Chapter III. EPA Correctly Determines That A Section 112(d)(4) Health-Based Standard Is Not Appropriate Or Lawful For EGU Acid Gases

EPA proposes to set what it calls a "conventional MACT standard" for hydrogen chloride ("HCl") and for HCl as a surrogate for all other acid gas HAPs [hydrogen fluoride ("HF"), hydrogen cyanide ("HCN"), chlorine ("Cl_2") and selenium dioxide ("SeO_2")] emitted by all new and existing units in all the solid fuel-fired EGU subcategories.[1] 76 Fed. Reg. 25,049-25,051; 76 Fed. Reg. 25,124-25,128 (Tables 1 and 2 to Subpart UUUUU of Part 63). For its liquid oil subcategory, EPA proposes "conventional MACT standards" for HCl and for HF. 76 Fed. Reg. 25,124-25,128 (Tables 1 and 2 to Subpart UUUUU of Part 63). While we do not concede that the Agency's surrogacy justification is adequate, *see infra* Chapter VII, we agree that the Agency must set MACT floors for the acid gases emitted by solid- and liquid-fueled coal- and oil-fired EGUs. The Agency correctly concludes, 76 Fed. Reg. at 25,049-25,050, that it does not have sufficient information to set a section 112(d)(4) "health-based" standard. Moreover, the statutory prerequisites for setting such a standard for the acid gases are simply not met, as we will show below. For, while EPA continues to describe its authority to issue section 112(d)(4) standards as "discretionary" in the broadest terms, in fact the statute narrowly cabins EPA's ability to invoke section 112(d)(4) to those situations in which an appropriate health threshold has already been established at the time of the MACT standard setting process. That is not the case here.

A. The Statute Does Not Permit EPA to Establish Standards under Section 112(d)(4) for any HAP for which there is no Existing Health Threshold Based on No Observable Adverse Effects

CAA section 112(d)(4) states that "[w]ith respect to pollutants *for* which a health threshold *has been established*, the Administrator may consider such

[1] In the alternative, EPA proposes limits on SO2 as a surrogate for the HCl "conventional MACT standard" but only for units equipped with flue gas desulfurization equipment. 76 Fed. Reg. 25,124-25,128 (Tables 1 and 2 to Subpart UUUUU of Part 63). This surrogacy approach is not adequately justified as discussed *infra* Chapter VII.

threshold level, with an ample margin of safety, when establishing emission standards under this subsection." 42 U.S.C. § 7412(d)(4) (emphasis added). Congress's use of the phrase "has been established," is evidence that EPA only may rely on this authority where an accepted health threshold already is in existence at the time of the MACT standard setting process. See Brief for Respondent Environmental Protection Agency in *National Lime Ass'n v. EPA,* (D.C. Cir. No. 99-1325, July 14, 2000). Furthermore, the legislative history for section 112(d)(4) clearly shows that an acceptable health threshold, at a minimum must be based on the "no observable effects level" for any health endpoint associated with the hazardous air pollutant for which the section 112(d)(4) "health based standard" is considered. *See* S. Rep. No. 101-228 at 171 (1990). Congress did not intend for EPA to spend time and resources during the standard setting exercise to evaluate or set health thresholds for HAPs, but rather to rely on previously established health thresholds.

Prior to 1990, the CAA required individual HAP listing and standard setting, based on public health protection with "an ample margin of safety." CAA of 1970 §112(b)(1)(B), P. Law 91-604 (amended in 1990). The 1990 revisions to the air toxics section of the Act were comprehensive, including a Congressionally-mandated list of HAPs to be regulated, and very specific statutory directives to EPA requiring the Agency to set technology-based standards for those HAPs for listed industries. Section 112(d)(4) also was included in the 1990 revisions. Section 112(d)(4), as finalized, authorized the Agency to "consider" an "establish*ed*" health threshold[2] in setting such standards. By contrast, an earlier bill, which was not adopted, would have made the authority to set a health-based standard contingent on a finding that a threshold "*can be* established"[3] – a forward looking construct that would accommodate investigation and establishment of the threshold as part of the MACT standard setting exercise, in a way that the final enacted language of section

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[2] In fact, the Senate Report describes the prerequisite as a "well-established" health threshold. See S. Rep. 101-228 at 171.

