

TABLE 9 TO SUBPART IIIII OF PART 63.—APPLICABILITY OF GENERAL PROVISIONS TO SUBPART IIIII—Continued

Citation	Subject	Applies to Subpart IIIII	Explanation
§ 63.10(d)(3)	Reporting Opacity or VE Observations	No	Subpart IIIII does not have opacity and visible emission standards.
§ 63.11	Flares	No	Subpart IIIII does not require flares.
§ 63.12	Delegation	Yes.	
§ 63.13	Addresses	Yes.	
§ 63.14	Incorporation by Reference	Yes.	
§ 63.15	Availability of Information	Yes.	

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 63

[FRL–7229–6]

National Emission Standards for Hazardous Air Pollutants: Chlorine and Hydrochloric Acid Emissions From Chlorine Production

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed decision not to regulate.

SUMMARY: EPA proposes not to regulate chlorine and hydrochloric acid (HCl) emissions for the Chlorine Production source category. We have determined that no further control is necessary because chlorine and HCl have well-defined health thresholds, and chlorine and HCl air emissions from chlorine producers result in human exposures in the ambient air that are below the threshold values with an ample margin of safety. This notice does not address mercury emissions from mercury cell chlor-alkali plants. Those emissions are addressed in a separate action in the proposed rule section of this **Federal Register**.

DATES: *Comments.* Submit comments on or before September 3, 2002.

Public Hearing. If anyone contacts the EPA requesting to speak at a public hearing by July 23, 2002, a public hearing will be held on August 2, 2002.

ADDRESSES: *Comments.* By U.S. Postal Service, send comments (in duplicate if possible) to: Air and Radiation Docket and Information Center (6102), Attention Docket Number A–2002–09, U.S. EPA, 1200 Pennsylvania Avenue, NW, Washington, DC 20460. In person or by courier, deliver comments (in duplicate if possible) to: Air and Radiation Docket and Information Center (6102), Attention Docket Number

A–2002–09, U.S. EPA, 401 M Street, SW., Washington, DC 20460.

Public Hearing. If a public hearing is held, it will be held at the new EPA facility complex in Research Triangle Park, North Carolina.

Docket. Docket No.

A–2002–09 contains supporting information used in developing the notice of proposed action for the Chlorine Production source category. The docket is located at the U.S. EPA, 401 M Street, SW., Washington, DC 20460 in Room M–1500, Waterside Mall (ground floor), and may be inspected from 8:30 a.m. to 5:30 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Mr. Iliam Rosario, Metals Group, Emission Standards Division (C439–02), U.S. EPA, Research Triangle Park, North Carolina 27711, telephone number: (919) 541–5308, facsimile: (919) 541–5600, electronic mail address: rosario.iliam@epa.gov.

SUPPLEMENTARY INFORMATION:

Comments. Comments and data may be submitted by electronic mail (e-mail) to: *a-and-r-docket@epa.gov*. Electronic comments must be submitted as an ASCII file to avoid the use of special characters and encryption problems and will also be accepted on disks in WordPerfect format. All comments and data submitted in electronic form must note the docket number: Docket No. A–2002–09. No confidential business information (CBI) should be submitted by e-mail. Electronic comments may be filed online at many Federal Depository Libraries.

Commenters wishing to submit proprietary information for consideration must clearly distinguish such information from other comments and clearly label it as CBI. Send submissions containing such proprietary information directly to the following address, and not to the public docket, to ensure that proprietary information is not inadvertently placed in the docket: OAQPS Document Control Office (C404–02), Attention: Iliam Rosario, Metals Group, Emission

Standards Division, U.S. EPA, Research Triangle Park, NC 27711. The EPA will disclose information identified as CBI only to the extent allowed by the procedures set forth in 40 CFR part 2. If no claim of confidentiality accompanies a submission when it is received by the EPA, the information may be made available to the public without further notice to the commenter.

Public Hearing. Persons interested in presenting oral testimony or inquiring as to whether a hearing is to be held should contact Cassie Posey, telephone number: (919) 541–0069. Persons interested in attending the public hearing must also call Cassie Posey to verify the time, date, and location of the hearing. The public hearing will provide interested parties the opportunity to present data, views, or arguments concerning the proposed emission standards.

Docket. The docket is an organized and complete file of all the information considered by the EPA in rule development. The docket is a dynamic file because material is added throughout the rulemaking process. The docketing system is intended to allow members of the public and industries involved to readily identify and locate documents so that they can effectively participate in the rulemaking process. Along with the proposed and promulgated standards and their preambles, the contents of the docket will serve as the record in the case of judicial review. (See section 307(d) (7)(A) of the Clean Air Act (CAA).) The materials related to this notice of proposed action are available for review in the docket or copies may be mailed on request from the Air Docket by calling (202) 260–7548. A reasonable fee may be charged for copying docket materials.

