ATTACHMENT A

PLACEHOLDER FOR CBI MATERIAL

ATTACHMENT B

Preliminary Risk Assessment of Manganese in Ambient Air at the S.H. Bell Company Facility in Chicago, Illinois

Prepared for
S.H. Bell Company
10218 S. Avenue O
Chicago, Illinois 60617

September 19, 2017

GRADIENT

www.gradientcorp.com
20 University Road
Cambridge, MA 02138
617-395-5000

Table of Contents

Page

Executive Summary ES-1

1 Manganese Air Monitoring Data and Exposure Evaluation 1
1.1 Mn Air Monitoring Data 1
1.2 Mn Exposure Concentrations 1
1.2.1 Consideration of Time Spent Indoors and Away from Home 2

2 Dose-Response Evaluation 4
2.1 Manganese Essentiality and Health Effects 4
2.2 Manganese Chronic Inhalation Toxicity Criteria and Application to Risk Assessment 4
2.2.1 Manganese Inhalation Toxicity Value 5

3 Risk Calculations 7

4 Preliminary Risk Evaluation Conclusion 8

References 9

List of Tables

Table 3.1 Mn PM_{10} Air Concentrations, Exposure Concentrations, and Hazard Indices

List of Figures

Figure ES.1 S.H. Bell Chicago Mn PM_{10} Concentrations Compared to Mn Air Concentrations with No Health Effects

Executive Summary

Gradient has reviewed the S.H. Bell Chicago manganese (Mn) PM_{10}[1] air monitoring data available on the US Environmental Protection Agency (US EPA) in Illinois website (US EPA, 2017). The website includes data from March-July 2017. S.H. Bell provided Gradient with additional Mn PM_{10} monitoring data from August 2017. Gradient conducted a preliminary risk evaluation from these Mn data, the results of which are summarized below.

- Gradient conducted a conservative screening-level risk evaluation, consistent with US EPA risk assessment guidelines (US EPA, 1989), from 6 months of Mn PM_{10} data collected at the S.H. Bell Chicago facility. Mn concentrations ranged from 0.018-1.23 $\mu g/m^3$, with an arithmetic mean (or average) of 0.292 $\mu g/m^3$ for the March -August 2017 data. The arithmetic mean Mn PM_{10} concentrations for the March-June and March-July 2017 data were 0.318 $\mu g/m^3$ and 0.310 $\mu g/m^3$, respectively, indicating that the mean Mn concentration has decreased over time.
- We compared the Mn air concentrations (adjusted for an exposure frequency of 350 days per year, per US EPA guidelines) to the Agency for Toxic Substances and Disease Registry (ATSDR) chronic Mn Minimum Risk Level (MRL) of 0.3 $\mu g/m^3$ (ATSDR, 2012). The MRL is a health-protective air concentration that is well below the level of Mn in air estimated to cause no adverse effects following continuous exposure (34 $\mu g/m^3$) and well below the threshold Mn concentration that is not expected to increase normal levels of Mn in the brain (10 $\mu g/m^3$). This comparison results in a hazard index (HI). HIs at or below 1 mean that there is no risk of adverse effects.[2] The results of this comparison are presented in Figure ES.1 below.
- HIs calculated from mean Mn PM_{10} concentrations from data from the three exposure periods (March-June 2017, March-July 2017, and March-August 2017) are all at or below 1 (see below), indicating there is no risk of adverse neurological effects, the most sensitive health endpoint for Mn, for the general population (including sensitive subpopulations) from continuous inhalation of Mn in ambient air in the vicinity of the S.H. Bell Chicago, Illinois facility.
  - HI for March-June 2017 data = 1
  - HI for March-July 2017 data = 1
  - HI for March-August 2017 data = 0.9
- In addition, the risk calculation is based on a high estimate of Mn exposure that assumes a resident inhales outdoor air at their home for 24 hours per day, for 350 days per year. Consistent with the US EPA exposure factor guidelines, it is likely that time spent indoors and away from home would effectively reduce the Mn exposures by about 50%, reducing the HIs further.
- Given the conservative and health-protective basis of the Mn risk calculations in our evaluation, Gradient concludes, based on the available data, that there is no evidence that Mn in ambient air near the S.H. Bell Chicago facility will cause adverse health effects in the nearby community.

[1] PM_{10} = Particulate matter ≤10 µm in diameter.
[2] This is based on US EPA's target HI of 1, meaning that no adverse effects are expected in the population if the HI is equal to 1 or lower (US EPA, 1989). An HI greater than 1 does **not** mean that adverse effects are likely to occur, but that more investigation may be necessary.

- Because ATSDR's chronic Mn MRL is derived for comparison to an exposure concentration averaged over 1 year or more, Mn PM_{10} data collection should continue at least through the end of February 2018 and the Mn risk should be re-evaluated at that point.

Sections 1-4 present the details of our risk evaluation.

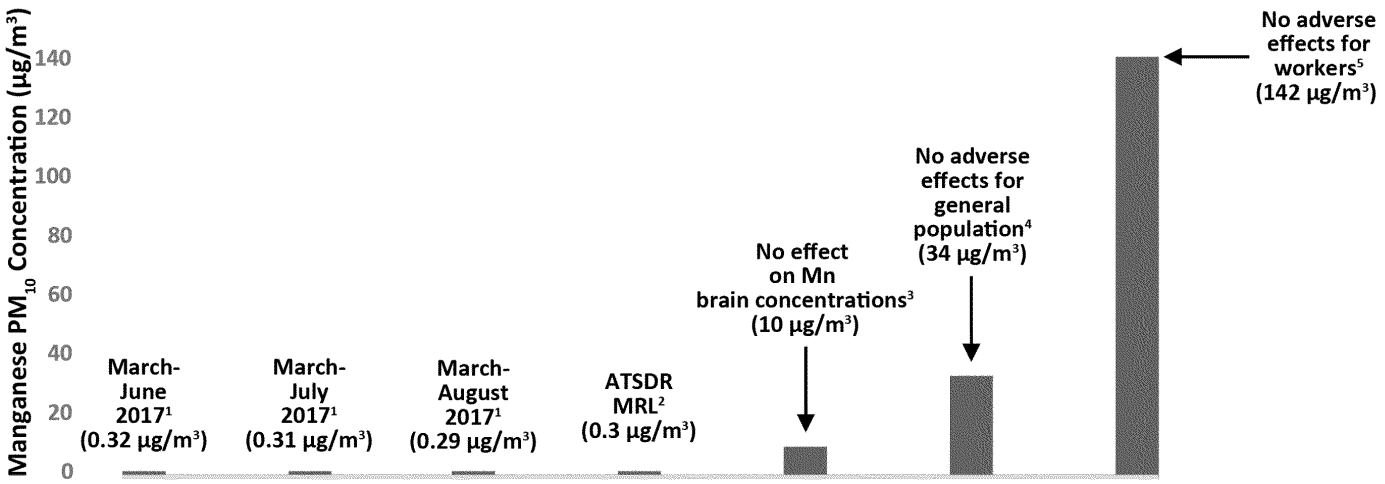


Figure ES.1 S.H. Bell Chicago Mn PM_{10} Concentrations Compared to Mn Air Concentrations with No Health Effects. ATSDR = Agency for Toxic Substances and Disease Registry; Mn = Manganese; MRL = Minimal Risk Level; PM_{10} = Particulate Matter ≤10 µm in Diameter. (1) Mn PM_{10} concentrations represent the arithmetic mean concentration over the exposure period listed. (2) ATSDR MRL = 0.3 µg/m^3 (ATSDR, 2012). (3) Exposure concentration at or below which Mn levels in the brain are not expected to increase above normal levels for fetuses, infants, children, and adults (Schroeter *et al.*, 2011, 2012; Yoon *et al.*, 2011). (4) No adverse effect for the general population (*i.e.*, continuous exposure) estimated from the no adverse effect worker exposure concentration (142 µg/m^3 x 5/7 days per week x 8/24 hours per day = 34 µg/m^3). (5) No adverse effect worker exposure concentration estimated from the Roels *et al.* (1992) study (*i.e.*, $BMDL_{10}$, or 95% lower confidence limit on the benchmark dose for a 10% extra risk compared to controls).

