Ex. 453). *See also* 2013 NAAQS Fed. Reg. at 3086 ("final rule is effective on March 18, 2013").

information on which to base decisions."[110] The EPA recognized that "the strongest evidence of associations occurs at concentrations around the long-term mean concentration."[111] "Thus, in earlier reviews, the EPA focused on identifying standard levels that were somewhat below the long-term mean concentrations reported in $PM_{2.5}$ epidemiological studies. The long-term mean concentrations represented air quality data typically used in epidemiological analyses and provided a direct link between $PM_{2.5}$ concentrations and the observed health effects."[112] "These data were available for all long- and short-term exposure studies analyzed and, therefore, represented the data set available for the broadest set of epidemiological studies."[113]

The EPA explored ways to take into account additional information from epidemiological studies, focusing on evaluating different statistical metrics, beyond the long-term mean concentration, to characterize the part of the distribution of $PM_{2.5}$ concentrations in which it continued to have confidence in the associations observed in epidemiological studies and below which there was a comparative lack of data such that confidence in the relationship was appreciably less.[114] This would also be the part of the distribution of $PM_{2.5}$ concentrations which had the most influence on generating the

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110 Tr. Vol. 7 at 85:10-17, 86:15-87:7, 87:25-88:2.

111 Ex. 444A at 3129.

112 *Id.*

113 *Id.*

114 *Id.*

health effect estimates reported in epidemiological studies.[115] The EPA's Policy Assessment concluded that focusing on concentrations within the lower quartile of a distribution, such as the range from the 25th to the 10th percentile, was reasonable to consider as a region within which to begin to have appreciably less confidence in the associations observed in epidemiological studies.[116]

Contrary to Drs. Marshall, Muller, and Desvousges, the EPA spent significant time and resources determining whether concentration-response functions should be trusted at all ambient-air concentrations. Following those studies, the EPA and its scientific and epidemiological advisors determined that considering $PM_{2.5}$ concentrations down to the lowest concentration observed in a study would be "a highly uncertain basis for selecting alternative standard levels."[117] Dr. Desvousges "would not disagree with" this approach to the reliability of the study data.[118] Notwithstanding this "restriction," the EPA took into consideration "studies that were very much towards the low end of the $PM_{2.5}$ ambient air concentrations."[119]

The EPA graphically displayed the most important studies in the Final Rule announcement, including the following graphic, taken from Exhibit 444A at page 3135

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115 Ex. 444A at 3129.

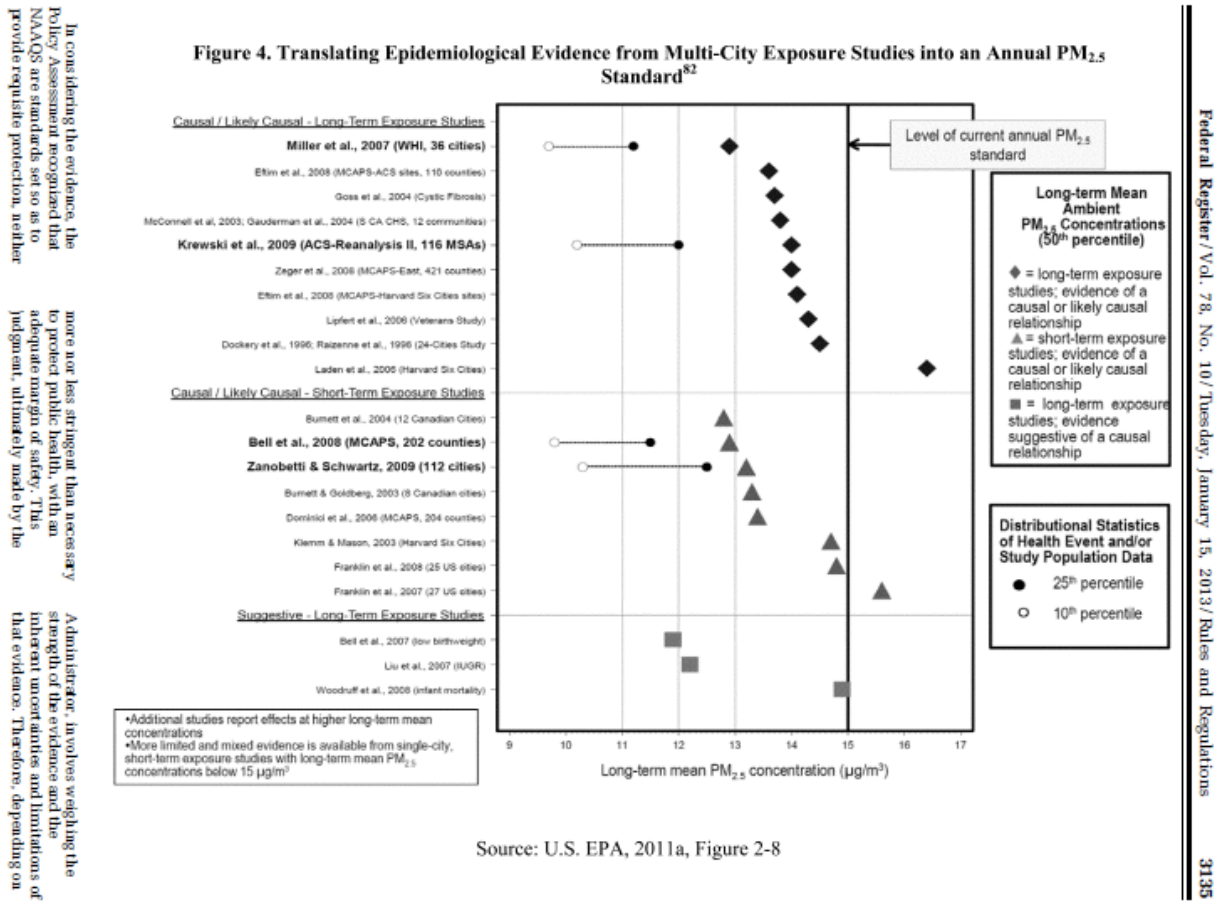
116 *Id.*; *see also* Tr. Vol. 7 at 102:24-103:3 (Desvousges); *see further* Tr. Vol. 7 at 52:8-16 (Dr. Desvousges explains why he relied on the inner quartile to address major uncertainties in the underlying data).