[3] 3 1990 Legislative History at 4425.

112(d)(4) clearly does not. In addition, the legislative history makes clear that any established health threshold that might form the basis for a section 112(d)(4) health-based alternative standard must be based on the "'no observable [adverse] effects level' ("NOAEL") below which human exposure is presumably 'safe'." S. Rep. No. 101-228 at 171 (1990). As will be shown below, there is no such established health threshold currently for HCl, or for any of the other acid gases (non-metal inorganic HAPs) EPA identifies as emitted by coal- or oil-fired EGUs.

As a threshold matter, then, section 112(d)(4) authority to set an alternative, health-based standard for HCl is simply not available to the Agency here, because as shown below, there is no currently established NOAEL-based health threshold for HCl, or for any of the other acid gases emitted by coal- and oil-fired EGUs. And, as EPA has admitted, the agency's information on the carcinogenicity of these substances is incomplete. The Agency notes that it has not evaluated the potential carcinogenicity of HCl, and does not know whether or not HCl causes cancer. 76 Fed. Reg. at 25,050, see also 71 Fed. Reg. 76,542, 76,553 (Dec. 20, 2006) ("The data are inadequate to make a determination as to whether HCl is carcinogenic in either humans or animals, so EPA has not developed an assessment for the carcinogenicity of HCl."). Obviously, if EPA does not know whether HCl causes cancer, the agency has not identified an established health threshold below which HCl does not cause cancer. For this reason alone, EPA cannot invoke section 112(d)(4) with respect to HCl.

B. EPA Is Not Authorized to Set Section 112(d)(4) Standards Based on A Surrogate Pollutant.

It is well established that when acting under section 112(d), EPA must set standards for each HAP emitted by a category or subcategory of sources. *National Lime Ass'n v. EPA*, 233 F.3d 625, 633-34 (D.C. Cir. 2000). Similarly, if the Agency invokes section 112(d)(4) authority to consider setting a health-based alternative standard, the Agency must conduct that evaluation on a pollutant-specific basis with respect to pollutants for which a health threshold is already established. See Fed. Reg. 25,049 n.162. Even if HCl could reasonably serve as a surrogate for the

other acid gases in setting MACT floors (a point which we do not concede), it cannot be a surrogate in health-based standard setting, because EPA must base any section112(d)(4) health based standard on a NOAEL threshold for the toxic pollutant in question. EPA seems to understand this point, *id.* & n. 163, at least as a technical matter. As EPA properly notes, 76 Fed. Reg. 25,049 n. 163, and as shown in Table III-1 below, for HCl, Cl_2, and HF, the primary health endpoint is respiratory irritation. For HCN, however, the primary health endpoint is neurological, and for HF, adverse bone density effects are also experienced with chronic exposures. Indeed, EPA notes that "[t]hese gases (*e.g.,* HF) can act on biological organisms in a different manner than HCl, and each of the acid gases affects human health with a different dose-response relationship." *Id.* It is not accurate or technically correct to select one acid gas (HCl) with one health endpoint to serve as a surrogate for another acid gas (HF or HCN) with a different health endpoint in health based standard setting "with an ample margin of safety" as resulting surrogate based health threshold simply will does not address or relate to the adverse health effects of the other HAP.

Additionally, acid gas health effects are based both on exposure and toxicity, and the degree of adverse affect due to these factors can vary significantly between the acid gas HAPs. The California standards for the acid gases Cl2, HF and HCN show that these pollutants are more toxic on a weight/volume basis than HCl (considering the respiration/inhalation pathway of exposure). And Cl2, HCN and HF are approximately 10 times more toxic than HCl for short-term exposures. Therefore, unless Cl2, HF, and HCN are *always* present at concentrations that are ten-fold lower than HCl, even for short durations (and EPA does not claim to posses such information), only pollutant-specific health-based thresholds could ever be technically supported.