1 Manganese Air Monitoring Data and Exposure Evaluation

Gradient has reviewed the S.H. Bell Chicago manganese (Mn) PM_{10}[3] air monitoring data available on the US Environmental Protection Agency (US EPA) in Illinois website (US EPA, 2017). The website includes data from March-July 2017. S.H. Bell provided Gradient with additional Mn PM_{10} data from August 2017.[4] This section describes the Mn data and exposure evaluation applied in the risk assessment.

1.1 Mn Air Monitoring Data

Mn PM_{10} air monitoring data from US EPA's S.H. Bell Chicago Air Monitoring Data website (US EPA, 2017) consist of approximately 10 samples per month (approximately 1 sample collected every 3 days), for a total of 61 samples collected from the beginning of March through the end of August 2017. Mn samples were collected from the S4 monitoring station, which is one of four monitoring stations located on the S.H. Bell property. The S4 monitoring station is located in the northern portion of the S.H. Bell property, as depicted on the US EPA website (US EPA, 2017).

Mn concentrations ranged from 0.018-1.23 $\mu g/m^3$, with an arithmetic mean of 0.292 $\mu g/m^3$ for the March-August 2017 data. The arithmetic mean Mn PM_{10} concentrations for the March-June and March-July 2017 data were 0.3 18 $\mu g/m^3$ and 0.31 0 $\mu g/m^3$, respectively, indicating that the mean Mn concentration has decreased over time. The arithmetic mean concentrations, described on the US EPA in Illinois website (US EPA, 2017), are used to derive the exposure point concentrations described below.

Note that, as described below, the risk evaluation applies the Agency for Toxic Substances and Disease Registry's (ATSDR) chronic Mn Minimal Risk Level (MRL) that is derived for application to an exposure concentration averaged over 1 year or more. The data used in this evaluat ion include 6 months of air samples during the spring and summer months, and, therefore, do not reflect Mn concentrations during other times of the year, when concentrations may differ (September-February). Sampling should continue during these months, and the risk evaluation should be conducted again with at least a full year of Mn PM_{10} data.

1.2 Mn Exposure Concentrations

The Mn inhalation exposure concentration (EC) is calculated as follows, per US EPA risk assessment guidelines (US EPA, 1989):

$$EC\ (\mu g/m^3) = CA \times EF \times ED \div AT$$

where:

CA = Average Mn PM_{10} Concentration in Air ($\mu g/m^3$) (US EPA, 2017)
EF = Exposure Frequency (days/year)

[3] PM_{10} = Particulate matter ≤10 µm in diameter.
[4] These data are preliminary and have not undergone quality assurance/quality control (QA/QC) review.

ED = Exposure Duration (years)
AT = Averaging Time (days)

US EPA typically considers a high-end residential exposure frequency of 350 days per year, an exposure duration of 30 years, and an averaging time of 30 years (or 10,950 days) for non-cancer risk evaluations (US EPA, 1989).

With these exposure assumptions, we calculate the following Mn ECs from the data for three exposure periods (March-June 2017, March-July 2017, and March-August 2017).

March-June 2017 data (40 samples) result in an EC of 0.305 μg/m^3:

$$EC = 0.318\ \mu g/m^3 \times 30\ \text{years} \times 350\ \text{days/year} \div 10{,}950\ \text{days}$$
$$EC = 0.305\ \mu g/m^3$$

March-July 2017 data (51 samples) result in an EC of 0.297 μg/m^3:

$$EC = 0.310\ \mu g/m^3 \times 30\ \text{years} \times 350\ \text{days/year} \div 10{,}950\ \text{days}$$
$$EC = 0.297\ \mu g/m^3$$

March-August 2017 data (61 samples) result in an EC of 0.280 μg/m^3:

$$EC = 0.292\ \mu g/m^3 \times 30\ \text{years} \times 350\ \text{days/year} \div 10{,}950\ \text{days}$$
$$EC = 0.280\ \mu g/m^3$$

1.2.1 Consideration of Time Spent Indoors and Away from Home

It is important to point out that the Mn ECs estimated above are for a resident who inhales Mn in outdoor air for 24 hours per day, for 350 days per year. The selection of 24 hours per day as the daily exposure duration implicitly assumes either that residents are outdoors for 24 hours per day, for 350 days per year, or that the concentration of indoor Mn particulates is the same as outdoor Mn particulates. Neither assumption is reasonable for the US population. The US EPA Exposure Factors Handbook (USEPA, 2011) reports that the 95th percentile time spent outdoors at a residence was 7.3 hours/day (30%) for adults (>18 years old) (16.7 hours/day indoors, or 70%). US EPA (2011) also indicates that the amount of time spent indoors for infants and children under the age of 2 is nearly the entire day (mean: 22 hours; 95th percentile: 24 hours). The US EPA Exposure Factors Handbook (US EPA, 2011) also indicates that the mean time spent away from home for adults who are 18-64 years old is approximately 7 hours/day (30% of time away from home).

Furthermore, a number of studies conducted in urban areas across the US and Canada have demonstrated that only a fraction of ambient particulates are capable of penetrating into homes (Ozkaynak *et al.*, 1996; Long *et al.*, 2001; Allen *et al.*, 2003; Williams *et al.*, 2003; Wallace and Williams, 2005; Sarnat *et al.*, 2006; Clark *et al.*, 2010). Particle infiltration is well-recognized to be highly variable, depending on particle properties (*e.g.*, size distribution, composition), season, home ventilation conditions, and home building characteristics (*e.g.*, age, construction type). The range of average particle infiltration factors (fraction of ambient particles remaining airborne indoors) from these studies is 0.48-0.74, with an overall average across studies of 0.60. For example, Sarnat *et al.* (2006) estimated an average particle infiltration factor of 0.48 for $PM_{2.5}$, based on 17 homes in Los Angeles, California. Long *et al.* (2001) estimated a $PM_{2.5}$ infiltration factor of 0.74 from 9 residential homes in Boston, Massachusetts. More recently, Clark *et al.*

(2010) estimated an infiltration factor of 0.52 from 46 residential homes in Toronto, Canada. Because the relative contribution of ambient Mn levels would be reduced in indoor air, as compared to outdoor air, it is scientifically appropriate to incorporate information on the apportionment of time between outdoor and indoor activities when estimating effective high-end exposure concentrations.

Consideration of these more realistic exposure assumptions about time spent indoors and away from home would effectively reduce the EC by about 50%. For example, if one assumes that the outdoor Mn air concentration is 0.3 $\mu g/m^3$, applying the adjustments discussed above would be as follows:

$$[(30\% \text{ time outdoors} \times 0.3\ \mu g/m^3) + (70\% \text{ time indoors} \times 60\% \text{ infiltration from outdoor air} \times 0.3\ \mu g/m^3)] \times 70\% \text{ of time spent at residence} = 0.151\ \mu g/m^3$$

2 Dose-Response Evaluation

2.1 Manganese Essentiality and Health Effects

Mn is a naturally occuring element and the fifth-most-abundant metal in the earth's crust. Mn is an essential nutrient that is necessary for the function of several enzyme systems and cell energy production in humans. A sufficient intake of Mn is needed for the formation of healthy cartilage and bone (ATSDR, 2012) and for neuronal health (Horning *et al.*, 2015; Chen *et al.*, 2015). Therefore, a deficiency of Mn can cause adverse health effects, including adverse neurological effects. In addition, because excess Mn accumulates in the brain, exposure to elevated levels of Mn *via* ingestion or inhalation can also cause adverse neurological effects (ATSDR, 2012; Horning *et al.*, 2015). Therefore, maintaining appropriate levels of Mn in the body is critical for human health.