WorldWide Web (www) Information. In addition to being available in the docket, an electronic copy of today's notice of proposed action will also be available through EPA's www site. Following signature, a copy of the rule will be posted on our policy and

guidance page for newly proposed or promulgated rules: <http://www.epa.gov/ttn/oarpg>. The web site provides information and technology exchange in various areas of air pollution control. If more information regarding the web site is needed, call our web site help line at (919) 541-5384.

Regulated entities. Entities potentially affected by this action include facilities engaged in the production of chlorine. Affected categories and entities include those sources listed in the primary Standard Industrial Classification code 2812 or North American Information Classification System code 325181.

This description is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding **FOR FURTHER INFORMATION CONTACT** section.

Outline. The information presented in this preamble is organized as follows:

I. Background

- A. What is the source of authority for development of NESHAP?
- B. What is the source category?
- C. What are the health effects of chlorine and hydrogen chloride?

II. Summary of Proposed Action

III. Rationale for Proposed Action

- A. What is our statutory authority under section 112(d)(4)?
- B. What is the basis for our proposed action?

IV. Solicitation of Comments and Public Participation

I. Background

A. What Is the Source of Authority for Development of NESHAP?

Section 112 of the CAA contains our authority for reducing emissions of hazardous air pollutants (HAP). Section 112(d) requires us to promulgate regulations establishing emission standards for each category or subcategory of major sources and area sources of HAP listed pursuant to section 112(c). Section 112(d)(2) specifies that emission standards promulgated under the section shall require the maximum degree of reductions in emissions of the HAP subject to section 112 that are deemed achievable considering cost and any non-air quality health and environmental impacts and energy requirements.

National emission standards for hazardous air pollutants (NESHAP) reflect the maximum degree of reduction in emissions of HAP that is achievable. This level of control is commonly referred to as maximum achievable control technology (MACT).

The CAA includes exceptions to the general statutory requirement to establish emission standards based on MACT. Section 112(d)(4) allows us to use discretion in developing risk-based standards for HAP "for which a health threshold has been established" provided that the standards achieve an "ample margin of safety."

B. What Is the Source Category?

The Chlorine Production source category was initially listed as a major source of HAP pursuant to section 112(c)(1) of the CAA on July 16, 1992 (57 FR 31576). At the time of the initial listing, we defined the Chlorine Production source category as follows:

* * * The Chlorine Production Source Category includes any facility engaged in the production of chlorine. The category includes, but is not limited to, facilities producing chlorine by the following production methods: diaphragm cell, mercury cell, membrane cell, hybrid fuel cell, Downs cell, potash manufacture, hydrochloric acid decomposition, nitrosyl chloride process, nitric acid/salt process, Kel-Chlor process, and sodium chloride/sulfuric acid process.

We know of no facilities that produce chlorine using hybrid fuel cells, the nitrosyl chloride process, the Kel-Chlor process, the sodium chloride/sulfuric acid process, or as a by-product from potash manufacturing. We have identified 45 facilities that produce chlorine using mercury cells, diaphragm cells, or membrane cells. Collectively, these facilities are referred to as chlor-alkali plants as they produce chlorine and alkali (sodium hydroxide) as co-products.

We have also identified three facilities that produce chlorine as a by-product: one from the production of sodium metal in Downs cell, another from the production of potassium nitrate fertilizer that uses the nitric acid/salt process, and a third that produces chlorine as a by-product from primary magnesium refining. In addition, we have identified a resin producer that produces chlorine both in a chlor-alkali plant and through the decomposition of HCl.

Of the 48 facilities that produce chlorine, we have identified 21 that are major sources, including 20 chlor-alkali plants and the one primary magnesium refining facility. The primary magnesium refining facility is itself a major source emitting on the order of 600 tons of chlorine and 3,000 tons of HCl yearly, and is, in fact, a separately listed source category. As such, it will be addressed on its own in a separate rulemaking.

None of the 20 chlor-alkali plants are major in and of themselves, but are major due to collocation. That is, they are part of a larger contiguous establishment that is a major source. These larger establishments include organic chemical manufacturers, polymer and resin producers, and pulp and paper mills, all of which are already subject to one or more NESHAP. For instance, the organic chemical manufacturers are subject to the Hazardous Organic NESHAP, or HON (40 CFR part 63, subparts F, G, and H). The HON is a comprehensive rule that covers process vent, transfer, storage tank, equipment leak and wastewater emissions from the production of almost 400 organic chemicals. More than 100 organic HAP are regulated under the HON.