117 Ex. 444A at 3129.

118 Tr. Vol. 7 at 104:1-15.

119 Tr. Vol. 7 at 106:1-4; Ex. 444A at 3135.

(*see also* at 3131-3133), which showed for all studies suggestive of a causal or likely causal relationship *and* all studies merely suggestive of a causal relationship a 3-year average mean ambient air $PM_{2.5}$ concentration well above 12 $\mu g/m^3$:




Source: U.S. EPA, 2011a, Figure 2-8

In considering the evidence, the Policy Assessment recognized that NAAQS are standards set so as to provide requisite protection, neither more nor less stringent than necessary to protect public health, with an adequate margin of safety. This judgment, ultimately made by the Administrator, involves weighing the strength of the evidence and the inherent uncertainties and limitations of that evidence. Therefore, depending on



Against the legal background that the requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting, and to provide a reasonable degree of protection against hazards that research has not yet identified, *see Lead Indus. Ass'n v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980); *Am.*

*Petroleum Inst. v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981); *Am. Farm Bureau Fed'n v. EPA*, 559 F.3d 512, 533 (D.C. Cir. 2009); *Ass'n of Battery Recyclers v. EPA*, 604 F.3d 613, 617–18 (D.C. Cir. 2010), and based on all of the available studies, materials, and scientific advice available to the federal government, the EPA found that it could protect public health with an adequate margin of safety in setting the NAAQS limit at 12 $\mu g/m^3$.[120] The EPA found no evidence of a reliable causal relationship between $PM_{2.5}$ exposure and human health risk below this standard.[121]

3. According to the California Air Resources Board, a $PM_{2.5}$ ambient-air concentration standard of 12 $\mu g/m^3$ adequately protects the health of the public with an adequate margin of safety

Similar to the EPA's NAAQS, the California Air Resources Board sets state ambient-air quality standards ("AAQS") for particulate matter.[122] Similarly to the EPA, the California Board is charged by statute with establishing the ambient-air standards "at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety."[123] In June of 2002, after study and a peer review process, California adopted new, revised PM AAQS for outdoor air, lowering the annual $PM_{10}$ standard from 30 $\mu g/m^3$ to 20 $\mu g/m^3$ and establishing a new annual standard for $PM_{2.5}$ of

120 Ex. 444A at 3088-3089.

121 Ex. 444A.

122 *See* California Health & Safety Code § 39606.

123 *See* California Health & Safety Code § 39606(d)(2).

12 μg/m$^3$.[124] The new California PM AAQS became effective on July 5, 2003.[125]

The MLIG respectfully submits that the unchallenged testimony of Dr. McClellan, the epidemiological evidence, and both the EPA's and the California Air Resources Board's setting of a 12 μg/m$^3$ $PM_{2.5}$ average mean ambient-air quality standard as "protective with an adequate margin of safety," prove that Dr. Marshall's, Dr. Desvousges' and Dr. Muller's failure to base their primary (or health) damages conclusions on local concentration-response data, rather than national data, and their failure to consider the community-exposure level render their methodology and opinions invalid with respect to impacts and damages resulting from Minnesota emissions of primary $PM_{2.5}$ and the formation of secondary $PM_{2.5}$ at ambient-air exposure levels below 12 μg/m$^3$.

