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Fond du Lac Environmental Program

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Richard:

I would like to submit to MPCA and the co-lead agencies for the PolyMet NorthMet Project some comments on a technical memorandum produced by Barr Engineering on behalf of their client on April 9, 2010. The subject of this technical memo was "Results from the Additional Baseline Monitoring for Sulfate and Methylmercury in the Embarrass River Watershed (July-November 2009)". This is an issue of prime concern to Fond du Lac and the other tribal cooperating agencies, as we have continually expressed over the course of the PolyMet EIS and SDEIS processes. On numerous occasions during IAP work group conference calls, cooperating agency coordination calls and tribal consultation meetings known as "sieve list" meetings, I have requested from the co-lead agencies any internal review comments of this technical memo from their mercury 'experts', but to date none have been provided.

Just prior to the March 29 sieve list meeting, we did receive a compilation of emails and documents related to this tech memo, including a string of emails beginning on February 24, 2010. The communications indicated that Barr was notifying the MPCA of the results of the additional baseline monitoring of sulfate, mercury and methylmercury in the area surrounding the NorthMet Project, and was requesting agency staff review from both the MPCA and MDNR. The email also noted that at the November 3, 2009 site meeting in Hoyt Lakes, which was also attended by tribal cooperating agency staff and included a field tour of the monitoring locations, the state agencies requested a meeting be held to discuss the draft findings. Tribal staff echoed that request, and asked to be allowed to participate, but there is no record of such a meeting having occurred. A second Barr email to MPCA provided to tribal staff, dated April 9, 2010, indicated that the technical memorandum in question was now posted to the PolyMet Project Management Website, and it was at this time that tribal staff were first made aware of its availability. The time lapse between the submission of the monitoring results on February 24, and the distribution of the technical memo on April 9, suggests that there was, at least, an opportunity for lead agency review.

Fond du Lac's overriding concern about this technical memo is its ultimate use and applicability to the ongoing analysis and predictions of potential environmental impacts of the proposed PolyMet NorthMet Project during the SDEIS process. I have reviewed the document, and requested outside review comments from two well-credentialed scientists I have worked with: John Sorensen of the UMD Physics Lab, and Sara Moses, environmental toxicologist with the Great Lakes Indian Fish and Wildlife Commission (GLIFWC). Following are some questions and concerns with the technical memo and Barr's interpretation of their results from this study.

