

3.2 Pollutants of Concern

3.2.1 Ambient Benchmark Concentrations

In 2006, the State of Oregon adopted ambient benchmark concentrations (ABCs) as goals, rather than as enforceable standards, to determine whether or not action is needed to reduce emissions of air toxics. Each air toxic of concern has a benchmark set based on its non-cancer or cancer causing effects, whichever level would be more protective. A carcinogen is any chemical for which there is sufficient evidence that exposure may result in cancer in humans or animals. Non-carcinogens are any chemicals that may cause non-cancer health effects such as respiratory irritation, nerve damage, or developmental problems. An ambient benchmark concentration is the annual average concentration of a toxic chemical in air that a person could breathe continuously for a lifetime without experiencing any non-cancer health effects or without increasing their excess cancer risk (i.e., their risk above the background cancer rate) by greater than one chance in a million.

Oregon's benchmarks were selected solely based on health protectiveness, without regard for economics or engineering feasibility. All of the benchmarks were set using several conservative assumptions, so that there is a large difference in concentration, a margin of safety, between where an effect was actually observed and the benchmark itself. Because of this margin of safety, all benchmarks are below concentrations at which adverse health effects in people most sensitive to air toxics are likely. Thus being above an ambient benchmark concentration does not mean an adverse health effect will occur; only that the margin of safety has been eroded and that additional work will be needed to identify, evaluate, and address a potential air toxics problem. Either modeled or monitored air toxic concentrations can be compared to the benchmarks. For more information on Oregon's air toxics benchmarks, please see [Appendix 12.1](#).

3.2.2 Selection of Pollutants of Concern for 2017 Modeling

In order to determine which pollutants should be included in the 2017 modeling, the PATS team first reviewed monitoring data from calendar year 2005 to identify which toxics were monitored near or above Oregon ambient benchmark concentrations. Then the Portland Air Toxics Assessment (PATA) and EPA national modeling data were reviewed to identify which toxics were modeled near or above Oregon ambient benchmark concentrations. Finally, the team reviewed if any toxics had any new emissions information that would indicate a potential risk; in addition, any developing toxicity data that would be cause for concern was reviewed. After reviewing all the data and information, 19 air toxics were identified for inclusion in the Portland Air Toxics model. A list of toxics, including the rationale for including them, was sent out to persons who chose to be alerted of any news regarding air toxics and specifically, the PATS project. In addition, the list of toxics was made available for review and comment via the DEQ website. DEQ did not receive any substantive comments regarding any of the toxics to be modeled in PATS.

The nineteen toxic air pollutants were selected for air dispersion modeling for one of three reasons:

- 1) 2005 monitoring data showed the pollutant was near or above Oregon ambient benchmark concentrations.** DEQ analyzed the 2005 monitoring data from the six sites in the area. The following pollutants were identified as being near or above the Oregon ambient benchmark concentration: benzene, polycyclic aromatic hydrocarbons, 1, 3-butadiene, acetaldehyde, naphthalene, arsenic compounds, manganese compounds, cadmium compounds, para-dichlorobenzene, and trichloroethylene. For information on monitoring locations and results, please see section 3.6.
- 2) PATA and EPA national modeling data showed the pollutant was modeled near or above Oregon ambient benchmark concentrations.** In addition to the 10 pollutants listed above, the following pollutants were selected based on NATA 2002 modeling results that demonstrated concentrations were near or above the Oregon ambient benchmark concentrations: diesel PM 2.5 (particulate matter 2.5 microns or less in diameter from combusting fuel in a diesel engine), methylene chloride, and 2,4-toluene diisocyanate.

3) Review of new emissions information or developing toxicity data indicated a potential risk. Lead compounds, ethylbenzene, formaldehyde, and perchloroethylene were reviewed by DEQ because of developing toxicity data that was cause for concern. Each pollutant's overall source, data collected and/or observed, and its relationship to the ambient benchmark concentration (ABC) are described below. For more details, please refer to the Air Toxics Pollutant Summary [Appendix 12.7](#).