The most common health effects associated with chronic inhalation of elevated levels of Mn in occupational environments are neuromotor deficits (*e.g.*, tremor, hand-eye coordination) (ATSDR, 2012). Chronic exposure to high levels of Mn (*i.e.*, greater than 2 mg/m^3) can cause a disabling syndrome called "manganism," which includes a dull affect, altered gait, fine tremor, headaches, and sometimes psychiatric disturbances (ATSDR, 2012). Studies suggest that chronic exposure to low levels of Mn in ambient air are unlikely to be associated with neurological effects. Typical levels of Mn in ambient air range from 0.02 μg/m^3 (mean in the US) to 0.3 μg/m^3, near industrial facilities (ATSDR, 2012).

2.2 Manganese Chronic Inhalation Toxicity Criteria and Application to Risk Assessment

US EPA and other regulatory agencies (*e.g.*, ATSDR) derive chronic inhalation toxicity criteria that are estimates of continuous inhalation exposure concentrations for individuals (including sensitive subpopulations) that represent negligible, if any, risk for adverse health effects during a lifetime. These toxicity criteria are derived from scientific studies in animals or humans, using either no observed adverse effect levels (NOAELs) (*i.e.*, exposure levels at which no statistically significant increases in adverse effects are observed between exposed and unexposed populations), or benchmark dose (BMD) concentrations (*e.g.*, $BMDL_{10}$ value, which is a 95% lower confidence limit on the BMD for a 10% extra risk compared to controls) as the point of departure (POD). The POD is typically divided by uncertainty factors (UFs) to account for various uncertainties in the underlying animal or human toxicity study (*e.g.*, sensitive subpopulations). Thus, inhalation toxicity criteria are developed to be well below concentrations that have been observed to cause adverse health effects. Regulatory agencies have different names for such criteria, although the values are derived using similar methodologies and are applied similarly in making decisions to manage risks from chemicals. For example, the US EPA inhalation criteria are termed as "reference concentrations" (or "RfCs"), and the ATSDR inhalation criteria are termed "minimal risk levels" (or "MRLs").

Exceedance of a chronic toxicity value does not indicate that any one individual is at elevated risk. That is, chronic toxicity values that include uncertainty factors and assumptions of continuous exposures, such as ATSDR MRLs and US EPA RfCs, are not intended to be an exact line above which toxic effects will occur and below which no effects will occur. US EPA has explained that toxicity criteria published in their

Integrated Risk Information System (IRIS) database cannot be used to predict whether or not an adverse health effect will occur:

> In general, **IRIS values cannot be validly used to accurately predict the incidence of human disease** or the type of effects that chemical exposures have on humans. This is due to the numerous uncertainties involved in risk assessment, including those associated with extrapolations from animal data to humans and from high experimental doses to lower environmental exposures. The organs affected and the type of adverse effect resulting from chemical exposure may differ between study animals and humans. In addition, many factors besides exposure to a chemical influence the occurrence and extent of human disease. (US EPA, 2005 [emphasis added])

ATSDR includes a similar discussion in describing MRLs:

> These substance-specific estimates [MRLs], which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that **MRLs are not intended to define clean-up or action levels**… MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are **below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. Exposure to a level above the MRL does not mean that adverse health effects will occur.** (ATSDR, 2012 [emphasis added])

2.2.1 Manganese Inhalation Toxicity Value

As discussed on the US EPA in Illinois website (US EPA, 2017), the arithmetic mean Mn concentration is compared to the ATSDR Mn MRL of 0.3 μg/m^3 (ATSDR, 2012). The ATSDR MRL is based on the most current science and, thus, is the most appropriate toxicity value to apply in a Mn inhalation risk assessment. The ATSDR Mn MRL is based on observations of subclinical neurological effects in workers exposed to Mn for an average of 5.3 years (Roels *et al.*, 1992). ATSDR applied US EPA's BMD software to derive a $BMDL_{10}$ POD of 142 μg/m^3 for abnormal eye-hand coordination in workers exposed to respirable Mn. ATSDR adjusted the 142 μg/m^3 POD to account for continuous exposure in the general population (*vs.* a worker population) (142 μg/m^3 × 5/7 days/week × 8/24 hours/day = 34 μg/m^3), and applied a UF of 10 for limitations/uncertainties and another UF of 10 for human variability, for a total UF of 100, resulting in an MRL of 0.3 μg/m^3.[5] Thus, the Mn MRL is 100-fold lower than the estimated continuous exposure concentration in the general population that would be expected to result in essentially no adverse effects.

Further, peer-reviewed studies suggest that Mn brain concentrations would not exceed normal levels in adults, children, neonates, or fetuses at Mn exposure concentrations as high as 10 μg/m^3 (Schroeter *et al.*, 2011, 2012; Yoon *et al.*, 2011), providing further support for the conservatism of the Mn MRL of 0.3 μg/m^3.

It is also important to consider that the Mn MRL is based on Mn concentrations with a mean particle aerodynamic diameter of ≤5 microns (μm) (PM_{05}) from the Roels *et al.* (1992) study. As discussed above, the Mn data for the S.H. Bell Chicago site are PM_{10} concentrations (*i.e.*, particle size ≤10 μm), which include the PM_{05} fraction and particles larger than 5 μm but less than or equal to 10 μm. Therefore, Mn

[5] It is noteworthy that the MRL is rounded down to one significant figure from 0.340 to 0.3 μg/m^3. Rounding the MRL to one significant figure provides support for rounding hazard indices to one significant figure. In addition, if one compares the unrounded numbers, all unrounded mean Mn PM_{10} concentrations for the S.H. Bell Chicago facility (0.297-0.318 μg/m^3) are below the unrounded Mn MRL of 0.34 μg/m^3.

PM_{10} concentrations likely overestimate Mn PM_{05} concentrations, and, therefore, comparison of Mn PM_{10} concentrations to the MRL likely overestimates the Mn risk.

3 Risk Calculations

Regulatory agencies typically present non-cancer risks as chronic hazard index (HI) estimates. HI estimates are calculated by dividing the exposure concentration by the chronic toxicity value. US EPA states that HI estimates should be rounded to and presented as one significant figure (US EPA, 1989). US EPA's target HI is 1, meaning that no adverse effects are expected in the population if the HI is equal to 1 or lower.

The Mn HI calculation is as follows:

$$HI = EC\ (\mu g/m^3) \div \text{Mn Inhalation Toxicity Value (MRL)}\ (\mu g/m^3)$$

where:

HI = Hazard Index
EC = Exposure Concentration
MRL = Minimum Risk Level

The Mn HI for the S.H. Bell Chicago air monitoring data (March-August 2017) is 0.9 (HI = 0.280 $\mu g/m^3$ ÷ 0.3 $\mu g/m^3$ = 0.9). The following table summarizes the Mn air concentrations, exposure concentrations, and HIs for the three exposure periods evaluated.

Table 3.1 Mn PM_{10} Air Concentrations, Exposure Concentrations, and Hazard Indices

Exposure Period	Mean Mn PM_{10} Air Concentration ($\mu g/m^3$)	Mn PM_{10} Exposure Concentration[1] ($\mu g/m^3$)	Hazard Index[2,3]
March-June 2017	0.318	0.305	1
March-July 2017	0.310	0.297	1
March-August 2017	0.292	0.280	0.9

Notes:
Mn = Manganese; PM_{10} = Particulate Matter ≤10 µm in Diameter; US EPA = US Environmental Protection Agency.
(1) Mn air concentrations adjusted for an exposure frequency of 350 days per year, per US EPA guidelines (US EPA, 1989).
(2) Note that had we calculated the HIs using the 95% upper confidence limit (UCL) on the mean, as opposed to the mean that is used on the US EPA in Illinois website, for the S.H. Bell Chicago Mn air monitoring data, the hazard indices (HIs) for all exposure periods would remain at 1.
(3) US EPA guidelines (1989) indicate that hazard indices should be reported to one significant figure. As stated in the guidelines (1989) in Exhibit 8-3, "All hazard indices and hazard quotients should be expressed as one significant figure."

