**Bill Pfanner - Eastshore Energy Center- acrolein cancer potential information**



**From:** Michael Toth
**To:**
**Date:** 10/10/2007 7:23 PM
**Subject:** Eastshore Energy Center- acrolein cancer potential information

Dear Bill,

I wish to alert the CEC and the Eastshore project staff to a scientific study from NYU published in a leading journal "Proceedings of the National Academy of Sciences". The study discusses the carcinogenicity of acrolein. This additional information regarding the carcinogenicity of acrolein elevates this toxin to a level of concern where it may be compared to second-hand tobacco smoke.

The CEC and the BAAQMD, upon which the CEC relies for performing emissions analysis, state that acrolein reporting has been suspended by CARB pending uncertainty regarding relevant levels of significance, ambient concentrations, and measurement methodology.

Since acrolein is the driver of the acute health risk for natural gas burning plants according to data published by CARB and the US EPA, the suspension of the reporting and analysis of acrolein emissions and their impact on public health by CARB in late 2006 would appear to preclude the the ability of the CEC and the BAAQMD to complete a health risk analysis for the Eastshore project as required under CEQA.

The suspension of reporting by CARB due to uncertainties necessitates a comprehensive, detailed analysis of the health impact of acrolein for each project under consideration, until general guidelines can be reinstated. The CEC and the BAAQMD appear to be doing the opposite and have been considering acrolein emissions as having zero (0) health impact by virtue of its omission. The deliberate omission of acrolein from the health risk analysis may result in avoidable harm to individuals and appears to be a violation of the spirit of CEQA.

Due to the high number of existing acrolein sources in the area such as highways and air traffic, the local area may already bear a significant and unfair burden of this toxin. Therefore, as a condition of certification, please require a thorough analysis of the health impact of acrolein on the local area surrounding this plant. This analysis should, at a minimum, include the following components to protect public health:

* Report the baseline acrolein emissions inventory
* Measure the maximum and average local concentrations over a period of several weeks from one or more neutral locations not in immediate proximity to emissions sources (ie. a large park or field)- the cumulative point of maximum acute impact for Russell City and Eastshore as published in your PSA appears to be suitable.
* Report the cumulative impact of both plants
* Use appropriate statistical confidence intervals for a conservative determination of risk to human health when applying emissions factors- ie. in the range of 95%.
* Use measurement techniques that are more conservative and source appropriate (ie. use FTIR as opposed to CARB-430 measurement method for Eastshore engines).
* Use only emissions factors derived from a statistically suitable population, and require direct measurement.

* Take into account factors of asthma, cancer risk, and irritation, especially for sensitive receptors, to the degree supported by available scientific studies.
* Publish all source data, calculations and measurements so they can be independently verified
* **Ensure that the local acrolein concentrations, taking into account the entire local inventory, will not exceed the acceptable daily intake for sensitive receptors or result in detectable odor or physical discomfort at any time.**

In addition to this analysis, as a condition of certification, please provide mechanisms to monitor the effects of acrolein emissions on the local population. This should include:

* Tracking hospitalizations for asthma related issues
* Providing an independently run hotline to report symptoms of acrolein exposure such as throat and eye irritation. Publish this information on-line on a daily or weekly basis. For obvious reasons, this hotline should not be managed by the plant operators.
* Monitoring local acrolein level in real time so that the population may avoid hazards or that hazards may be correlated with reported symptoms.

Please forward this information to Dr. Greenberg. The following is a reference to the PubMed citation for the study I've referred to.

Thanks,
Mike Toth

http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&list_uids=17030796&dopt=AbstractPlus&holding=f1000%2Cf1000m%2Cisrctn

1: Proc Natl Acad Sci U S A. 2006 Oct 17;103(42):15404-9. Epub 2006 Oct 9.

Comment in:
Proc Natl Acad Sci U S A. 2006 Oct 24;103(43):15725-6.

Acrolein is a major cigarette-related lung cancer agent: Preferential binding at p53 mutational hotspots and inhibition of DNA repair.

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The tumor suppressor gene p53 is frequently mutated in cigarette smoke (CS)-related lung cancer. The p53 binding pattern of carcinogenic polycyclic aromatic hydrocarbons (PAHs) found in CS coincides with the p53 mutational pattern found in lung cancer, and PAHs have thus been considered to be major culprits for lung cancer. However, compared with other carcinogenic compounds, such as aldehydes, the amount of PAHs in CS is minute. Acrolein (Acr) is abundant in CS, and it can directly adduct DNA. Acr-DNA adducts, similar to PAH-DNA adducts, induce predominantly G-to-T transversions in human cells. These findings raise the question of whether Acr-DNA adducts are

responsible for p53 mutations in CS-related lung cancer. To determine the role of Acr-DNA adducts in p53 mutagenesis in CS-related lung cancer we mapped the distribution of Acr-DNA adducts at the sequence level in the p53 gene of lung cells using the UvrABC incision method in combination with ligation-mediated PCR. We found that the Acr-DNA binding pattern is similar to the p53 mutational pattern in human lung cancer. Acr preferentially binds at CpG sites, and this enhancement of binding is due to cytosine methylation at these sequences. Furthermore, we found that Acr can greatly reduce the DNA repair capacity for damage induced by benzo[a]pyrene diol epoxide. Together these results suggest that Acr is a major etiological agent for CS-related lung cancer and that it contributes to lung carcinogenesis through two detrimental effects: DNA damage and inhibition of DNA repair.

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