1. While I understand the focus of this study was to better understand potential enhanced mercury methylation from high sulfate seepage from the LTV tailings basin, it is not clear why the study only sought to capture methyl mercury export during storm events. Many studies have demonstrated that a significant portion of the mercury transported from a watershed occurs during spring runoff, often in excess of 50% of the annual loading (e.g., http://www.agu.org/pubs/crossref/2011/2010JG001330.shtml. US Forest Service studies in this region have shown that approximately 25% of the total and meHg loading to a stream is a result of snowmelt and spring rains. Earlier sampling would have been desirable.
2. It is ultimately more important to understand meHg fluxes per unit area, and even more specifically, per unit area of wetlands within the watershed, than simply concentrations. I'm not certain why there would be so much uncertainty in delineating wetlands within the watersheds, given the field validation work that has been done since the November 2009 site visit. Barr's pairwise comparisons for meHg in stream sites with elevated and background sulfate concentrations do not account for differences in watershed size or % wetlands in watersheds, both factors that are typically positively correlated with mercury and meHg in water.
3. If there are more hydrologically connected wetlands within a watershed, you would expect more meHg. The appendix provides information about the percent wetlands (control watersheds had 45% and 23% wetlands, high sulfate watersheds had 47% and 15%), but there is no information about hydrologic connectivity of the wetlands to the streams. DOC can be a good indicator of this, and DOC measured in the two high sulfate streams was 12mg/l and 14 mg/l respectively, whereas DOC in the control streams was 19mg/l and 22 mg/l. To some degree, this predisposes an interpretation of no differences between control and high sulfate streams (detection of any sulfate effect is potentially confounded by the differences in DOC).
4. Mercury studies by the USGS (George Aitken, presentation to the National Water Quality Monitoring Conference, Denver 2010) show that meHg and DOC concentrations are highly correlated in stream measurements, and track closely with the stream hydrograph. In fact, the DOC peak precedes the stream hydrograph peak during storm events. Barr sampled meHg on the falling limb of the hydrograph, potentially missing the main 'flush' of meHg.
5. What is the rationale for using only filtered samples for the stream meHg monitoring? It would have been useful to have at least some filtered/unfiltered samples at various sites for comparison, to ensure that they were not missing a potentially large mass of meHg flowing through the system.
6. It appears that Barr used data from PM 21, a mid-basin sampling station in the deepest part of Sabin Lake, as representing the concentration of meHg in water flowing out of Sabin Lake, rather than establishing a station at the outflow. If the average meHg concentration drops from 0.31 ng/l to 0.23 ng/l in the distance between PM 23 (inlet) and PM 21, then why would we not assume that it would drop even more by the time it reaches the outlet? Likewise, PM 22, the mid-basin sampling station for Wynne Lake was used to represent what is leaving Wynne Lake, rather than an actual outflow station.
7. J. Sorensen identified several apparent errors in calculations. Regarding the input to Sabin Lake, on page 31 of the technical memo it is stated that "MeHg concentration in the inlet to Sabin Lake (PM 23) is fairly stable; average = 0.31 $\pm$ 0.19 (s.d.) ng/l. The Embarrass River flow at PM 23 is more variable (average = 55 $\pm$ 36 (s.d.) cubic feet/second (cfs), and when coupled with the average concentration, the average mass input to Sabin Lake is approximately 1.5 $\pm$ 4.0 (s.d.) grams per month (g/mo – average flow X average mass). When J. Sorensen recalculated the mass input to Sabin Lake, his result was 1.25 g/mo, and his sd estimation using propagation of errors resulted in 1.6. Also, the deposition

calculations for Sabin Lake may be inaccurate. Table 6 states that Sabin Lake loses 0.74 g/mo, but it is not clear how Barr converted that to g/yr – perhaps by multiplying by 12? But depending upon the depth of ice cover, it may preclude significant flow for several months each year. Sorensen calculated an annual yield of 5.9 g/yr based upon an assumption of eight months of flow. The surface area of the lake is about 1,200,000 m2, which yields about 4.9 ug/m2/yr. Barr calculated 0.0073 ug/m2/yr, and noted that was substantially less than what Brezonik observed (0.32 ug/m2/yr), but according to Sorensen's calculation it is substantially greater. It might be important to check all calculations to ensure accuracy.

8. On page 32, it is stated "It is currently assumed that the difference between the inputs and outputs presented in Table 6 are accounted for mostly by burial of meHg to sediments". Why is there no discussion or consideration of biologic uptake? Sabin Lake is recognized has having relatively higher primary productivity than Wynne Lake, which could impact meHg bioaccumulation rates in the two lakes (the "dilution effect" of more algal biomass taking up meHg than can be consumed by secondary producers and higher trophic levels). This is a critical missing element for being able to describe the fate of meHg produced in this stream/lake system, and to be able to make defensible conclusions about differences in meHg in biotic endpoints from this particular study. Sediment cores from the two lakes could reveal some of this important information about Hg, meHg, and the various sulfur species.
9. There are three steps that affect meHg levels in biota, the endpoint Fond du Lac is most specifically concerned about: 1) mercury methylation; 2) its initial incorporation at the base of the food web, and 3) its subsequent trophic transfer and bioaccumulation. The Barr study only examined the first step in this process; even more narrowly, only at the relationship between this process and sulfate levels in the water. Barr concludes: "In summary, while the tailings basin may be contributing sulfate to what is considered by the MPCA and MDNR to be a high risk environment (i.e., wetlands to the north/northwest of the basin that contribute their water to the Embarrass River and the downstream chain of lakes), the 2009 additional baseline monitoring data does not indicate that the elevated sulfate concentrations are resulting in elevated meHg or "% that is meHg" compared to background conditions. Based on these finding, and noting that the tailings basin has been a watershed feature for some 40+ years, it is unlikely that continued operation of the tailings basin by PolyMet will have an effect on the sulfate and meHg dynamics in the Embarrass River watershed".