Note that if we adjust for more realistic exposure assumptions regarding time spent indoors and away from home, the HIs would be even lower.

4 Preliminary Risk Evaluation Conclusion

The results of our conservative preliminary risk evaluation, conducted in a manner consistent with US EPA risk assessment guidelines, indicate that there is no risk of adverse neurological effects for the general population (including sensitive subpopulations) from continuous inhalation of Mn in ambient air (collected from March-August 2017) in the vicinity of the S.H. Bell Chicago facility (HI = 0.9). Hazard indices calculated from March-June and March-July 2017 data also do not exceed 1 and , therefore, indicate no adverse effects. This conclusion is based on comparison of the Mn ECs to the ATSDR chronic Mn MRL of 0.3 $\mu g/m^3$ that is well below the level of Mn in air estimated to cause no adverse effects following continuous exposure (34 $\mu g/m^3$), and well below the threshold Mn concentration that is not expected to increase normal levels of Mn in the brain (10 $\mu g/m^3$). In addition, the risk calculation is based on a high estimate of Mn exposure that assumes a resident inhales outdoor air at their home for 24 hours per day, for 350 days per year. As discussed above, it is likely that time spent indoors and away from home would effectively reduce the Mn exposures and risk estimates by about 50% in accordance with the US EPA exposure factor guidelines.

Given the conservative and health-protective basis of the Mn risk calculations in our evaluation, Gradient concludes, based on the available data, that there is no evidence that Mn in ambient air near the S.H. Bell Chicago facility will cause adverse health effects in the nearby community.

Note that although the average Mn PM_{10} air concentrations for the three exposure periods all round to 0.3 $\mu g/m^3$, the concentrations have decreased slightly over time from 0.318$\mu g/m^3$ (March-June 2017), to 0.310 $\mu g/m^3$ (March-July 2017), to 0.297 $\mu g/m^3$ (March-August 2017). Since the ATSDR MRL is derived for comparison to an exposure concentration averaged over one year or more, Mn PM_{10} data collection should continue at least through the end of February 2018 and the Mn risk should be re-evaluated at that point.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2012. "Toxicological Profile for Manganese (Final)." 556p., September.

Allen, R; Larson, T; Sheppard, L; Wallace, L; Sally Liu, LJ. 2003. "Use of real -time light scattering data to estimate the contribution of infiltrated and indoor generated particles to indoor air." *Environ. Sci. Technol.* 37:3484-3492.

Chen, P; Chakraborty, S; Mukhopadhyay, S; Lee, E; Paoliello, MM; Bowman, AB; Aschner, M. 2015. "Manganese homeostasis in the nervous system." *J. Neurochem.* 134(4):601-610. doi: 10.1111/jnc.13170.

Clark, NA; Allen, RW; Hystad, P; Wallace, L; Dell, SD; Foty, R; Dabek -Zlotorzynska, E; Evans, G; Wheeler, AJ. 2010. "Exploring variation and predictors of residential fine particulate matter infiltration." *Int. J. Environ. Res. Public Health* 7(8):3211-3224.

Horning, KJ; Caito, SW; Tipps, KG; Bowman , AB; Aschner, M. 2015. "Manganese is essential for neuronal health." *Annu. Rev. Nutr.* 35:71-108. doi: 10.1146/annurev-nutr-071714-034419.

Long, CM; Suh, HH; Catalano, PJ; Koutrakis, P. 2001. "Using time - and size-resolved particulate data to quantify indoor penetration and deposition behavior." *Environ. Sci. Technol.* 35(10):2089-2099. doi: 10.1021/es001477d.

Ozkaynak, H; Xue, X; Spengler, J; Wallace, L; Pellizzari , E; Jenkins, P. 1996. "Personal exposure to airborne particles and metals: Results from the particle team study in Riverside, California." *J. Expo. Anal. Environ. Epidemiol.* 6(1):57-78.

Roels, HA; Ghyselen, P; Buchet, JP; Ceulemans, E; Lauwerys, RR. 1992. "Assessment of the permissible exposure level to manganese in workers exposed to manganese dioxide dust." *Br. J. Ind. Med.* 49:25-34.

Sarnat, SE; Coull, BA; Ruiz, PA; Koutrakis, P; Suh, HH . 2006. "The influences of ambient particles composition and size on particle infiltration in Los Angeles, CA, residences." *J. Air Waste Manag. Assoc.* 56:186-196.

Schroeter, JD; Dorman, DC; Yoon, M; Nong, A; Taylor, MD; Andersen, ME; Clewell, H J. 2012. "Application of a multi -route physiologically-based pharmacokinetic model for manganese to evaluate dose-dependent neurological effects in monkeys." *Toxicol. Sci.* 129(2):432-446.

Schroeter, JD; Nong, A; Yoon, M; Taylor, MD; Dorman, DC; Andersen, ME; Clewell, HJ III. 2011. "Analysis of manganese tracer kinetics and target tissue dosimetry in monkeys and humans with multi - route physiologically based pharmacokinetic models." *Toxicol. Sci.* 120(2):481-498. doi: 10.1093/toxsci/kfq389.

US EPA. 1989. "Risk Assessment Guidance for Superfund (RAGS). Volume I: Human Health Evaluation Manual (Part A) (Interim final)." Office of Emergency and Remedial Response, NTIS PB90155581, EPA-540/1-89-002, 287p., December.

US EPA. 2005. "Integrated Risk Information System (IRIS): Additional Information." July 26. Accessed at https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=2776.

US EPA. 2011. "Exposure Factors Handbook: 2011 Edition." Office of Research and Developmen t, National Center for Environmental Assessment (NCEA), EPA/600/R-090/052F, 1,436p., September.

US EPA. 2017. "S.H. Bell Chicago Air Monitoring Data." Ac cessed at https://www.epa.gov/il/sh -bell-chicago-air-monitoring-data#locations.

Wallace, L; Williams, R. 2005. "Use of personal -indoor-outdoor sulfur concentrations to estimate the infiltration factor and outdoor exposure factor for individual homes and persons." *Environ. Sci. Technol.* 39:1707-1714.

Williams, R; Suggs, J; Rea, A; Leovic, K; Vette, A; Croghan, C; Sheldon, L; Rodes, C; Thornburg, J; Ejire A; Herbst, M; Sanders, W. 2003. "The Research Triangle Park particulate matter panel study: PM mass concentration relationships." *Atmos. Environ.* 37:5349-5363.

Yoon, M; Schroeter, JD; Nong, A; Taylor, MD; Dorman, DC; Andersen, ME; Clewell, HJ III. 2011. "Physiologically based pharmacokinetic modeling of fetal and neonatal manganese exposure in humans: Describing manganese homeostasis during development." *Toxicol. Sci.* 122(2):297-316. doi: 10.1093/ toxsci/kfr141.