Polymers and resins producers are subject to four separate NESHAP (40 CFR part 63, subparts U, W, JJJ, and OOO) and must control process vent, transfer, storage tank, equipment leak and wastewater emissions. Chlor-alkali facilities that are collocated with pulp and paper mills are covered by 40 CFR part 63, subpart S (Pulp and Paper MACT III) and 40 CFR part 63, subpart KK (Printing and Publishing MACT). Chlor-alkali production facilities are also collocated with the following source categories: hazardous waste pesticide active ingredients production (subject to 40 CFR part 63, subpart MMM), polyether polyols production (subject to 40 CFR part 63, subpart PPP), and polycarbonates production (subject to 40 CFR part 63, subpart YY). There is also the Miscellaneous Organic Chemical Products and Processes NESHAP, currently under development, which will cover a variety of smaller, specialty chemical manufacturing processes, many that utilize chlorine. Therefore, most major processes at the sites where chlor-alkali facilities are located are subject to, or will be subject to, NESHAP to reduce HAP emissions. In addition to NESHAP, the chlorine production facilities are themselves subject to rules pursuant to section 112(r) of the CAA for the prevention of accidental releases of chemicals (40 CFR part 68).

The primary HAP emitted from chlorine production facilities processes are chlorine and HCl.¹ In each of the three chlor-alkali electrolytic cell processes, an electric current is passed through a salt solution (brine) causing the dissociation of salt to produce

¹ The mercury cell chlor-alkali process also emits mercury. Those emissions are addressed in a separate proposal elsewhere in today's **Federal Register**.

chlorine gas and an alkaline solution. Chlorine is collected from the cell room and is cooled, dried, and condensed in the purification process. The dried, gaseous chlorine then may be cooled further and compressed and liquified using multiple-stage condensers in the compression/liquefaction operation. Chlorine can be emitted from the tail gas stream from the final liquefier, the cell room, and equipment in chlorine service. Hydrochloric acid is used to pretreat feed brine prior to entering a chlor-alkali cell and at other locations throughout the process to adjust pH. It can also be emitted from storage tanks and equipment in HCl service.

Since chlor-alkali processes produce both chlorine and hydrogen, it is common for a direct synthesis HCl production unit to be incorporated into a chlor-alkali facility. This is the situation at four of the 20 chlor-alkali facilities at major source plant sites. In the direct synthesis process, chlorine and hydrogen are burned together to produce HCl. The gaseous HCl stream is then routed to an absorber and concentrated to produce a liquid HCl product. In many instances at chlor-alkali facilities, gaseous chlorine-containing waste streams (such as the tail gas from the liquifiers) provide chlorine to the HCl production unit. Therefore, we consider these direct synthesis HCl production units to be a part of the chlor-alkali facilities. These direct synthesis HCl production units can emit HCl from the absorber vent and associated storage vessels and transfer racks.

C. What Are the Health Effects of Chlorine and Hydrogen Chloride?

Acute (short-term) exposure to high levels of chlorine in humans can result in chest pain, vomiting, toxic pneumonitis, and pulmonary edema. At lower levels, chlorine is a potent irritant to the eyes, the upper respiratory tract, and lungs. Chronic (long-term) exposure to chlorine gas in workers has resulted in respiratory effects including eye and throat irritation and airflow obstruction. Animal studies have reported decreased body weight gain, eye and nose irritation, non-neoplastic nasal lesions, and respiratory epithelial hyperplasia from chronic inhalation exposure to chlorine. No information is available on the carcinogenic effects of chlorine in humans from inhalation exposure. We have not classified chlorine for potential carcinogenicity.

Hydrogen chloride is corrosive to the eyes, skin, and mucous membranes. Acute inhalation exposure may cause eye, nose, and respiratory tract irritation and inflammation and pulmonary

edema in humans. Chronic occupational exposure to HCl has been reported to cause gastritis, bronchitis, and dermatitis in workers. Prolonged exposure to low concentrations may also cause dental discoloration and erosion. No information is available on the reproductive or developmental effects of HCl in humans. In rats exposed to HCl by inhalation, altered estrus cycles have been reported in females and increased fetal mortality and decreased fetal weight have been reported in offspring. We have not classified HCl for carcinogenicity.

II. Summary of Proposed Action

We are proposing not to regulate chlorine and HCl emissions from chlorine production processes. Under the authority of section 112(d)(4), we have determined that no further control is necessary because chlorine and HCl are "health threshold pollutants," and chlorine and HCl levels emitted from chlorine production processes are below their threshold values within an ample margin of safety. Further, due to the fact that these two pollutants are the only HAP emitted in significant quantities from chlorine production plants, we are proposing not to develop any NESHAP for the Chlorine Production source category, with the exception of a NESHAP for mercury emissions from mercury cell chlor-alkali plants.

III. Rationale for Proposed Action

This section explains the statutory basis for considering health thresholds when establishing standards, and the basis for today's proposed action, including a discussion of the risk assessment conducted to support the ample margin of safety decision.