The pollutants selected for modeling were:

- 1,3 butadiene
- 1,4 Para-Dichlorobenzene
- 15 PAH
- Acetaldehyde
- Acrolein
- Arsenic compounds
- Benzene
- Cadmium compounds
- Chromium VI
- Diesel PM 2.5
- Ethylbenzene
- Formaldehyde
- Lead compounds
- Manganese compounds
- Methylene chloride
- Naphthalene
- Nickel compounds
- Perchloroethylene
- Trichloroethylene

After modeling was completed, methylene chloride was dropped from data summaries because of data quality concerns. Two other pollutants, perchloroethylene and trichloroethylene were also dropped from the summaries after modeling showed they would be below the benchmark throughout the PATS study area in 2017.

3.2.3 Description of Pollutants of Concern

This section describes the emission sources, health effects, benchmarks, and 2005 monitoring results for pollutants of concern in the PATS study area. For more information on monitoring, please see [section 3.6](#) and the [Monitoring Appendix 12.5](#).

3.2.3.1 1,3-Butadiene

1,3-butadiene is a pollutant resulting from incomplete combustion from on-road engines, nonroad engines (like lawn and garden equipment), and marine recreational vehicles. Additional sources include petroleum refining, production of rubber and copolymer plastics, forest fires, and cigarette smoke. Epidemiological studies have reported a possible association between 1,3-butadiene exposure and cardiovascular diseases. Epidemiological studies of workers in rubber plants have shown an association between 1,3-butadiene exposure and increased incidence of leukemia. Animal studies have reported tumors at various sites from 1,3-butadiene exposure. EPA has classified 1,3-butadiene as carcinogenic to humans by inhalation.

The ambient benchmark concentration (ABC) for this pollutant is 0.03 ug/m³ (micrograms per cubic meter); however, 99% of the monitoring data from 2005 was below the minimum detection limit of 0.221 ug/m³, so an accurate assessment for this pollutant is unknown, which is why DEQ chose to model this pollutant.

3.2.3.2 1,4-Dichlorobenzene

1,4-dichlorobenzene (also para- or p-dichlorobenzene) is used mainly as a fumigant for the control of moths, molds, and mildews, and as a space deodorant for toilets and refuse containers. It is also used as an intermediate in the production of other chemicals, in the control of tree-boring insects, and in the control of mold in tobacco seeds. The general population is mainly exposed through breathing vapors from para-dichlorobenzene products used in the home, such as mothballs and toilet deodorizer blocks. Chronic (long-term) 1,4-dichlorobenzene inhalation exposure in humans results in effects on the liver, skin, and central nervous system. No information is available on the reproductive, developmental, or carcinogenic effects of 1,4-dichlorobenzene in humans. A National Toxicology Program study reported that 1,4-dichlorobenzene caused kidney tumors in male rats and liver tumors in both sexes of mice by gavage (experimentally placing the chemical in their stomachs). EPA has classified 1,4-dichlorobenzene as a Group C, possible human carcinogen.

The monitoring data for this pollutant was below the minimum detection limit, which is 0.6 ug/m^3 . Since the ABC is 0.09 ug/m^3 , DEQ must rely on modeling data for evaluation.

3.2.3.3 Polycyclic Aromatic Hydrocarbons (15 PAH)

Polycyclic aromatic hydrocarbons: PAHs, like benzene, result primarily from incomplete combustion of carbon-containing materials and so sources again include industrial processes, on-road and nonroad engines, and residential wood combustion. Other sources include commercial wood burning, coal-fired power plants, and municipal waste incineration. Cancer is the major concern from exposure to PAHs. Epidemiologic studies have reported an increase in lung cancer in humans exposed to coke oven emissions, roofing tar emissions, and cigarette smoke; all of these mixtures contain PAH compounds. Animal studies have reported respiratory tract tumors from inhalation exposure to benzo[a]pyrene stomach tumors, leukemia, and lung tumors from oral exposure to benzo[a]pyrene. EPA has classified seven PAHs (benzo[a]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) as Group B2, probable human carcinogens.