S.H. Bell Company
Chicago S. Ave "O" Terminal

ATTACHMENT C

March 2, 2017

S1: µg/m 3
S2: µg/m 3
S3: µg/m 3
S4: µg/m 3

Mn: 0.397 µg/m 3

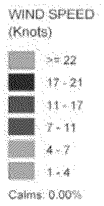
S.H. Bell Company
Chicago S. Ave "O" Terminal

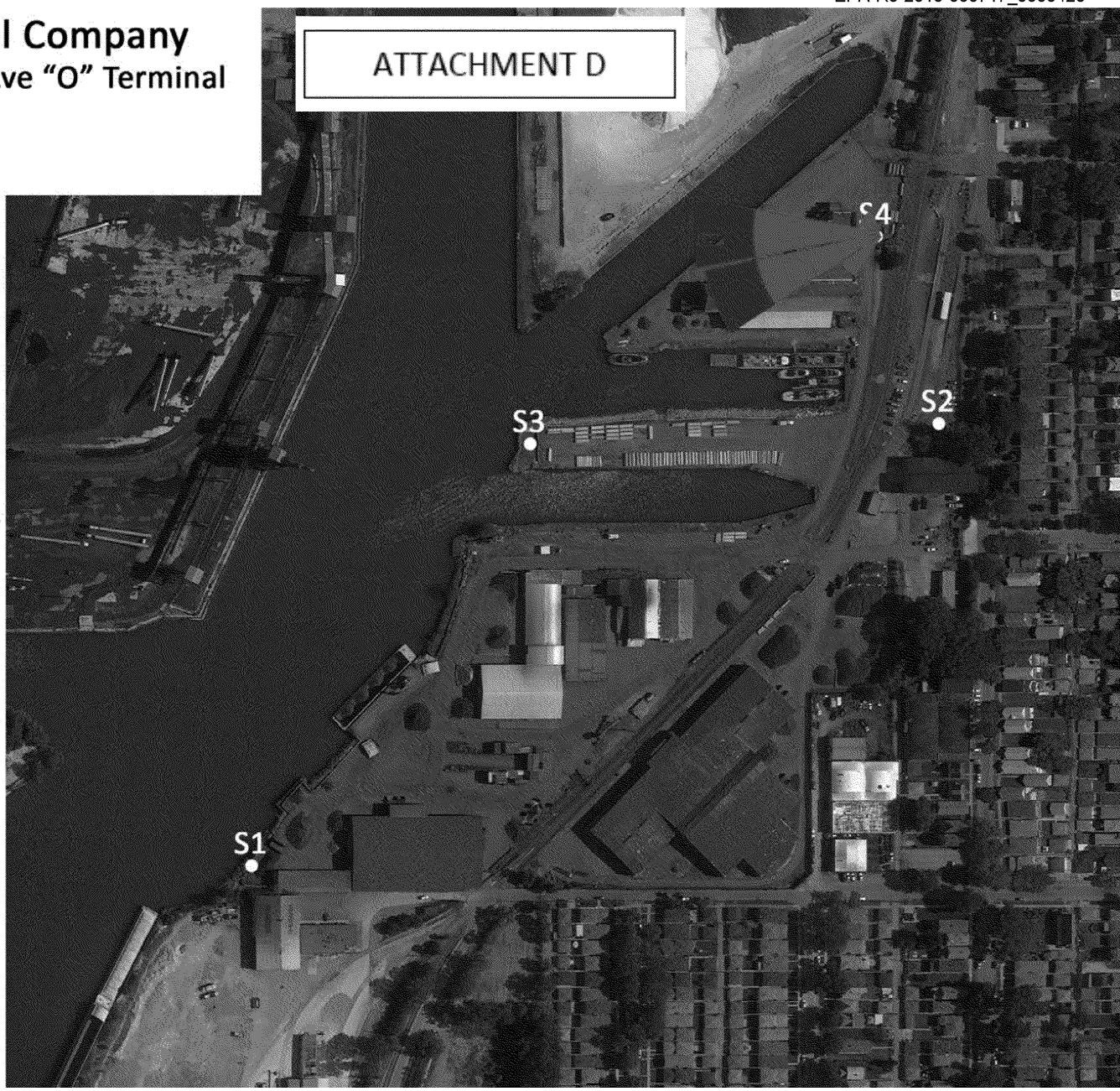
ATTACHMENT D

March 8, 2017

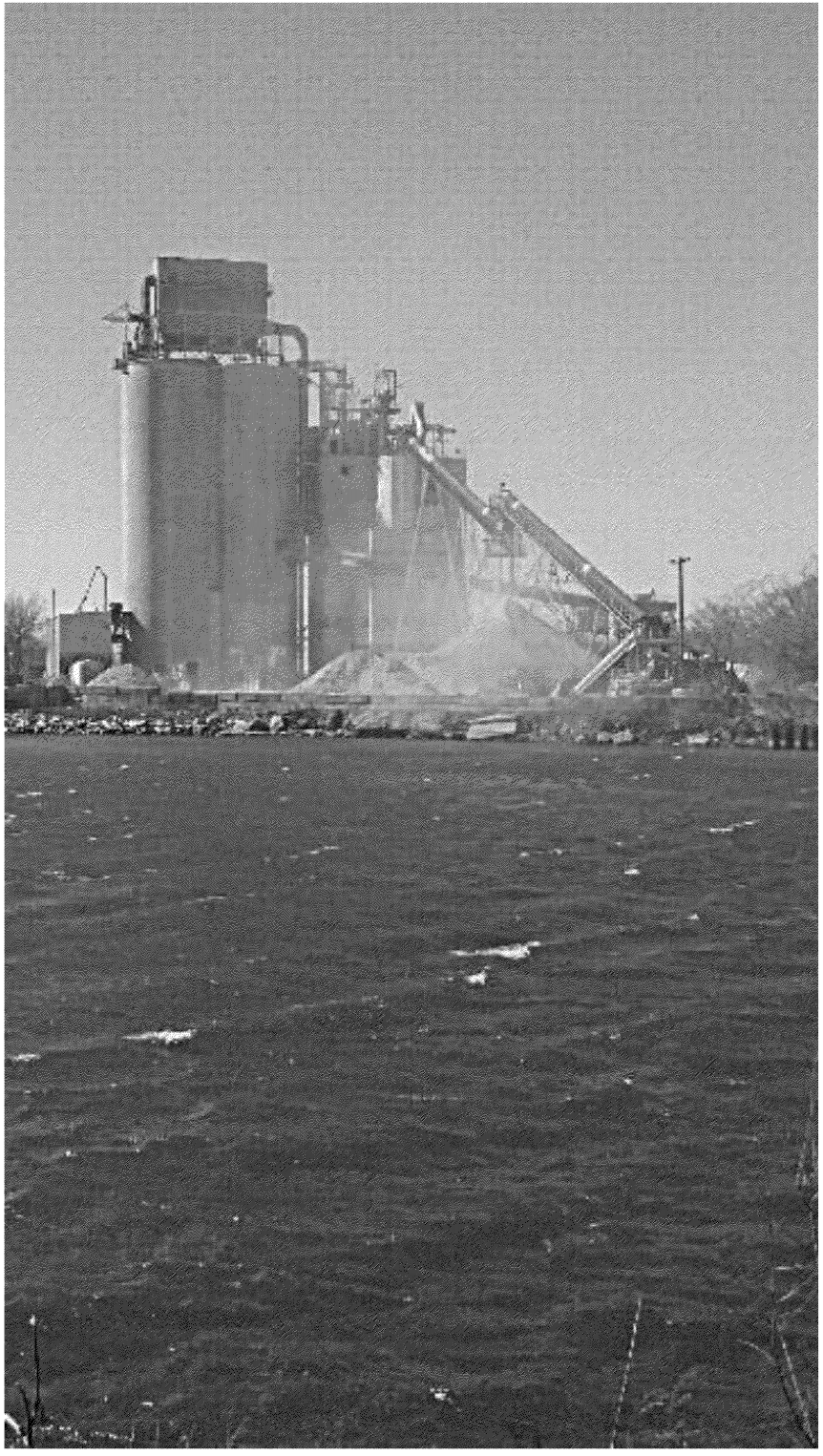
S1: µg/m 3
S2: µg/m 3
S3: µg/m 3
S4: µg/m 3