C. The requirement that Section 112(d)(4) standards must incorporate "an ample margin of safety" prohibits EPA from acting under this section where it lacks evidence on certain dimensions of health risk

The "ample margin of safety" language in section 112(d)(4) means at the very least that any standard that is set under this authority must be sufficient to protect against significant unforeseen consequences, particularly where the Agency is aware that those consequences may occur, but simply does not have enough evidence about them. *See, e.g. EDF v. EPA*, 598 F.2d 62, 81 (D.C.Cir. 1978)(holding that the phrase 'ample margin of safety' in the Clean Water Act's toxic provisions required EPA to protect against as yet unidentified risks to human health, including those "which research has not yet identified."). The fact that EPA has asserted in previous rulemakings that it was appropriate to exercise section 112(d)(4) discretion in the absence of evidence of carcinogenic risk, and on the limited understanding of the health risks it did have,[4] does not make that interpretation correct.[5] The absence of evidence of risk is not sufficient to demonstrate that an "ample margin of safety" exists. In fact, EPA's prior view turns the statutory requirement, as interpreted by the D.C. Circuit, on its head. Because the 'ample margin of safety' requirement is meant to protect against risks that have not yet been identified in research, a section 112(d)(4) standard simply cannot be justified on grounds that EPA does not have sufficient evidence about the health risks posed by a HAP.

Additionally, the Administrator must evaluate the potential for environmental impacts when considering whether to exercise her discretion under section 112(d)(4). As the legislative history indicates, and EPA has correctly noted here that it has authority to consider adverse environmental effects as well in

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[4] See 76 Fed. Reg. 25,050 (citing the Agency's previous section 112(d)(4)decisions, which it characterizes as unit-specific, under the 1998 Pulp and Paper MACT, 63 Fed. Reg. at 18,765 (April 15, 1998) and Lime Manufacturing MACT, 67 Fed. Reg. at 78,054 (Dec. 20, 2002)).

[5] Indeed, EPA seems to recognize that its earlier decisions were in error, and recently has opted not to finalize section 112(d)(4) standards for acid gas HAPs emitted by Portland Cement Facilities, 75 Fed. Reg 54,970 (Sept. 9, 2010).

evaluating whether it may set a section 112(d)(4) standard. Fed. Reg. at 25,050. As EPA previously has said it understands, "employing a section 112(d)(4) standard rather than a conventional MACT standard '*shall not* result in adverse environmental effect which would otherwise be reduced or eliminated.' " See proposed Industrial Boiler MACT, 75 Fed. Reg. 32,006, 32,031 (June 4, 2010)(emphasis added) (quoting S. Rep. No. 228, 101st Cong., 1st Sess. (1989) at 171). It is therefore not only "*appropriate* to consider potential adverse environmental effects in addition to adverse health effects when setting an emission standard … under 112(d)(4)," 76 Fed. Reg. at 25,050 (emphasis added), EPA *must* do so, and must show that any resulting health threshold-based standard does not cause adverse environmental effects in excess of those that would result from a MACT standard.

D. Existing RfCs for the acid gases are insufficient to form the basis for a Section 112(d)(4) standard for EGU emissions of these pollutants.

EPA now distances itself from previous actions in previous rulemakings, in which it relied on the RfC for HCl as the basis for essentially exempting HCl from the statutory requirements by invoking section 112(d)(4). 76 Fed. Reg. at 25,050. Commenters assert that EPA acted unlawfully in doing so in those previous actions, and that it would be equally incorrect here to suggest that the existing RfC is an "established health threshold" that could as a legal or technical matter offer sufficient support for an alternative regulatory approach for HCl, whether as a surrogate or not. Nor can the existing RfCs for the other EGU acid gases (where they exist) be used in this way. And EPA correctly notes that it has never implemented a NESHAP that used section 112(d)(4) with respect to the acid gases HF, CL2, SeO2 or HCN. *Id.*