The 2005 data shows that the annual average for PAHs is below the ABC, which is 0.0009 ug/m^3 for total (32) PAHs. PAHs are a group of compounds that have different levels of toxicity and may pose diverse health risks. Toxic equivalent factors were used to scale the individual hydrocarbons to the toxicity level of benzo[a]pyrene, which is the chemical more commonly monitored out of the PAHs.

3.2.3.4 Acetaldehyde

Like the pollutants listed above, acetaldehyde's is primarily from incomplete combustion. It is also an intermediate in the synthesis of other chemicals (e.g. perfumes, dyes), food preservative, and a solvent in rubber and paper industries. Secondary sources include oxidation of hydrocarbons. Acetaldehyde's destruction processes include photolysis, reaction with hydroxyls (OH), and wet deposition. Acetaldehyde is considered a probable human carcinogen (Group B2) based on inadequate human cancer studies and animal studies that have shown nasal tumors in rats and laryngeal tumors in hamsters. For 2005, acetaldehyde was measured at three times over the ABC, which is 0.45 ug/m^3 .

3.2.3.5 Acrolein

Acrolein is primarily from wood burning, structural fires, and construction. Acrolein is extremely toxic to humans from inhalation and dermal exposure. Acute (short-term) inhalation exposure may result in upper respiratory tract irritation and congestion. No information is available on its reproductive, developmental, or carcinogenic effects in humans. The animal cancer data are limited, but one inhalation study resulted in nasal lesions in rats. EPA considers acrolein data are inadequate for an assessment of human carcinogenic potential. There is no mechanism for measuring acrolein at this time, so there is no monitoring data for 2005, so this pollutant was selected by DEQ for modeling. The ABC for this pollutant is 0.02 ug/m^3 .

3.2.3.6 Arsenic

Sources of arsenic are both anthropogenic and natural. Our soils in the Pacific Northwest are naturally high in arsenic because of their volcanic origins. Nationally, sources of arsenic include coal combustion and copper smelting. In Oregon, metal processing, agricultural pesticides, and soil dust are sources of arsenic. An increase in lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. EPA has classified inorganic arsenic as a Group A, human carcinogen. Monitoring data showed North Roselawn and the Kelly and Curry monitoring sites to have peaks of uncertain origin. Since industrial activity is localized, perhaps an anthropogenic influence is present at these sites; however, due to the same reasoning, it is unlikely that the observed regional patterns may be explained by anthropogenic (i.e. industry) sources alone. Oil and natural gas combustion and on-road and nonroad engines are important sources of arsenic. The 2005 annual average was measured four to five times over the ABC, which is 0.0002 ug/m^3 .

3.2.3.7 Benzene

Benzene is primarily from incomplete combustion from any burning, including industrial processes, on-road and nonroad engines and from residential wood combustion. Chronic (long-term) inhalation exposure has caused various disorders in the blood, including reduced numbers of red blood cells and aplastic anemia, in occupational settings. Reproductive effects have been reported for women exposed by inhalation to high levels, and adverse effects on the developing fetus have been observed in animal tests. Increased incidences of leukemia (cancer of the tissues that form white blood cells) have been observed in humans occupationally exposed to benzene. EPA has classified benzene as a Group A, human carcinogen. In 2005, Benzene was measured at concentrations six times over the ABC of 0.13 ug/m^3 . Benzene has been measured over the ABC at the North Roselawn site since monitoring began in 1999.

3.2.3.8 Cadmium

Cadmium is usually found as a mineral combined with other elements. It is produced by refining zinc ores. Most cadmium is used in batteries, pigments, metal coatings, and plastic. Although there is limited human data, an association between cadmium exposure and an increased risk of lung cancer has been reported from human studies, in particular from occupational studies of smelter workers. Cadmium has been shown to be a developmental toxicant in animals, resulting in fetal malformations and other effects. Animal studies have demonstrated an increase in lung cancer from long-term inhalation exposure to cadmium. EPA has classified cadmium as a Group B1, probable human carcinogen. The annual average for cadmium was measured below the ABC, which is 0.0006 ug/m^3 for 2005; but levels have exceeded the ABC on occasion, at both the North Roselawn and Kelly and Curry stations.