Mn: 0.765 µg/m 3





ATTACHMENT E



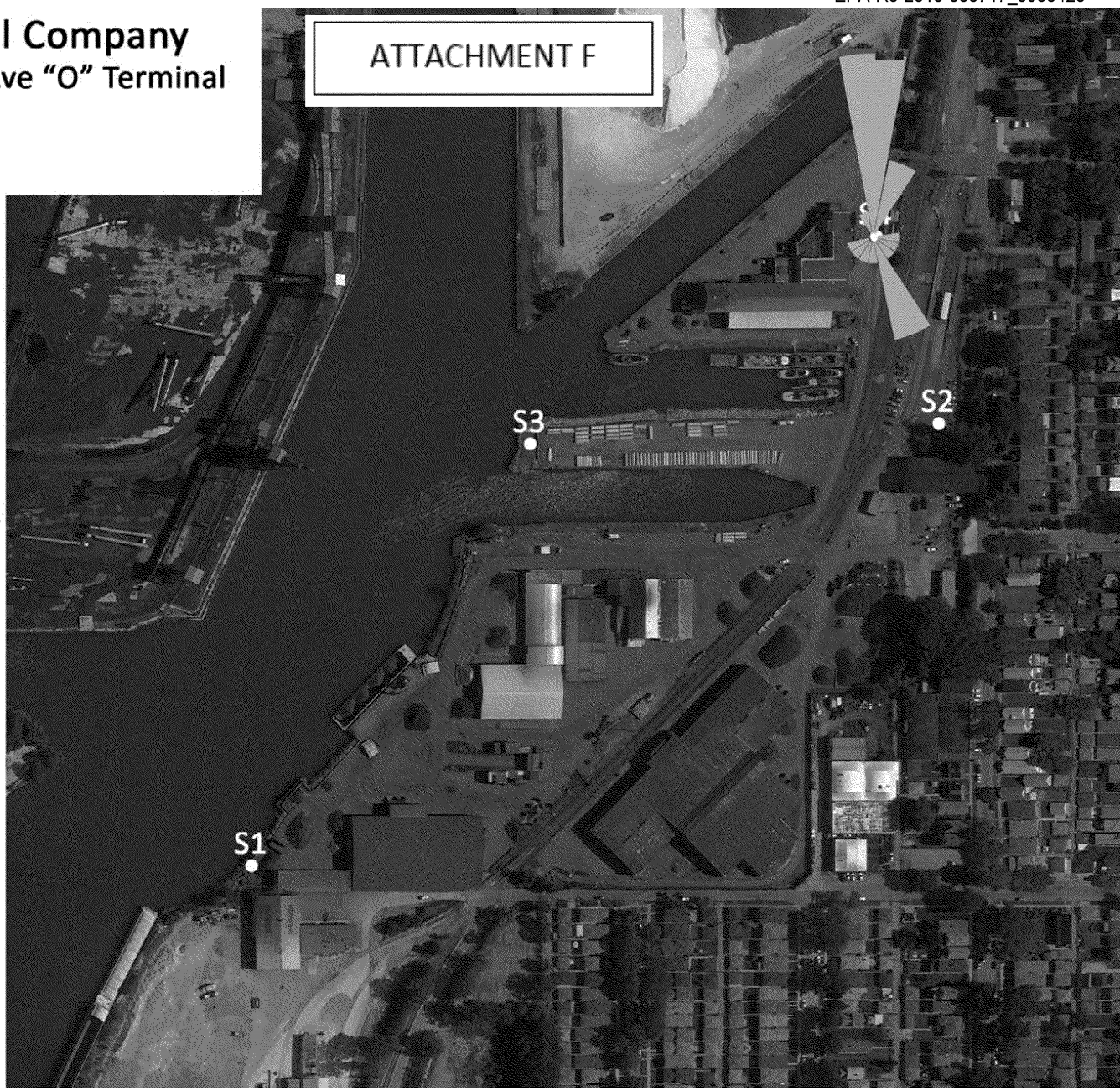
S.H. Bell Company
Chicago S. Ave "O" Terminal

ATTACHMENT F

March 20, 2017

S1: µg/m 3
S2: µg/m 3
S3: µg/m 3
S4: µg/m 3

Mn: 0.504 µg/m 3



ATTACHMENT G

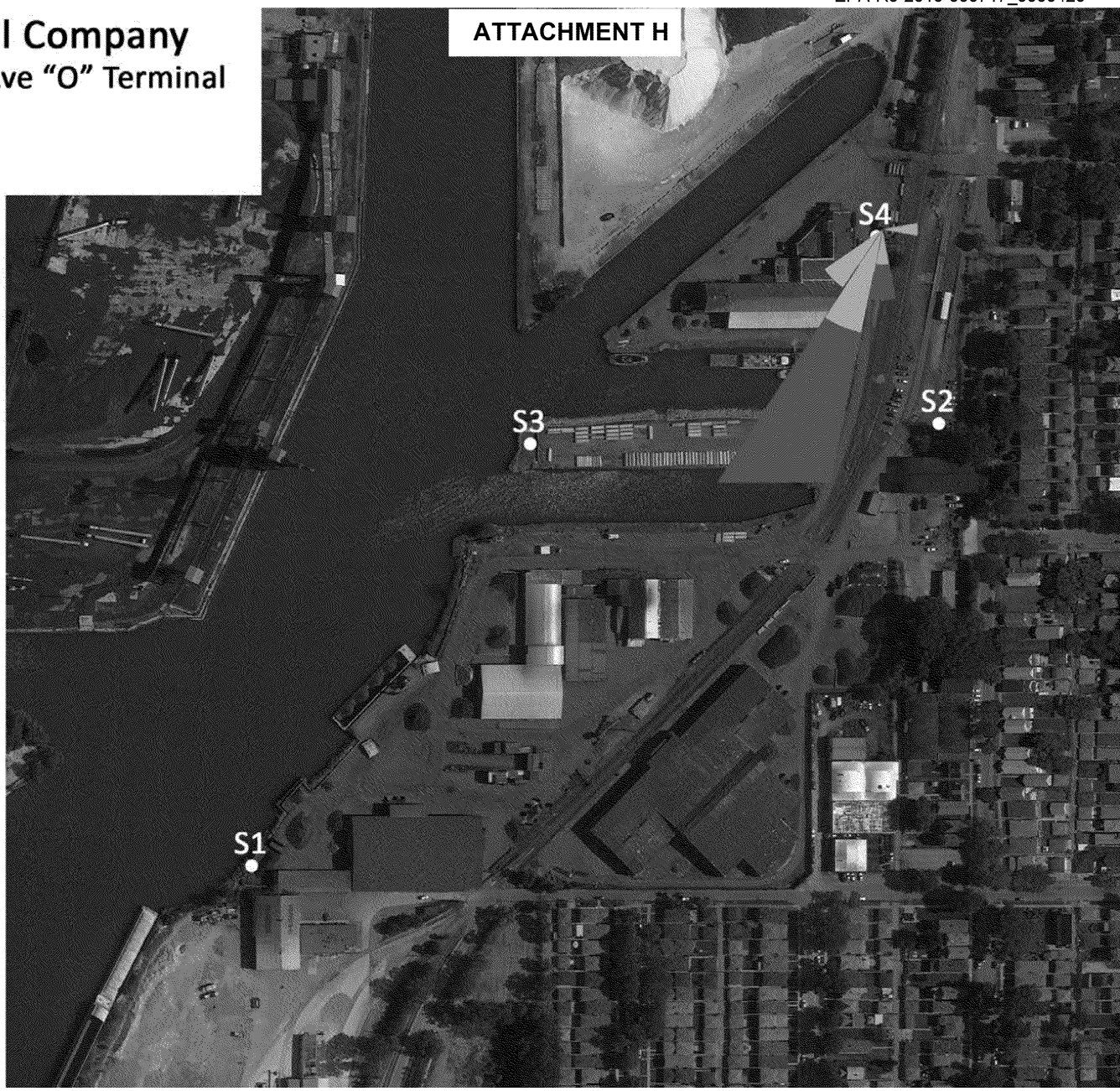
S.H. Bell Company
Chicago S. Ave "O" Terminal

ATTACHMENT H

May 16, 2017

S1: µg/m 3
S2: µg/m 3
S3: µg/m 3
S4: µg/m 3

Mn: 1.23 µg/m 3



WIND SPEED
(Knots)