The inhalation Reference Concentrations, or RfCs, for Cl2, HF, HCN and HCl are shown in Table III-1. An inhalation RfC represents the air-related toxicity value for a noncancer health endpoint associated with exposure to an air toxic, and

is expressed in weight of the toxic per volume of air (mg/m^3).[6] The inhalation RfC provides a continuous inhalation exposure estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The inhalation RfC considers toxic effects for both the respiratory system (a portal of entry) and effects peripheral to the respiratory system (extra-respiratory or systemic effects). An RfC can be derived from a 'no observed adverse effect level' (NOAEL), 'lowest observed adverse effect level' (LOAEL), or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used.[7] Reference values may also be derived for acute (≤24 hours), short-term (>24 hours, up to 30 days), and subchronic (>30 days, up to approximately 10% of the life span) exposure durations, all of which are derived based on an assumption of continuous exposure throughout the duration specified. RfDs and RfCs are generally used in noncancer health assessments.

Table III-1 summarizes U.S. EPA (via the Integrated Risk Information System, IRIS[8]) and California Environmental Protection Agency (Cal EPA) toxicity values[9] for the acid gases HCl, Cl_2, HF, and HCN. IRIS is a human health assessment program that provides high-quality science-based human health assessments to support EPA's regulatory activities. IRIS is prepared and maintained by the EPA's National Center for Environmental Assessment within the Office of Research and Development.[10] IRIS contains toxicity values for noncancer

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[6] See U.S. EPA, "A Review of the Reference Dose and Reference Concentration Processes. Risk Assessment Forum, Washington, DC" (2002). EPA/630/P-02/002F. Available at: http://www.epa.gov/ncea/iris/RFD_FINAL1.pdf.

[7] *Id.*

[8] Available at http://www.epa.gov/ncea/iris/index.html.

[9] Available at http://www.oehha.ca.gov/air/allrels.html.

[10] Animal studies can form the basis for these thresholds, as is clear from Table III-1. To account for the fact that humans may be more or less sensitive than the test animal, a 10-fold uncertainty factor is usually applied to the NOAEL. This uncertainty factor is called the "interspecies uncertainty factor." An additional 10-fold uncertainty factor, the "intraspecies uncertainty factor," is usually applied to account for the fact that some humans may be substantially more sensitive to the

and cancer endpoints. In addition, Table III-1 contains Cal EPA toxicity values, which are promulgated for use in California by the state's environmental agency, but which are not necessarily endorsed or adopted ("established") by U.S. EPA. Thus, while Cal EPA values are presented here to indicate the acid gases for which one well-regarded governmental agency has determined that enough toxicity information is available to set an exposure threshold, that fact does not mean that the Cal EPA values are "established" for the purposes of section 112(d)(4), as discussed below.

Table III-1: Regulatory toxicity values from IRIS and Cal EPA.

Acid Gas	Regulatory Agency	Type of Inhalation Toxicity Value	Value	NOAEL/LOAEL basis? Confidence?	Study Population/ Exposure Type	Target Organ	References
HCl	U.S. EPA	Chronic Reference Concentration	0.02 mg/m^3	LOAEL: 15.0 mg/m^3 (10 ppm) Low confidence in RfC.	Rats/ Chronic	Respiratory tract	Sellakumar et al., 1985; Albert et al., 1982
	U.S. EPA	Carcinogenicity	This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.				
	Cal EPA	Chronic Reference Exposure Level	0.009 mg/m^3		Rats/ Chronic	respiratory tract	Sellakumar, et al., 1985
	Cal EPA	Acute 1-hour Reference Exposure Level	2.1 mg/m^3		Asthmatics aged 18-25/ Acute (45 minutes)	respiratory system, eyes	Stevens et al., 1992
	Cal EPA	Carcinogenicity	No cancer potency value listed.				
Cl_2	U.S. EPA	Chronic Reference Concentration	No chronic inhalation value determined at this time.				
	U.S. EPA	Carcinogenicity	Not available at this time.				
	Cal EPA	Chronic Reference Exposure Level	0.0002 mg/m^3		Rats/ Chronic	upper respiratory epithelial lesions	Wolf et al., 1995
	Cal EPA	Acute 1-hour Reference Exposure Level	0.21 mg/m^3		Adult volunteers/ Acute (30 minutes)	itching or burning of throat	Anglen, 1981
	Cal EPA	Carcinogenicity	No cancer potency value listed.				
HF	U.S. EPA	Substance not listed					
	CalEPA	Chronic Reference Exposure Level	0.014 mg/m^3		Adult occupational/ Chronic (5 to 26 yrs)	Bone density effects	Derryberry et al., 1963
	CalEPA	Acute 1-hour	0.24		Adult	Upper	Lund et al.,