3.2.3.9 Chromium VI

Chromium VI is primarily from fossil fuels. It is also released from chemical manufacturing (paint dyes, rubber, and plastics), metal finishing, cement plants, and decomposition of brake linings. The respiratory tract is the major target organ for chromium (VI) toxicity, for acute (short-term) and chronic (long-term) inhalation exposures. Shortness of breath, coughing, and wheezing were reported from a case of acute exposure to chromium (VI), while perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, and other respiratory effects have been noted from chronic exposure. Human studies have clearly established that inhaled chromium (VI) is a Group A, human carcinogen, resulting in an increased risk of lung cancer. Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure. The ABC for this pollutant is 0.00008 ug/m^3 ; however, 94% of the monitoring data from 2005 was below the minimum detection limit of 0.0032 ug/m^3 , so an accurate assessment for this pollutant is unknown, which is why DEQ chose to model this pollutant.

3.2.3.10 Diesel Particulate Matter

Diesel PM 2.5 (particulate matter 2.5 microns or less in diameter) is primarily from on-road and nonroad diesel engines, including cars and trucks, construction, marine, and rail sources. EPA states on their website that they have “concluded that diesel exhaust ranks with the other substances that the national-scale assessment suggests pose the greatest relative risk. First, a large number of human epidemiology studies show increased lung cancer

associated with diesel exhaust. Furthermore, exposures in these epidemiology studies are in the same range as ambient exposures throughout the United States. In addition to the potential for lung cancer risk, there is a significant potential for non-cancer health effects as well, based on the contribution of diesel particulate matter to ambient levels of fine particles. Exposure to fine particles contributes to harmful respiratory and cardiovascular effects, and to premature mortality.” EPA has not developed a cancer potency factor for diesel. There is no monitored data for diesel particulate. The ABC for this pollutant is $0.1 \mu\text{g}/\text{m}^3$.

3.2.3.11 Ethylbenzene

Ethylbenzene is mainly used in the manufacturing of styrene. Occupational exposure to ethylbenzene occurs in factories that use ethylbenzene to produce other chemicals; in operations that include gas, oil, and varnish, workers, spray painters, and persons involved in gluing operations. Exposure to ethylbenzene occurs from the use of consumer products, gasoline, pesticides, solvents, carpet glues, varnishes, paints, and tobacco smoke. Chronic (long-term) exposure to ethylbenzene by inhalation in humans has shown conflicting results regarding its effects on the blood. Animal studies have reported effects on the blood, liver, and kidneys from chronic inhalation exposure to ethylbenzene. Limited information is available on the carcinogenic effects of ethylbenzene in humans. In a study by the National Toxicology Program, exposure to ethylbenzene by inhalation resulted in an increased incidence of kidney and testicular tumors in rats, and lung and liver tumors in mice. EPA has classified ethylbenzene as a Group D, not classifiable as to human carcinogenicity. At the beginning of the PATS study, this pollutant did not have an ABC, but EPA’s NATA 2002 modeling showed the pollutant in the Portland Metro area’s air shed. DEQ wanted to model for this pollutant to assess if there is a cause for concern. The ABC for this pollutant is $0.4 \mu\text{g}/\text{m}^3$, however due to an error, the benchmark should be $2000 \mu\text{g}/\text{m}^3$, an adjustment DEQ will propose during the next phase of benchmark updates.