## IV. ANY CONSIDERATION OF DAMAGES SHOULD BE LIMITED TO A LOCAL GEOGRAPHIC SCOPE

The extraterritorial damages issue, like damages within Minnesota, is a function of downwind damages. Dr. Desvousges has credibly testified that the uncertainty already present in the damages calculations for Minnesota and a 100 mile rectangular grid around Minnesota, even using the complex CAMx photochemical grid model, becomes significantly greater as the distance from the source increases.[126] While the EPA used

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124 *See* 17 California Code of Regulation §§ 70100, 70100.1, and 70200. *See also* Ex. 444A at 3110.

125 *See* 17 California Code of Regulation §§ 70100, 70100.1, and 70200.

126 *See, e.g.*, Ex. 609 at at 35:8-14, 45:26-46:2; Tr. Vol. 7 at 115:2-116:6, 133:24-134:13, and 135:16-18.

CAMx for analysis under the Cross-State Air Pollution Rule ("CSAPR"),[127] Dr. Desvousges explained that there is a difference between the way CAMx was used by the EPA in the CSAPR process and the way the model is used here, "[b]ecause in this particular proceeding what we are trying to do is to come up with reliably estimated externality values that involve combining information with a lot of different uncertainties. What EPA [was] looking at [in the CSAPR process] [was] trying to predict various changes in air emissions that would happen under different regulatory scenarios."[128] Accordingly, EPA's correct use of CAMx for the CSAPR analysis has no relevancy to endorsing that, or any other model, for national calculations of the sort made here.

It is noteworthy that the EPA has limited use of reduced form models (or at least AP2, as InMap has not even been evaluated yet) to 50 kilometers from the source,[129] and that both appear to create significantly higher damages outside of Minnesota than within Minnesota, and according to Dr. Desvousges "yield questionable results that should not be relied on for the establishment of externality values in this docket."[130]

Where the Commission appropriately declined to review outdated EPA data in the Original Proceeding because it was not reliable,[131] the same can no longer be said. Taking into consideration the EPA's up-to-date analysis of epidemiological studies of

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127 Tr. Vol. 8 at 68:5-9.

128 Tr. Vol. 7 at 61:15-62:9 (Desvousges).

129 *See, e.g.*, Ex. 604 at 10:11-13; Tr. Vol. 7 at 56:8-15.

130 Ex. 604 at 10:16-19.

131 *See* Order Establishing Environmental Cost Values, MPUC Docket No. 93-583, Jan. 3, 1997 at 16-17.

$PM_{2.5}$ impacts, and Dr. McClellan's testimony regarding the insufficiency and invalidity of the health-impact assumptions and thus the damages calculations of Drs. Marshall, Desvousges, and Muller, the MLIG respectfully submits that extrapolating the problems of those reports with an extra-territorial and unreliable damages prong would yield arbitrary and capricious results. The MLIG accordingly respectfully submits that any consideration of damages should be limited to a local geographic scope, such as was performed by Dr. Desvousges.

## CONCLUSION

At the outset of this proceeding, the MLIG urged the Commission and the Administrative Law Judge to proceed in this proceeding in a statistically sound, evidence-based approach.[132] The MLIG submitted that the outcome of this proceeding should be based on empirical evidence, sound analysis, that it should avoid undue speculation, and that it should be respectful of Minnesota and Minnesota commerce and industry.[133]

This phase of the case begins and ends with the burden of proof. Neither the CEOs, nor the Agencies, nor Xcel can meet their burden of proof, because each of their experts has failed to make the required proximate-cause connection between primary $PM_{2.5}$ emission and secondary $PM_{2.5}$ formation in and above Minnesota on the one hand and human-health damages in a low-$PM_{2.5}$ ambient-air environment on the other hand, as

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132 Tr. Vol. 6 at 24.

133 *Id.*

testified to by Dr. McClellan and recognized by the EPA and the State of California, and based on epidemiological literature and studies. In the absence of a breakout of the remaining (non-health) damages studied by Dr. Desvousges and Dr. Muller, no evidence as to any admissible damages exists in the record.

The MLIG additionally submits that any consideration of other damages, such as agricultural, materials, and visibility damages, should be limited to a local geographic scope due to the significant uncertainties and unreliability of national scope calculations by the models, as testified to by Dr. Desvousges. Acceptance of a national geographic scope would accordingly be neither statistically sound nor based upon reliable evidence.

Respectfully submitted,

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