effects of substances than others. Additional uncertainty factors may also be applied. If studies using human subjects are the basis of a RfC, then the interspecies uncertainty factor can be reduced to as low as 1, but generally the 10-fold intraspecies uncertainty factor is retained.

		Reference Exposure Level	mg/m^3		volunteers/ Acute (1 hour)	respiratory tract irritation	1997
	Cal EPA	Carcinogenicity	No cancer potency value listed.				
HCN	U.S. EPA	Chronic reference concentration	0.003 mg/m^3	LOAEL: 7.07 mg/m^3 (6.4 ppm). Low confidence in RfC.	Adult occupational/ Chronic	CNS and thyroid	El Ghawabi et al., 1975
	U.S. EPA	Carcinogenicity	This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.				
	Cal EPA	Chronic Reference Exposure Level	0.009 mg/m^3		Adult occupational/ Chronic	CNS and thyroid	El Ghawabi et al., 1975
	Cal EPA	Acute 1-hour Reference Exposure Level	0.34 mg/m^3		Monkeys/ Acute (30 minutes)	CNS depression	Purser, 1984; Purser et al., 1984
	Cal EPA	Carcinogenicity	No cancer potency value listed.				

ppm=parts per million

To serve as the basis for a section 112(d)(4) standard, the "established health threshold" must be based on a NOAEL, as described *supra*. A NOAEL is the highest concentration where no adverse effect is observed in the most sensitive health endpoint among all studies examined. As Table III-1 indicates, there are no NOAELs forming the basis for existing RfCs for HCl, HF, Cl_2 or HCN. A NOAEL relates to a specific adverse health endpoint and cannot be assumed to apply to all health endpoints. The existence of a NOAEL set for a particular health endpoint for a specific pollutant cannot be interpreted to mean that there are no other health endpoints adversely affected by exposure to that pollutant; rather only that the other health endpoints do not occur at the concentration seen for the NOAEL of the most sensitive endpoint. If effects are observed at all dose levels tested, then the smallest dose tested, the Lowest Observed Adverse Effect Level (LOAEL) is used to calculate the RfC. An additional uncertainty factor usually is applied in these cases, since the NOAEL, by definition, would be lower than the LOAEL had it been observed.

As table III-1 demonstrates, however, the existing RfCs for both HCN and HCl are based on studies providing 'LOAEL values, as no appropriate studies providing NOAEL values are available. These RfCs also are "inhalation RfCs" – that is they represent the health risk and toxicity associated with the inhalation pathway of exposure only. But for these pollutants, there are other exposure

pathways (the skin and eyes for example) by which health effects can occur. So, even if these RfCs were set on the basis of a NOAEL (which they are not), they would be an inadequate basis for section112(d)(4) standard setting. Additionally, no RfC is available for Cl_2 at all, and HF is not among the 540 substances listed within IRIS, so no RfC is available for that acid gas. Finally, in evaluating the evidence that is available, for HCl and HCN, EPA states it has "low confidence" in the RfC values.

Indeed, EPA has acknowledged that exposure to HCl does damage people's health by conceding that it causes "corrosive tissue damage." 71 Fed. Reg. 76,542. Although EPA has claimed in the past that such damage does not constitute "adverse effects" because the tissue damage "does not exceed an organism's ability to repair it" — *i.e.*, is not permanent or fatal, *id.*, that argument was capricious – even preposterous – at the time, and commenters hope that EPA no longer even entertains it. Damage to an "organism's" tissue — *e.g.* the lung tissue of a child — *is* an adverse health effect. By contrast, EPA regularly relies on the American Thoracic Society's Statement on "What Constitutes an Adverse Health Effect of Air Pollution?" in reviewing the effects of pollutants under Sections 108 and 109.[11] That statement[12] identifies adverse respiratory effects to include clearly reversible health endpoints such as "acute upper respiratory tract infections that do not interfere with normal activity" and "increased prevalence or incidence fo cough/phlegm production requiring medical attention." Congress did not intend EPA to invoke section 112(d)(4) unless it was established that there would be no adverse health effects and, *a fortiori*, did not intend the agency to do so when it knew that there would be any adverse health effects.