3.2.3.12 Formaldehyde

Formaldehyde is primarily from incomplete combustion from industry, on-road and nonroad engines, construction equipment, diesel fuel combustion, railroads, and airports, as well as from wood burning. It is used as a concrete and plaster additive, as a disinfectant, and as a wood preservative. Secondary sources include oxidation of hydrocarbons. Formaldehyde’s destruction processes include photolysis, reaction with hydroxyls (OH), and wet deposition. Chronic (long-term) inhalation exposure to formaldehyde in humans can result in respiratory symptoms, and eye, nose, and throat irritation. 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. EPA considers formaldehyde as a Group B1, probable human carcinogen. All 2005 annual averages for formaldehyde were measured below the ABC, which is $3 \mu\text{g}/\text{m}^3$ and is based on non-cancer effects due to, at the time of this report, unresolved questions regarding the degree to which formaldehyde is a carcinogen. The highest annual average measured at the NW Post Office monitoring station.

3.2.3.13 Lead

Lead’s primary use is in the manufacture of batteries and in the production of metal products, such as sheet lead, solder (but no longer in food cans), and pipes, and in ceramic glazes, paint, ammunition, cable covering, and other products. Lead is toxic, causing a variety of effects at low dose levels. Chronic (long-term) exposure to lead in humans results in effects on the blood, central nervous system, blood pressure, kidneys, and Vitamin D metabolism. Children are particularly sensitive to the chronic effects of lead, with slowed cognitive development, reduced growth and other effects reported. Reproductive effects, such as decreased sperm count in men and spontaneous abortions in women, have been associated with high lead exposure. The developing fetus is at particular risk from maternal lead exposure, with low birth weight and slowed postnatal neurobehavioral development noted. Human studies are inconclusive regarding lead exposure and cancer. The 2005 annual average for lead, $0.0038 \mu\text{g}/\text{m}^3$, measured below the current ABC, which is $0.15 \mu\text{g}/\text{m}^3$. The annual average for 2005 is also well below this new proposed standard.

3.2.3.14 Manganese

Manganese is a naturally occurring metal found in rocks. Organic manganese compounds include pesticides and fuel additive for some gasolines. Compounds can enter the air from iron, steel, power plants, and coke ovens; as well as from dust from mining operations. Chronic (long-term) exposure to high levels of manganese by inhalation in humans may result in central nervous system effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. A syndrome named manganism may result from chronic exposure to higher levels; manganism is characterized by feelings of weakness and lethargy, tremors, a mask-like face, and psychological disturbances. Respiratory effects have also been noted in workers chronically exposed by inhalation. Impotence and loss of libido have been noted in male workers afflicted with manganism. The 2005 annual average for manganese was measured below the ABC, which is 0.09 ug/m^3 . However, monitoring data shows that levels have, on occasion, exceeded the ABC at the Post Office monitoring station.

3.2.3.15 Methylene Chloride

Methylene chloride is predominantly used as a solvent in paint strippers and removers; as a process solvent in the manufacture of drugs, pharmaceuticals, and film coatings; as a metal cleaning and finishing solvent in electronics manufacturing; and as an agent in urethane foam blowing. It is also used as a propellant in aerosols for products such as paints, automotive products, and insect sprays. The effects of chronic (long-term) exposure to methylene chloride suggest that the central nervous system is a potential target in humans and animals. Human data is inconclusive regarding methylene chloride and cancer; EPA has classified methylene chloride as a Group B2, probable human carcinogen. Animal studies have shown increases in liver and lung cancer and benign mammary gland tumors following the inhalation of methylene chloride. The 2005 annual average for methylene chloride was measured below the ABC, which is 2.1 ug/m^3 .

3.2.3.16 Naphthalene

Naphthalene's primary use is in the production of phthalic anhydride, but other uses of naphthalene include carbamate insecticides, surface active agents and resins, as a dye intermediate, as a synthetic tanning agent, as a moth repellent, and in miscellaneous organic chemicals. Naphthalene is released to the air from the burning of coal and oil and from the use of mothballs. Chronic (long-term) exposure of workers and rodents to naphthalene has been reported to cause cataracts and damage to the retina. Hemolytic anemia has been reported in infants born to mothers who "sniffed" and ingested naphthalene (as mothballs) during pregnancy. Available data are inadequate to establish a causal relationship between exposure to naphthalene and cancer in humans. EPA has classified naphthalene as a Group C, possible human carcinogen. The 2005 annual average for naphthalene is below the ABC, which is 0.03 ug/m^3 .