A. What Is Our Statutory Authority Under Section 112(d)(4)?

As stated previously in this notice, section 112 of the CAA includes exceptions to the general statutory requirement to establish emission standards based on MACT. Of relevance here, section 112(d)(4) allows us to develop risk-based standards for HAP "for which a health threshold has been established" provided that the standards achieve an "ample margin of safety." Therefore, we believe we have the discretion under section 112(d)(4) to develop risk-based standards which may be less stringent than the corresponding floor-based MACT standards for some categories emitting threshold pollutants.

In deciding standards for this source category, we seek to assure that emissions from every source in the category or subcategory are less than the

threshold level for an individual exposed at the upper end of the exposure distribution. The upper end of the exposure distribution is calculated using the "high end exposure estimate," defined as a plausible estimate of individual exposure for those persons at the upper end of the exposure distribution, conceptually above the 90th percentile, but not higher than the individual in the population who has the highest exposure. We believe that assuring protection to persons at the upper end of the exposure distribution is consistent with the "ample margin of safety" requirement in section 112(d)(4).

We emphasize that the use of section 112(d)(4) authority is wholly discretionary. As the legislative history indicates, cases may arise in which other considerations dictate that we should not invoke this authority to establish less stringent standards, despite the existence of a health effects threshold that is not jeopardized. For instance, we do not anticipate that we would set less stringent "risk-based" standards where evidence indicates a threat of significant or widespread environmental effects, although it may be shown that emissions from a particular source category do not approach or exceed a level requisite to protect public health with an ample margin of safety. We may also elect not to set less stringent risk-based standards where the estimated health threshold for a contaminant is subject to large uncertainty. Thus, in considering appropriate uses of our discretionary authority under section 112(d)(4), we consider other factors in addition to health thresholds, including uncertainty and potential "adverse environmental effects," as that phrase is defined in section 112(a)(7).

B. What Is the Basis for Our Proposed Action?

We are proposing in today's notice not to develop NESHAP for the Chlorine Production source category other than the mercury standards being proposed elsewhere in today's **Federal Register** for mercury cell processes. This decision is based on the following. First, we consider chlorine and HCl to be threshold pollutants. Second, we have defined threshold values in the form of Inhalation Reference Concentrations (RfC) and acute exposure guideline levels (AEGl). Third, chlorine and HCl are emitted from chlorine production plants (in the absence of additional control) in quantities that result in human exposure in the ambient air at levels well below the threshold values with an ample margin of safety. Finally, there are no adverse environmental

effects associated with these pollutants. The bases and supporting rationale for these conclusions are provided in the following sections.

1. Threshold Pollutants

For the purposes of section 112(d)(4), several factors are considered in our decision on whether a pollutant should be categorized as a health threshold pollutant. These factors include evidence and classification of carcinogenic risk and evidence of noncarcinogenic effects. For a detailed discussion of factors that we consider in deciding whether a pollutant should be categorized as a health threshold pollutant, please see the April 15, 1998 **Federal Register** document (63 FR 18766).

In the April 15, 1998 action cited above, we determined that HCl, a Group D pollutant, is a health threshold pollutant for the purpose of section 112(d)(4) of the CAA (63 FR 18753). We also believe that it is reasonable to classify chlorine as a Group D pollutant. There have been limited animal studies and observations of human occupational inhalation exposure for chlorine. There has been no evidence of a carcinogenic response in chronic, subchronic, or acute inhalation exposures in laboratory animal studies or from occupational inhalation exposure. Based on the limited negative carcinogenicity data, and on our knowledge of how chlorine reacts in the body and its likely mechanism of action, we presumptively consider chlorine to be a threshold pollutant.

2. Health Effects Exposure Assessment

We conducted a risk assessment to determine whether the emissions of chlorine and HCl from chlorine production plants at the current baseline levels are in quantities that are below the threshold values for chlorine and HCl within an ample margin of safety. The summary of this assessment is organized as follows: (1) Hazard identification and dose-response assessment, (2) emissions and release information, and (3) exposure assessment.

It is important to note that the risk assessment methodology applied here should not be interpreted as a standardized approach that sets a precedent for how EPA will analyze application of section 112(d)(4) in other cases. The approach presented here, including assumptions, models, and worst-case of sensitivity analysis, was selected to meet the unique needs of this particular case, to provide the appropriate level of detail and margin of safety given the data availability,

chemicals, and emissions particular to this category.

Hazard Identification and Dose-Response Assessment

The RfC is a "long-term" threshold, defined as an estimate of a daily inhalation exposure that, over a lifetime, would not likely result in the occurrence of noncancer health effects in humans. We have determined that the RfC for HCl of 20 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) is an appropriate threshold value for assessing risk to humans associated with exposure to HCl through inhalation (63 FR 18766, April 15, 1998). Therefore, we used this RfC as the threshold value in our exposure assessment for HCl emitted from chlorine production plants.