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[11] See, for example, U.S.EPA. 2008. *Integrated Science Assessment for Sulfur Oxides: Health Criteria*, p. 1-12 – 1-13 and U.S, EPA, 2009. *Integrated Science Assessment for Particulate Matter*, p.1-23.

[12] American Thoracic Society. 2000. What Constitutes and Adverse Health Effect of Air Pollution, Am J Respir Crit Care Med. 161: 665-673.

As noted above, in evaluating human health risk for noncancer endpoints, it is equally important to consider short-term exposures as well as long-term/chronic exposure to these emissions. Moreover, health effects depend upon both exposure and toxicity, and for acute effects, HF and HCN are more toxic on a weight/volume basis than HCl. The Cal EPA sets an acute reference exposure level (1 hour exposure) (REL) as 2.1 mg/m^3 for HCl, 0.21 mg/m^3 for Cl_2, 0.24 mg/m^3 for HF, and 0.34 mg/m^3 for HCN. Therefore, Cl_2, HF, and HCN are approximately 10-fold more toxic than HCl on a weight-standardized basis for short-term exposures. For these reasons, as well, unless Cl_2, HF, and HCN are *always* present at concentrations that are at least 10-fold lower than HCl, even for short (1-hour) durations (a point on which EPA does not have information in the record for this rulemaking), only separate health-based thresholds, established for each acid gas, could ever be used to support a section 112(d)(4) standard.

Respiratory effects (the endpoint of most concern for HCl, Cl_2, and HF but not HCN) are likely after short-term exposures to high concentrations of acid gases. EPA asserts that it has little information on the peak short-term emissions of HCl from boilers, however. Were the existing RfCs used as the basis for a section 112(d)(4) alternative standard (and we do not concede they could be used in this way), compliance with the health-based threshold would therefore be based on long-term average exposures. Lack of data on exactly what these intermittent peak exposures might be, however, is not sufficient reason to adapt a threshold based solely on chronic exposures. As discussed above, inhalation RfCs exist only for two of the acid gases emitted by EGUs, HCl and HCN, and both of these RfCs reflect only studies of chronic exposures.

It is notable that none of the four acid gases examined, HCl, CL_2, HF, or HCN, has undergone a complete evaluation and determination of human carcinogenic potential under the IRIS program. As described above, this absence of information does not provide evidence that there is an absence of risk. Because section 112(d)(4) requires any alternative to a MACT standard to be based on both

"no adverse effects" and an "ample margin of safety," the incomplete nature of this evaluation makes a section 112(d)(4) standard unavailable for these pollutants. EPA's IRIS evaluates cancer risks through a two-step process, which first evaluates whether a pollutant is carcinogenic, and then, if so further describes its toxicity. The first step uses a cancer weight-of-evidence descriptor to describe a substance's potential to cause cancer in humans, and the conditions under which the carcinogenic effects may be expressed. Under the EPA's 2005 guidelines for carcinogen risk assessment, a narrative approach is used to characterize carcinogenicity.[13] Five standard weight-of-evidence descriptors (*Carcinogenic to Humans, Likely to Be Carcinogenic to Humans, Suggestive Evidence of Carcinogenic Potential, Inadequate Information to Assess Carcinogenic Potential,* and *Not Likely to Be Carcinogenic to Humans*) are used as part of the narrative.

