

work practice standards for the PCWP NESHAP.

#### *C. What Criteria Are Used in the Development of NESHAP?*

Section 112 of the CAA requires that we establish NESHAP for the control of HAP from both new and existing major sources. The CAA requires the NESHAP to reflect the maximum degree of reduction in emissions of HAP that is achievable. This level of control is commonly referred to as the MACT.

The MACT floor is the minimum control level allowed for NESHAP and is defined under section 112(d)(3) of the CAA. In essence, the MACT floor ensures that the standard is set at a level that assures that all major sources achieve the level of control at least as stringent as that already achieved by the better-controlled and lower-emitting sources in each source category or subcategory. For new sources, the MACT floor cannot be less stringent than the emission control that is achieved in practice by the best-controlled similar source. The MACT standards for existing sources can be less stringent than standards for new sources, but they cannot be less stringent than the average emission limitation achieved by the best-performing 12 percent of existing sources in the category or subcategory (or the best-performing 5 sources for categories or subcategories with fewer than 30 sources).

In developing MACT, we must also consider any control options that are more stringent than the floor. We may establish standards more stringent than the floor based on the consideration of cost of achieving the emissions reductions, any health and environmental impacts, and energy requirements.

#### *D. How Was This Proposed Rule Developed?*

We used several resources to develop this proposed rule, including questionnaire responses from industry, emissions test data, site visits to PCWP facilities, telephone contacts, and operating permits. We consulted representatives of the PCWP industry, State and Federal representatives, and emission control device vendors in developing this proposed rule. Industry representatives provided emissions test data, arranged site visits, reviewed draft questionnaires, and identified issues and provided information to help resolve issues in the rulemaking process. State representatives provided emissions test data and copies of permits.

We identified the MACT floor level of control with information obtained from the questionnaire responses, emission test reports, site visits, telephone contacts, and operating permits.

#### *E. What Are the Health Effects of the Pollutants Emitted From the PCWP Industry?*

This proposed rule protects air quality and promotes the public health by reducing emissions of some of the HAP listed in section 112(b)(1) of the CAA. The HAP emitted by PCWP facilities include, but are not limited to, acetaldehyde, acrolein, formaldehyde, methanol, phenol, and propionaldehyde. Exposure to these compounds has been demonstrated to cause adverse health effects when present in concentrations higher than those typically found in ambient air.

We do not have the necessary data on each PCWP facility and the people living around each facility to determine the actual population exposures to the HAP emitted from these facilities and the potential health effects. Therefore, we do not know the extent to which the adverse health effects described in the following subsections occur in the populations surrounding these facilities. However, to the extent the adverse effects do occur, today's proposed rule would reduce emissions and subsequent exposures.

##### 1. Acetaldehyde

Acetaldehyde is ubiquitous in the environment and may be formed in the body from the breakdown of ethanol (ethyl alcohol). Acute (short-term) exposure to acetaldehyde results in effects including irritation of the eyes, skin, and respiratory tract. In humans, symptoms of chronic (long-term) exposure to acetaldehyde resemble those of alcoholism. Long-term inhalation exposure studies in animals reported damage to the nasal epithelium and mucous membranes, growth retardation, and increased kidney weight. We have classified acetaldehyde as a probable human carcinogen (Group B2) based on animal studies that have shown nasal tumors in rats and laryngeal tumors in hamsters.

##### 2. Acrolein

Acute (short-term) inhalation exposure to acrolein may result in upper respiratory tract irritation and congestion. The major effects from chronic (long-term) inhalation exposure to acrolein in humans consist of general respiratory congestion and eye, nose, and throat irritation. Acrolein is a strong dermal irritant, causing skin burns in humans. We consider acrolein a

possible human carcinogen (Group C) based on limited animal cancer data suggesting an increased incidence of tumors in rats exposed to acrolein in the drinking water.

##### 3. Formaldehyde

Both acute (short-term) and chronic (long-term) exposure to formaldehyde irritates the eyes, nose, and throat, and may cause coughing, chest pains, and bronchitis. Reproductive effects, such as menstrual disorders and pregnancy problems, have been reported in female workers exposed to formaldehyde. Limited human studies have reported an association between formaldehyde exposure and lung and nasopharyngeal cancer. Animal inhalation studies have reported an increased incidence of nasal squamous cell cancer. We consider formaldehyde a probable human carcinogen (Group B2).

##### 4. Methanol

Acute (short-term) or chronic (long-term) exposure of humans to methanol by inhalation or ingestion may result in blurred vision, headache, dizziness, and nausea. No information is available on the reproductive, developmental, or carcinogenic effects of methanol in humans. Birth defects have been observed in the offspring of rats and mice exposed to methanol by inhalation. A methanol inhalation study using rhesus monkeys reported a decrease in the length of pregnancy and limited evidence of impaired learning ability in offspring. We have not classified methanol with respect to carcinogenicity.

##### 5. Phenol

Acute (short-term) inhalation and dermal exposure to phenol is highly irritating to the skin, eyes, and mucous membranes in humans. Oral exposure to small amounts of phenol may cause irregular breathing, muscular weakness and tremors, coma, and respiratory arrest at lethal concentrations. Anorexia, progressive weight loss, diarrhea, vertigo, salivation, and a dark coloration of the urine have been reported in chronically (long-term) exposed humans. Gastrointestinal irritation and blood and liver effects have also been reported. No studies of developmental or reproductive effects of phenol in humans are available, but animal studies have reported reduced fetal body weights, growth retardation, and abnormal development in the offspring of animals exposed to phenol by the oral route. We have classified phenol in Group D, not classifiable as to human carcinogenicity.