- >= 22
- 17 - 21
- 11 - 17
- 7 - 11
- 4 - 7
- 1 - 4

Calms: 0.00%

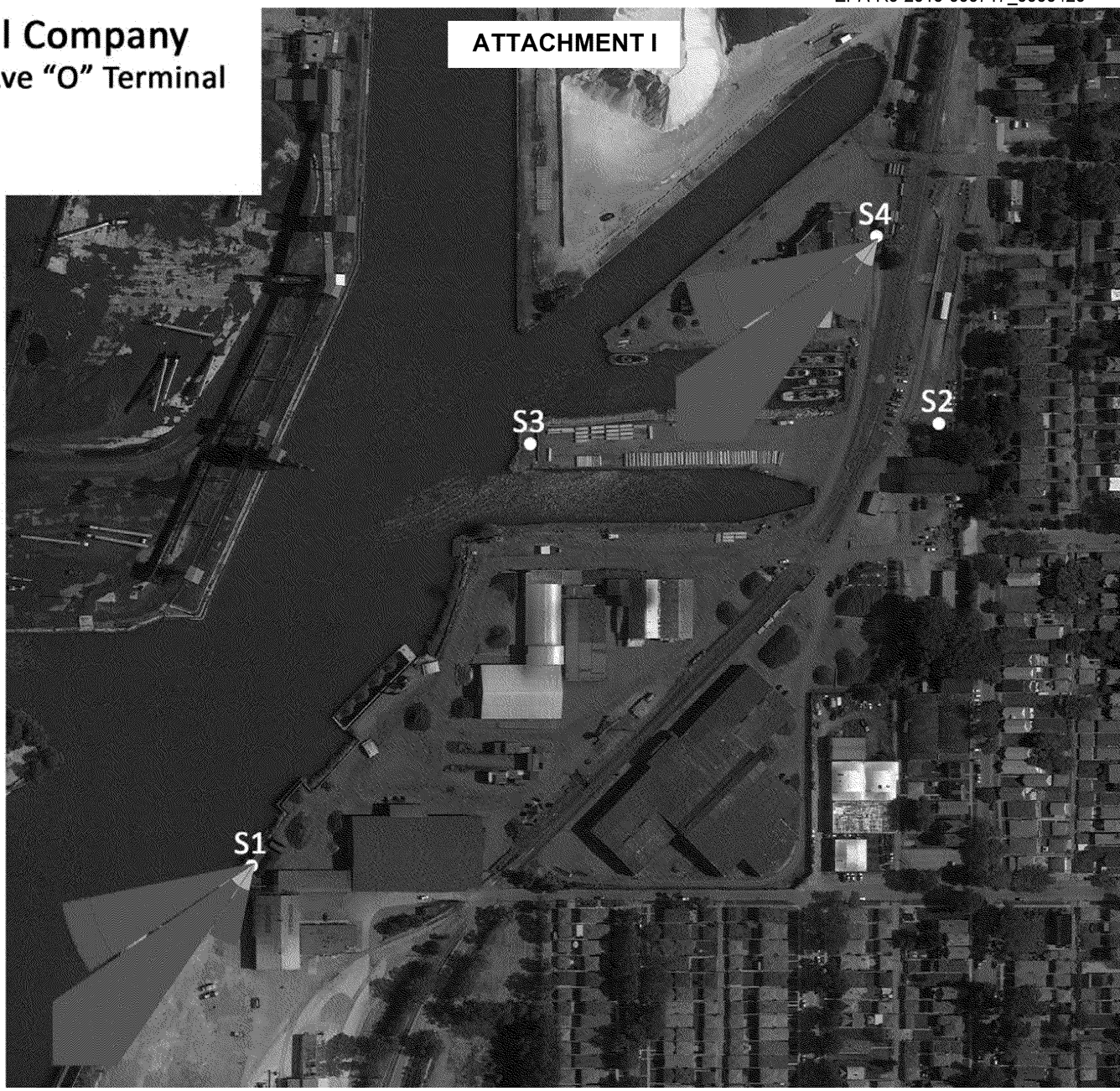
S.H. Bell Company
Chicago S. Ave "O" Terminal

ATTACHMENT I

May 22, 2017

S1: 47 µg/m 3
S2: 21 µg/m 3
S3: 24 µg/m 3
S4: 24 µg/m 3

Mn: 1.10 µg/m 3



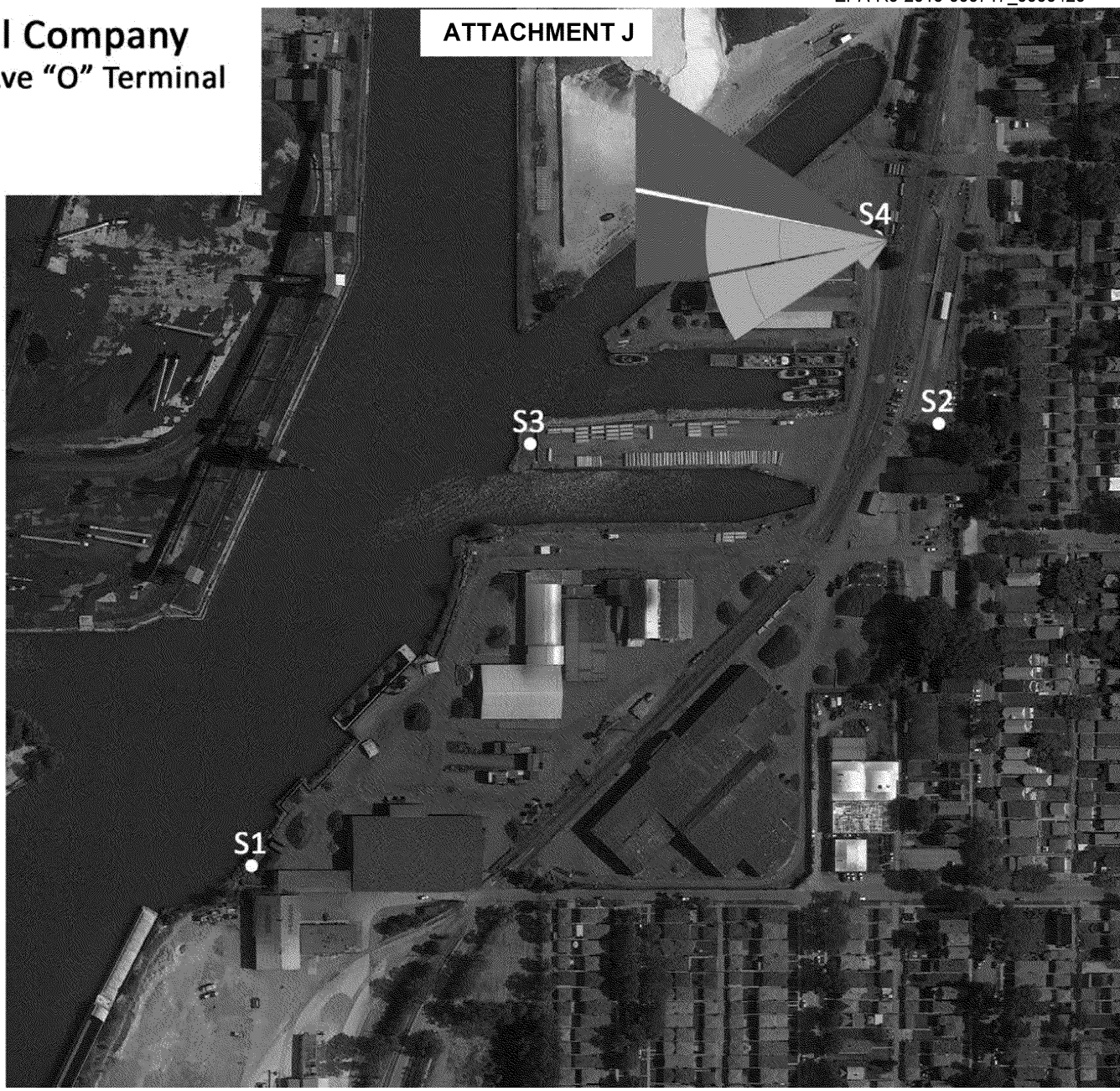
S.H. Bell Company
Chicago S. Ave "O" Terminal