   Yet, the fact is that fish in Sabin and Wynne Lakes have stricter mercury based consumption advice than the general advice for the state of Minnesota. There is an obvious disconnect between the report findings and the mercury levels in fish. S. Moses described three possible explanations for this:

   1) The conclusions of the Barr report are incorrect, and the sulfate from the tailings basin is in fact resulting in higher fish mercury levels in the lakes by increasing mercury methylation.
   2) The increased meHg in the fish is due to increased mercury methylation, but this methylation is due to factors other than elevated sulfate inputs from the tailings basin.
   3) The increased meHg in fish is due to processes occurring subsequent to mercury methylation (i.e., the processes of methylmercury incorporation at the base of the food web and/or its subsequent movement and accumulation through the food web.

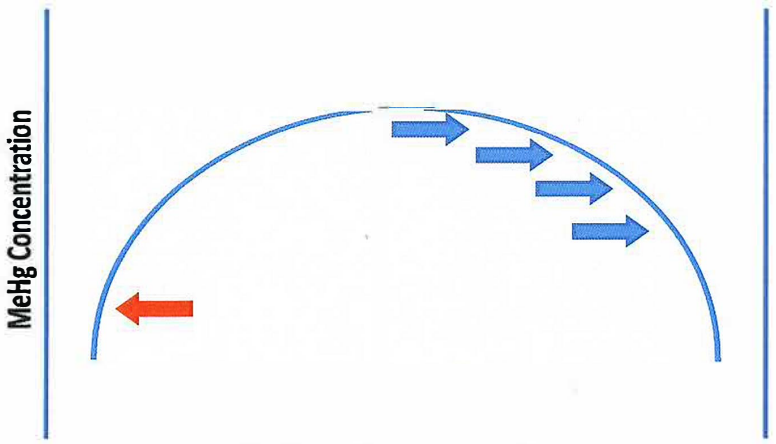
   She discussed each of these scenarios in greater detail, below:

Option 1: Barr's conclusions were incorrect

There are some issues that could have weakened the report conclusions. First, the sample size is very small. Additionally, as explained on page 12, the relative percent difference between duplicate samples was considered high (>30%). The combination of small sample size and high analytical variability would make any true difference between sites very difficult to detect. This is further illustrated by the power values reported in Table 2 (pages 18-19). In footnotes, Barr states these values represent "the percent of times a true difference of one standard deviation would be identified given significance levels of 0.05 and 0.1 for the 95% and 90% CI respectively." These values are as low as 18%. So the fact that the analysis did not find significant differences in meHg levels or % meHg does not provide much confidence that a difference may not actually exist.

Barr points out another issue: they measured sites along the river system at the same time, yet the water takes time to move downstream through the system. So, ideally, to link the measurements upstream with those downstream, the measurements should have been staggered in time.

On page 29, Barr discusses an inverse relationship in the lakes between sulfate and meHg concentration. They conclude that this indicates that elevated sulfate does not result in elevated meHg concentration. But the relationship between sulfate and meHg concentration is non-linear, with sulfate concentrations above a certain level actually inhibiting meHg production.



It is possible that, since they are working in a system with relatively high sulfate levels, they were sampling along the part of the curve where the blue arrows are placed. You would in fact see an inverse relationship between sulfate and meHg within this portion of the curve. But this does not in fact prove that if sulfate were very low, such as where the red arrow is placed, the meHg concentrations wouldn't be lower than the current levels.

In this same section, it is not clear why Barr only compared sulfate with meHg concentrations and not also with % meHg, as they did in the rest of the sections.