We also considered using the RfC for chlorine. In cases where we have not studied a chemical itself, we rely on the studies of other governmental agencies, such as the Agency for Toxic Substances and Disease Registry (ATSDR) or the Office of Health Hazard Assessment of California's Environmental Protection Agency (CAL EPA), for RfC values. The CAL EPA developed an RfC value of 0.2 $\mu\text{g}/\text{m}^3$ for chlorine based on a large inhalation study with rats.

Since chlorine does not generally persist in the atmosphere, we evaluated the appropriateness of using this chlorine RfC for this assessment. Chlorine in the atmosphere photolyzes to chloride ions (Cl^-) and then quickly reacts with methane to form HCl in bright sunshine. The estimated chlorine lifetime under these conditions is approximately 10 minutes. Even though emissions of chlorine in the absence of sunshine (e.g., at nighttime) remain as chlorine in the atmosphere until sunlight emerges, we do not believe that use of the chlorine RfC was appropriate for this assessment since long-term exposure to significant levels of chlorine is unlikely. EPA requests comments on the appropriateness of using a chlorine RfC to assess impacts of long-term exposure in this case.

However, we did conclude that the health effects of the long-term exposure to the HCl formed from the chlorine emitted from chlorine production plants should be considered. Therefore, we calculated the amount of HCl that would be formed from the emitted chlorine and used the HCl RfC of 20 $\mu\text{g}/\text{m}^3$ for determining the long-term noncarcinogenic effects of the chlorine emissions.

In addition to these effects of long-term inhalation of HCl, we also considered whether thresholds for short-term exposure to chlorine and HCl should be considered in this

assessment. Acute exposure guideline level toxicity values are estimates of adverse health effects due to a single exposure lasting 8 hours or less. The confidence in the AEGL (a qualitative rating or either low, medium, or high) is based on the number of studies available and the quality of the data. Consensus toxicity values for effects of acute exposures have been developed by several different organizations, and we are beginning to develop such values. A national advisory committee organized by the EPA has developed AEGL for priority chemicals for 30-minute, 1-hour, 4-hour, and 8-hour airborne exposures. They have also determined the levels of these chemicals at each exposure duration that will protect against discomfort (AEGL1), serious effects (AEGL2), and life-threatening effects or death (AEGL3). Hydrogen chloride has been assigned a 1-hour AEGL2 of 33,000 $\mu\text{g}/\text{m}^3$. Above this level, it is predicted that the general population, including sensitive individuals (such as asthmatics, children, or the elderly), could experience irreversible or other serious, long-lasting adverse health effects, or an impaired ability to escape. This value is a medium confidence value based on the severe nasal or pulmonary histopathology observed in rats exposed to a high concentration of 1,950,000 $\mu\text{g}/\text{m}^3$ HCl for 30 minutes. The AEGL2 value for HCl is displayed in an EPA internal database, the Air Toxics Health Effects Database (ATHED), as the appropriate value to use in short-term modeling.

Chlorine has been assigned a 1-hour AEGL2 toxicity value of 5,800 $\mu\text{g}/\text{m}^3$. This value is based on a human inhalation exposure study that included a sensitive individual, and this AEGL value has a high confidence value (62 FR 58839). This AEGL2 value is also contained in EPA's ATHED as the appropriate value to use in short-term modeling.

We used these AEGL values as threshold values for assessing the inhalation health effects of short-term exposures to chlorine and HCl. While chlorine does photolyze and eventually form HCl, we concluded that it was appropriate to use the chlorine AEGL value of 5,800 $\mu\text{g}/\text{m}^3$ for this assessment since it would be possible for individuals to be exposed to chlorine for 1-hour periods at night or on cloudy days.

Emissions and Release Information

Under the authority of section 114, we collected chlorine and HCl emissions information for all chlorine production facilities at the 20 major source sites.

Chlorine and HCl emissions were reported for point sources and fugitive emissions from the chlorine production units at each site. For the four sites where direct synthesis HCl production units are part of the chlorine production facility, emissions were also reported.

Respondents provided maximum annual and hourly chlorine and HCl emissions (typically, permitted emission rates were provided) and release characteristics. According to the information submitted, plantwide annual chlorine emissions from chlorine production processes ranged from less than one kilogram per year to over 6 Megagrams per year (Mg/yr). Of the 20 plant sites, 11 reported HCl emissions from chlorine production (and for four sites, HCl production processes), which ranged from less than one kilogram per year to around 32 Mg/yr.