ATTACHMENT J

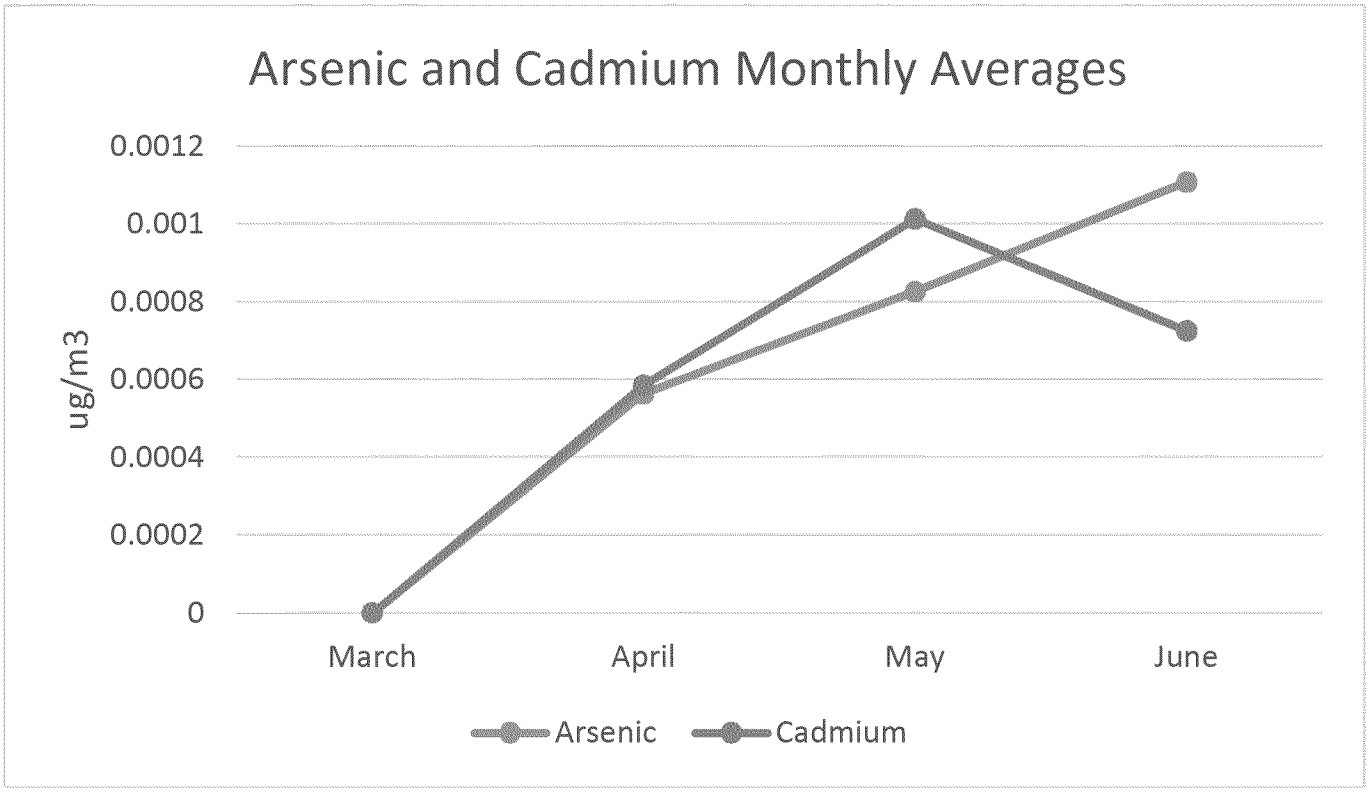
May 31, 2017

S1: 41 µg/m 3
S2: 34 µg/m 3
S3: 37 µg/m 3
S4: 39 µg/m 3

Mn: 0.45 µg/m 3

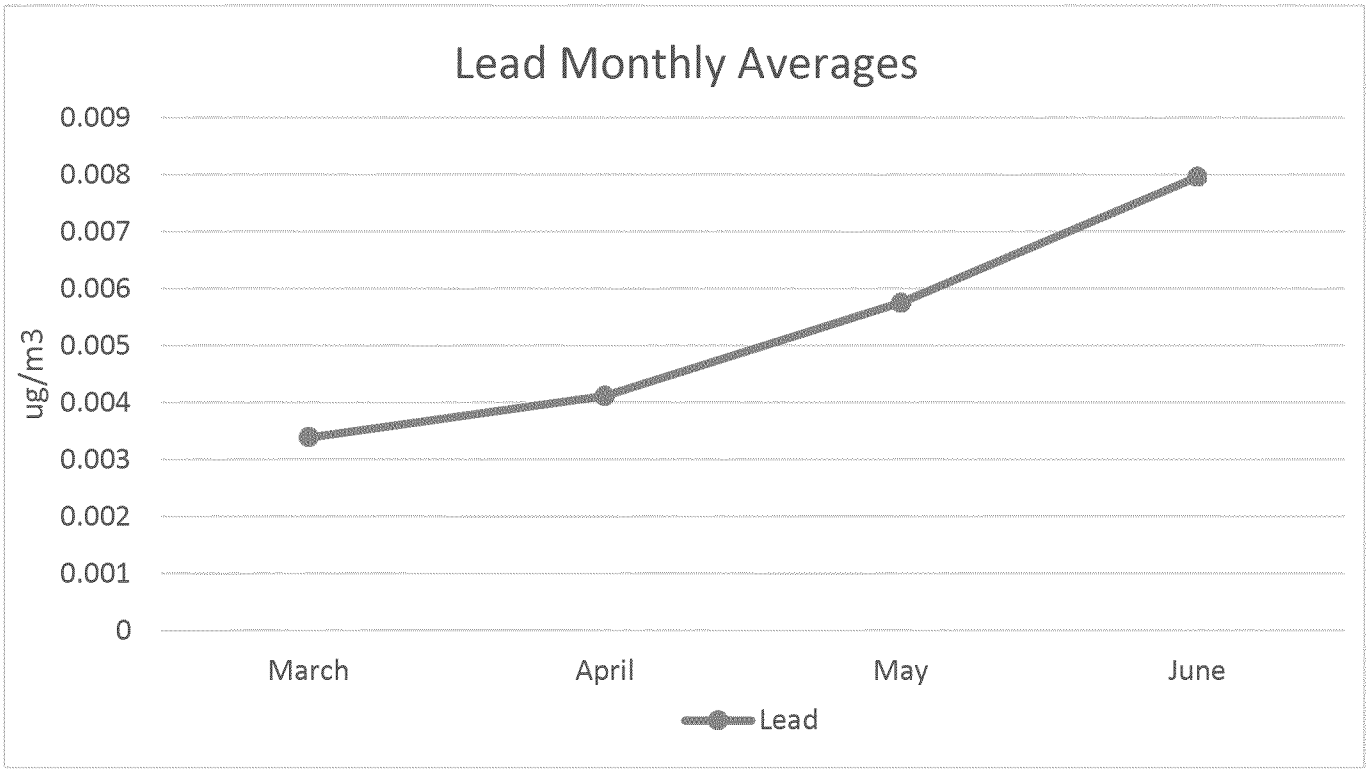


ATTACHMENT K



*The March average for cadmium and arsenic is non-detect based on the laboratory reports.

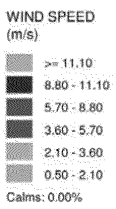
ATTACHMENT L



S.H. Bell Company
Chicago S. Ave "O" Terminal

July 24, 2017

Mn: 0.304 µg/m 3



S.H. Bell Company
Chicago S. Ave "O" Terminal

Aug 2, 2017

Mn: 0.382 µg/m 3

WIND SPEED
(m/s)

- >= 11.10
- 8.80 - 11.10
- 5.70 - 8.80
- 3.60 - 5.70
- 2.10 - 3.60
- 0.50 - 2.10

Calms: 0.00%