3.2.3.17 Nickel Compounds

Nickel is an abundant natural element found in soil and emitted from volcanoes. It can combine with other metals to form alloys for heat exchangers, along with other items. Nickel is most often used to make stainless steel and nickel compounds are used for nickel plating, to make some batteries, and as catalysts. Nickel is released into the air by industries that make or use nickel or nickel compounds. It is also released by oil-burning power plants and trash incinerators. Only two insoluble forms of nickel - refinery dust and the subsulfide from smelters - are considered to be known (Class A) human carcinogens. Soluble forms of nickel are more toxic to the respiratory track than less soluble forms but are not carcinogenic. Serious health effects from exposure to nickel, such as chronic bronchitis, reduced lung function, and cancer of the lung and nasal sinus, have occurred in people who have breathed dust containing certain nickel compounds while working in nickel refineries or nickel-processing plants. The levels of nickel in these workplaces are much higher than usual levels in the environment. Oregon has 3 ambient benchmark concentrations for nickel compounds: (1) 0.004 ug/m^3 for nickel refinery dust; (2) 0.002 ug/m^3 for nickel sub-sulfide; (3) 0.05 ug/m^3 for soluble nickel compounds. DEQ has performed monitoring and modeling only for soluble nickel compounds and modeling shows that some local areas of Portland may be above the benchmark for these compounds. There are no nickel smelters or refineries in the Portland area.

3.2.3.18 Perchloroethylene (tetrachloroethylene):

Perchloroethylene is widely used for dry-cleaning fabrics and metal degreasing operations. The main effects of perchloroethylene in humans are neurological, liver, and kidney effects following acute (short-term) and chronic (long-term) inhalation exposure. Adverse reproductive effects, such as spontaneous abortions, have been reported from occupational exposure to perchloroethylene; however, no definite conclusions can be made because of the limitations of the studies. Results from epidemiological studies of dry-cleaners occupationally exposed to perchloroethylene suggest increased risks for several types of cancer. Animal studies have reported an increased incidence of liver cancer in mice, via inhalation and gavage (experimentally placing the chemical in the stomach), and kidney and mononuclear cell leukemia in rats. In the mid-1980s, EPA considered the epidemiological and animal evidence on perchloroethylene as intermediate between a Group B and a Group C, probable and possible human carcinogen, respectively. EPA is currently reassessing its potential carcinogenicity. The monitoring data for this pollutant was below the minimum detection limit, which is 0.678 ug/m^3 . Since the ABC is 35 ug/m^3 , modeling and monitoring data, indicate that this pollutant is not as serious a concern as others may be. Although the ABC is currently based on a non-cancer value, perchloroethylene is considered a weak carcinogen.

3.2.3.19 Trichloroethylene

Trichloroethylene's main use is in the vapor degreasing of metal parts. Trichloroethylene is also used as an extraction solvent for greases, oils, fats, waxes, and tars, as a chemical intermediate in the production of other chemicals, and as a refrigerant. Trichloroethylene is used in consumer products such as typewriter correction fluids, paint removers/strippers, adhesives, spot removers, and rug-cleaning fluids. Chronic (long-term) inhalation exposure to trichloroethylene can affect the human central nervous system, with symptoms such as dizziness, headaches, confusion, euphoria, facial numbness, and weakness. Liver, kidney, immunological, endocrine, and developmental effects have also been reported in humans. A recent analysis of available epidemiological studies reports trichloroethylene exposure to be associated with several types of cancers in humans, especially kidney, liver, cervix, and lymphatic system. Animal studies have reported increases in lung, liver, kidney, and testicular tumors and lymphoma. EPA is currently reassessing the cancer classification of trichloroethylene. The 2005 monitoring data for trichloroethylene was below the minimum detection limit of 0.1 ug/m^3 , which is below the benchmark of 0.5 ug/m^3 . Modeling data estimates levels below the benchmark.