The hourly plantwide chlorine emissions from chlorine production processes ranged from less than 2 grams per hour (g/hr) to around 10 kilograms per hour (kg/hr). For the 11 sites reporting HCl emissions, the hourly HCl emissions ranged from less than 1 g/hr to around 1 kg/hr.

Ten of the plant sites did not report any fugitive emissions. We believe that it is reasonable to expect that all chlorine production facilities would have some fugitive emissions. Therefore, we developed emission factors based on the reported fugitive emissions and related capacities for those plant sites that did report fugitive emissions. These factors ranged from 6.3×10^{-8} to 2.88 pounds per ton of chlorine production capacity. We used the maximum emission factor to conservatively estimate fugitive emissions for the 10 facilities that did not report fugitive emissions.

The release characteristics needed for the dispersion model included stack height, stack diameter, temperature, and exit velocity for point sources. For approximately 98 percent of the point sources reported, these parameters were provided in the section 114 responses. If release characteristics were not provided, we assigned default parameters based on data for the chlorine production industry in national emission databases and other data reported in response to the survey. The release characteristics needed for fugitive emission sources are release height and area. Release heights were provided for about 17 percent of the fugitive emission sources. For those fugitive emission sources for which information on release heights were not provided, we assumed that they were at 1 meter. No information was provided regarding the area of the fugitive

emission sources. Therefore, we assumed an area of 2,000 square meters for every fugitive emission source, which is a standard default used in modeling.

Exposure Assessment

The exposure assessment was conducted for chlorine and HCl emissions from all chlorine production processes in the source category (*i.e.*, from the chlorine production processes at the 20 sites that are major sources of HAP). As discussed above, the emissions data and release characteristics provided directly from all 20 plants were used as inputs to the assessment.

The Industrial Source Complex—Short Term Dispersion Model, Version 3 (ISCST3), was used for this exposure assessment. Receptors were placed at the center of census blocks (based on the 2000 Census) within 2 kilometers of the site and in the population-weighted centers of census block groups or census tracts out to 50 kilometers. Meteorological data from the nearest representative meteorological station were used. EPA requests comments on how to consider locations of receptors in assessing potential impacts on an individual exposed at the upper end of the exposure distribution for a large number of diverse facilities.

To determine the impacts of long-term exposure to chlorine and HCl emissions from chlorine plants, we used the maximum annual emission values provided by the plants. As discussed above, we converted the chlorine emissions to HCl since chlorine only persists in the atmosphere for a short amount of time. Therefore, we modeled the annual average HCl concentration at each receptor that was the result of the combination of the HCl emissions and the chlorine emissions that were converted to HCl through photolysis and subsequent reaction with methane.

As noted earlier, ten of the plants did not report any fugitive emissions. For these plants, we modeled the reported point source emissions and then modeled the estimated fugitive emissions separately. We added the highest concentration resulting from point source emissions with the highest concentration resulting from the fugitive emissions to obtain a conservative estimate of the highest HCl concentration that would be expected.

The highest modeled annual average HCl concentration from any chlorine production plant was $0.6 \mu\text{g}/\text{m}^3$. This is less than 3 percent of the HCl RfC of $20 \mu\text{g}/\text{m}^3$. Over 15 million people live in the areas around these 19 plant sites. Of these people, only around 1,300 were

exposed to annual average HCl concentrations greater than 1 percent of the RfC. In fact, well over 99 percent were exposed to annual average HCl concentrations less than 0.1 percent of the RfC.

To determine the impacts of short-term exposures to chlorine and HCl emissions from chlorine production plants, we used the maximum hourly emission values provided by the plants and obtained the highest individual hourly concentrations from the ISCST3 model. Separate runs were conducted for chlorine and HCl. The same process described above was used for plants that did not report any fugitive emissions.

The highest 1-hour chlorine concentration modeled was $346 \mu\text{g}/\text{m}^3$, which is less than 6 percent of the AEGL2 1-hour threshold value for chlorine ($5,800 \mu\text{g}/\text{m}^3$). This highest 1-hour HCl modeled concentration was $120 \mu\text{g}/\text{m}^3$, which is less than 1 percent of the AEGL2 1-hour threshold value for HCl ($33,000 \mu\text{g}/\text{m}^3$). We modeled these short-term concentrations for 5 years for each plant, which means concentrations were obtained for over 830,000 hours. Only around 75 hours (less than one hundredth of one percent) had modeled chlorine concentrations greater than 5 percent of the AEGL2 value, and no hours had modeled HCl concentrations greater than the AEGL2 value.

Given the fact that the highest modeled concentrations were so far below the threshold values, we elected to primarily evaluate the uncertainty and variability of this assessment qualitatively, coupled with a few basic sensitivity analyses. These sensitivity analyses focused on evaluating the uncertainties for the "worst-case" situations, as we were not concerned with uncertainties that resulted in even lower estimated risks.