In the second IRIS step, for pollutants found to be carcinogenic at step 1, cancer slope factors (for oral exposures) and unit risks (for inhalation exposures) are used to estimate the risk of cancer associated with exposure to a carcinogenic or even a potentially carcinogenic substance. A unit risk is defined as the upper-bound, approximating a 95% confidence limit, of excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration 1 mg/m^3 in air. The interpretation of unit risk for a substance in air would be as follows: if unit risk = 2×10^{-6} per mg/m^3, one might expect, as an upper bound estimate of risk, that based on a lifetime daily exposure to 1 mg/m^3 of the substance in air, up to 2 excess cancer cases may develop per 1,000,000 exposed individuals.

The California EPA, under its Office of Environmental Health Hazard Assessment, conducts health risk assessments of chemical contaminants found in air, including those identified as toxic air contaminants under California's Air Toxics Hot Spots Act. Assessments can include development of Cancer Potency

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[13] U.S. EPA (2005). Guidelines for Carcinogen Risk Assessment. U.S. Environmental Protection Agency, Washington, DC, EPA/630/P-03/001F, 2005. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=116283.

Factors[14] to assess the cancer risk from carcinogens in air, and development of Reference Exposure Levels (RELs) to assess noncancer health impacts. Cal EPA has set both chronic RELs and acute RELs for the four acid gases considered in this rulemaking (see Table III-1). These limits are not U.S. EPA limits, however, and just as for the EPA RfCs, they do not include cancer risk assessments for these pollutants.

E. Section 112(d)(4)'s Requirement to Set Any Health-Based Standards With "An Ample Margin of Safety" Requires Evaluation of Synergistic Health Effects.

EPA correctly notes that the fact that EGUs can be located in close proximity to a wide variety of industrial facilities makes predicting and assessing all possible mixtures of HCl and other acid gases difficult, if not impossible. 76 Fed. Reg. 25,050. By the same token, the fact that EGUs are often located in close proximity to other industrial facilities means that the cumulative effect of such interactions is a necessary component to any evaluation of health thresholds "with an ample margin of safety." EPA's comment that it could set such standards at a level that "at lease assures that persons exposed to emissions of the pollutant would not experience the adverse health effects on which the threshold is based due to source in the controlled category or subcategory," 76 Fed. Reg. at 25,050-51, ignores these synergistic effects between the emissions from the source in the controlled category and other sources in close proximity to it. EPA may not disregard the fact that there are other sources of air toxics, or other air pollutants located in the vicinity, in setting section 112(d)(4) standards. Such an approach would discount potential toxicologic interactions. Since all of the acid gases are respiratory irritants, one way to account for potential toxicologic interactions of these pollutants would be the use of the hazard index (HI) approach, as described in EPA's "Guideline for the Health

[14] Available at http://www.oehha.ca.gov/air/hot_spots/tsd052909.html.

Risk Assessment of Chemical Mixtures" and the Supplement thereto.[15] 76 Fed. Reg. 25,050.

EPA asserts that in the absence of information on interactions, the Agency assumes an "additive cumulative effect" citing the Supplementary Guidance. Based on its Hazard Index approach,[16] and in the absence of studies explicitly addressing the toxicity of mixtures of HCl with other respiratory irritants, EPA is taking the position that *if* the different acid gases affect health through the same health endpoint, they can be assumed to interact additively. However, this fundamental assumption is not correct. At least one of the acid gases emitted by EGUs, HCN, is a known neurotoxin. Its health effects therefore must not be considered additive with the health effects of other acid gases for which the health endpoint is different. Additionally, although Table III-1 shows the effects for the target organs and pathways studied, there are other pathways of exposure affecting other target organs, and the combined effects are not additive just as the effects of HCN are not additive with HCl. For these reasons, the Agency should not assume additive cumulative effects among these HAP.