Another issue of note is that Barr compares meHg and sulfate within the lakes after they have already shown that the meHg is not likely being methylated in the lakes (lack of

vertical meHg stratification), but entering the lakes already methylated from the river. The methylation is likely taking place in the wetlands upstream, then transported downstream into the lakes. What they really needed to show is that increased sulfate upstream does not correlate with the meHg levels in the lakes.

In their transect analysis (pg 35-36), Barr seems to play down some of their findings. They see an overall decrease in %MeHg from upstream of the tailings basin to the most downstream sites. This makes sense, since it appears that there are more wetlands in the upstream portion of the river. You would expect a lot of methylation to be occurring there naturally, with a decrease as you move downstream. But, they do not see a simple linear trend. They have some overall evidence that %MeHg peaks just downstream of the tailings basin. This evidence is actually fairly strong on certain sampling dates (e.g. Fig. 19, 8/20 and 9/16). So, it seems plausible that there is methylation occurring upstream in the low sulfate wetlands and then additional methylation occurring when the sulfate input occurs. I would need to see a better integration of the MeHg, %MeHg, % wetlands, and sulfate information to really form an opinion about what might be occurring here. But I do think they play down the fact that it is not a simple linear decrease in %MeHg across the system (upstream to downstream) and that the peak is actually partway downstream, immediately after the input from the tailings basin.

Also, considering the high % wetlands throughout this system, it is hard to establish a background in this system. It seems that methylation by wetlands has the potential to overshadow the in-stream effects the sulfate is having, especially considering the small sample size.

<u>Option 2: Increased mercury methylation due to factors other than elevated sulfate levels</u>
This was difficult to assess from the report. There are several things that can affect mercury methylation rate and extent: sulfate concentration, inorganic mercury concentration, temperature, pH, DOC, etc. It seems these were measured previously at the sites, but not tied into the analysis in the current report. The way the analysis was done was simply to compare MeHg concentrations or %MeHg between sites with high versus low sulfate levels. I can envision a scenario where sulfate levels were actually increasing mercury methylation, but that it was being masked by a change in some other water quality characteristic that was having the opposite effect. It seems that a better way to analyze this data would have been to create a model that considers all of these criteria together and determine which were affecting MeHg and % MeHg.

<u>Option 3: Increased fish tissue mercury concentrations related to factors other than mercury methylation</u>
There has been no sampling of the biota, other than existing game fish tissue data from the state, to provide information about meHg uptake and bioaccumulation. I presume that Barr is proceeding under the assumption that if there is not additional methylmercury in the water as a result of sulfate loadings from the tailings basin, then there should not be higher mercury in the biota. I do not believe that this is necessarily true. Mercury levels in fish could be higher due to greater bioavailability of mercury at the base of the foodweb or changes in foodweb structure (prey species distribution, trophic level). Can sulfate affect these sorts of processes, independently of its effect on mercury methylation? There is evidence from EPA's comprehensive study on the effects of mountaintop removal coal mining in the Appalachian region that demonstrates impacts of high salinity discharge

(including sulfate) on biological condition and aquatic toxicity, measured through benthic macroinvertebrate and fish community data.
http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=233809
http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=225743

In terms of changes in methylmercury bioavailability at the base of the food web, I am not sure how the presence of sulfates might have an effect; pH and DOC can affect this process: http://www.globalmercuryproject.org/database/Upload/Global%201998%20Watras%20Review%20bioaccum%20Hg%20in%20pelagic.pdf.

See also:
http://pubs.acs.org/doi/abs/10.1021/es8027567

10. Thus far, the results of this study have been referenced in several work products associated with the SDEIS: the Cumulative Mercury Deposition Analysis Work Plan, several versions of the PolyMet Cumulative Mercury Deposition Report, and in the human health risk analysis. We have not yet seen any of the water quality or aquatic resources draft chapters to know how EMR plans to reference the results and interpretation from this study.

I look forward to further discussions about these issues, and greater resolution before the release of the draft SDEIS.

Sincerely,
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