We identified four potential areas of uncertainty/variability in the exposure assessment described above. These are emissions, the fate and transport model, exposure estimates, and toxicological dose response. Each of these areas is briefly discussed in the following.

As emission rates increase, exposure and risk increase. As noted earlier, the facilities reported maximum annual and maximum hourly emission rates. Most often, the reported rates were the facility's permitted emission rates. In addition, for those facilities that did not report any fugitive emissions, we estimated and modeled fugitive emissions based on the highest emission factor. Therefore, we would expect actual emissions to be less than those modeled, and thus, we believe that the results are biased high.

The primary uncertainties identified that are associated with the fate and transport modeling were the inherent uncertainty associated with the trying to represent complex atmospheric processes with a series of equations in the ISCST3 model (which is beyond the scope of this assessment) and missing release parameters, particularly for fugitive emission sources.

For the point sources, around 2 percent of the parameters were missing. For each missing parameter, we assigned a default parameter that was within the ranges provided by the other respondents. Since the actual release characteristics could be either higher or lower than these defaults, the results could be biased either way for this small percentage of the point sources.

Release heights were only provided for 17 percent of the fugitive emission sources, which ranged from 1.8 meters to 9.1 meters. For the fugitive sources without heights provided, we used a default height of 1 meter, which is more conservative than any reported value. Therefore, we anticipated that this could bias the results high.

There was considerable uncertainty associated with the size and location of fugitive emission sources. We used a default area of 2,000 m² for every fugitive emission source, with dimensions approximately 45 meters by 45 meters. This is a generic default value that we typically use for modeling fugitive emission sources, and it is not based on information provided by actual chlorine production facilities. The southwest corner of this area was placed at the mid-point of the locations for all reported point sources for the facility. The lack of information regarding the true size and location of chlorine production facilities could bias the concentration estimates high or low.

Uncertainty and variability also exist in the exposure estimates and the toxicological dose response, most of which result in the overestimation of risk. The RfC and AEGL2 values used in the assessment, which were discussed above, may contain multiple uncertainty factors whose impact is to add degrees of conservatism resulting in an overestimation of noncancer effects. In addition, the RfC assumes that individuals would be continuously exposed to the modeled concentration. As we believe these factors would only decrease the risk estimates, we did not evaluate their impact.

As noted above, our focus was only on those uncertainties that might increase the risk estimates and, thus, impact our decision not to regulate HCl and chlorine emissions from this source category. Of the basic uncertainties

discussed above, the factors that we believe could result in underestimated HAP concentrations (and, therefore, underestimated risks) include the default stack parameters for point sources and the default size and location of the fugitive emission sources.

We conducted a worst-case analysis for both long-term and short-term exposures to evaluate the potential upper-end impact of these uncertainties. For this analysis, we selected the single point source location from all plants that resulted in the highest estimated concentration people would be exposed to when run using a uniform emission rate. We then modeled the highest total facility emissions (maximum annual emissions for the long-term analysis and maximum hourly emissions for the short-term analysis) of chlorine and HCl at that point source location and used the most conservative stack parameters. We then chose the highest of these totals for chlorine and for HCl to put at the single point location. We also modeled a fugitive emission source using the highest reported emission factor coupled with the highest production capacity.

The results of this analysis show that, even with these worst-case conditions, the modeled concentrations were well below the threshold values. For the long-term impacts of the chlorine and HCl emissions (modeled as HCl, as discussed previously), the highest modeled annual HCl concentration was less than 5 µg/m³, which is less than 23 percent of the HCl RfC. The highest modeled maximum 1-hour chlorine and HCl concentrations were around 2,500 µg/m³ and 230 µg/m³, respectively. These values represent around 44 percent of the 1-hour chlorine AEGL2 threshold value and less than 1 percent of the 1-hour HCl AEGL2.

3. Environmental Effects

The standards for emissions must also protect against significant and widespread adverse environmental effects to wildlife, aquatic life, and other natural resources. We did not conduct a formal ecological risk assessment. However, we have reviewed publications in the literature to determine if there would be reasonable expectation for serious or widespread adverse effects to natural resources.

We consider the following aspects of pollutant exposure and effects: Toxicity effects from acute and chronic exposures to expected concentrations around the source (as measured or modeled), persistence in the environment, local and long-range transport, and tendency for bio-

magnification with toxic effects manifest at higher trophic levels.

As discussed above, the evidence available to date indicates that chlorine and HCl are threshold pollutants for the purposes of section 112(d)(4). Since chlorine is converted to HCl in the atmosphere, we did not perform a separate evaluation of chlorine exposure in this analysis.