For example among the pollutants that may be emitted by other emissions units in the vicinity is particulate matter ("PM"), including PM in the respirable size range, PM_{10} (PM that are 10 micrometers [µm] in aerodynamic diameter) and smaller. Under atmospheric conditions volatile HAPs that are emitted by boilers can condense onto or react with the surface of these PM, allowing the HAPs to travel along with the particles. Such particles can serve as "carriers" to bring the adhered HAPS deep within the lung, where the HAPS can interact with the

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[15] US EPA 2000. Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures. U.S. Environmental Protection Agency, Washington DC. EPA/630/R-00/002. Available at: http://www.epa.gov/nceawww1/pdfs/chem_mix/chem_mix_08_2001.pdf. This guidance replaced previous U.S. EPA "Guidelines for the Health Risk Assessment of Chemical Mixtures," 51 Fed. Reg. 34,014 (Sept. 24, 1986), available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=22567.

[16] 75 Fed. Reg. at 32032 referencing "Guidance for the Health Risk Assessment of Chemical Mixtures" (no citation given), but see previous note.

respiratory system directly or be leached off of the particle surface and become available systemically. Other HAPS and criteria pollutants may also be present on PM, including transition metals, ions (sulfate, nitrate), organic compound, carbonaceous material, minerals, reactive gases, and materials of biologic origin.[17]

The wide variety of HAPs and other pollutants EGUs emit, plus the proximity of other industrial and mobile sources of HAPS provide multiple opportunities for reaction and makes predicting and assessing all possible mixtures of HCl and other emitted air pollutants difficult, if not impossible. Because the statute requires standard setting with an "ample margin of safety" when section 112(d)(4) is invoked, as discussed above, these synergies make section 112(d)(4) standard setting practicably impossible to do lawfully for this industrial category.

F. Section 112(d)(4)'s Requirement to Set Any Health-Based Standards With "An Ample Margin of Safety" Requires Evaluation of Health Effects Beyond the EGU Fenceline. Environmental Justice Concerns Mitigate Against Anything Other than MACT-based Standards for this Reason.

Existing coal- and oil-fired EGUs are disproportionately located in industrial areas that are lower income or areas in which concentrations of persons of color reside. 76 Fed. Reg. 25,089-90 & Table 30. Because as EPA notes, "the fact that EGUs can be located in close proximity to a wide variety of industrial facilities makes predicting and assessing all possible mixtures of HCl and other emitted air pollutants difficult, if not impossible," 76 Fed. Reg. at 25,050, that fact also means that it is impossible for EPA fully to assess the health effects "with an ample margin of safety" of the combination of the air pollutants from these facilities on the people living in the vicinity. This fact pattern alone illustrates why national MACT standard setting is and must be the default requirement in the 1990 Clean Air Act, rather than "health-based" standard-setting under section 112(d)(4). The fact that

[17] *See, e.g.*, Chapter 3, U.S. EPA. Integrated Science Assessment for Particulate Matter (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-08/139F, 2009, available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546.

EGUs and other major industrial sources of air toxics are often located together with other sources of HAPs, including area sources and mobile sources, is a key reason (although not the only reason) that the former, exclusively health-based scheme for standard setting, was so unworkable. Not only are the physical interrelationships between the HAPs synergistic, making specific health effects very difficult to predict, but each situation will involve HAPs with different characteristics with respect to spatial distributions and health endpoints. Defining the geographic scope will not be possible on a nationwide basis.

These issues obviously implicate questions related to environmental justice concerns. As EPA reports, its own analysis demonstrate that "coal-fired EGUs are located in areas where [the] minority share of the population living within a 3-mile buffer is higher than the national average. For these same areas, the percent of the population below the poverty line is also higher than the national average." 76 Fed. Reg. at 25,090. EPA's analysis further shows that "some EGUs emit enough Ni or Cr to cause elevated lifetime cancer risks greater than 1 in a million in nearby communities, and that there are localized areas with elevated levels of [mercury] deposition around most U.S. EGUs." *Id.* at 25,089. As mentioned in Chapter 1, mercury hot spots will be of particular concern for subsistence fishers who must eat their catch either for economic, or in some instance (as is the case for some Native American tribes) cultural reasons. Information gathering and better understanding of the cumulative health impacts in areas where many sources of HAP are located together (for example, in industrial areas) would be beneficial. But we assert that the issues EPA raises here amply demonstrate that it is impossible to set a national health-based standard for these air toxics that incorporates an "adequate margin of safety."