No research has been identified for effects on terrestrial animal species beyond that cited in the development of the HCl RfC. Modeling calculations indicate that there is little likelihood of chronic or widespread exposure to HCl at concentrations above the threshold around chlorine production facilities. Based on these considerations, we believe that the RfC can reasonably be expected to protect against widespread adverse effects in other animal species as well.

Plants also respond to airborne HCl levels. Chronic exposure to about 600 µg/m³ can be expected to result in discernible effects, depending on the plant species. Plants respond differently to HCl as an anhydrous gas than to HCl aerosols. Relative humidity is important in plant response; there appears to be a threshold of relative humidity above which plants will incur twice as much damage at a given dose. Effects include leaf injury and decrease in chlorophyll levels in various species given acute, 20-minute exposures of 6,500 to 27,000 µg/m³. A field study reports different sensitivity to damage of foliage in 50 species growing in the vicinity of an anhydrous aluminum chloride manufacturer. American elm, bur oak, eastern white pine, basswood, red ash and several bean species were observed to be most sensitive. Concentrations of HCl in the air were not reported. Chloride ion in whole leaves was 0.2 to 0.5 percent of dry weight; sensitive species showed damage at the lower value, but tolerant species displayed no injury at the higher value. Injury declined with distance from the source with no effects observed beyond 300 meters. Maximum modeled long-term HCl concentrations (0.6 µg/m³) are well below the 600 µg/m³ chronic threshold, and the maximum short-term HCl concentration (346 µg/m³) are far below the 6,500 µg/m³ acute exposure threshold. Therefore, no adverse exposure effects are anticipated.

Prevailing meteorology strongly determines the fate of HCl in the atmosphere. However, HCl is not considered a strongly persistent pollutant, or one where long range transport is important in predicting its ecological effects. In the atmosphere, HCl can be expected to be absorbed into

aqueous aerosols, due to its great affinity for water, and removed from the troposphere by rainfall. In addition, HCl will react with hydroxy ions to yield water plus chloride ions. However, the concentration of hydroxy ions in the troposphere is low, so HCl may have a relatively long residence time in areas of low humidity. No studies are reported of HCl levels in ponds or other small water bodies or soils near major sources of HCl emissions. Toxic effects of HCl to aquatic organisms would likely be due to the hydronium ion, or acidity. Aquatic organisms in their natural environments often exhibit a broad range of pH tolerance. Effects of HCl deposition to small water bodies and to soils will primarily depend on the extent of neutralizing by carbonates or other buffering compounds. Chloride ions are essentially ubiquitous in natural waters and soils so minor increases due to deposition of dissolved HCl will have much less effect than the deposited hydronium ions. Deleterious effects of HCl on ponds and soils, where such effects might be found near a major source emitting to the atmosphere, likely will be local rather than widespread, as observed in plant foliage.

Effects of HCl on tissues are generally restricted to those immediately affected and are essentially acidic effects. The rapid solubility of HCl in aqueous media releases hydronium ions, which

can be corrosive to tissue when above a threshold concentration. The chloride ions may be concentrated in some plant tissues, but may be distributed throughout the organism, as most organisms have chloride ions in their fluids. Leaves or other tissues exposed to HCl may show some concentration above that of their immediate environment; that is, some degree of bioconcentration can occur. However, long-term storage in specific organs and biomagnification of concentrations of HCl in trophic levels of a food chain would not be expected. Thus, the chemical nature of HCl results in deleterious effects, that when present, are local rather than widespread.

In conclusion, acute and chronic exposures to expected HCl and chlorine concentrations around the source are not expected to result in adverse toxicity effects. These pollutants are not persistent in the environment. Effects of HCl and chlorine on ponds and soils are likely to be local rather than widespread. Finally, chlorine and HCl are not believed to result in biomagnification or bioaccumulation in the environment. Therefore, we do not anticipate any adverse ecological effects from chlorine and HCl.

4. Summary of Basis for Proposed Action

The results of the exposure assessment showed exposure levels to

chlorine and HCl emissions from chlorine production facilities are well below the health threshold values. Furthermore, the threshold values, for which the RfC and AEGL values were determined to be appropriate values, were not exceeded when taking into account an ample margin of safety. Finally, no significant or widespread adverse environmental effects from chlorine and HCl are anticipated. Therefore, under authority of section 112(d)(4), we have determined that further control of chlorine and HCl emissions from chlorine production facilities is not necessary.

IV. Solicitation of Comments and Public Participation

We seek full public participation in arriving at final decisions and encourage comments on all aspects of this notice of proposed action from all interested parties. You need to submit appropriate supporting data and analyses with your comments to allow us to make the best use of them. Be sure to direct your comments to the Air and Radiation Docket and Information Center, Docket No. A-2002-09 (*see ADDRESSES*).

Dated: June 5, 2002.

Christine Todd Whitman,
Administrator.

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